Министерство здравоохранения Республики Беларусь
УО «Витебский государственный медицинский университет»

PREVENTIVE DENTISTRY: METHODICAL GUIDANCE FOR DENTAL STUDENTS

ПРОФИЛАКТИЧЕСКАЯ СТОМАТОЛОГИЯ

учебно-методическое пособие

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Предназначено для студентов 2 курса, изучающих дисциплину «Профилактика стоматологических заболеваний» на английском языке, для студентов стоматологического факультета, субординаторов, магистрантов, аспирантов, клинических ординаторов, слушателей факультета повышения квалификации.

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CONTENT

I. PREVENTION OF MAIN ORAL DISEASES.

1. Health and disease in dentistry

Health is a state of complete physical, social and mental wellbeing (World Health Organization's Constitution). Many factors combine together to affect the health of individuals and communities. Whether people are healthy or not, is determined by their circumstances and environment. The factors which have been found to have the most significant influence – for better or worse – are widely known as the **determinants of health (**gender, culture, social status, social environments, employment, education, physical environments, childhood development, individual health practices, healthcare services, social support, genetic giftedness).

Diseases is any deviation from or interruption of the normal structure or function of any body part, organ, or system that is manifested by a characteristic set of symptoms and signs and whose etiology, pathology, and prognosis may be known or unknown.

Oral pathology includes infectious diseases (caries, periodontitis, stomatitis, etc.), maldevelopments (malocclusion, cleft palate, hypoplasia, etc.), trauma of maxillofacial area (teeth or soft tissues injuries) and tumors.

Oral health is a functional, structural, aesthetic, physiologic and psychosocial state of well-being and is essential to an individual's general health and quality of life. (2014, ADA)

Medical prevention is a system of economic, social, hygienic and medical measures undertaken by the state, public organizations and individual citizens in order to ensure a high level of public health and disease prevention.

According to the WHO classification, disease prevention can be divided to primary, secondary and tertiary (basing on preventive measures application terms).

Levels of prevention:

a. **Primary prevention** is devoted to prevent disease or injury before it occurs. Example of prevention methods is education about healthy habits. Primary prevention consists of traditional and specific health promotion.

b. **Secondary prevention is aimed to reduce** progress of a disease at early stage and prevents complications. Examples of prevention methods are rehabilitation regular exams and screening tests to detect disease in its earliest stages).

c. **Tertiary prevention** provides in-time disease treatment and prevention of disease recurrence, eliminating disease and its irreversible complications. It also includes measures to reduce or limit impairment and disability. Examples of prevention methods are rehabilitation programs, chronic disease management programs.

Risk factor, cause, background of disease occurrence. Causal and pathogenetic prevention of oral diseases.

Theories of disease causation include

- Germ theory of disease (monocausal): Work of Koch and Pasteur revealed that the prevailing health problems of the time were the products of living organisms. Isolation of bacillus causing tuberculosis and identification of the organism responsible for 22 infectious diseases between 1880 to 1900, gave rise to the idea that each disease had a single and a specific cause. A set of rules was formulated by Koch (Koch postulates) for establishing causal relationship between a microorganism and a disease states. In brief, it was essential that to be ascribed a causal role, the agent must always be found with the disease in question and not with any other disease.
- Epidemiological triad: The germ theory could not explain why not all those exposed to pathogen become ill: an organism or other noxious agent is a necessary, but not a sufficient cause of disease. The epidemiological triangle approach sees disease as the product of an interaction between an agent, a host, and the environment. The epidemiological triangle is useful in understanding infectious disorders, but is less useful with respect to chronic and degenerative disorders such as stroke arthritis and heart disease.

A **risk factor** is any attribute, characteristic or exposure of an individual that increases the likelihood of developing a disease or injury (**WHO).**

Cause of diseases is the reason or origination of disease occurrence.

Background pathology is pathological condition which accompanied main current disease and can increase severity of it.(e.g., disease-caries, cause-microbial plaque, containing Str. Mutans, risk factor-sugar-containing diet, background pathology – chronic gastritis).

Modes of Intervention

Primary Prevention (Prepathogenesis) Primary preventive services are those that prevent the initiation of disease.

a. Health promotion: It is process of enabling people to increase control over and to improve health. This can be achieved by

- Health education; instruction on proper plaque removal, daily tooth brushing and flossing
- Environment modification such as safe water, control of insects and rodents.
- Nutritional interventions: improvement of nutrition in vulnerable group. iv. Lifestyle and behavioural changes; which favor health

Health education is any combination of learning experiences designed to help individuals and communities improve their health, by increasing their knowledge or influencing their attitudes.(WHO)

Promotion of preventive measures among the population (health education) is performed in the active and passive forms (Table 1).

Methods are called active, if it due to public participation. Their advantage is a direct relationship and interaction, and a specialist audience, which ensures the best effect of exposure.

Methods that do not require the active participation of the population are called passive. They do not require the presence of medical personnel and exposed for a long time and for a large audience. The drawback is the lack of feedback between patients and specialists.

Table 1 «Contemporary forms of dental health education»

Organized population		**Unorganized population**	
Active forms	**Passive forms**	**Active forms**	**Passive forms**
Interviews, lectures, health lessons,	Stands, exhibitions, posters, educational games, booklets, commercials, films, cartoons	Interviews	Stands, exhibitions, posters, educational games, booklets, commercials, films, cartoons

Hygienic education is a system of useful skills and habits formation on the basis of healthy lifestyle knowledge and belief in their implementation. To give knowledge to patients is the easiest part of the job. The most difficult is to convert knowledge into beliefs and their implementation.

In the prevention of any disease, especially primary, you should start with the parents first and foremost children. There are no preventive methods without the participation of parents, because they will not be effective. Doctors and parents must work together.

Oral health education of the population is one of the professional duties of the dentist, hygienist.

The ultimate goal of education is to ensure that high-quality oral hygiene has become an integral part of the lifestyle, habits of each individual.

Oral hygiene education used both approaches:

1) specialists motivate and educate students and adults;

2) motivating parents teach their children.

3. Instilling of hygienic habits in children.

Education has a gradual stages:

1 - knowledge,

2 - understanding,

3 - development of skills and abilities,

4 – formation of skills (habit).

Therefore, to make use of the skills necessary to provide not only knowledge ("what and how to do it"), but also an understanding ("why do we need to do"). In other words, the patient needed motivation of the child and his parents to preserve dental health.

The basic training scheme of oral hygiene requires adaptation:

1) social conditions (the level of cultural and economic opportunities for the audience);

2) the age of the audience (mental and physical ability to learn); priorities in motivating, the choice of means and methods of oral hygiene;

3) the possibility of technical equipment of the learning process (the presence of visual aids, water supply, indicators of dental plaque, brushes, pastes, etc.).

Health educational project is the form of health promotion which provides by complex plan of preventive measures and possibility of its complex simultaneous implementation for patients considering their risk factors, dental and somatic status.

The scheme of health educational project preparing.

1. Determination of a subject (person, group, population).
2. Determination of the project content:

A. Risk factors in subjects

B. Psychological and material-economic status of subjects

3. Choice of implementation methods (communication, education, training)
4. Project organization
5. Implementation
6. Efficiency analysis
7. Correction and resumption of the project

5. Main oral diseases includes diseases of hard dental tissues, periodontal tissues, oral mucosa, soft tissues, malocclusion and etc. Contemporary, oral diseases are classified by ICD-10 classification. ICD-10 is an international statistical classification used in health care and related industries. Produced by the World Health Organization, it is used in several countries around the world. Some have gone on to develop their own national enhancements, building off the international version of the classification. Chapter XI of ICD-10 deals with conditions effecting the digestive system:

Principles of primary prevention management (organization)

High-risk Strategy Here the population is classified in relation to the degree of risk which individuals or groups of individuals exhibit, or are exposed to. It aims to bring preventive care to individuals or a group at special risk, which will reduce their risk factors.

Advantages

a. Doctor-patient relationship.
b. Better motivation.
c. Cost benefits.
d. Individual appropriateness.

Disadvantages

a. Criteria for ‘at risk’ not clear.
b. Cost of screening.
c. Does not tackle cause of disease.
d. Misses transitional populations.

Mass Strategy “Population strategy” is directed at the whole population irrespective of individual risk levels. This approach does not differentiate between individuals in any defined population and is directed towards the whole population. It treats all individuals as at equal risk. Underlying factors which contribute to the etiology of the disease, or themselves are causative factors, are targeted for alteration.

The population approach is directed towards socio-economic, behavioral and lifestyle changes.

Advantages
a. Easier.
b. Behaviorally appropriate for whole population.
c. Gets to the cause of the disease.
d. Reaches all who may become high risk / sufferers.

Disadvantages
a. Requires a lot of motivation.
b. Dilution of efforts.
c. Dilution of effects.
d. Less specific.
e. Higher rate of failure.
f. Lessened benefit to individuals.

There are 3 levels of preventive methods basing on the coverage of subjects:

- **Individual level** (for 1 certain patient/person), e.g. professional oral hygiene.
- **Group level** (for group of patients/persons with similar risk factors of dental disease occurrence), e.g. hygiene lesson.
- **Population** (for population of country or region), e.g. centralized fluoridated water.

Classification of preventive procedures basing on their executors:

- **Home** procedures, e.g. daily brushing routine.
- **Office** (professional) procedures, e.g. fissure sealing.
- **Public** measures, e.g. National dental health program.

II. DENTAL EXAMINATION

Table №2 "Questioning"

Points of questioning	**Clinical importance of answers**
1. Name, Surname.	- for control and future dental appointment. - to determine the effectiveness of prevention programs
2. The place of residence, place of work (study)	- for control and future dental appointment. - to determine the effectiveness of prevention programs - to identify the risk factors associated with water sources, environmental conditions, industrial and social factors - to assess the possibility of implement the activities of primary prevention
3. Age	- to take into account age mental and physical development of a patient to choose the proper methods of prevention - To assess the psycho-physiological characteristics to choose the proper methods of prevention
4.Pregnancy and childbirth antenatal and postnatal periods clinical course, type and features of feeding (diet)	- To identify the risk factors occurred during organs and tissues formation - To identify the risk factors related to diet, chewing load, etc.
5. Somatic diseases (old and present)	- To identify the risk factors associated with immunity, metabolism, etc. - To assess the psycho-physiological characteristics to choose the methods of prevention
6. Harmful habits	- To identify risk factors
7. Oral diseases (old and present)	- To assess a local immunity, caries resistance, etc.
8. Complaints about maxillofacial region status	- To assess a dental status
9. Daily oral care	- To identify risk factors - To assess the level of hygienic education and motivation - To recommend dental hygiene correction
10. Other dental preventive measures	- To assess the effectiveness of the previous preventive program - To avoid the duplication of useless prescriptions.

Table №3 "Assesment of general status"

Points of assesment	**Norm**	**Deviation**	**The clinical sense of deviation**
1. Psychoemotional state	Calm and ready to cooperate	Excited or has a delay in mental development	- Examination is difficult - It is important for selecting of primary prevention methods
2. Physical development	Corresponds to the actual age	- A delay or advance in age relation.	- Risk factors (especially metabolism) - It is important for selecting of primary prevention methods
3. Posture	Straight	- A violation of posture, curvature of	- Risk factors (of violation of the musculoskeletal system)

		the spine	
4. Gait	Free, active	- Difficult movement	- Risk factors (of violation of the musculoskeletal system)
5. Type of constitution	Harmonic	- Hyposthenic - Hyperthenic	- Risk factors (especially metabolic)

Extraoral examination

Table №4 "Assessment of maxillofacial functional status"

Points of assesment	**Norm**	**Deviation**	**The clinical sense of deviation**
1. The symmetry and proportionality of face and neck	The symmetry and proportionality	Acquired facial asymmetry	-Tissues are swelling (inflammation symptom) - Displacement of tissue resulting from trauma or hemorrhage - Offset or growth of tissue because of tumor
2. Pinna	The correct anatomical shape	The lack or deformation of pinna	- Malformation of the branchial arches as a signal of possible defects of the jaw bones - trauma
3. Facial skin	Clear, physiologically colored	With lesions and elements of skin and mucosal diseases	- the symptom of different diseases
4. Red border	Moderately moist	Dry with labial fissures, squama and crusts.	- the symptom of red border disease as a result of the habit of lip licking or mouth breathing
5. Joining closure of lips	Klein's line is the area of lips' joining.	Mouth is open	- incorrect nasal breathing (ENT pathology) - the symptom of the bundle of circular muscles of the mouth and masticatory muscles - the symptom of the malocclusion
6. Nose	The narrow nose bridge, oval or round nostrils.	The wide nose bridge, split-like nostrils.	- the symptom of permanent mouth breathing
7. Submandibular lymph nodes	Not palpable or under 1 cm in diameter, flexible, mobile, painless, with a smooth surface, not soldered to surrounding tissues.	More than 1 cm in diameter, dense (solid), soldered to surrounding tissues, painful on palpation, with a bumpy surface	- the symptom of inflammation in regional lymphatic nodes. - the symptom of tumor growth

Intraoral examination

Inspection of the oral cavity is executed at the dentist's chair. Parents can keep on hand children under 3 years old. The patient sits or lay in a chair, a doctor is from "7" to "10-12" hours.

The dental tools are needed for inspection of the oral cavity:

1. Dental Mirror: focuses light; gives the enlarged image; allows you to see inaccessible areas. A doctor holds it in right hand, if it is the only tool for examination or in the left, if there is probe in right hand. The mirror must hold by I and II fingers in the top of the handle. To obtain an image of various points of the oral cavity mirror is tilted to 20 ° (a pendulum motion) or rote (circular movement),but the main position does not change.

2. Dental Probe: to evaluate the mechanical properties of the object (teeth), find the cavities and defects. The probe is held by I, II, III fingers of the right hand in the middle or lower third of the handle, perpendicular to the examining surface. Movements should be soft, not aggressive. Be aware of possible dangers:

- The possibility of pain, especially in probing cavity;
- The possibility of damage to the structure of the enamel in the zone of low salinity, i.e. the just erupted teeth and in the area of initial caries;
- The ability to push infected dental plaque deeper in healthy fissures.

The algorithm of dental intraoral examination:

1. Inspection of oral mucosa
- Mucosa of lips, cheeks, palate;
- The state of excretory ducts of the major salivary glands;
- Tongue mucosa.

2. Architectonic of soft tissues assessment (inspection can be executed during mucous examination)
- The oral vestibulum depth;
-Labial frenulum;
- Buccal cords (mucosal folders);
-Tongue frenulum.

3. Examination of periodontal status.

4. Occlusion assessment

5. Dental hard tissues examination

Table №5 «Oral mucosa examination»

Point of examination	Metods	Norm	Deviation	Clinical sense of deviation
1. Labial, buccal and palatal mucosa.	Visual examination	Pink, clear, moderately moist without elements of lesion.	-elements of lesion -dry	-symptom of mucosal, salivary glands or somatic diseases.
2. Ducts of the salivary glands	Visual examination, salivation stimulation with	Salivation is free, saliva is pure and liquid.	Saliva is turbid and viscous, lack of salivation.	-symptom of salivary glands diseases; -caries risk factor

	massage of glands.			
3. Lingual mucosa	Visual examination	All types of papilla are presence, keratinization is normal.	Elements of lesion, desquamation, hyperkeratosis.	- symptom of oral and somatic pathology. - functional disorders.

Table №6 «Oral architectonic examination»

Point of examination	Metods	Norm	Deviation	Clinical sense of deviation
1.Oral vestibulum	Withdraw a lip in horizontal position, measure the height of gingival attachment.	5-10 mm medium	Under 5 mm - shallow, More than 10 mm - deep	Risk factor of periodontal pathology
2. Labial frenulum	Withdraw the lips (upper –up, lower - down), examine visually (or ischemization test)	Long, thin, woven to papilla base	Woven in the gingival papilla; at the moment of tension -papillae replacement or color change (positive ischemization)	Risk factor of periodontal pathology and malocclusion.
3. Buccal cords	Move aside a cheeks and examine a dome of vestibulum.	Medium or non-expressed, non-traumatic for periodontium	Strong, short, deforming gingival papilla in the woven point.	Risk factor of periodontal pathology
4. Lingval frenulum	Lift tongue with mirror or request to push it toward or lift by himself.	Long, thin, woven to medium third of tongue.	Strong, short, woven to anterior third of tongue or central gingival papilla, tongue movement is limited and difficult, the top of tongue fold or bifurcate at the protrusion moment.	Risk factor of periodontal pathology, functional disorders.

Examination of periodontium includes assessment of the gum mucosa (papillae, marginal and attached gingiva) and gingival groove.

Normally papilla are well defined, have a triangular (or trapezoidal) shape, close fitting to the teeth, have a pink color. Marginal gingiva has the same color. Attached gum can be paler.

Gingival groove (determined with light sensing between the gum and the tooth neck) is no deeper 1-1.5 mm.

Deviations includes redness, swelling, bleeding, elements of lesions, abscesses, fistulas, the destruction of the gingival groove (symptoms of periodontal disease).

Occlusion examination.

The bite is characterized by three positions:

- The spatial ratio of the jaws
- The form of the dental arches
- The position of individual teeth

1. The spatial ratio of the jaws in the position of central occlusion.

Determine the ratio of the key opposing teeth in three planes a) sagittal, b) vertical c) horizontal.

2. Form of the dental arches

Dental arches are investigated by wide open mouth. In the tprimary occlusion dental arches have the shape of a semicircle, in a permanent bite upper dental arch are shaped like a semi-ellipse, lower - parabola.

3. Evaluation of the position of individual teeth

The teeth should take the place of an appropriate its functional group, does not displace the adjacent teeth of the dentition.

Dental Health for preventive inspection assessed by examination of each tooth using dental mirrors and probing the fissures, cavities, etc.

Inspection begins with the right upper last molar in the dental arch (from left side to right), inspecting each tooth on all sides in the upper jaw, down to the left last tooth and consistently progressing to the last right tooth. It should examine all five tooth surfaces:

1) oral (palatal - for the upper teeth, lingual - for lower teeth)
2) vestibular
3) medial
4) distal
5) occlusal (for molars and premolars).

Table №7 «The stages of dental examination»

Stage	Norm	Pathology
Determination of dental sizes (visually, with mirror)	Teeth corresponds to age and group anatomical characteristics.	Bigger or smaller size.

Determination of enamel maturity and teeth form (visually, with mirror)	Teeth corresponds to age and group anatomical characteristics. Enamel is smooth.	Deformation because of caries lesion or trauma. Hypoplasia, hyperplasia, cracks, increasing enamel abrasion.
Determination of enamel color, presence of spots and plaque (visually, with mirror)	For primary teeth – white blue (cold shade), for permanent – white yellow (warm shade). The absence of spots and plaque.	Caries- chalky white, brown, black; Hypoplasia and fluorosis – chalky and yellow spots; Color changes because of medicaments; Hereditary disorders of hard tissues – from yellow to brown; Complicated caries – from pink to grey; Soft and mineralized plaque.
Determination of enamel luster (with mirror)	Expressed luster	In case of caries and fluorosis luster is absence
Examination of enamel surfaces (probing)	Enamel surface is smooth. Probing detects only natural fissures and gaps	In case of caries surface is rough with softened areas; probing detects cavities, in case of non-carious lesion enamel can be thick, rough or bumpy.
Determination of hard dental tissues properties (with mirror and probe)	Enamel and dentine are complete	In case of caries they can be damaged forming a cavity, in case of non-carious lesion – gaps, spots, fissures and pathological abrasion of hard dental tissues.

Dental formula

18	17	16	15	14	13	12	11	21	22	23	24	25	26	27	28
			55	54	53	52	51	61	62	63	64	65			
			85	84	83	82	81	71	72	73	74	75			
48	47	46	45	44	43	42	41	31	32	33	34	35	36	37	38

International Teeth Identification System (in accordance with ISO).

The International Standards Organization Designation System (ISO System) by the World Health Organization notation system is widely used by dental professionals internationally to associate information with a specific tooth. Based on the Fédération Dentaire Internationale (FDI), it is also known as ISO 3950 notation. Thus the ISO System uses a two-digit numbering system in which the first digit represents a tooth's quadrant and the second digit represents the number of the

tooth from the midline of the face. For permanent teeth, the upper right teeth begin with the number, "1". The upper left teeth begin with the number, "2". The lower left teeth begin with the number, "3". The lower right teeth begin with the number, "4". For primary teeth, the sequence of numbers goes 5, 6, 7, and 8 for the teeth in the upper right, upper left, lower left, and lower right respectively. When speaking about a certain tooth such as the permanent maxillary central incisor, the notation is pronounced “one, one”. Beware of mixing up the teeth in written form such as 11, 12, 13, 14, 15, 16, 17, 18 between the Universal and ISO systems. For example: retention of a primary molar tooth in the otherwise regular intact lower right jaw, position 5, would be noted as: 41, 42, 43, 44, 85, 46, 47, 48.

Registration of dental status.

Dental health is described using dental formula. To record the dental formula graphics and digital system is used. For permanent teeth there are quadrants I-IV, for the primary - V-VIII quadrants.

Table №8 «Registration codes»

Point of record	Symbols (codes):	
	Primary teeth	Permanent teeth
Healthy tooth	A	0
Dental caries and its complications	B	1
Filling	D	3
Extracted tooth	E	4
Artificial crown	G	7
Impacted tooth	-	8

III CONCEPTS OF CAUSAL PREVENTION OF DENTAL CARIES

The accumulation of knowledge about dental caries etiology has caused a number of theories of its origin. At different times, various theories of the caries nature (general, infectious and / or chemical) used to dominate. This fact defines the strategy of management of the disease.

Theories originated long before A.C. gradually evolved to the beginning of the XIX century. And they have following chronological order:

• version of the primacy of the human organism : from the humoral theory of Galen (III c. BC) to the vital theory («gangrene = caries», XVII c.);

• version of the infectious causes - from arguments about imaginary worms (VI cent. BC) to the documentary findings in caries micrococci - «denticolae» (1850s.);

• chemical version - from assumptions about the dangers of the rotting remains of adhesive sweet fruit on the teeth (III c. BC) to the conclusions about chemical damage of decomposing food (1819.), dissolution of enamel by acid (1830), including sugars and enzyme role (1867).

At the turn of the XIX and XX centuries the modern concept of dental caries formation has begun. Opening Pasteur microbial fermentation of carbohydrates allowed to unite infectious and chemical theories.

Miller reproduced in vitro decalcification of enamel and dentin by lactic acid, produced by several dozen species of microorganisms containing in the food (Chemical-parasitic theory, 1890.).

Williams said in 1897 that the microbiological production of acid simulated by Miller, in real life takes place in dental plaque,

In 1924, Streptococcus mutans was named the dominant cariogenic microorganism , in 1955 the leading etiologic role of microorganisms (not carbohydrates) was determined.

In 1960 Case’s experiment has been proven carious diseases accordance with all Koch's postulates characterizing infectious disease:

- caries doesn’t develop without microorganisms,
- it is contagious,
- there is possible to stop the activity of caries with antimicrobials.

During these years chemical version has been enriched by arguments about proteolysis (previous demineralization 1940s gg.) and chelation (excretion of calcium from the enamel in neutral conditions, 1954.). There were studies about phosphate(1960s gg.) and fluoride (1940s gg.) participation in the caries process. The version of the human organism dominant role at the caries development enriched with notions of circulation violation of fluids in the tooth (1928), the imbalance of same effects on the pulp (1960.), an autoimmune nature of caries (2000).

Contemporary concept can be represented by the following definition: caries - an infectious process, which is initiated by a specific microflora of plaque fermenting food carbohydrate components of plaque with forming an acid for quite a long time in low caries resistance conditions.(tab.1)

Tab.1 Contemporary concept of caries development

Acidogenic microorganisms become cariogenic, i.e. those which are able to ferment carbohydrates acid (Streptococcus mutans, Lactobacillus, Actinomyces).

The most modern version of the caries concept - a "theory of ecological shift": normal acidogenic microflora of dental biofilm becomes dangerous only if selective conditions in the oral cavity is formed by the person himself or those who control his diet and oral care. And the existence of dental biofilm can be maintained mainly through reproduction of acidogenic microflora. Carbohydrates (mono- and disaccharides and starches under certain conditions) - the substrate which long (often) presence in the teeth environment and provides such selective conditions.

Chemical essence of the initial caries is associated with an imbalance of constantly changing dissolution of enamel apatite calcium phosphate crystals and its precipitation. Initially, the surface layer of the enamel is demineralized, then, if the situation doesn’t stabilize, in mineral exchanges involved deeper layers of tissue. The dissolution of the inorganic phase is provided by acids plaque chelates linking calcium. Proteolytic enzymes cause the destruction of dentin organic matrix after demineralization.

The modern concept of dental caries prevention is considering caries as an infectious disease associated with food and dependent on a number of local conditions ("resistance"). Such a conception of nature makes possible to diagnose caries on the preclinical stages, to use successfully non-invasive treatment methods, etiotropic and pathogenic preventive measures.

Contemporary prevention of dental caries is directed:

• to prevent environmental shift in plaque (the control of colonization of the oral cavity, elimination of acidogenic microflora domination conditions by health diet and proper oral hygiene, prevention of microflora isolation in brushing unavailable places, the chemotherapeutic control of cariogenic flora, etc. .);

• to maintain the mineral balance in the area of pH decreasing to preserve the clinical integrity of the dental tissues (the creation of conditions for the formation and maturation of dental tissues before its eruption, support the protective properties of the oral fluid, the introduction of minerals in the teeth environment, etc.).

The main purpose of the prevention - maintaining the health of teeth. It can be achieved with a combination and implementation of different preventive methods in organizational schemes due to the needs and possibilities of the patient (groups of the population), dental services, and the state in general.

IV. CARIES EPIDEMIOLOGY AND STATISTICS.

The Decayed, Missing, Filled (DMF) index is the key measure of caries experience in dental epidemiology. The DMF Index is expressed as the total number of teeth or surfaces that are decayed (D), missing (M), or filled (F) in an individual. When the index is applied to dental surfaces, it is called the DMFT index.

Primary bite – df (t) or df (s)
Mixed bite – DMF (T)+df(t) or DMF(S)+df (s)
Permanent bite – DMF (T) or DMF (S)

WHO recommends for DMF registration:
"D"
1) tooth having a lesion (in one or more surfaces) in the grooves, fissures, smooth surfaces with definite a caries cavity.
2) tooth with a temporary filling;
3) tooth with a permanent filling and tooth decay.

"F"
1) tooth having one or more permanent filling (not decay)
2) tooth with a crown, set in relation to the previous carious destruction

However in the category "P"(and thus DMF)
1. teeth covered with sealant, 2. teeth with temporary crowns or tooth crowns restoring after injuries, etc. carious lesions does not include

"M"
1) in patients younger than 30 years - a tooth removed because of tooth decay. (The component "Y" for people under 30 years old do not include teeth removed for reasons related to orthodontics, periodontics, injury, etc.)
2) in patients aged 30 years and older - a tooth lost for any reason.
With age the individual DMF can:
1) be stable (no progression of the former intact teeth)
2) increase (in the range of 0 to 32)

Calculation: DMF(T) = D(T)+M(T)+F(T)
DMF(S) = D (all surfaces with lesion) + M (4or 5*missing tooth) + F(all filling surfaces)

Caries intensity assessment according to the T.F.Vinogradova, Leus methods.

Table №9 «DMF interpretation»

age	index	Assessment of caries intensity			
		low	medium	high	very high
1	df	-	-	1	2+
2	df	-	1	2	3+
3	df	1	2	3	4+
4	df	1	2-3	4	5+
5	df	1-2	3-4	5-6	7+
6	DMF+ df	0+(1+2)	0+(3+5)	0+(6+7)	(1+)+(8+)
7	DMF+ df	0+(1+3)	0+(4+5)	1+(6+8)	(2+)+(8+)
8	DMF+ df	1+(1+3)	0+(4+6)	2+(7+9)	(3+)+(9+)
9	DMF	1+	2	3	4+
10	(etc.)	1	2-3	4	5+
11		1	2-3	4-5	6+
12		1-2	3-4	5-6	7+
13		1-2	3-5	6-7	8+
14		1-3	4-5	6-8	9+
15		1-3	4-6	7-9	10+
16		1-3	4-6	8-10	11+
17		1-4	5-7	8-11	12+
18		1-4	5-8	9-12	13+
19		1-4	5-9	10-13	14+
20		1-3	4-6	7-12	13+
21		1-3	4-6	7-12	13+
22		1-3	4-6	7-13	14+
23		1-3	4-7	8-13	14+
24		1-3	4-7	8-14	15+
25		1-3	4-7	8-15	16+
26		1-4	5-7	8-15	16+
27		1-4	5-8	9-16	17+
28		1-4	5-8	9-16	17+
29		1-4	5-8	9-17	18+
30		1-4	5-9	10-18	19+
40		1-6	7-12	13-24	25+
50		1-7	8-15	16-30	31+
60		1-8	9-18	19	Not found

Group DMF = (DMF +DMF2+….)/N (the number of group members)

Table №10 «WHO interpretation of group DMF in 12-year-old patients»

DMF value	**Interpretation**
from 0,0 to 1,1	extremely low
from 1,2 to 2,6	low
from 2,7 to 4,4	medium
from 4,5 до 6,5	high
6,6 and upper	extremely high

The prevalence of dental caries and its interpretation

Prevalence - is the proportion of people with tooth decay and total number of examined patients.

Example: in a group - 100 people, 90 of them have a DMF> 0.

The prevalence are:

90 pers. / 100 people. x 100% = 90%

WHO drew attention to the proportion of "free"persons from decay (in this example = 10%) are changed and the following interpretation of prevalence of dental caries in 12-year-olds:

Table №11 «Caries prevalence interpretation»

The proportion of caries-free individuals (%)	Appropriate caries prevalence
under 5%	LOW
from 5% to 20%	MEDIUM
20% and upper	HIGH

Assessment of the dynamic indicators of the caries intensity.

1. Growth of caries intensity

As already noted, primary prevention programs aim to reduce (ideally - stop) the progression of caries .The dynamical progression of caries use the term caries increment (ΔDMF). It is calculated as the difference of final and initial values of the caries intensity.

ΔDMF = DMF2 - DMF1 (DMF2 recorded after some time (a year or two or more) after the registration DMF1). Usually ΔDMF calculated in a group or population. To evaluate the effectiveness of two prevention methods is to compare ΔDMF in two groups.

2. Reduction of caries is calculated to compare the growth of caries in different groups, as a relative value and expressed as a percentage.

Example: in group A with comprehensive prevention program ΔDMF(A) = 1.0; in group B with only dental health educational measures ΔDMF(B) = 2.5.

The maximum gain of - in group B, and this value is taken as 100%. Next, determine what portion of the increase was ΔKPUB in Group A:

X% = 1.0 / 2.5 x 100% = 40%

In group A there are only 40% intensity index increase (comparing with possible in group B).

Reduction - is the proportion of "prevention" and "failed" caries increment in the group with the possible maximum:

Reduction = 100% - 40% = 60%

In this case we say that in group A the preventive program provided 60% of caries reduction.

V. CARIES RESISTANCE AND REMINERALIZATION OF DENTAL HARD TISSUES.

Teeth form in close connection with the child's general development. In the formation period they are influenced by various factors (external and internal), which affect both the rate of growth, salinity, and the timing of teething. Induction of temporary teeth buds formation at 6-8 weeks of fetal development.

Calcification of the enamel of deciduous teeth begins at 4-5 months of embryonic development. By the birth of a child there are non-mineralized fissures and most part of primary molars crowns, temporary canines 2/3 crowns, cervical part of the incisors. This fact explains the most frequent caries localization in this area.

Non-eruptive teeth mineralization continues after baby birth for 1,5 -11 months (Schroeder, 1991). The final enamel mineralization takes place only for 2-2.5 years after teeth eruption (with maximum intense in the 1st year after eruption).

The process of formation and mineralization of hard dental tissues during non-eruptive development is affected by the mother health (diseases, smoking, alcohol abuse, etc.). The mineral composition of saliva plays main role in post-eruptive teeth mineralization. During formation teeth react to all changes influencing on the child body.

Eruption of deciduous teeth begins at 5-6 months of postnatal development and ends to 2-2.5 years. During the teeth eruption tissue surrounding it and forming teeth roots change, alveolar bone and periodontium rebuild. After final dental hard tissues mineralization and root formation there is a period of relative stability, which last in average 2.5-3 years.

Caries resistance is the ability to resist to caries process. The main causative agent of dental caries is microbial plaque. Caries resistance is provided by enamel properties and mineralization capabilities, therefore it was divided into organism caries resistance, which is determined by physiological levels of general health, and local resistance associated with acid resistance of enamel and oral fluid cariesprotective properties. Stages of enamel caries resistance formation can be divided into pre-eruptive and post-eruptive periods.

Critical periods of the formation of caries, i.e. periods of development (prenatal and postnatal), which the body is most sensitive to changes in external and internal environment are presented in Table №3.

Table №14 «Critical periods of the formation of caries»

PERIOD	TEETH FORMATION STAGES
6-8 weeks of prenatal development	foundation of primary teeth buds
15-16 weeks of prenatal development	differentiation of dental tissues
17-20 weeks of prenatal development	beginning of primary teeth mineralization foundation of 6^{th} teeth buds
23-25 weeks of prenatal development	mineralization of primary teeth crowns foundation of $1^{st} – 3^{rd}$ teeth buds
30 weeks of prenatal development	beginning of 6^{th} teeth mineralization
3-8 month of life	beginning of 1^{st} -3^{rd} teeth mineralization
2-3 years	foundation of 4^{th} , 5^{th} , 7^{th} teeth buds beginning of 4^{th} teeth mineralization
2,5-3,5 years	beginning of 5^{th},7^{th} teeth mineralization
4-5 years	foundation of 8^{th} teeth buds

Common factors defining caries resistance include:

1. Pregnancy and childbirth situation (complicated, with accompanying diseases)
2. Disease during mother's pregnancy and early years of a child's life (infectious, chronic somatic, allergic, etc.).
3. Common resistance (immunity) of the organism.
4. The complete and balanced nutrition for child and mother during pregnancy period (adequate intake of foods containing vit. A, D, E, C, compounds of Ca, Mg, P).
5. The content of fluoride in drinking water.
6. Environmental conditions, etc.

Local factors include:

1. Structure and biochemical composition of the enamel.
2. The local immunity status.
3. The properties of the oral fluid.
4. Conditions of the oral hard and soft tissues.
5. Bite.

In the clinical practice specific tests are used to evaluate the enamel resistance to acid (CRT, TER, TERI), the density of dental tissue structure (electrometry, laser reflectometry), which allow to draw conclusions about the resistance to dental caries and provide programs of dental caries prevention The density, permeability and solubility of enamel depends on enamel maturity and mineral metabolism. It is known that natural apatites, which contain six or more calcium atoms Ca8H2 (P04) 6 • 6H2O, Ca10 (P04) 6 • (OH) 2 and have a Ca: P

share in apathy of 1: 3 or more and apatite fluoride Ca10 (P04) 6F2 have the highest acid resistance.

Generally caries is determined by the composition and structure of enamel and other dental tissues, specific and nonspecific factors of oral immunity protection, quantitative and qualitative indicators of saliva, a diet features, bad habits, substance abuse and plaque characteristics. In turn, each of these factors depends on the organism general status, reactance and resistance.

Table №15 « Methods of erupted teeth enamel caries resistance determination»

Name and authors	Theoretical foundation	Materials and equipment	Methodology for test	Results registration	Results interpretatio
1	2	3	4	5	6
CRT (coloures Reaction time) Maiwald, 1978	The study of solubility in the acid: the higher solubility of enamel apatite is corresponded to faster apatite destruction and acid neutralization.	1) 1N hydrochloric acid solution; 2) Micropipette to deposit acid; 3) 3 mm diameter filter paper disc impregnated by the indicator - 0.02% aqueous crystal violet (in an acidic environment - yellow, in neutral - purple)	• 1.2 is isolated from the saliva, cleaned from plaque and dried. • Disk is placed on the vestibular surface of the tooth, and it was dropped with 1 mkl of acid. • Note: After the test remineralization therapy is required	To mark the time of indicator color change from yellow to violet	CRT≥60 sec – low solubility; CRT<60 sec – high solubility
TER (test of enamel resistance) Okushko V., 1984	The study of solubility in the acid. The higher solubility of enamel is corresponded to the greater degree of enamel demineralization under the acid influence and the higher colorant absorption	1) 1N hydrochloric acid solution; 2) Pipette or glass rod for applying acid; 3) 1% solution of methylene blue 4) 10-point scale of methylene blue solution shades, corresponding to its serial dilutions of 1: 2 - from 100% to 0.18%	• 1.2 is isolated from saliva, cleaned and dried. • It is applied on the vestibular surface with a 1.5-2 mm diameter droplet of acid. • After 5 seconds a drop of acid is relieved with dry swab. • Matt enamel surface is colored for 60 second with methylene blue • The indicator is removed from the surface with dry swab.	To compare the intensity of the colored spot with a scale, expressing the result as a percentage	From 0,18% to 3,1% (1-5 points) –low enamel solubility; ≥3,1% - high enamel solubility (6 and more points)
Laser reflectome try	Studying the degree of mineralization: the lower enamel mineralization level corresponds to its more loose structure, worse waveguide properties of the enamel, less enamel light transmission and light reflectance	1) Helium-neon laser LGN-105 with a wavelength of 0.63 microns; 2) A reflected light camera; 3) Measurer the reflected light	• Clean enamel surface from plaque and dry it • Directs the beam of coherent light from a laser source • Photograph reflected by the enamel light	To assess the value of the reflected light and compare with standard	Value of reflected light: 0,24 – caries resistant enamel; 0,3 - caries labile enamel

VI. TYPES, COMPOSITION AND PROPERTIES OF DENTAL DEPOSITS.

Classification of dental plaque:

1. by localization
- supragingival
- subgingival

2. by pathogenic influence:
- physiological
- pathological

3. by origin:
- acquired
- innate

4. by mineralization:
- non-mineralized (soft) dental plaque:

a) Pellicle;

b) Dental plaque;

c) Soft plaque (white matter)

d) Food remains (debris)
- mineralized (hard) dental plaque

a) supragingival tartar;

b) subgingival calculus.

The primary enamel **cuticle** – is a product of ameloblasts activity during amelogenesis. Reduced enamel epithelium is the layer on the surface of just erupted tooth created by ameloblasts.

The structure has 2 levels:

1) subsurface layer

2) surface layer (in some places on the enamel surface in the form of microscopic film - 0.2 um).

Embryonic origin structures are lost shortly after tooth eruption and consequently their role in tooth physiology is not important.

Pellicle is thin organic film (from 1 to 10 microns), a structural element of the surface enamel layer, which consist of selectively adsorbed salivary glycoproteins and components of lysed bacteria (aminoacids of derivative bacterial membranes). Pellicle free from microorganisms. Pellicle has 3 layers. Two of them are located on the enamel surface, and the third is the surface layer. Morphological pellicle feature is niches which are receptacles for bacterial cells.

Pellicle, as a membrane, gives enamel selective permeability. Due to this fact the diffusion of various solutions from saliva into the tooth enamel and backwards

can be significantly changed. Solutions must either penetrate through the pores of the membrane or dissociate into ions to cross the barrier. This barrier controls the rate of remineralization and demineralization process.

Pellicle plays an important role in the selective attachment of bacteria. If enamel is covered with pellicle, Str.Salivarius adsorption slows and Str.Sangues adsorption accelerates. Thus, the most significant role of pellicle is to create initial microbial colonization conditions on the enamel surface.

Dental plaque (DP) is a dense, non-mineralized, highly organized biofilm of microbes, organic and inorganic material derived from the saliva, gingival crevicular fluid, and bacterial byproducts.

Dental plaque formation.

From the moment a baby passes through the birth canal and takes its first breath, microbes begin to reside in its mouth. Later on, as teeth erupt, additional bacteria establish colonies on the tooth surfaces. Dental bacterial plaque is a bio‑film that adheres tenaciously to tooth surfaces, restorations, and prosthetic appliances.

The pattern of plaque biofilm development includes three phases:

1. Attachment of bacteria to a solid surface;
2. Formation of microcolonies on the surface;
3. Formation of mature, subgingival plaque biofilms

Tab.№16 The phases of dental plaque development

1	**Pellicle formation**	A thin bacteria free layer form within minutes of cleaned tooth
2	**Attachment** • Electrostatic (by electrostatic forces, van der Waals, providing close contact of the tooth surface with microbial cells) • Mechanical (with pills, fimbria) • Chemical (connection between linkage groups (adhesins) located on the surface of the microbial cells and complementary groups (receptors) located on the surface of the macroorganism.)	Within hours, bacteria attach to the pellicle and a slime layer is formed around the attached bacteria
3	**Plaque growth and uploading** (Young supra-gingival plaque)	It consists mainly of gram-positive cocci and rods, some gram-negative cocci

		and rods.
4	**Plaque growth and uploading** (Aged supra-gingival plaque formation)	There is the increase of percentage of gram-negative anaerobic bacteria
5	**Plaque growth and uploading** (Sub-gingival plaque formation)	Tooth-attached plaque, mostly gram-positive bacteria, with some gram-negative cocci and rods. Epithelial attached and unattached plaque, mostly gram-negative rods and spirochetes.

Tab.№17 Microbial plaque composition

	gram-positive		*gram-negative*	
	aerobes, facultative anaerobes	**anaerobes**	**aerobes, facultative naerobes**	**anaerobes**
cocci	Streptococcus	Peptokokki	Neisseria	Veylonelly
rods	Actinomyces, Lactobacillus, Corynebacterium	Bifidobacteria, propionbakterii		Bacteroides, fuzobakterii, leptotrihii
spirochetes			Leptospire	Treponema Borrelia

Bacterial populations of supra and subgingival DP composition is very similar. But there are more vibrio and fuzobakterium in the subgingival DP .

The role of dental plaque at caries and periodontum diseases development.

The higher rate of dental plaque formation there is the more cariogenic it becomes. The most important plaque component for caries development is Str.Mutans (there are 5 types a, в, c, d, e spreading among world population). Str.Mutans are adsorbed selectively on the aproximal surfaces and fissures, Str.Sangues locate on the «smooth surfaces».

Producing the organic acids by the oral microorganisms plays the main role at the caries development occurrence. Saliva influences on the acid production process.

But with accumulation of dental plaque its influence is reduced. And conversely the effect of dental plaque functional features increases. Cariogenicity level increases in the cases of carbohydrates consumption. Neither excessive

salivation nor active chewing helps to increase the pH of the DP. The presence of mineral components in DP defines its cariogenic status either. There are Ca, P, and F (with concentration of 2 mg/l) mostly (the more concentration, the less cariogenic potential).

Gingivitis is the result of gum irritation with lysis products of DP bacterial cells. Periodontal pockets flora is a source of the common intoxication of organism.

Pathogenic components of dental plaque

1. Substances possessing a straight crippling influence

- Organic acids
- Idol
- Ammonium
- Hydrogen sulfide
- Toxic amines

Proteolytic enzymes
Collagenase
Hyaluronidase
Glucuronidase
Neuraminidase

2. Substances which induct inflammation

- Chemotactic substances
- Activators of complement system
- Histamine

3. Substances which induct inflammation from organism immunity reaction.

- Endotoxins
- Peptidoglycans
- Polysaccharides
- Bacterial agents

Dental plaque indication.

Identification of the supra- gingival dental plaque is difficult for both patient and dentist, because of the color similarity between the tooth surface and dental plaque. Plaque identification may be done either by screening the plaque directly from the tooth surface, changing its color with a disclosing solution , or by using the ability of natural teeth to fluoresce under blue light. Disclosing dyes work by changing the color of dental plaque so that it contrasts with the white tooth surface. Dental plaque has the ability to retain a large number of dye substances which can be used for disclosing purposes. This property is related to interaction, because of the polarity difference between the components of the plaque and the dyes. The particles are bound to the surface by electrostatic interaction (proteins) and hydrogen bonds (polysaccharides). Over the years, different staining agents have been used. The first chemical reported to stain plaque was iodine (unstable) but, over the time, a variety of dyes have been used resistant colorants, such as:

• fuchsine

• erythrosine
• merbromin
• methylene blue
• brilliant blue
• crystal violet
• gentian violet
• fluorescein.

Tab. №18 Methods of dental plaque detection

METHODS OF PLAQUE DETECTION	
Visual examination	Drying
Probing	Coloration
Blue light fluoresce	

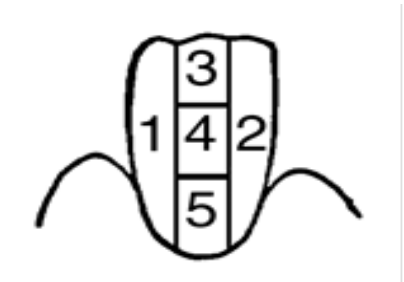
Methods of oral hygiene index assessment.

Dental plaque is the pathogenic factor for the teeth and periodontum. The control of dental plaque is the one of the main tasks of dental prophylaxis. For a quantitative diagnosis of dental plaque, the patient's motivation, assess the dynamics of the oral health level, the implementation of prevention programs need to use the standard "health index".

Patient Hygiene Performance Index (PHP, Podshadley and Haley ~)

This index was developed to evaluate patients'hygiene performance following toothbrush instructions.

It uses the same six tooth surfaces as in the OHI-S but divides each tooth surface into nine principal areas. After resistant colorant application within each surface area the debris is scored on a yes or no basis, where if any debris is present, a score of one is assigned, and where a surface is free of debris a score of 0 is given. The PHP score is the total of the score for each surface divided by the number of tooth surfaces examined.



1 - medial sector
2 - distal sector
3 - mid-occlusive sector
4 - central sector
5 - mid-Cervical sector

Pic. № 1

Calculation:
PHP oral= ΣPHP teeth/ n teeth, if
Σ – sum of examined teeth scores
n - number of examined teeth (6)
INTERPRETATION TABLE

Value	Interpretation
0	exelent hygiene
0,1-0,6	good hygiene
0,7-1,6	satisfactory hygiene
1,7 and more	unsatisfactory hygiene

The Simplified Oral Hygiene Index OHI-S (Greene and Vermillion, 1964) differs from the original OHI (The Oral Hygiene Index) in the number of the tooth surfaces scored (6 rather than 12), the method of selecting the surfaces to be scored, and the scores, which can be obtained. The criteria used for assigning scores to the tooth surfaces are the same as those use for the OHI (The Oral Hygiene Index).

The OHI-S has two components, the Debris Index and the Calculus Index. Each of these indexes, in turn, is based on numerical determinations representing the amount of debris or calculus found on the preselected tooth surfaces.

The six surfaces examined for the OHI-S are selected from four posterior and two anterior teeth.

In the posterior portion of the dentition, the first fully erupted tooth distal to the second bicuspid (15), usually the first molar (16) but sometimes the second (17) or third molar (18), is examined. The buccal surfaces of the selected upper *molars and the lingual surfaces of the selected lower molars are inspected. In the* anterior portion of the mouth, the labial surfaces of the upper right (11) and the lower left central incisors (31) are scored. In the absence of either of this anterior teeth, the central incisor (21 or 41 respectively) on the opposite side of the midline is substitted.

Criteria for classifying debris

Scores	Criteria
0	No debris or stain present
1	Soft debris covering not more than one third of the tooth surface, or presence of extrinsic stains without other debris regardless of surface area covered
2	Soft debris covering more than one third, but not more than two thirds, of the exposed tooth surface.
3	Soft debris covering more than two thirds of the exposed tooth surface.

Criteria for classifying calculus

Scores	Criteria
0	No calculus present
1	Supragingival calculus covering not more than third of the exposed tooth surface.
2	Supragingival calculus covering more than one third but not more than two thirds of the exposed tooth surface or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth or both.
3	Supragingival calculus covering more than two third of the exposed tooth surface or a continuos heavy band of subgingival calculus around the cervical portion of the tooth or both.

Calculation:

INTERPRETATION TABLE

Value	Interpretation
0-0,6	good hygiene
0,7-1,6	satisfactory hygiene
1,7-2,5	unsatisfactory hygiene
2,5 and more	poor hygiene

Plaque Index (PLI Silness and Loe)

Index PLI is intended to determine the thickness of the plaque.

We study the four surfaces of the tooth: vestibular, oral, distal, medial. The presence of plaque is determined visually or with probe, without coloring. It is important that compressed air be used to dry the tooth surfaces prior to the evaluation of the unstained plaque deposits.

The amount of plaque on the surface is estimated on the scale:

0 - No plaque in the gingival area.

1 - A film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may only be recognized by running a probe across the tooth surface, not visible by the naked eye.

2 - Moderate accumulation of soft deposits within the gingival pocket, on the gingival margin and/or adjacent tooth surface, which can be seen by the naked eye.

3 - Abundance of soft matter within the gingival pocket and/or on the gingival margin and adjacent tooth surface.

Calculation of the index PLI for one tooth:

PLI tooth = Σ (sum) score of four surfaces / 4

Calculation of the oral index PLI:

oral PLI = Σ score of examined teeth / n (number of such teeth).

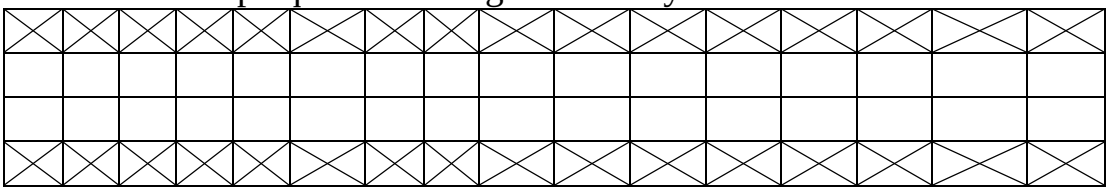
The Plaque Control Record - (O' Leary T, Drake R, Naylor, 1972)

The Plaque Control Record was developed to give the therapist, hygienist, or dental educator a simple method of recording the presence of the plaque on individual tooth surfaces. These surfaces are:

- Mesial
- Distal
- Buccal
- Lingual

At the control appointment a suitable disclosing solution such as Bismarck Brown, Diaplac or similar is painted on all exposed tooth surfaces. After the patient has rinsed, the operator (using an explorer or a tip of a probe) examines each stained surface for soft accumulations at the dentogingival junction. When found, they are recorded by making a dash/red colour in the appropriate spaces on the record form. Those surfaces, which do not have soft accumulations at the dentogingival junction, are not recorded.

After all teeth are examined and scored, the index is calculated by dividing the number of plaque containing surfaces by the total number of available surfaces.



Example: all teeth examined 32 x 4 = 128 surface surfaces.

40 Surface - 100%

70 contaminated surfaces - x%

X = 70/128 * 100% = 31%

VII. INDIVIDUAL ORAL HYGIENE PRODUCTS

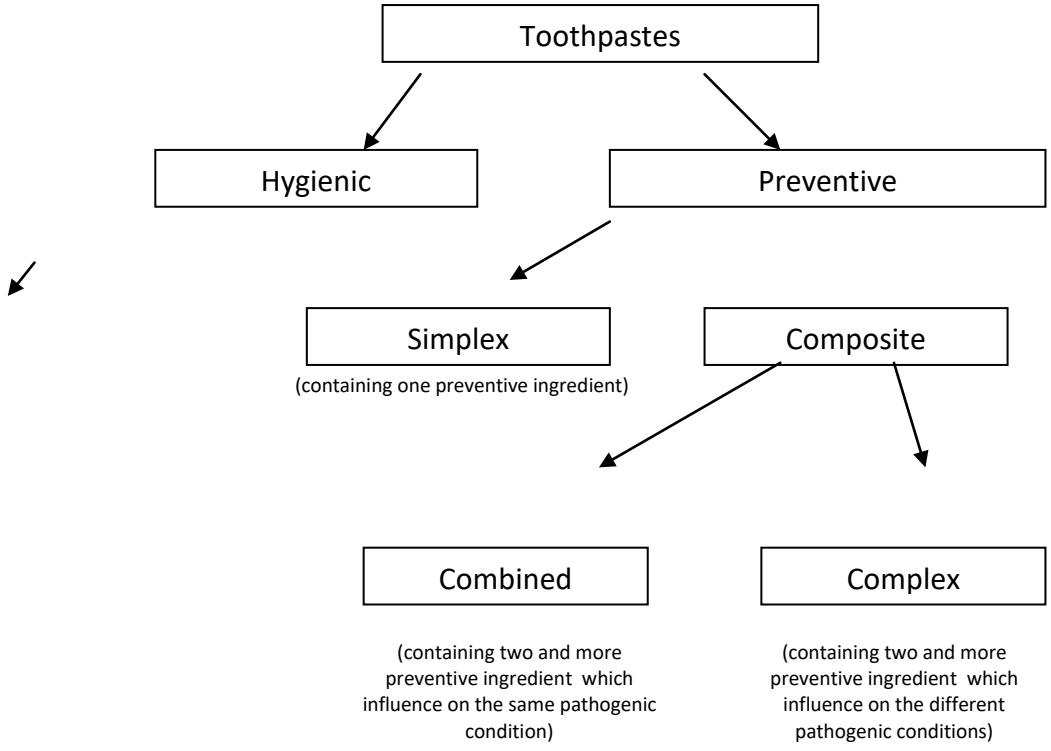
CLASSIFICATION OF IOHP (by S.B. Ulitovskiy)

1. Manual toothbrushes.
2. Toothpastes.
3. Liquid IOHP (mouthwashes, oral elixirs, oral deodorizers).
4. Interdental IOHP (floss, tooth pick, interdental stimulator, etc.).
5. Electric IOHP (electric toothbrush, electric floss, oral center, etc.).
6. Tooth-powder.
7. Chewing gums.
8. IOHP for tooth whitening.

TOOTHPASTES

Toothpaste is a dental hygiene product used with a toothbrush as an accessory to clean and maintain oral health by abrasive removing the dental plaque and food from dental surfaces.

CLASSIFICATION OF TOOTHPASTES (BY S.B. ULITOVSKIY)



Preventive toothpastes includes:

- anticaries toothpastes;
- paste affecting the oral soft tissue;
- antitartar pastes;

- antiplaque pastes;
- pastes desensitizing dental hard tissues;
- whitening pastes;
- odor control (desodoration) pastes.

Toothpastes contain active ingredients or additives that perform specific functions. These additives are abrasives, fluorides, desensitizing agents, antiplaque agents, and antitartar ingredients. Toothpastes also contain detergents, humectants, thickeners, preservatives, flavoring agents, sweeteners, and coloring agents. The exact composition of a particular toothpaste varies with each manufacturer, but a typical formulation is abrasive 10-40%, humectant 20-70%, water 5-30%, binder 1-2%, detergent 1-3%, flavour 1-2%, preservative 0.05-0.5% and therapeutic agent 0.1-0.5%.

1. **Abrasives** (SiO_2, $CaCO_3$, $NaHCO_3$) perform the primary functions of removing plaque and stain from teeth.

2. **Common fluorides** in toothpastes include stannous fluoride, sodium monophosphate fluoride, organic compounds of fluoride (amine fluor) and sodium fluoride. Fluoride's primary action is to be incorporated into the tooth substrate (enamel and dentin) making the tooth more resistant to acid attack by cariogenic bacteria. Fluoride is also bactericidal and has additional antiplaque effects.

3. **Desensitizing agents** are active ingredients, usually potassium nitrate, in toothpaste that reduce dentin hypersensitivity through a depolarizing effect on the odontoblastic processes in the dentinal tubules. The nerve endings of the odontoblastic processes then repolarize and have a reduced pain sensing ability. Also, desensitizing effects of arginine bicarbonate calcium carbonate complex and stabilized stannous fluoride have been demonstrated to provide dentin desensitizing effect.

4. **Antiplaque agents** reduce plaque growth. This can have a positive effect in reducing plaque on teeth, reducing gingivitis, and potentially reducing caries. Some antiplaque agents include triclosan, papain, and sanguinaria extract. Triclosan has been accepted by FDA as an antiplaque-antigingivitis therapeutic additive to toothpastes.

5. **Antitartar ingredients** that reduce calculus buildup on teeth include tetrapotassium pyrophosphate, tetrasodium pyrophosphate, disodium pyrophosphate, papain and citroxaine.

6. **Remineralizing agents** have recently been added to toothpastes. These remineralizing agents are based upon amorphous calcium phosphate. This soluble calcium and phosphate are described as enhancing remineralization, preventing dental caries, reducing enamel and/or dentin erosion, and reducing dentin hypersensitivity. The mode of action that has been hypothesized for these agents is the calcium and phosphate in soluble form which allows it to bind to enamel and dentin and to dental plaque. While there are laboratory studies demonstrating these effects, there is little clinical evidence to support these claims.

6. **Detergents** (foaming agents) are responsible for the foaming action of toothpastes. Sodium lauryl sulfate (SLS) the most widely used detergent in toothpastes, has been reported to cause adverse effects on oral soft tissues. SLS in toothpastes significantly increased the incidence of desquamation of the oral mucosa compared with toothpastes containing the detergent cocoamidopropyl-betaine (CAPB). Patients with a history of recurrent apthous ulcers should use toothpastes that are SLS-free.

7. **Humectants** provide for toothpaste texture and help the toothpaste maintain its moisture. Some common humectants in toothpastes are glycerine, sorbitol and water. Xylitol is also a humectant. Thickeners are added to a toothpaste to provide body to the toothpaste. Some thickeners are carrageenan and xanthan gum.

8. **Preservatives** prevent growth of microbes in the toothpaste. Some common preservatives used in toothpastes are methyl paraben and sodium benzoate.

9. Flavoring agents are added to improve the taste of toothpastes. They can range from minty flavors to fruity flavors.

10. **Herbal agents** such as aloe vera, sodium carrageenan, Echinacea, goldenseal and bee propolis have been added to toothpastes. Currently there are no controlled, long-term studies that demonstrate the efficacy of these agents.

11. **Sweeteners** also improve the taste of toothpaste. Most toothpaste sweeteners are artificial and are not able to be used by cariogenic bacteria.

12. **Coloring agents** are added to provide toothpastes with a pleasing appearance.

MOUTHWASHES

Mouthwash (mouth rinse, oral rinse or mouth bath) is additional IOHP using before, after and instead of teeth brushing. It consist topical agent to prevent oral disease (caries, periodontitis, gingivitis, stomatitis, halitosis, etc.) such as antiseptics, flavor, fluoride, SAS, antitatar and antiplaque components. There are recommended to use compatible on ingredients mouthwashes with patient toothpastes.

CHEWING GUM Chewing gum is preventive preparation for chewing, usually made of flavoured and sweetened chicle or such substitutes as polyvinyl acetate to increase salivation and self-cleaning of hard and soft oral tissues surfaces after meal. Chewing time per serving for 3-5 minutes in order to avoid cumulative depletion of the salivary glands. The main active ingredients are calcium compounds, xylitol and antiseptics.

CLASSIFICATION OF IOHP (by S.B. Ulitovskiy)

1. Manual toothbrushes.
2. Toothpastes.

3. Liquid IOHP (mouthwashes, oral elixirs, oral deodorizers).
4. Interdental IOHP (floss, tooth pick, interdental stimulator, etc.).
5. Electric IOHP (electric toothbrush, electric floss, oral center, etc.).
6. Tooth-powder.
7. Chewing gums.
8. IOHP for tooth whitening.

TOOTHBRUSHES

Practical classification of toothbrushes

By age of use: children, adolescent, adults.

By purpose of use: hygiene, preventative (periodontal), additional (special purpose).

By mechanism type: manual, mechanical, electric.

By design: straight, angular.

By indication: with indication and without it.

By bristle type: natural, artificial.

By bristle class (the material of the bristle): nylon, setron, perlon, derolon, silicon, mixed (a combination of different stiffness degrees bristles), combined (coated), microtextural (with twisted bristle)

By bristles stiffness:

"Sensitive" (for patients with pathological sensitivity)

"Soft" (for children, patients with hemophilia and postoperative oral wounds)

"Medium" (for main group of patients)

"Hard" (whitening and toothbrushes for smokers)

"Extra Hard" (toothbrushes for orthopedic appliances)

"HS"; mixed (a combination of bristles of different degrees of hardness), combined (combination of several kinds of materials, changing the stiffness of the bristles).

By bristle beams position:

single-level,
two-level,
three-level,
multi-level;

By bristle tip processing:

rifled,
polished,
rounded,
honed,
combined.

By form of handle: flat, thin, narrow, round and combined (combination of several materials), mixed (a combination of different forms), resilient, tough.

By size of handle: children, teenager, adult ("Small","Medium", "Large").

By type of handle holding : corrugated, flat, convex, concave, mixed, horizontal, vertical, circular, combined, universal, special capture, mixed (a combination of several types of capture), combined (combination of several types of materials).

Toothbrushes for special purpose

"Ortho" toothbrushes (with V-shaped bristle recess) for patient with braces and

"Sulcus" toothbrushes have long narrow head on the surface of which there are two longitudinal rows of bristle beams. Their function is to support as an extra brush and contribute to a better cleaning of the teeth from plaque, food residues crowded teeth, single crowns or implants and other orthopedic and orthodontic constructions.

Electric toothbrush

An electric toothbrush is a toothbrush that makes rapid, automatic bristle motions, either circular, reciprocating, pulsating motion (where the brush heads alternates clockwise and counterclockwise rotation), in order to clean teeth. Electric toothbrushes can be classified according to the frequency (speed) of their movements as power, sonic or ultrasonic toothbrushes, depending on whether they make movements that are below, at or above the audible range (20–20,000 Hz or 2400–2,400,000 movements per minute), respectively.

Studies have shown that electric toothbrushes do not have any advantages over manual toothbrushes in cleaning efficiency. In this regard, the use of the electric toothbrush can be recommended to patients with physical disabilities or poor manual possibilities.

Ionic toothbrush

Use of devices with ionic action in the oral cavity is not a new concept. The terms ionophoresis, electrophoresis, and electrolyzing have been used in dentistry for many years. Ionic toothbrush is the manual toothbrush, in common sense, with replaceable brush-heads, and works on the principle of changing surface charge of tooth to repel plaque even from inaccessible areas of teeth. It is also speculated that the activated anions might inhibit coupling between the pellicle and bacteria, mediated by calcium bridges. The important ionic exchange with normal mechanical action of the bristles on the tooth surface enhances plaque removal.

Ultrasonic toothbrush

An ultrasonic toothbrush is an electric toothbrush that operates by generating ultrasound in order to aid in removing plaque and rendering plaque bacteria harmless. It typically operates on a frequency of 1.6 MHz, which translates to 96,000,000 pulses or 192,000,000 movements per minute.

DENTAL FLOSSES

Dental floss is a cord of thin filaments used to remove food and dental plaque from between teeth in areas a toothbrush is unable to reach (from interdental space mostly).

Classification of flosses:

By form

- round (wide interdental spaces)
- flat floss (narrow interdental spaces)
- flat tape (crowded teeth)

By surface treatment type

- waxed (has a high sliding ability to easily penetrate into the interdental spaces; recommended for patients with tight interdental contacts and a large number of proximal restoration.)
- unwaxed (has better cleaning ability as compared with waxed because swelling in gingival liquid fibers effectively remove plaque from the interdental space.)

By impregnation

- without impregnating
- impregnated (with chlorhexidine, sodium fluoride, menthol)

Factors to be considered when choosing the right floss or whether the use of floss as an interdental cleaning device is appropriate may be based on:

- The tightness of the contact area: determines the width of floss
- The contour of the gingival tissue
- The roughness of the interproximal surface
- The client's manual dexterity and preference: to determine if supplemental device is required

Specialized plastic wands, or floss picks, have been produced to hold the floss. These may be attached to or separate from a floss dispenser. While wands do not pinch fingers like regular floss can, using a wand may be awkward and can also make it difficult to floss at all the angles possible with regular floss. These types of flossers also run the risk of missing the area under the gum line that needs to be flossed. On the other hand, the enhanced reach of a wand can make flossing the back teeth easier.

Dental floss is the most frequently recommended cleaning aid for teeth sides with a normal gingiva contour in which the spaces between teeth are tight and small. The dental term ‘embrasure space’ describes the size of the triangular-shaped space immediately under the contact point of two teeth. The size of the embrasure space is useful in selecting the most appropriate interdental cleaning aid. There are three interproximal embrasure types or classes as described below:

The classification of the amount of gum that fills the spaces between the teeth

- Type I – the gums fills embrasure space completely
- Type II – the gums partially fills embrasure space
- Type III – the gums do not fill embrasure space

Floss stick (floss pick)

A floss pick is a disposable oral hygiene device generally made of plastic and dental floss. The instrument is composed of two prongs extending from a thin plastic body of high-impact polystyrene material. A single piece of floss runs between the two prongs. The body of the floss pick generally tapers at its end in the shape of a toothpick. Floss picks are manufactured in a variety of shapes, colors and sizes for adults and children. The floss can be coated in fluoride, flavor or wax.

INTERDENTAL BRUSHES

An interdental or interproximal ("proxy") brush is a small brush, typically disposable, either supplied with a reusable angled plastic handle or an integral handle, used for cleaning between teeth and between the wire of dental braces and the teeth. There is evidence that, after tooth brushing with a conventional tooth brush, interdental brushes remove more plaque than dental floss.

Classification of interdentalbrushes:

By form - conical and cylindrical

By stiffness - hard and soft (for patient with dentine sensitivity and implants)

By size (size depends on interdental space measured by calibration probe)

Method of usage: brush is introduced into each interdental space and cleaned it by rotation clockwise.

TOOTHPICK

A toothpick is a small stick of wood, plastic, bamboo, metal, bone or other substance used to remove detritus from the teeth, usually after a meal. A toothpick usually has one or two sharp ends to insert between teeth. Toothpicks are classified as plastic, wooden and bone (by material), round and triangular (by section form). Disadvantage of toothpick using is trauma of gingival papillae.

INTERDENTAL STIMULATORS

Interdental stimulators - rubber or plastic cones attached to the handle. Rubber stimulants are more safety. They are used to massage gums and clean interdental spaces and cervical dental areas. Patient should make circular movements in the interdental space with light pressure on the papilla.

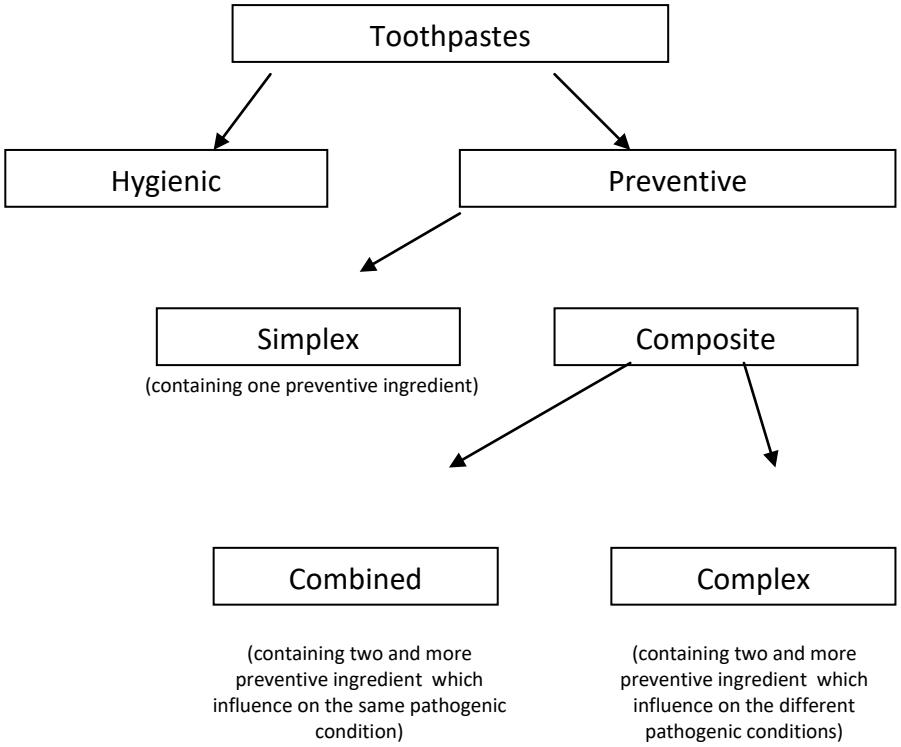
ORAL IRRIGATORS

An oral irrigator (also called a dental water jet) is a home care device that uses a stream of pulsating water to remove plaque and food debris between teeth and below the gumline and improve gingival health with massage and increasing of local blood circulation.

TOOTHPASTES

Toothpaste is a dental hygiene product used with a toothbrush as an accessory to clean and maintain oral health by abrasive removing the dental plaque and food from dental surfaces.

CLASSIFICATION OF TOOTHPASTES (BY S.B. ULITOVSKIY)



Preventive toothpastes includes:

- anticaries toothpastes;
- paste affecting the oral soft tissue;
- antitartar pastes;
- antiplaque pastes;
- pastes desensitizing dental hard tissues;
- whitening pastes;
- odor control (desodoration) pastes.

Toothpastes contain active ingredients or additives that perform specific functions. These additives are abrasives, fluorides, desensitizing agents, antiplaque agents, and antitartar ingredients. Toothpastes also contain detergents, humectants, thickeners, preservatives, flavoring agents, sweeteners, and coloring agents. The exact composition of a particular toothpaste varies with each manufacturer, but a typical formulation is abrasive 10-40%, humectant 20-70%, water 5-30%, binder 1-2%, detergent 1-3%, flavour 1-2%, preservative 0.05-0.5% and therapeutic agent 0.1-0.5%.

1. **Abrasives** (SiO_2, $CaCO_3$, $NaHCO_3$) perform the primary functions of removing plaque and stain from teeth.

2. **Commonfluorides** in toothpastes include stannous fluoride,sodium monophosphate fluoride, organic compounds of fluoride (amine fluor) and sodium fluoride. Fluoride's primary action is to be incorporated into the tooth substrate

(enamel and dentin) making the tooth more resistant to acid attack by cariogenic bacteria. Fluoride is alsobactericidal and has additional antiplaque effects.

3. **Desensitizing agents** are active ingredients, usually potassium nitrate, in toothpaste that reduce dentin hypersensitivity through a depolarizing effect on the odontoblastic processes in the dentinal tubules. The nerve endings of the odontoblastic processes then repolarize and have a reduced pain sensing ability. Also, desensitizing effects of arginine bicarbonate calcium carbonate complex and stabilized stannous fluoride have been demonstrated to provide dentin desensitizing effect.

4. **Antiplaque agents** reduce plaque growth. This can have a positive effect in reducing plaque on teeth, reducing gingivitis, and potentially reducing caries. Some antiplaque agents include triclosan, papain, and sanguinaria extract. Triclosan has been accepted by FDA as an antiplaque-antigingivitis therapeutic additive to toothpastes.

5. **Antitartar ingredients** that reduce calculus buildup on teeth include tetrapotassium pyrophosphate, tetrasodium pyrophosphate, disodium pyrophosphate, papain and citroxaine.

6. **Remineralizing agents** have recently been added to toothpastes. These remineralizing agents are based upon amorphous calcium phosphate. This soluble calcium and phosphate are described as enhancing remineralization, preventing dental caries, reducing enamel and/or dentin erosion, and reducing dentin hypersensitivity. The mode of action that has been hypothesized for these agents is the calcium and phosphate in soluble form which allows it to bind to enamel and dentin and to dental plaque. While there are laboratory studies demonstrating these effects, there is little clinical evidence to support these claims.

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VIII. METHODS OF INDIVIDUAL ORAL HYGIENE

The basis of oral hygiene is the mechanical removing of dental plaque from dental surfaces, gingival margin, as well as prostheses and orthodontic appliances.

Methods of mechanical cleaning of oral cavity include:

1. **Self-cleaning** (by saliva and during rough food chewing)
2. **Rising**

The easiest way to mechanically cleanse the mouth and remove food debris and soft deposits, but not dental plaque.

Method is used in case of blood clotting disorders (decompensated form), acute inflammation of the oral soft tissues, severe general condition of the patient, after snacks during the day, before and after brushing, after vomiting episodes (toxemia of pregnancy, bulimia etc.).

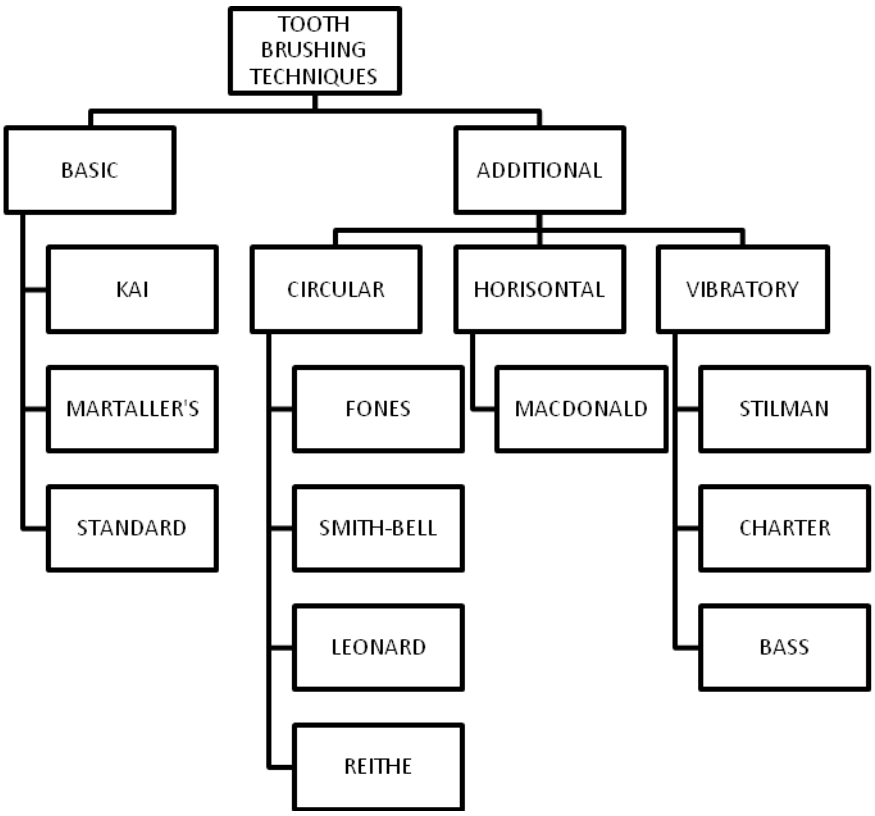
3. **Wiping**

Wiping is more effective than rinsing because part of dental plaque is removed with it. Tools to wipe teeth include gauze moistened with water, antiseptic or hygienic foam, special wipes and silicone fingertip according to indications. Wiping is the first hygienic procedure for child. Parents should start to wipe child's oral mucosa of cheeks, tongue and alveola after first feeding 2 times per day.

4. **Brushing** implementation begins from first tooth eruption. The algorithm of all types of brushing techniques includes the of dividing of tooth arch to segments and 10 times repetition of cleaning motions in every segments (from upper jaw S1 to lower jaw S6). There are 4 segments (S1-S4 excluding premolars) in primary dentition and 6 (S1-S6) in permanent dentition.

The dental arch divides into 6 parts*:*

- right molars (S1)
- right premolars (S2)
- right canines and incisors (S3)
- left canines and incisors (S4)
- left premolars (S5)
- left molars (S6)



Scheme №2 «Toothbrushing techniques»

1. KAI (german abbreviation from occlusial-vestibular-oral surfaces names according to sequence of brushing). This method is indicated for children under 6 years and persons with a low level of psycho-physiological ability.

Occlusial surfaces are cleaned with ***horizontal reciprocating*** motions. ***Vestibular*** surfaces in closed jaws position are brushing with ***circular*** motions. Motions for ***oral*** surface are ***sweeping.***

2. PR. MARTALLER's METHOD

Used for junior schoolchildren. Look like simplified standard method starting from occlusal surfaces because of children make maximum efforts at the beginning of the teeth cleaning procedures and can injure a periodontium. ***Occlusial-vestibular-oral*** sequence of brushing.

Occlusial surfaces are cleaned with ***horizontal reciprocating*** motions. ***Vestibular*** surfaces in closed jaws position are brushing with ***zigzag*** motions. Motions for ***oral*** surface are ***sweeping.***

If a patient has orthodontic braces he should divide the brushing motion to short strokes cleaning the space between the gingival margin andnon-removable elements.

3. STANDARD METHOD

This method is suitable for adolescences and adults without dental disease and physic and psychological specialties.

Vestibular-oral-occlusial sequence of brushing including 10 times repetition of all types of motion in every segment (S1-S6) of upper and lower jaw.

On all surfaces sequentially they make the motions:

1) sweeping vertical - to remove plaque from the cervical areas and smooth surfaces of crowns, including contact;

2) horizontal – to brush natural enamel recesses;

3) circular - to cleanse natural enamel recesses and massage the gingival margin.

4. BASS METHOD:

- Most effective in cleaning cervical 1/3, gingival margin and sulcus;
- Includes light periodontal massage.

Vestibular-oral-occlusial sequence of brushing. Direct bristles apically to sulcus at 45° to the long axis of the tooth. Use gentle force to insert bristles into sulcus. On the lingual surfaces of front teeth, brush is placed vertically and apply gentle vibratory motion of up-and-down strokes. On the chewing surface apply a motion of back-and-forth brushing strokes.

5. STILLMAN'S METHOD:

- Provides gingival stimulation (vibratory motion);
- Suitable for gingival recession (Toothbrush bristle ends not directed into sulcus);
- Less traumatic to the gingiva.

Vestibular-oral-occlusial sequence of brushing. Direct bristles apically to sulcus at 45° to the long axis of the tooth . Place bristles partly on the cervical part and on the gingiva. Apply vibratory motion with slight pressure to stimulate gingiva. Repeat for the lingual surface of the tooth. Use short back-and-forth strokes on the occlusal surfaces gently. Place toothbrush perpendicular to the tooth surface.

6. CHARTER'S METHOD:

- Efficiently cleans interproximal areas;
- Able to clean areas between fixed appliances (prosthetic and orthodontic) and gingival margins
- Useful for persons after periodontal surgery (temporary cleaning of surgical wounds).

Place bristles horizontally and parallel to the arch at 45°at the gingival margin. Direct bristles toward the crown of the tooth rather than the root. Bristles are directed occlusally and vibrate into the interdental spaces. Use short back-and-forth strokes for activation. Repeat for other parts of the mouth until all areas are cleaned.

7. MACDONALD METHOD (method of 3 arches).

Most common method but has no benefits because it causes gingival recession. Move bristles horizontally using gentle scrubbing motions.

8. FONES' (CIRCULAR) METHOD:

- An easy-to-learn first technique for young children.

Teeth are held in occlusion. Bristles are activated in a circular motion.

9. LEONARD METHOD

Vestibular and oral surfaces are cleaned by vertical motions, for chewing surfaces there are used horizontal motions (without extreme force). In contrast to the **Reithe** method, vestibular surfaces are cleaned with closed teeth arches, and the bristles are always perpendicular to the surface of teeth. The advantage of this method is low risk of gum injury.

The choice of methods of dental plaque mechanical removal for particular patient should be based on the knowledge of his mental and physical condition, age, somatic, dental and periodontal status (risk factor, disease, health maintenance, postoperative condition), mature of enamel, level of hard tissues abrasion, quantity and quality of dental plaque. The level of mental development, coordination of movements is important to know to determine the maximum technical complexity of methods, which is available to the patient.

In case of periodontal disease in remission toothbrush with medium hardness bristles is recommended to use a, during acute period - with soft bristles. For patients with initial signs of periodontal disease the main method of brushing is Bass techniques, with gingival recession - Steelman method, in the presence of pathological pockets and tremas - Charter method.

IX. ENDOGENUOUS PREVENTION OF CARIES.

Types of preventive measures:

1 Endogenous (common, systemic)
2 Exogenous (local, topical)

1. Pre-eruptive (prenatal, postnatal)
2. Post-eruptive

1. Medicinal (tablets, solutions for systemic and local application)
2. Non-medicinal (diet, resistance improving, etc.)

Pre-eruptive prevention of dental diseases is the complex of measures directed to prevent caries in children during natal development and early life (before teeth eruption).

Pre-eruptive caries prevention includes prenatal and postnatal (post-birth to teething) preventive measures:

1. A complete, balanced diet (a sufficient amount of vitamins and minerals) and regular meal regime;

2. Prevention of infectious and somatic maternal diseases during pregnancy and the early childhood diseases;

3. Fluoride-containing endogenous sources (water, salt, milk, tablets) according to indications and natural content;

4. Additional sources of vitamins and minerals for optimal daily intake (vitamins D, A and C, calcium, phosphorous, magnesium, etc.);

5. Management of organism resistance according to indications (adaptogens, immunostimulators).

Norm of fluoride consumtion – 0.5-4.0 mg/day: south climate (warm area – 0,5-0,7), north climate (cold, moderate – 0,8 – 1,2)

0-6 month - 0.01
7-12 month - 0,5
1-3 years - 0,7
4-8 years - 1,0
9-13 years - 2,0
14-18 years 3,0
Men older than 18 years - 4,0
Women older than 14 years - 3,0

Natural fluoride sources are water, air, food. Additional endogenous sources for areas with fluoride concentration in water <0,3 mg/l include:

- fluoridated water (40-60% of caries reduction efficiency)
- fluoridated salt (40-60%)
- fluoridated milk (20-30%)

- fluoride medcine: tablets, drops (80-90%)

Fluoride tablets prescription features:

- 9 month per year excluding summer period
- from 3 years of life
- 3-6 years – 1,1 g per day (1 tablet)
- 6-15 years – 2,2 g per day (2 tablet)
- chewing up, without drinking with diary product

Monitoring of fluoride consumption

1. Urinary fluoride excretion test (more accurate): Twenty-four-hour urine samples were collected from patient under customary conditions of fluoride intake, determine the fluoride concentration with ionometer, calculate the total load and compare with standard fluoride doses.
2. Analysis (calculation) of daily fluoride load

Diet is a main source of calcium consumption. Additional sources can be composed by: calcium monotherapy, complex of calcium and vitamin D (other minerals, for example, Mg), complex of vitamins and minerals (including calcium)

It is should to use soluble calcium salts: lactate gluconate (smaller), because of calcium is absorbed poorly from calcium chloride and calcium caseinate (from dairy products).

Vitamin D (ergocalciferol) regulates phosphorus and calcium metabolism, facilitating their absorption in the intestine and its temporary deposition in bone tissues. Vitamin D, which daily dosage including natural sources for a pregnant woman is 400 IU, plays an important role at caries prevention and proper dental and bone tissues formation

In case of insufficient intake of vitamins from food or digestive diseases it is necessary to prescribe multivitamin complex with mineral additives (for example, "Oligovit"," Revit "," Multitabs "," Alphabet "," Vitus "," Supradin " and others). Multivitamin supplements with minerals, vitamin supplements should be approved by therapist or pediatrician.

The prescription of endogenous caries prevention sources is possible only in case of a doctor entering information on the micro, macro elements and vitamins in daily consumption load, physical health and other pharmacological admissions of a patient. The main purpose is to prevent overdose of fluoride and vitamins and also not to harm the patient.

METHODS OF PRE-ERUPTIVE PREVENTION

1. Medical education of pregnant woman, future and young parents and children (at dental offices, women clinics).

2. Prevention of somatic and infectious diseases during pregnancy and early life (especially, with teratogenicity influence -rubella, toxoplasmosis, etc.). Treatment of current diseases.

3. Refuse a harmful habits during pregnancy.

4. Healthy diet and lifestyle.

5. Preventive dental appointment for future mother (no less than 3 per pregnancy) and her child.

6. Early indication of risk factors of oral diseases.

7. Proper oral hygiene in children before teeth eruption to eliminate pathological microflora.

X. CALCIUM-, PHOSPHATE- AND FLUORIDE-CONTAINING SOURCES FOR EXOGENOUS PREVENTION OF CARIES.

Calcium\phosphate-containing products for topical (post-eruptive) prevention of caries.

High levels of calcium and phosphate induces fast precipitation of calcium compounds and phosphate to the enamel surface pores thereby increase remineralization. There is a direct relationship between calcium concentration in the products and saliva and the level of enamel remineralization.

The need for long-term contact with the enamel of the Ca-P compounds is based on the low concentration usage. In order to increasing of calcium in demineralized enamel from 0,1mmol / l to normal level 30 mmol / L it requires 10,000 volumes of saliva or optimal concentration of calcium\phosphate solution.

Levels of pH influences on enamel demineralization-remineralization process:

1) Dissolving the most of calcium and phosphate compounds in supplements is required acidic environment;

2) For the precipitation of ionized components from supplements and saliva is favorable alkaline environment.

At the end of the 1990s it began to develop a basic "formula" of "calcium-phosphate technology" is widely used to prevent and treat caries and non-carious diseases (erosion, abrasion and hypersensitivity of teeth enamel).

Ca-P compounds are used in tooth pastes, vanishes, mouthrinses, professional gels (Belagel Ca\P, ROCS medical minerals), chewing gums (Orbit, Xylitol).

Calcium\phosphate compounds in preventive supplies:

1. ***ACP (amorphous calcium phosphate), ACP and ACP-CPP.*** Casein phosphopeptides (CPP) from the major protein of milk have the ability to stabilize calcium, phosphate and fluoride ions as water-soluble amorphous complexes that provide bioavailable calcium, phosphate and fluoride ions to the tooth. These complexes of amorphous calcium phosphate and amorphous calcium fluoride phosphate stabilized by the CPP have been demonstrated in a range of laboratory, animal and short-term human clinical trials to repair (remineralise) early stages of tooth decay by replacing the calcium and phosphate ions lost due to decay. CPP-ACP in saliva, significantly reduced caries experience in children compared with a control toothpaste. (Tooth mousse, MI paste)

2. Calcium gluconate. 10% calcium gluconate solution is the form of calcium most widely used in the treatment of hypocalcemia in medcine. But this form of calcium is not as well absorbed as calcium lactate, and it only contains 0.93% (930 mg/dl) calcium ion. Calcium gluconate is not efficient as topical preventive supplement in dentistry. (10% solution for professional prosedures)

3*. Hydroxiapatite (HAP).* Synthetic hydroxyapatite is similar in composition to the mineral component of bone and teeth and the most efficient preventive calcium\phosphate compound in contemporary dentistry. (Biorepair, SPLAT)

4. Calcium glycerophosphate.(CaGP) Calcium glycerophosphate has the potential to reduce the progression of caries via all of these mechanisms if it is applied frequently and at a sufficiently high concentration. Reduction of plaque mass has also been proposed as a cariostatic mechanism but this seems less likely. Animal studies have shown that the calcium glycerophosphate/sodium monofluorophosphate system can have a greater anti-caries effect than sodium monofluorophosphate alone and this was subsequently confirmed in a caries clinical trial. (ROCS)

5. MINERALIN (ROCS)

6. SensiStat® - complex calcium carbonate and arginine bicarbonate (Colgate)

7. Calcis- derived from the eggshell component of cosmetic products, include water-soluble calcium phosphate, acetate, lactate, succinate, citrate, and tartrate. (Splat)

8. Calcium lactate (President)

9. Calcium phosphate (Threecalcium phosphate -TCF) (SPLAT)

III Combined methods of post-eruptive caries prevention (Ca-P-F technologies)

I Fluoride-containing supplements for topical (post-eruptive) prevention of caries.

Fluoride is a mineral that occurs naturally in all water sources, even the oceans. The fluoride ion comes from the element fluorine. Fluorine, the 17th most abundant element in the earth's crust, is never encountered in its free state in nature. It exists only in combination with other elements as a fluoride compound.

Fluoride is effective in preventing and reversing the early signs of dental caries (tooth decay). Researchers have shown that there are several ways through which fluoride achieves its decay-preventive effects. It makes the tooth structure stronger, so teeth are more resistant to acid attacks. Acid is formed when the bacteria in plaque break down sugars and carbohydrates from the diet. Repeated acid attacks break down the tooth, which causes cavities. Fluoride also acts to repair, or remineralize, areas in which acid attacks have already begun. The remineralization effect of fluoride is important because it reverses the early decay process as well as creating a tooth surface that is more resistant to decay.

Fluoride is obtained in two forms: topical and systemic. Topical fluorides strengthen teeth already present in the mouth making them more decay-resistant. Topical fluorides include toothpastes, mouthrinses and professionally applied fluoride therapies.

Fluoride function in dentistry based on:

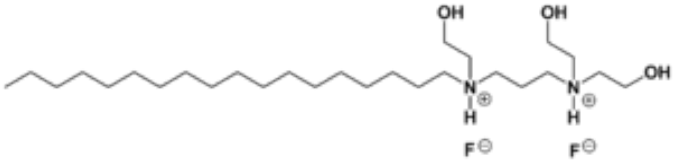
- remineralization

- bactericidal action
- violation of glycolysis in dental plaque

Fluoride compounds in preventive supplies:

1. ***Amine fluoride (Olaflur)*** is a fluoride-containing substance that is an ingredient of toothpastes and solutions for the prevention of dental caries. It has been in use since 1966. Especially in combination with dectaflur, it is also used in the form of gels for the treatment of early stages of caries, sensitive teeth, and by dentists for the refluoridation of damaged tooth enamel. Olaflur is a salt consisting of an alkyl ammonium cation and fluoride as the counterion. With a long lipophilic hydrocarbon chain, the cation has surfactant properties. It forms a film layer on the surface of teeth, which facilitates incorporation of fluoride into the enamel. The top layers of the enamel's primary mineral, hydroxylapatite, are converted into the more robust fluorapatite. The fluoridation reaches only a depth of a few nanometres, which has raised doubts whether the mechanism really relies on the formation of fluorapatite.

Formula 1:



Examples of supplies: «El-mex», «Lacalut», «Dentavit», «ROCS».

2. ***Stannous fluoride (Fluorostan)***

Stannous fluoride is the only fluoride source that provides protection against all 3 oral health conditions of plaque/gingivitis, tooth sensitivity, and cavities.

Formula 2:

Examples of supplies: «President », «Colgate », «Silca».

3. ***Sodium fluoride***

Formula 3: **Na-F**

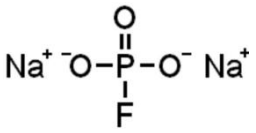
Examples of supplies: «Blend-a-med», «Crest», «»

4. ***Sodium monofluorophosphate (MFP).*** It functions as a source of fluoride via the following hydrolysis reaction:

$PO3F2^{-} + OH^{-} \rightarrow HPO4^{2-} + F^{-}$

Available literature on sodium monofluorophosphate and sodium fluoride suggests that sodium monofluorophosphate is hydrolysed in fluoride and phosphate ions. Monofluorophosphate is better tolerated than other common sources of fluoride such as sodium fluoride and stannous fluoride.

Formula 4:



Examples of supplies: «Colgate» (Pr&G), «SPLAT», «Sesodyne».

Concentration of active compounds in dental preventive products and supplies:

1. Tooth pastes – 500 – 1500 p.p.m

2. ***Professional gels and mousses («ROCS medical 5000 p.p.m.», «Fluoresse», «Belagel F»)–*** from 5000 – 20000 p.p.m.

3. ***Varnishes (Fluoride varnish, Multifluoride, Bifluoridine, Fluoresse)*** **–** 20000 – 60000 p.p.m.

4. Solutions for rinsing (0,05, 0,1,0,2 % sodium fluoride, mouthrinses with fluoride) **-** under 500 p.p.m.

5. Solutions for application (1-2% sodium fluoride)– 10000 – 20000 p.p.m.

6. Foams – under 500 p.p.m.

7. Home self-applied gels and mousses («Elmex gel», « ROCS medical 5000 p.p.m.»)– 1500 – 5000 p.p.m.

8. Filing and other dental materials (GIC, fluoride containing resins, professional hygiene pastes) – 10 -25% by all material weight.

Supplements and methodics for local complex Ca-P-F-mineralizing prevention.

Solutions (classical methods):

• application of 10% calcium gluconate solution (15 min) + application of 2% sodium fluoride solution (by Borovsky - Leus);

• application of 2% sodium fluoride solution (3-5 min) + calcium gluconate (by Suntsov).

• application of 10% calcium gluconate solution (2-4 min) + rinse of 0.2% sodium fluoride or fluoride varnish application (by T.F.Vinogradova);

3-4 times per year for 30-40% caries reduction. The effectiveness of calcium and phosphates increases with electrophoresis combining.

Pastes and mousses for Ca-P-F remineralization. Pastes based on CPP-ACP and amine fluoride 900 ppmF («MI paste», GC, Japan) are recommended for children older than 6 years for application after evening tooth brushing. Treatment course: at least 1 month 2 times per year.

Gels containes 1,23% F and the ACP is not harmful to composite restorations and ceramics.

Varnishes.

Cleanpro, 3M; EnamelPro® Gel 1.23%, Enamel ProVarnish 5% are recommended for tooth tissues remineralization in case of caries and hypersensitivity. Treatment course: 2-3 applications with several days intervals repeated 2-6 times a year

according to the caries intensity of. Indications and usage rules of Ca-P-F-varnish do not differ from those of the fluorine-containing gels.

Deep fluoridation systems.

The system includes a set of two liquids (by Knapvost). One of them contains the fluoride compound and another is based on the calcium compound. After sequential application to the tooth surface insoluble compounds precipitates in enamel structure. («Enamel and dentine sealing liquid», Humanchemie, Germany; «Gluftored», Vladmiva, Russia; «Triftorid», Omegadent, Russia)

XI. ORAL FLUID: CARIESPROTECTIVE CAPABILITIES AND THEIR ASSESSMENT.

Condition of tooth enamel is largely determined by the characteristics of saliva and oral fluid.

Oral fluid contains mixed (of the three pairs of large and many small salivary glands) saliva with various inorganic and organic impurities (microbial and epithelial cells, food debris, and toxins al.).

Salivation during the day (≈ 500 ml.):

• ≈ 300 ml - basic (unstimulated), pruduced without meals

• ≈ 200 ml - stimulated saliva (mainly the parotid gland), during meals

All time in the oral cavity there is about 0.5 ml of saliva and tissue is covered with film (0.1 ml. / min. and fully renovated for 4 - 5 minutes after swallowing action). The trigger for salivary glands is the irritation of periodontal mechanoreceptors, masticatory muscles proprioceptors and taste buds.

The composition of saliva. Mixed saliva is an solution of organic and mineral substances:

- 99.5% of water.

- 0.22% of inorganic components (H, K, Na, Ca, Mg, Fe, Cu, P, S, Cl, F et al.). In an oral liquid, they can be in the form of simple and complex ionized ions and in the contain of organic compounds (proteins, protein salts, chelates etc.).

- 0.28% of organic substances: simple proteins (albumins, globulins), complex proteins (glycoproteins), non-protein nitrogen components (amino acids, urea), monosaccharaides and their products (organic acid).

Functions of saliva.

1. Protective

• Buffer function

• Moistening of oral mucosa and teeth surfaces, glycoproteins (mucin), electrolytes

• Antimicrobial immunity and protection (lysozyme, lactoferrin, lactoperoxidase, IgA, glycoproteins)

• Oral mucosa and teeth surfaces washing

2. Trophic - mineralization and remineralization of enamel (calcium, phosphate, anionic protein and other minerals)

3. Digestive -mechanical and chemical food processing, preparing for flavor assessment (mucin,water, gustin, amylase, maltase)

4. Speech -lubrication (water, mucin)

The salivary glands have different composition and quality of their secrets:

1) parotid

• phosphates (high level);

• carbonate buffer (average level);

• amylase, catalase (high level).

2) submandibular and sublingual

- phosphates (high level)
- carbonates and phosphates (high level);
- amylase (low level).

3) small salivary glands

- phosphates (low level);
- lack of buffer capacity.

Properties of saliva.

Table №17 «Properties of saliva»

Options	Unstimulated saliva	Stimulated saliva
Rate of salivation ml/min	0,25 – 0,31	1 – 3
pH	6,5 – 6,9	7,0 – 7,5
Buffer capacity	4,25 – 4,75	5,75 – 6,5

The functional structure of the saliva.

The structural unit of the oral fluid is a micelle.

1. The core is Ca3 (PO4) 2
2. The adsorption layer:

a) the potential-determining ions (HPO42-)

b) counter ions (Ca2 +)

3. The diffuse layer (water, glycoproteins) protects the micelles from the agra-navigation.

Tree form crystals can be explained by the presence of salivary mucin having a branched structure. Such structure allows to isolate ions from each other and save their reactivity.

The viscosity of the saliva.

The viscosity of saliva (due to mucin) promotes the formation of a protective film on the surface of the enamel and mucosa (pellicle), but makes it difficult to penetrate into the narrow space (fissures, contact points, the elements of orthodontic appliances).

The enamel mineralization by saliva.

Saliva nourishes the tooth as blood nourishes body. Enamel is crystals in the solution of its own ions. The properties of these crystals (dissolving) depend on the concentration of ions (calcium, phosphate, hydroxyl groups) in the saliva and its acidity. Ion concentration is controlled by homeostatic factor and neurohumoral mechanisms. The average concentration of calcium

- in saliva - 1.15 x 10-3 mmol / L.
- in blood serum - 2.5 x 10-3 mmol / L.

The average concentration of phosphorus

- in saliva - 1.10 x 10-3 mmol / L.
- in blood serum - 6.03 x 10-3 mmol / L.

Thus, due to the different concentrations of calcium and phosphorus, blood supersaturated with hydroxyapatite in 2 - 3 times, saliva – in 4 - 5 times. Consequently, saliva has a large mineralizing potential. Protein-bond ions are involved in the micelles (45% of calcium and 6% of phosphates) and therefore not precipitate. Ions from a supersaturated solution introduced into the hydration layer of enamel apatite creating a depot and penetrate from it into enamel. In the presence of fluoride ions the rate of these processes increases. Enamel appetites change qualitatively (ions Mg, Cl, hydroxyls are replaced with fluoride) forming less soluble crystals. With age the degree of mineralization of the enamel (calcium content) increases under favorable conditions:

• 6 years - the ratio of Ca: P in the first permanent enamel painters 1.51;

• 10 years - 1.71.

The acidity of saliva (concentration of H +) defines the parameters of colloidal systems (osmosis, dispersion, precipitation of salts or their suspended state and so on). By increasing the pH of the saliva H + ions react with phosphate ions, transforming it into HPO_4^{2-}. Consequently the concentration of free ions PO_4^{3-} decreases.

The saliva pH is 6.2 - 7.4 (in children it is more alkaline, in aged people is more acidic). The optimal pH of saliva for reminiralisation 7 or more. If pH <7 demineralization occurs. The pH is maintained by numerous of saliva buffer systems (bicarbonates, phosphate ions, glutamate, ammonia create chemical bonds with food substances and neutralized acidic of oral liquid and even plaque).

Antimicrobial protection.

• Immunoglobulins;

• Enzymes provide bacteriostatic and bactericidal effects:

- Lysozyme dissolves walls of bacteria;
- Lactoferrin binds iron required for the growth of many bacteria;
- Lactoperoxidase forms the bactericidal compound (gipotiotsianat).

1**. The rate of salivation**. (T.L.Redinova, A.R.Pozdeev, 1994).

It is necessary graduated tube and stopwatch to study the rate of salivation. Patients are asked to tilt his head, open his mouth and allow saliva to flow freely into the tube without swallowing the oversight of the lower lip. Start and end of saliva collection are noted with stopwatch (usually 5 - 15 min.). The rate of salivation is calculated as $RS = V / t$,

if RS - the rate of salivation;

V - volume of released saliva in ml .;

t - time of collecting saliva in min.

Normal speed unstimulated salivation - 0.31 - 0.6 ml / min., hyposecretion - 0.03 - 0.3 ml / min., hypersecretion - 0.61 - 2.40 ml / min .

2. The viscosity of saliva (T.L.Redinova, A.R.Pozdeev, 1994). It is necessary 1,0 ml micropipette and stopwatch to study viscosity of saliva.

$Vs / Vw = Vis / Viw$, which means $Vis = (Vw \times Viw) / Vs$,

where Vw - volume of water expired in ml .; Vs- volume of expired saliva in ml .; Vis - the viscosity of saliva.; Viw - water viscosity

Optimal value is 1.0 - 4.0 (in specific units)

3. Determining of saliva acidity

1) I*ndicatory method*: a standard test strip impregnated with saliva and its pH is determined by comparative school colors.

2) *Potentiometric method* (with ionomer using*)* is more accurate.

Normal pH is 6.5 - 7.7.

4. Determination of buffering capacity of saliva requires special paper strips soaked in acid and acid-base indicator. A drop of saliva from the pipette is applied to the strip. Saliva neutralize the acidic impregnating strips (demonstrated by a color change indicator). In most cases, the initial level of acid pH = 4.5 corresponds to yellow-brown color. pH = 4.5 - 5.5 - green, pH = 7 - blue.

5. The method of determining of the **mineralizing potential of saliva** assess form and structure of crystals formed by slowly drying droplets of saliva.

From the bottom of the oral cavity with a pipette gaining unstimulated saliva and applied to a glass slide. Saliva dries in air with room temperature or in a thermostat. Dried drop microscope with low magnification (2 x 6). Interpretation of evaluated results is following:

1 point - a scattering of randomly arranged structure of irregular shape;

2 points - a fine grid of lines across the field of view;

3 points - some crystals of irregular shape on the background grid and clumps;

4 points - a medium-sized tree crystals;

5 points - the crystal structure is clear, large, like a fern or parquet.

The value divided into 3 and interpret:

0 - 1 - very low, 1.1 - 2.0 - low, 2.1 - 3.0 satisfactory 3.1 - 4.0 -high, 4.1 - 5.0 - very high.

Crystal properties of saliva depend on the physical condition, so they are quite volatile.

6. **The clinical definition of enamel remineralization speed** by saliva Assessment due to the enamel dissolving by acid. It is required a standard acid solution for enamel etching, dye (2% solution of methylene blue) and a stopwatch.

A drop of the acid solution is applied on cleaned, dried and isolated enamel of central incisor for 60 seconds. Then the solution is washed, dried and enamel etched zone is applied with colorant for 60sec. The colorant is removed with a cotton swab, the color intensity is evaluated by10-point scale. Daily determine the intensity of staining (without re-ecting). Notes how many days it took to enamel complete reminiralisation with saliva. For good mineralizing properties of saliva it needs 1 - 3 days, for bad - more than 5 days.

XII. PREVENTION OF PIT-AND-FISSURE CARIES.

The problem of fissure caries (FC) is the leading in cariesology as FC is the most frequent among all carious lesions sites.

The high prevalence of FC associated with the anatomical shape of fissures, long period of maturation in comparison with the smooth surfaces of the teeth, retention of food debris, lack of high-quality dental care possibilities, complicated diagnosis, in most cases.

FC prevention is closely linked with the enamel maturity level. It is established that FC prevention efficiency depend on:

- determining the optimal timing of prophylaxis implementation,
- remineralization supplement composition,
- regularity of complex preventive measures.

Fissure caries (FC) has the most frequent lesion location. 80% of fissure cavities occur in 5 - 15 years old children. The prevalence of the initial FC occurence during 1-3 years after tooth eruption and enamel maturation is 99.03%. Most fissures (51.31%) decaded during the first year of maturation, 22.68% - during the second year. Progression of the initial caries into cavity lesion occurs in 20.37%of cases.

Risk factors and causes of fissure caries:

1. Hypomineralization and increasing hard tissue solubility of fissures during first years after the eruption;
2. Formation of high acid concentration zones near the fissure;
3. Decreasing of self-cleaning in the fissures;
4. Configuration of fissures;
5. Difficulties of fissure caries diagnosis;
6. Small thickness of the enamel in the fissure;
7. Poor oral hygiene.

Fissure classification:

I. Localization

1. Fissures of premolars and molars occlusal surfaces.
2. Fissures of the lower molars buccal surfaces and the upper molars palatal surfaces.
3. Blind pits on the palatal surfaces of the upper lateral incisors.

II. Depth (the depth of the fissure width may vary from 0.006 to 3.0 mm., An average diameter of 0.17 mm)

1. Small (shallow) – less than l / 2 enamel thickness.
2. Medium – more than1/2 the thickness of the enamel.
3. Deep - separate to the enamel-dentine junction less than 2 mm.

4. Complete (separate across the all enamel thickness)

III. Form
1. Funnel
2. Cone
3. V-shaped
4. U- shaped
5. Hemisphere
6. Test-tube form
7. Sack -shaped
8. Droplet
9. Polypoid
10. Multiple horns
11. Flask-shaped
12. Inverse Y- shaped

Open type fissures (flat, funnel, cone, V-shaped, U- shaped, hemisphere***):***

- good ability of visual inspection by the agency of probing
- well washing with oral liquid;
- low accumulation of dental plaque,
- no requirement of sealing

Closed type fissures:

- low caries resistance of fissure enamel
- pure ability of self-cleaning and tooth brushing
- high accumulation of dental plaque
- low rapid of mineralization (provided by the pulp, not saliva!)

IV. Level of mineralization

- Low
- Medium
- High

V. The shape of the external fissure orifice (the diameter in the average is 0.17 mm):

• Round
• Oval
• Triangular
• Other

Diagnosis of fissure status

Methods:
1. visual (examination, TER-test);
2. tactile (probing);

3. X-ray;
4. electrometry;
5. transillumination;
6. laser fluorescence (DIAGNODDENT (Kavo)).

Electrometric method is based on the ability of hard dental tissues to conduct electric current. Teeth with varying degrees of maturity hard tissue have different conductivity (electrometry value):

1) High ILM - (EM is less than 8 mA). Fissure enamel is shiny and smooth, probe slides across surfaces.

2) Medium ILM - (EM = 9 - 20 mA). Single fissures with chalky opaque shade, probe sticks in 1-2 deep fissures). Chance of caries – 80%.

3) Low ILM- (EM is more than 20 mA). Enamel without gloss, fissures are whitish, opaque, probe sticks in 2-3 deep fissures). Chance of caries – 100%

Fissure maturation terms:

- Upper and lower premolars –5 years after eruption,
- Upper molars - 4-6 years after eruption,
- Lower molars - 5-6 years after eruption (it is depended on the individual oral care and frequency of carbohydrate intake).

In every clinical case for proper prevention strategy implementation a dentist should determine:

1. The level of caries occurence risk in patient.
2. The level of caries occurence risk for certain tooth.
3. The tissue condition in pits and fissures.

<u>Assessment of caries occurence risk in patient includes examination:</u>

* Common health
* PEC parents, brothers and sisters
* Rate of salivation **
* Frequency of carbohydrates consumption**
* Regularity of dental visits **
*Caries intensity (DMF)**
* Presence of caries / fillings in fissures
* Fluoride using (topical and endogenous)**
* Level of oral hygiene **

<u>Assessment of caries occurence risk for certain tooth:</u>

* Morphology of the pits and fissures
* Hard tissues condition**
* Ability to cleanse the teeth (self-cleaning or with toothbrush)
* Clinical signs of dental caries in fissures**

Signs of high risk in children:

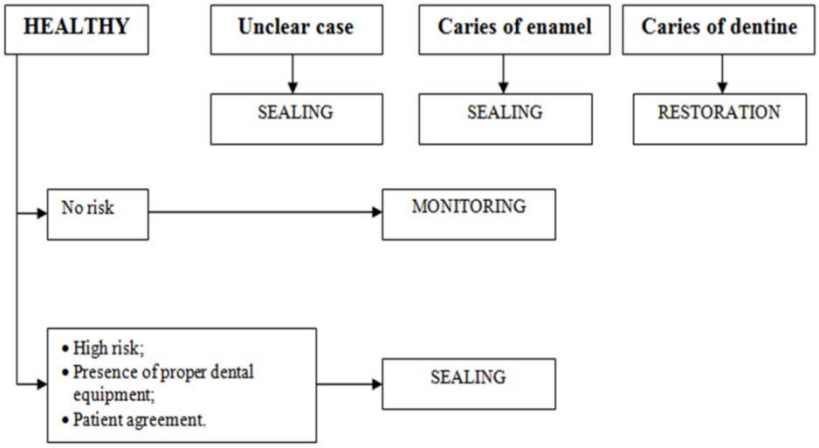
manifestation of ≥2 acute or recurrent caries in the last year, and / or the presence of ≥2 marked * risk factors

Signs of high risk in adults:

a manifestation of ≥2 carious lesions during the past 3 years, and / or the presence of ≥2 marked **risk factors

Status of dental tissues in the pits and fisssures:

- healthy
- doubtful
- with enamel caries
- with dentine caries



Scheme №3 «Fissure caries management»

Signs of a high chance of caries occurence:

• high intensity of teeth / surfaces caries;
• carious other tooth surface;
• poor oral hygiene;
• incorrect diet and poor quality of food;
• change in the color of tissues;
• significant post-eruptive age of the tooth (2-3 years).

Fissure sealing is fissure caries prevention method consist in:

Covering of immature fissure hard tissues by dental material with remineralizing properties;

Forming a mechanical barrier between the immature dental tissues and aggressive factors of the oral cavity.

The purpose of sealing is to:

1. form a barrier against cariogenic bacteria
2. remineralize immature or hypomineralized enamel
3. reduce a dental caries

Sealing Methods:

1. Non-invasive sealing (with full preservation of tooth tissues)
2. Invasive sealing (with significant excision of healthy and diseased tissues)
3. Preventive fissure sealing.

Materials for fissure sealing include:

- Glass ionomer cements (all types besides lining GIC)
- Resine composite (flowable composites, composite sealants)
- Compomer (polyacid modified resin) sealants

GIC materials bond both to enamel and dentine after being cleaned with polyacrylic acid conditioner. Some other advantages GIC's have is that they contain fluoride and are less moisture sensitive, with suggestions being made that despite having poor retention, they may prevent occlusal caries even after the sealant has fallen out due to their ability to release fluoride.

Indications for GIC sealants:

1. Unsatisfactory oral hygiene
2. Inability of proper isolation
3. Non invasive sealing procedures
4. High caries intensity level

Examples: "Vitremer", "Fuji Triage", "Ketak Molar", etc.

Resin based sealants classification:

- First generation: set with UV curing.
- Second generation: chemical-curing (autopolymerized).
- Third generation: visible light-cured.
- Fourth generation: contain fluoride.

Indications for resin based sealant:

1. Good oral hygiene
2. Possibility of good isolation
3. Invasive sealing procedures
4. Low caries intensity level

Non-invasive fissure sealing is the caries prevention method which consists in forming of covering sealant barrier on tooth occlusal surface.

Indications:

- Intact fissures;
- Low activity of dental caries;
- Good oral hygiene
- Early term after eruption of teeth (not more than 1 - 2 years);
- Available informative diagnostic procedures;

- The absence of demineralizing areas in the fissure.

Technique stages:

1. Detection of occlusal contacts
2. Cleaning
3. Isolation
4. Antiseptic application
5. Drying
6. Filling
7. Test of occlusal contacts
8. Coating (postbonding)
9. Fluoride application

Invasive fissure sealing is the treatment and prevention method which includes preparation opening of fissures with special atraumatic ("safe") instruments and their filling with sealant when non-invasive measures aren't efficient.

Indications:

- Optional method in case of long term (>2 years) after eruption;
- Dark (coloring) and deep fissures;
- High intensity of caries;
- Good and satisfactory hygiene level

Recommended matherials: flowable composite resin, glass-ionomer cement, BisGMA-based sealant.

Instruments:

✓ Fissurotom (spetial carbide burs)

Developed in collaboration with Temple University, the Fissurotomy® instrument head shape allows early diagnosis and conservative preparation of fissure caries, often without the use of anesthesia.

✓ Low abrasive diamond conical burs

Technique stages:

1. Detection of occlusal contacts
2. Cleaning
3. Preparation
4. Isolation
5. Antiseptic application
6. Drying
7. Filling
8. Test of occlusial contacts
9. Coating (postbonding)
10. Fluoride application

Preventive fissure filling (sealing) is the methods of caries treatment which includes combining of:

- filling of cavity (in one or several decayed fissures and pits);
- invasive sealing of others fissures (health or pigmenting fissures and pits of the same tooth)

Indication:

- High caries intensity.
- Caries lesion in immature tooth.
- Possibility of MI (minimal invasive) treatment providing.

Technique stages:

1. Detection of occlusal contacts
2. Cleaning
3. Preparation
4. Isolation
5. Antiseptic application
6. Drying
7. Filling (lining + restoration+ sealing)
8. Test of occlusal contacts
9. Coating (postbonding)
10. Fluoride application

Assessment of the sealant status is held not less than 1 time in 6 months.

XIII. PRIMARY PREVENTION OF NON-CARIOUS LESIONS OF HARD DENTAL TISSUES, TEETH TRAUMA.

Definitions of tooth wear

Tooth wear is usually due to a combination of processes, the 'triumvirate' of abrasion, attrition, and erosion. It is unusual for wear to be solely attributed to one of these. Rather, tooth wear is due to all three processes with perhaps one of these predominating.

Abrasion is loss of tooth substance from the friction of a foreign body, often a toothbrush. There are case reports of many quite bizarre causes, including occupational hazards. *Attrition* is a loss of tooth substance due to tooth-to-tooth contact. It was very marked in predynastic Egyptians, prior to 3000 BC, and considered to be due to the sand content of the diet, and therefore, could be regarded as partly attrition and partly abrasion. Parafunctional activities such as nocturnal bruxism are probably a more common cause with the type of diet in the twenty-first century AD. *Erosion* is very different from these two physical types of tooth wear; it is the loss of tooth structure by chemical means, usually acidic, and not associated with mechanical or traumatic factors, or dental caries.

Although these three terms are well known and in common usage, there are other terms describing non-carious destructive processes involved in tooth wear, but these are much less wellknown and not universally accepted. These are demastication and abfraction. *Demastication* is a term used for wearing away of tooth substance during mastication. This could be specifically applied to the type of wear shown by the ancient Egyptians, and would depend on the abrasivity of the food consumed. *Abfraction* has been defined as the wedge-shaped defect observed at the cemento-enamel junction. Axial forces on the tooth tend to concentrate stress in this region, and cause microfractures and tooth tissue loss. This is sometimes confused with cervical abrasion and requires further investigation to establish it as a separate entity.

Types of tooth wear

• Abrasion: wearing away of tooth substance through physical or mechanical processes with a foreign body

• Attrition: loss of tooth substance due to tooth-to-tooth contact

• Erosion: irreversible loss of dental hard tissue due to a chemical process not involving bacteria, and not directly associated with mechanical or traumatic factors or with dental caries

• Demastication: loss of tooth substance during mastication, influenced by the abrasivity of the food. This should be regarded as a combination of abrasion and attrition

• Abfraction: stress concentrations near to the gingival margins leading to microfractures. This probably increases the susceptibility to abrasion and erosion.

Excessive tooth wear is still not a public dental health problem that dental caries and periodontal disease are. However, for a significant and increasing number of individuals, it is a serious oral condition, which can require very extensive and expensive treatment. It is vital that the existence of tooth wear is identified early and appropriate preventive measures put into place.

Abrasion

Prevention of abrasion is largely common sense. If the main aetiology is incorrect toothbrushing and/or the use of an overly abrasive toothpaste, then the technique and the paste can be changed.

Other aetiological factors need to be identified and then appropriate advice given. This can occasionally be quite problematic if the cause of the abrasion is related to professional activities, such as in wind instrument players.

Abrasive foods are uncommon in developed countries, but may be significant factors in certain areas of the developing world.However, there are usually many health and social problems that may render tooth wear somewhat insignificant compared with these other public health concerns.

Attrition

If attrition related to parafunctional activity is found to be progressing to unacceptable levels, then prevention of further tooth surface loss must be considered. Although males consistently show more attrition than females, they experience far fewer symptoms of temporo-mandibular dysfunction or myofascial pain than females. The severity of attrition has not been shown to be strongly associated with the development of signs and symptoms. It has proved to be impossible to totally stop nocturnal bruxing activity; although drug therapy such as tricyclics will reduce REM (Rapid Eye Movement) sleep, which is when the majority of this activity takes place. The most realistic method of controlling attrition is not in the prevention of the parafunctional activity itself, but rather in the prevention of the damage it causes. An occlusal splint (mouthguard) can be constructed, which will prevent the tooth to tooth contact which results in attrition. For long term use, and to try to minimize the amount of bruxing activity, a hard occlusal guard to cover the maxillary teeth such as a ‘Michigan Splint’ is an option to consider. This, may require the use of a face-bow so that the models can be correctly articulated, and the occlusal surfaces constructed to a predetermined occlusal scheme. There is no published evidence from clinical trials to show thatalteration of the occlusion will eliminate bruxism, but it may have an effect on the site of the attrition. Occlusal management (adjustment) aims to direct the forces generated during bruxism through areas of the teeth and restorations that are best suited to accept them. This will, therefore, reduce mechanical failure, mobility, and wear.

Erosion

The first step in the prevention of erosion is making a diagnosis. Once suspicions have been raised, it is essential to record accurately the severity and extent. This will establish the clinical baseline so that any progression can be assessed, and the effects of preventive measures monitored. It may also be necessary to undertake these procedures in patients with marked abrasion and attrition.

Prevention

• Abrasion: good oral hygiene instruction relating to toothbrushing procedures and use of toothpaste with minimal abrasivity

• Attrition: it is impossible to prevent parafunctional activity, but the consequent damage may be minimized by providing occlusal protection

• Erosion: the most important step in prevention is the determination of primary aetiological factors

• Any reflux activity

• Dietary assessment and counselling

• Adequate follow-up to check whether the dentition is stable or deteriorating

Enamel hypoplasia

Etiology

Tooth development can be disturbed by constitutional disturbances. Maternal illness during pregnancy can affect all primary teeth and first permanent molar teeth. Childhood febrile illness or gastroenteritis can affect the adult dentition. These disturbances produce a linear pattern of hypoplasia corresponding to the site of amelogenesis at the time (chronological hypoplasia). Infection or trauma of a primary tooth may cause hypoplasia of the underlying permanent germ.

Clinical features

Hypoplasia related to medical, dental and trauma history.

Prevention

Measures for healthy pregnancy maintenance and early diagnostics of genetic diseases.

Fluorosis

Etiology

Amelogenesis can be disturbed by excessive chronic ingestion of fluoride either from naturally occurring sources in drinking water or from overdosage by fluoride supplements and toothpastes or by a combination of the two. It can occur in the primary dentition but is largely confined to the permanent dentition. It commonly affects the outer enamel layers.

Clinical features

May vary from diffuse white opaque lines to scattered white flecking, or a more opaque and confluent dense white chalky mottling that may contain brown discoloration, or all the above with pitting hypoplasia.

Other causes of intrinsic discoloration.

Prevention

Daily control of fluoride load in risk factors group, use of water of good quality.

Amelogenesis imperfecta

Etiology

Genetic with different 'modes' of inheritance as well as a wide variety of presentations.

Clinical features

There are three main types of enamel anomaly:

- Hypoplastic. There is a deficiency of matrix but normal calcification of matrix which is present. The enamel is pitted and irregular and retains extrinsic stain.
- Hypocalcified. The enamel matrix is normal but there is inadequate calcification. The enamel may be normal in the gingival third of the tooth. Affected enamel is often opaque and retains stain. It is soft and easily lost.
- Hypomature. The enamel matrix is normal but there is little maturation or calcification of the enamel and the enamel is soft and porous.

Prevention

Measures for healthy pregnancy maintenance and early diagnostics of genetic diseases.

Dentinogenesis imperfecta

Etiology

Autosomal dominant inheritance.

Clinical features

Dentine is abnormal in structure and is translucent. Three main types exist:

• Type I (associated with osteogenesis imperfecta).

• Type II (hereditary opalescent dentine).

• Type III (brandywine type).

Types I and II are similar: primary teeth are more severely affected than permanent teeth. In the permanent dentition, teeth which develop first may be more severely affected than those which develop later. The teeth are translucent and vary in colour from grey to blue or brown. Enamel is poorly adherent to abnormal dentine and easily chips and wears. Crowns are bulbous with pronounced cervical constriction. Radiographically there are shortened roots, progressive pulp chamber and canal obliteration, and spontaneous periapical abscess formation.

Prevention

Measures for healthy pregnancy maintenance and early diagnostics of genetic diseases.

XIV. PRIMARY PREVENTION OF DENTAL TRAUMA

Epidemiology

Trauma to children's teeth occurs quite frequently. However, it is evident from the world literature that dental trauma is a global entity. At the age of 5 years some 31–40 per cent of boys and 16–30 per cent of girls will have suffered dental trauma. By the age of 12 years the corresponding figures are 12–33 per cent of boys and 4–19 per cent of girls.Traumatic injuries are twice as common in boys in both the permanent and the primary dentitions. The major causes of these injuries vary considerably and include accidents in and around the home, falls during normal play, injuries sustained during sport, and injuries as a direct result of violence. The main 'peak periods' for dental injury are described as being between the ages of 1 and 3, and again between the ages of 7 and 10. For children under 3 years of age, who are usually both unsteady on their legs and lacking in a proper sense of caution, falls are the most common cause of injury. In school-age children, bicycle, skateboard, micro-scooters, and road accidents are the most significant factors, while in adolescence there is another, although less marked, peak largely due to sports injuries. Most of these sports injuries result from participation in contact sports such as American football, rugby, soccer, boxing, wrestling, diving or stick sports. However, other sports like skiing, skating, cycling, and horse riding, which do not necessarily involve player contact, may also place the participant at risk.

The Fédération Dentaire International (FDI) have recently classified organized sport into two categories:

(1) high-risk sports that include American football, hockey, ice-hockey, lacrosse, martial sports, rugby, football, and skating; and

(2) medium-risk sports that include basketball, diving, squash, gymnastics, parachuting, and waterpolo (FDI 1990).

In childhood a small percentage of injuries can be attributed to violence, but once adulthood is reached, violence is a commoner cause of dental trauma than sports. An iatrogenic cause of trauma, particularly in younger patients where the anterior teeth are only partially erupted and root length is not complete, is avulsion reportedly caused by excessive pressure from a laryngoscope during intubation anaesthesia.

Broadly speaking, approaches to unintentional injury prevention can be divided into education (provision of information and training), environmental change (modification of products/environment, or use of additional safety devices), and enforcement (usually through regulation or legislation).

Primary prevention

Playground surfaces

The most common cause of tooth injury in children is falling on a hard surface. The British Standard for new play equipment for permanent installation outdoors, strongly recommends that any organization responsible for the purchase

of play equipment should ensure that an impact-absorbing surface is provided around the items from which children are most likely to fall. Studies of accidents to children in playgrounds have shown that the majority of the more serious cases were head injuries caused through striking hard ground. Playgrounds should be all about fun and be as safe as practically possible; however, no matter how safe the equipment or the playground's layout, there is always a risk that children will trip or stumble, run into each other or a piece of equipment, miss their footing or loose their grip, or more seriously, fall from a height. If a child falls from an item of play equipment, then he or she falls subject to the forces of gravity. This acceleration throughout a 2.5 m fall will result in a fall speed of 7 m/s or 15.7 mph at the point of ground contact. The purpose of safer surfacing in a play area is to absorb the impact of such a fall and to prevent a child suffering a head impact, which could be life threatening. The ability of a surface to absorb an impact is measured by its Critical Fall Height (CFH). British Standard 7188 (1991) gives details for CFH testing criteria as well as tests for a surface's resistance and ease of ignition. BS 7188 uses the Severity Index (SI) as a means of calculating CFH, but a new European standard for playground surfacing is currently being drafted, which will use Head Injury Criteria (HIC) as its means of calculating CFH. The test, which determines a human's tolerance to an impact SI, is based on research into road vehicle design and the NASA manned space programme.They estimated the severity of a blow to the head by mathematical integration of the area under a plot of deceleration versus time for the entire duration of the impact event (Wayne State University Curve). This curve produces a theory of 'short duration, high acceleration' tolerance. The deceleration suffered by a child's head as it is brought to a stop, and the period of time over which the deceleration acts must be considered. For brain damage not to occur, it is summated that the child's head should not be subject to a prolonged deceleration of more than 50 g.

Impact-absorbing surfaces are tested by dropping a head form representation of a child's head from a series of heights on to the surface. Accurate electronic deceleration measurements are taken during the period of impact in order to obtain the SIs for these falls, which are then plotted. A surface's CFH represents the greatest height of a head-first fall from which a child, landing on a surface, could be expected to avoid sustaining a critical head injury. The height of the curve at which the SI or HIC is 1000 represents the surface's CFH. In addition to the measurement of a surface CFH, BS 7188 also describes the measurement of four other parameters:

1. the ability of the surface to resist abrasive wear;
2. the slip resistance of the material;
3. the resistance to indentation by part landing and recovery from sustained landing; and
4. the response of the material to one particular source of ignition.

The resilient or compliant elastomeric composition of impactabsorbing surfaces is expensive. A cheaper alternative is tree bark chippings, but these have

the disadvantage of needing daily raking to remove, for example, broken glass and dog faeces. In addition to consideration of the playground surface, all playground equipment should meet British Standard Safety Criteria. Slides should not be free standing, but should be built into earth mounds. Climbing frames should be no higher than 2.5 m high and built over an acceptable surface. Supervision of small children at play (parental or professional) is very important, and probably the most effective way of preventing serious injury.

Early (mixed dentition) treatment of large overjets

The incidence of accidental damage to permanent incisors significantly increases with overjets greater than 9 mm. Even though the proportion of children with an overjet of 7 mm or more, never exceeds 9 per cent, this is still a significant number of children at high risk from traumatic injury. However, the relationship between overjet size and the concomitant risk of trauma remains unclear. A recent systematic review has shown that overjets of >3mm may pose a significant risk for dental trauma, and there is support for including such a measurement in an orthodontic treatment index (Nyugen et al. 1999). However, the roles of some confounders (lip posture, sports participation, tendency for accidents) remains to be elucidated. The same authors concluded that overjet may actually play less of a role as a risk factor in boys, probably because the nature of their activities overrides any effect of overjet!

It should be the aim of any caring society to prevent disfigurement from loss of or damage to a permanent incisor and for this reason alone early treatment of large overjets is justified.

Orthodontic treatment in the early mixed dentition is classically carried out in uncrowded arches using functional appliances or extra oral traction. Both treatments work best during active growth and may have a favourable influence on growth in some cases. An early start to treatment does not always mean an early finish, and treatments can be prolonged. However, if treatment is done in crowded arches then it is inevitably longer and involves two stages:

1. Primary canine extraction and overjet reduction.
2. Relief of crowding in the permanent dentition by extraction followed by arch realignment with fixed orthodontic appliances.

Therefore, while it may be feasible to correct incisor oral relationship in the early mixed dentition, a number of problems may arise, and treatment should not be attempted unless there are strong indications for doing so, and certainly not without a precise orthodontic diagnosis and treatment plan (Richardson 1989).

Dentoalveolar trauma

Prompt intervention following accidental damage to teeth can have a secondary preventive effect by reducing the complications of trauma. The development of both the acid-etch technique, dentine bonding agents, and more recently compomer technology, where a bonded restoration can be achieved without washing and drying, means that there is no excuse for leaving exposed dentine for any length of time in coronal fractures. The recognition that non-setting

calcium hydroxide is capable of allowing continued root growth and apexification in non-vital immature incisors has made both treatment and long-term prognosis more predictable for these teeth. Recently, however, a new product, Mineral Trioxide Aggregate (MTA), promises to replace the time spent achieving an apical barrier with non-setting calcium hydroxide by creating a barrier in one appointment. This would save considerable patient and operator time. The avulsed tooth is now a viable proposition, and if stored correctly and replanted soon after injury may be retained as a functioning member of the dentition with a healthy periodontal ligament for life. Even the avulsed tooth with an extraalveolar dry time of greater than 60 min, which has had its necrotic periodontal ligament removed, may grow a new periodontal ligament with the help of Emdogain Gel. Such advances in the field of dental traumatology are exciting, and enable the clinician to retain teeth which would previously have been extracted. These advances in the diagnosis, treatment, and prognosis of dental traumatic injuries have been most significant over the last 25 years and current knowledge is essential to treat appropriately (Welbury 2001).

The role of the dental practitioner in child protection

The incidence of orofacial injuries in children who have been physically abused is in excess of 65%. In all types of abuse, the incidence of orofacial injuries which are visible to the dental practitioner is of the order of 35%. The dental practitioner may be the first professional to see or suspect abuse. Injuries may take the form of contusions and ecchymoses, abrasions and lacerations, burns, bites or dental trauma.

The following eleven points should be considered by the dentist whenever doubts or suspicions are aroused:

- Could the injury have been caused accidentally and if so how?
- Does the explanation for the injury fit the age and the clinical findings?
- If the explanation of cause is consistent with the injury, is this itself within normally acceptable limits of behaviour?
- If there has been any delay seeking advice, are there good reasons for this?
- Does the story of the accident vary?
- The nature of the relationship between parents and child.
- The child’s reaction to other people.
- The child’s reaction to any medical or dental examination.
- The general demeanour of the child.
- Any comments made by the child and/or parents that cause concern about the child’s upbringing or life-style.
- History of previous injury.

XV. PREVENTION OF PERIODONTAL PATHOLOGY

Periodontium is the tissues that surround and support the teeth, including the gingivae, cementum, periodontal ligament, alveolar and supporting bone.

Functions of periodontium:

1. protective
2. supporting
3. homeostatic
4. sensory
5. trophic

Risk factors of periodontal disease.

Common:

1. General health (presence or absence of somatic, infectious or allergic diseases)
2. Immunity status
3. Harmful habits (alcohol abuse, smoking, gluttony etc.)
4. Environmental conditions
5. Professional harmfulness
6. Genetic factors

Local:

The main risk factors of the periodontal diseases and their diagnosis and correction are performed in Tab.№19

Tab. № 19 "Local risk factors of periodontal diseases"

Risk factors	Mechanism of influence	Diagnosis	Correction
Supragingival dental plaque	Intoxication with endotoxin, exotoxin and microbial side products	Drying Probing Coloration	Individual and professional oral hygiene
Subgingival dental plaque	Intoxication with endotoxin, exotoxin and microbial side products, mechanical trauma	Probing	Professional oral hygiene
Malocclusion	Increasing or decreasing of functional load	Examination of occlusion	Orthodontic and orthopedic treatment
Anomaly of soft tissue architectonic:	-	-	-
1) short lingual and labial frenulum	Dystrophy of tissues in the area of frenulum attachment	Examination (positive ischemia test result)	Surgical treatment
2) small oral vestibulum	Dystrophy of tissues in the area of influence	Examination (depth of the vestibulum)	Surgical treatment
Traumatic factors:			

1) lengthened orthopedic crown cervical edges;	Dystrophy in the area of no- physiological pressure	Examination of the gingival egde	Correction of the crowns and dentures
2)restorations «laying on the gum»;	Dystrophy in the area of no- physiological pressure	Examination with main and additional methods (flossing, transillumination etc.).	Correction of restorations
3) incorrect restoration changing the occlusial plane	Increasing or decreasing of functional load	Examination	Correction of restorations
Conditions of increasing dental plaque formation:	-	-	-
1)malocclusion, orthodontic appliances and dentures;	Retention of dental plaque, chemical and mechanical trauma	Examination	Orthodontic treatment, oral hygiene training
2)carious cavities;	Retention of dental plaque, chemical and mechanical trauma	Examination	Dental treatment
3) defects and uneven restorations in cervical area	Retention of dental plaque, chemical and mechanical trauma	Examination	Restoration polishing
Human behavior:	Injury	Oral hygiene training, teeth brushing control	Teaching of proper interdental hygiene products using
1)incorrect flossing and using of other interdental hygiene products;			
2) smoking	Suppression of the immune response and tissue regeneration of periodontium	Anamnesis, examination	Health education, elimination of harmful habit
Somatic factors	Reducing of adaptive periodontal capacities	Anamnesis	Main pathology treatment, primary preventive measures

Strategy of periodontal disease prevention consists of pathogenic dental plaque control and eliminating or minimizing of local risk factors.

Gingival indexes

PMA Index (Schour, Masler, 1948) is used to determine the clinical periodontal status on the prevalence of visual signs of inflammation - redness and swelling of the gum tissue.

The title of the index P – papillae, - 1 point (the early stages of the inflammation), M –marginal gum - 2 points and A –attashed gingiva -3 points).

Examine the medial papilla, attached and marginal gingiva of all teeth (or selected by researcher). The individual index is defined by the formula:

PMA = total score / number of examined teeth

The popular modification Rarma PMA index value is calculated using the formula:

*PMA = score / 3 * n * 100%,*

if *n* - the number of examined teeth;

3 - the maximum score for periodontal inflammation of 1 tooth.

PMA interpretation

1-33% - mild periodontal inflammation

34-66% - moderately,

more than 67% - heavy.

Gingival index GI (Loe, SiIpes 1963) was created for the assessment of the gingival condition and records qualitative changes in the gingiva. It scores the marginal and interproximal tissues separately on the basis of 0 to 3.

Key teeth: 16, 21,24,36,41,44 (in 4 areas: medial and distal papillae, oral and vestibular gingiva)

The criteria are:

0 - Normal gingiva;

1 - Mild inflammation – slight change in color and slight edema but no bleeding on probing;

2 - Moderate inflammation – redness, edema and glazing, bleeding on probing;

3 - Severe inflammation – marked redness and edema, ulceration with tendency to spontaneous bleeding.

The bleeding is assessed by probing gently along the wall of soft tissue of the gingival sulcus. The scores of the four areas of the tooth can be summed and divided by four to give the GI for the tooth. The GI of the individual can be obtained by adding the values of each tooth and dividing by the number of teeth examined. The GI may be used for the assessment of prevalence and severity of gingivitis in populations, groups and individuals.

Interpretation

0.1-1.0 - mild inflammation;

1.1-2.0 - moderate inflammation

2.1-3.0 - signifies severe inflammation.

GI index is also used to assess the effectiveness of preventive and treatment procedures. This index is used in children with periodontal formed!

Periodontal indexes

Community periodontal index CRITN (WHO 198O)

Three indicators of periodontal status are used for this assessment:

1. presence or absence of gingival bleeding
2. supra- or subgingival calculus
3. periodontal pockets-subdivided into shallow (4-5mrn) and deep (6mm or more).

The mouth is divided into sextants defined by teeth numbers 18-14, 13-23 24-28, 38-34, 33-43, and 44-48. A sextant should be examined only if there are two or more teeth present and not indicated for extration. When only one tooth remains in a sextant, it should be included in the adjacent sextant.

The two molars in each posterior sextant are paired for recording, and if one is missing, there is no replacement. If no index teeth or tooth is present in a sextant qualifying for examination, all the remaining teeth in that sextant are examined. For young people up to the age of 19 years, only six teeth - 16,11, 26, 36, 31 and 46 - are examined. This modification is made in order to to avoid classifying the deepened crevices associated with eruption as periodontal pockets. For the same reason, when examining children under the age of 15, recording for pockets should not be attempted, i.e., only bleeding and calculus should be considered. If no index tooth is present in a sextant qualifying for examination, single fully erupted incisors or premolars may be substituted.

Tab №20 "Codes and interpretation of CPITN"

Code	Description	Trigger
0	No disease (no gingival pockets < 3 mm)	No action required
1	Bleeding on probing (no gingival pockets < 3 mm)	Oral hygiene instruction because bleeding on probing usually indicates the presence of plaque induced gingivitis
2	No periodontal pocketing < 3mm, but calculus present with or without plaque retentive factors such as "overhanging" restorations	Oral hygiene instruction, remove plaque retentive factors (e.g. replace ledged restoration with correct use of matrix band, remove calculus with professional tooth cleaning)
3	Shallow periodontal pockets 4 - 5 mm (i.e. first band on probe partially visible)	More detailed examination of periodontal condition indicated
4	Deep periodontal pockets > 6 mm (first band on probe disappears)	More detailed examination of periodontal condition indicated

Etiology and pathogenesis of periodontal diseases.

The oral epithelium is keratinized but the crevicular (sulcular) epithelium and the junctional epithelium are not. The junctional epithelium is attached to the enamel surface and underlying connective tissue by a basal lamina and hemi-desmosomes, and its free surface (from which desquamation takes place) forms the bottom of the gingival crevice. The crevice is only about 0.5 mm deep—as seen in histological section. Clinically, however, the crevice depth is considered to be the distance to which a blunt probe will penetrate and, because it will readily disrupt the fragile junctional epithelium, the probing depth of the healthy gingival crevice reaches about 2 mm.

When plaque is allowed to accumulate freely, there is an acute exudative inflammatory response within 2–4 days in the connective tissue underlying the

coronal portion of junctional epithelium. After 10–21 days of persistent plaque accumulation, marked collagen destruction and a dense infiltrate of chronic inflammatory cells can be observed in this zone. The clinical changes of chronic gingivitis can now be detected: redness, swelling, reduced resistance to probing, and an increased tendency of the gingiva to bleed on probing or when the teeth are brushed.

Bacterial deposits do not extend below the gingival margin in the subclinical stages of developing gingivitis. The process of gingival enlargement, however, helps to create a subgingival flora and, gradually, apical advancement of subgingival plaque occurs as the junctional epithelium separates from the tooth surface to become ‘pocket epithelium’, characterized by the formation and lateral extension of rete pegs, and by areas of micro-ulceration.

As chronic gingivitis develops, an equilibrium is usually established between the increased mass of bacteria and the host defences, maintaining a state of chronic gingivitis indefinitely. If and when periodontitis does supervene, it is thought to be precipitated either by a proportional increase in pathogenic microorganisms within the subgingival bacterial flora, by impaired host resistance, or by both factors in combination.

As soon as the destructive process extends apically to affect the alveolar bone and fibre attachment of the root surface, periodontitis is said to have developed. Thus, periodontitis is characterized by loss of (connective tissue) attachment. Junctional epithelium proliferates apically to maintain an epithelial barrier at the base of the deepening pocket, and the denuded cementum becomes contaminated by micro-organisms and their products. Periodontitis is detected most readily with a probe, a bloodstained or purulent exudate being elicited by probing to the base of the pocket beyond the amelo-cemental junction.

Chronic gingivitis is a condition that can be largely reversed by plaque control. On the other hand, the loss of fibre attachment, which is the principal feature of periodontitis, is virtually irreversible. Destruction may occur at a linear rate, proceeding very slowly, consistent with tooth survival, or progressing more quickly, leading eventually to tooth loss. Attachment loss may also occur at a continuous, but exponential rate. Alternatively, progression may be episodic, acute episodes being interspersed with periods of remission or repair. Different patterns of progression may affect the same site at different times, and prolonged remission may not be uncommon. Furthermore, the rate of periodontal destruction may vary at different stages of the disease, between single tooth surfaces and between individuals.

Periodontitis is treated by removal of plaque and calculus, together with pathologically altered cementum, and by establishing effective, daily plaque

control. Following treatment of periodontitis, repair processes take place in which the junctional epithelium is re-established by involution of pocket epithelium, and supported by new gingival connective tissue, consisting of functionally orientated (but not tooth-attached) collagen fibres. More advanced lesions may not respond to treatment without surgical intervention.

Epidemiological studies are carried out to determine population trends in the occurrence and distribution of periodontal disease. However, the interpretation and comparison of data are fraught with difficulties. Fundamentally, there has been a lack of uniformly applied diagnostic criteria. Instead, epidemiological surveys have used a wide variety of disease markers. These include gingivitis levels, probing depths, clinical attachment level scores, and radiographically assessed alveolar bone loss, all expressed in a variety of different ways. Attachment loss with gingival recession may occur due to trauma from oral hygiene devices rather than inflammatory processes, making attachment loss on facial surfaces difficult to interpret. Partial recording systems which may not reflect full mouth conditions are commonly employed. Furthermore, there is great variation in choice of threshold values used to assign an individual subject as a ‘case’, that is, as suffering from periodontal disease. Finally, the periodontal disease experience of older populations with substantial tooth loss has been necessarily, but falsely, based on their surviving healthier teeth. The narrative which follows should be read with these methodological and analytical considerations in mind.

Professional oral hygiene (POH) is a health involving theory and evidence-based practice drawing on biomedical, social, and behavioural sciences. The practice of dental hygiene involves collaboration with clients, other health professionals, and society to achieve and maintain optimal oral health, an integral part of well-being. (***in the broad sense***). **Professional oral hygiene** is the complex professional dental procedure to provide and maintain the proper level of oral hygiene(***strictly***)

POH goals:

1. Identification and timely elimination (minimization) of periodontal disease risk factors.
2. Providing of high-level individual (home) oral hygiene.
3. Removal of dental deposits using professional measures and techniques.
4. Elimination of local traumatic factors (restoration and dentures correction, bad habits).

Types of POH:

1. ***Preventive*** (traditionally called a “teeth cleaning,” prophylaxis removes plaque build-up that cannot be removed by brushing, and includes tooth polishing. New data suggest that teeth should be cleaned every 8-16 weeks).

2. ***Full mouth debridement*** (when teeth have not been professionally cleaned for more than a year and there is heavy tartar build-up, a full mouth debridement is often necessary).

3. ***As a part of the complex periodontal therapy.***

4. ***Periodontal maintenance*** (to help maintain the results of treatment for periodontal disease, this specialized cleaning is performed every 8, 10, or 12 weeks).

POH stages:

1. ***motivation*** on risk factors of oral diseases, oral health education (psyhological concept of involving the person in his health problem; internal and external factors that stimulate desire and energy in people to keep health.);

2. ***prescribtion of proper oral care products and supplements***;

3. ***instruction*** (the caregiver and child or adolescent) in proper oral hygiene techniques and IOHP using;

4. ***control of oral hygiene*** by:

- index assessment (OHI-S, PLI, PHP, O'Leary)
- dye using
- supervision (controlled) brushing;

5. ***professional tooth cleaning*** (brushing, flossing, scaling, root planning, polishing) is a part of oral hygiene and involves the removal of dental plaque from teeth with the intention of preventing cavities (dental caries), gingivitis, and periodontal disease and hardened deposits (tartar) not removed by routine cleaning;

6. ***remineralization*** (application of remineralizing supplements):

- ca\phosphate – containing supplements (solutions, gels, mousses, varnishes)
- fluoride – containing supplements (solutions, gels, mousses, varnishes)
- complex (combined) supplements (deep fluoridation sistem, mousses, varnishes, gels)

Possibilities of poh in children and adolescence:

- using of conservative (non-invasive) measures is determined by the low prevalence of destructive periodontal disease (except for rapidly progressive periodontitis) in children;
- immature periodontal tissue determined the methods choice of dental plaque removing (up to the full maturity in 12-15 years);
- regularity of professional oral hygiene procedures is caused by a form (compensated, sub- or decompensated) of caries course and severity of periodontitis in children.

XVI. PREVENTION OF HALITOSIS

Halitosis, colloquially called bad breath, or fetor oris, is a symptom in which a noticeably unpleasant odor is present on the exhaled breath. Concern about halitosis is estimated to be the third most frequent reason for people to seek dental care, following tooth decay and gum disease; and about 20% of the general population are reported to suffer from it to some degree.

Not all who think they have halitosis have a genuine problem. Of those who feel they have halitosis, significant percentages (5- 72%) have been reported to have no genuine halitosis when professionally examined. Of those who have genuine halitosis, often the odor is caused by bacteria present below the gumline and on the back of the tongue. The remaining 10% is accounted for by many conditions, including disorders in the nasal cavity, sinuses, throat, lungs, esophagus, stomach or elsewhere. See the related article on tonsilloliths for another possible cause of halitosis.

Very rarely, halitosis can be one of many symptoms of a serious underlying medical condition such as liver failure; but, in the vast majority of cases, the cause is minor and can often be reduced by adjustments to oral hygiene, including brushing or gently scraping the back of the tongue and improving the health of the gums by using dental floss. Occasionally, however, especially if the origin of the odor is not in the mouth, halitosis can be more difficult to diagnose and to manage successfully. Bad breath is a social taboo; and, as a result, perceived or genuine halitosis can sometimes trigger social anxiety.

Classification

The Miyazaki et al. classification was originally described in 1999 in a Japanese scientific publication, and has since been adapted to reflect North American society, especially with regards halitophobia. The classification assumes three primary divisions of the halitosis symptom, namely genuine halitosis, pseudohalitosis and halitophobia. This classification has been suggested to be most widely used, but it has been criticized because it is overly simplistic and is largely of use only to dentists rather than other specialties.

1. Genuine halitosis

A. Physiologic halitosis

B. Pathologic halitosis

Oral

Extra-oral

2. Pseudohalitosis

3. Halitophobia

The Tangerman and Winkel classification was suggested in Europe in 2002.This classification focuses only on those cases where there is genuine

halitosis, and has therefore been criticized for being less clinically useful for dentistry when compared to the Miyazaki et al. classification.

Intra-oral halitosis

Extra-oral halitosis

A. Blood borne halitosis

Systemic diseases

Metabolic diseases

Food

Medication

B. Non-blood borne halitosis

Upper respiratory tract

Lower respiratory tract

The same authors also suggested that halitosis can be divided according to the character of the odor into 3 groups:

"Sulfurous or fecal" caused by volatile sulfur compounds (VSC), most notably methyl mercaptan, hydrogen sulfide and dimethyl sulfide.

"Fruity" caused by acetone, present in diabetes.

"Urine-like or ammoniacal" caused by ammonia, dimethyl amine and trimethylamine (TMA), present in trimethylaminuria and uremia.

Based on the strengths and weaknesses of previous attempts at classification of halitosis, an etiologic classification has now been proposed:

Type 0 (physiologic)

Type 1 (oral)

Type 2 (airway)

Type 3 (gastroesophageal)

Type 4 (blood-borne)

Type 5 (subjective)

Any halitosis symptom is potentially the sum of these types in any combination, superimposed on the physiologic odor present in all healthy individuals.

Differential diagnosis

Oral cavity case

In about 90% of genuine halitosis cases, the origin of the odor is in the mouth itself. This is known as intra-oral halitosis, oral malodor or oral halitosis.

There is an extensive list of possible causes of halitosis in the mouth alone, however by far the most prevalent causes reported are halitogenic biofilm on the posterior dorsal tongue and within gingival crevices and periodontal pockets (i.e. bacteria living on the back of the tongue and below the gumline, and in the pockets created by gum disease between teeth and the gums). Both of these main causes share a common theme of proteolytic putrefaction of sulfur containing amino acids

in dietary and salivary protein by mostly anaerobic, Gram negative bacterial species. There are over 600 types of bacteria found in the average mouth. Some of these can produce high levels of foul odors when incubated in the laboratory. The odors are produced mainly due to the breakdown of proteins into individual amino acids, followed by the further breakdown of certain amino acids to produce detectable foul gases. For example, the breakdown of cysteine and methionine produce hydrogen sulfide and methyl mercaptan, respectively. Volatile sulfur compounds have been shown to be statistically associated with oral malodor levels, and usually decrease following successful treatment. Other parts of the mouth may also contribute to the overall odor, but are not as common as the back of the tongue. These locations are, in order of descending prevalence: inter-dental and sub-gingival niches, faulty dental work, food-impaction areas in between the teeth, abscesses, and unclean dentures. Oral based lesions caused by viral infections like Herpes Simplex and HPV may also contribute to bad breath.

The intensity of bad breath may differ during the day, due to eating certain foods (such as garlic, onions, meat, fish, and cheese), smoking, and alcohol consumption. Since the mouth is exposed to less oxygen[medical citation needed] and is inactive during the night, the odor is usually worse upon awakening ("morning breath"). Bad breath may be transient, often disappearing following eating, drinking, tooth brushing, flossing, or rinsing with specialized mouthwash. Bad breath may also be persistent (chronic bad breath), which affects some 25% of the population in varying degrees.

Tongue

Normal appearance of the tongue, showing a degree of visible white coating and normal irregular surface on the posterior dorsum. The most common location for mouth-related halitosis is the tongue. Tongue bacteria produce malodorous compounds and fatty acids, and account for 80 to 90% of all cases of mouth-related bad breath. Large quantities of naturally-occurring bacteria are often found on the posterior dorsum of the tongue, where they are relatively undisturbed by normal activity. This part of the tongue is relatively dry and poorly cleansed, and the convoluted microbial structure of the tongue dorsum provides an ideal habitat for anaerobic bacteria, which flourish under a continually-forming tongue coating of food debris, dead epithelial cells, postnasal drip and overlying bacteria, living and dead. When left on the tongue, the anaerobic respiration of such bacteria can yield either the putrescent smell of indole, skatole, polyamines, or the "rotten egg" smell of volatile sulfur compounds (VSCs) such as hydrogen sulfide, methyl mercaptan, Allyl methyl sulfide, and dimethyl sulfide. The presence of halitosis-producing bacteria on the back of the tongue is not to be confused with tongue coating. Bacteria are invisible to the naked eye, and degrees of white tongue coating are present in most people with and without halitosis. A visible white tongue coating does not always equal the back of the tongue as an origin of halitosis, however a "white tongue" is thought to be a sign of halitosis. In oral medicine generally, a white tongue is considered a sign of several medical conditions. Patients with

periodontal disease were shown to have sixfold prevalence of tongue coating compared with normal subjects. Halitosis patients were also shown to have significantly higher bacterial loads in this region compared to individuals without halitosis.

The normal anatomy of the dorsal tongue surface consists of a posterior third and an anterior two thirds. The posterior third constitutes the lingual tonsil, with cryptolymphatic units roughening the surface of this area. Between the anterior and posterior portions of the dorsal tongue is the V shaped line of vallate papillae. Anteriorly, the surface is even more irregular, with filiform, fungiform and foliate papillae. This irregular surface is a perfect habitat for bacteria and it can also trap debris. The roughly 25 cm2 area carries the heaviest bacterial loads in the oral cavity. The posterior dorsum of the tongue is the site of greatest generation of VSC and hence usually the greatest contributor to oral malodor. Hence, this irregular surface usually forms a coating which can be easily removed. This coating consists of desquamated epilethia (shed skin cells from the tongue), food debris, bacteria and their extracellular matrix. Essentially, a layer of living and non living components referred to as a biofilm. Two conditions that may further predispose to halitogenic biofilm on the dorsal tongue are lingua plicata (fissured or scrotal tongue), a common condition where there are many grooves or fissures on the tongue, and lingua villosa (hairy tongue) where the normal hair-like projections of the specializeds oral mucosa on the dorsal tongue are longer than usual, and may even be discolored.

Gingiva

Gingival crevices are the small grooves between teeth and gums, and they are present in health, although they may become inflamed when gingivitis is present. The difference between a gingival crevice and periodontal pocket is that former is <3mm in depth and the latter is >3mm. Periodontal pockets usually accompany periodontal disease (gum disease). There is some controversy over the role of periodontal diseases in causing bad breath. However, advanced periodontal disease is a common cause of severe halitosis. Waste products from the anaerobic bacteria growing below the gumline (subgingival) have a foul smell and have been clinically demonstrated to produce a very intense bad breath. Removal of the subgingival calculus (i.e. tartar or hard plaque) and friable tissue has been shown to improve mouth odor considerably. This is accomplished by subgingival scaling and root planing and irrigation with an antibiotic mouth rinse. The bacteria that cause gingivitis and periodontal disease (periodontopathogens) are invariably gram negative and capable of producing VSC. Methyl mercaptan is known to be the greatest contributing VSC in halitosis that is caused by periodontal disease and gingivitis. The level of VSC on breath has been shown to positively correlate with the depth of periodontal pocketing, the number of pockets, and whether the pockets bleed when examined with a dental probe. Indeed, VSC may themselves have been shown to contribute to the inflammation and tissue damage that is characteristic of periodontal disease. However, not all patients with periodontal disease have

halitosis, and not all patients with halitosis have periodontal disease. Although patients with periodontal disease are more likely to suffer from halitosis than the general population, the halitosis symptom was shown to be more strongly associated with degree of tongue coating than with the severity of periodontal disease. Another possible symptom of periodontal disease is a bad taste, which does not necessarily accompany a malodor that is detectable by others.

Other potential causes in the oral cavity.

There have been myriad other reported causes of oral malodor, however these are all much less common than the two above causes.

Deep carious lesions (dental decay) – which cause localized food impaction and stagnation

Recent dental extraction sockets – fill with blood clot, and provide an ideal habitat for bacterial proliferation

Interdental food packing – (food getting pushed down between teeth) - this can be caused by missing teeth, tilted, spaced or crowded teeth, or poorly contoured approximal dental fillings. Food debris becomes trapped, undergoes slow bacterial putrefaction and release of malodorous volatiles. Food packing can also cause a localized periodontal reaction, characterized by dental pain that is relieved by cleaning the area of food packing with interdental brush or floss.

Acrylic dentures (plastic false teeth) – inadequate denture hygiene practises such as failing to clean and remove the prosthesis each night, may cause a malodour from the plastic itself or from the mouth as microbiota responds to the altered environment. The plastic is actually porous, and the fitting surface is usually irregular, sculpted to fit the edentulous oral anatomy. These factors predispose to bacterial and yeast retention, which is accompanied by a typical smell.

Oral infections

Oral ulceration

Fasting

Stress/anxiety

Menstrual cycle – at mid cycle and during menstruation, increased breath VSC were reported in women.

Smoking – Smoking is linked with periodontal disease, which is the second most common cause of oral malodor. Smoking also has many other negative effects on the mouth, from increased rates of dental decay to premalignant lesions and even oral cancer.

Alcohol

Volatile foodstuffs – e.g. onion, garlic, durian, cabbage, cauliflower and radish. Volatile foodstuffs may leave malodorous residues in the mouth, which are the subject to bacterial putrefaction and VSC release. However, volatile foodstuffs may also cause halitoisis via the blood borne halitosis mechanism.

Medication – often medications can cause xerostomia (dry mouth) which results in increased microbial growth in the mouth.

Nasal cavity and sinuses

In this occurrence, the air exiting the nostrils has a pungent odor that differs from the oral odor. Nasal odor may be due to sinus infections or foreign bodies.

Halitosis is often stated to be a symptom of chronic rhinosinusitis, however gold standard breath analysis techniques have not been applied. Theoretically, there are several possible mechanisms of both objective and subjective halitosis that may be involved.

Tonsils

There is disagreement as to the proportion of halitosis cases which are caused by conditions of the tonsils. Some claim that the tonsils are the most significant cause of halitosis after the mouth. According to one report, approximately 3% of halitosis cases were related to the tonsils. Conditions of the tonsils which may be associated with halitosis include chronic caseous tonsillitis (cheese-like material can be exuded from the tonsillar crypt orifi), tonsillolithiasis (tonsil stones), and less commonly peritonsillar abscess, actinomycosis, fungating malignancies, chondroid choristoma and inflammatory myofibroblastic tumor.

Esophagus

The lower esophageal sphincter, which is the valve between the stomach and the esophagus, may not close properly due to a Hiatal Hernia or GERD, allowing acid to enter the esophagus and gases to escape to the mouth. A Zenker's diverticulum may also result in halitosis due to aging food retained in the esophagus.

Stomach

The stomach is considered by most researchers as a very uncommon source of bad breath (except in belching). The esophagus is a closed and collapsed tube, and continuous flow (as opposed to a simple burp) of gas or putrid substances from the stomach indicates a health problem—such as reflux serious enough to be bringing up stomach contents or a fistula between the stomach and the esophagus—which will demonstrate more serious manifestations than just foul odor.[9]

In the case of allyl methyl sulfide (the byproduct of garlic's digestion), odor does not come from the stomach, since it does not get metabolized there.

Systemic diseases

There are a few systemic (non-oral) medical conditions that may cause foul breath odor, but these are extremely infrequent in the general population. Such conditions are:

1. Fetor hepaticus: an example of a rare type of bad breath caused by chronic liver failure.
2. Lower respiratory tract infections (bronchial and lung infections).
3. Renal infections and renal failure.
4. Carcinoma.

5. Trimethylaminuria ("fish odor syndrome").
6. Diabetes mellitus.
7. Metabolic conditions, e.g. resulting in elevated blood dimethyl sulfide.
8. Individuals afflicted by the above conditions often show additional, more diagnostically conclusive symptoms than bad breath alone.
9. Uncommon Origins (According to research done by Theodoor Hendrik van de Velde in the 1920s, orgasm lead to bad breath in females for about an hour after climax).

Halitophobia (delusional halitosis)

One quarter of the patients seeking professional advice on bad breath suffer from a highly exaggerated concern of having bad breath, known as halitophobia, delusional halitosis, or as a manifestation of Olfactory reference syndrome. These patients are sure that they have bad breath, although many have not asked anyone for an objective opinion. Halitophobia may severely affect the lives of some 0.5–1.0% of the adult population.

Diagnostic methothods

Self diagnosis

Scientists have long thought that smelling one's own breath odor is often difficult due to acclimatization, although many people with bad breath are able to detect it in others. Research has suggested that self-evaluation of halitosis is not easy because of preconceived notions of how bad we think it should be. Some people assume that they have bad breath because of bad taste (metallic, sour, fecal, etc.), however bad taste is considered a poor indicator. Patients often self-diagnose by asking a close friend.

One popular home method to determine the presence of bad breath is to lick the back of the wrist, let the saliva dry for a minute or two, and smell the result. This test results in overestimation, as concluded from research, and should be avoided. A better way would be to lightly scrape the posterior back of the tongue with a plastic disposable spoon and to smell the drying residue. Home tests that use a chemical reaction to test for the presence of polyamines and sulfur compounds on tongue swabs are now available, but there are few studies showing how well they actually detect the odor. Furthermore, since breath odor changes in intensity throughout the day depending on many factors, multiple testing sessions may be necessary.

Professional diagnosis

If bad breath is persistent, and all other medical and dental factors have been ruled out, specialized testing and treatment is required. Hundreds of dental offices

and commercial breath clinics now claim to diagnose and treat bad breath. They often use some of several laboratory methods for diagnosis of bad breath:

Halimeter is a portable sulfide monitor used to test for levels of sulfur emissions (to be specific, hydrogen sulfide) in the mouth air. When used properly, this device can be very effective at determining levels of certain VSC-producing bacteria. However, it has drawbacks in clinical applications. For example, other common sulfides (such as mercaptan) are not recorded as easily and can be misrepresented in test results. Certain foods such as garlic and onions produce sulfur in the breath for as long as 48 hours and can result in false readings. The Halimeter is also very sensitive to alcohol, so one should avoid drinking alcohol or using alcohol-containing mouthwashes for at least 12 hours prior to being tested. This analog machine loses sensitivity over time and requires periodic recalibration to remain accurate.

Gas chromatography: portable machines, such as the Oral Chroma, are currently being introduced. This technology is specifically designed to digitally measure molecular levels of the three major VSCs in a sample of mouth air (hydrogen sulfide, methyl mercaptan, and dimethyl sulfide). It is accurate in measuring the sulfur components of the breath and produces visual results in graph form via computer interface.

BANA test: this test is directed to find the salivary levels of an enzyme indicating the presence of certain halitosis-related bacteria.

β-galactosidase test: salivary levels of this enzyme were found to be correlated with oral malodor.

Although such instrumentation and examinations are widely used in breath clinics, the most important measurement of bad breath (the gold standard) is the actual sniffing and scoring of the level and type of the odor carried out by trained experts ("organoleptic measurements"). The level of odor is usually assessed on a six-point intensity scale.

Treatment and prevention

Chronic halitosis is not well understood by most physicians and dentists, so effective treatment is not always easy to find.

The following strategies may be suggested:

Gently cleaning the tongue surface twice daily is the most effective way to keep bad breath in control; that can be achieved using a tooth brush, tongue cleaner or tongue brush/scraper to wipe off the bacterial biofilm, debris, and mucus. An inverted teaspoon may also do the job. Scraping or otherwise damaging the tongue should be avoided, and scraping of the V-shaped row of vallate papillae found at the extreme back of the tongue should also be avoided. Brushing a small amount of antibacterial mouth rinse or tongue gel onto the tongue surface will further inhibit bacterial action.

Diet. Eating a healthy breakfast with rough foods helps clean the very back of the tongue.

Chewing gum: Since dry-mouth can increase bacterial buildup and cause or worsen bad breath, chewing sugarless gum can help with the production of saliva, and thereby help to reduce bad breath. Chewing may help particularly when the mouth is dry, or when one cannot perform oral hygiene procedures after meals (especially those meals rich in protein). This aids in provision of saliva, which washes away oral bacteria, has antibacterial properties and promotes mechanical activity which helps cleanse the mouth. Some chewing gums contain special anti-odor ingredients. Chewing on fennel seeds, cinnamon sticks, mastic gum, or fresh parsley are common folk remedies.

Gargling right before bedtime with an effective mouthwash. Several types of commercial mouthwashes have been shown to reduce malodor for hours in peer-reviewed scientific studies. Mouthwashes may contain active ingredients that are inactivated by the soap present in most toothpastes. Thus it is recommended to refrain from using mouthwash directly after toothbrushing with paste.

Maintaining ***proper oral hygiene***, including daily tongue cleaning, brushing, flossing, and periodic visits to dentists and hygienists. Flossing is particularly important in removing rotting food debris and bacterial plaque from between the teeth, especially at the gumline. Dentures should be properly cleaned and soaked overnight in antibacterial solution (unless otherwise advised by one's dentist).

Probiotic treatments, specifically Streptococcus salivarius has been claimed to suppress malodorous bacteria growth, however well designed randomised control clinical trails are needed to assess this. Certainly there is more evidence for mechanical tongue cleansing and to a lesser extent specific antimicrobial mouthwashes.

Cleaning the tongue. The most widely known reason to clean the tongue is for the control of bad breath. Methods used against bad breath, such as mints, mouth sprays, mouthwash or gum, may only temporarily mask the odors created by the bacteria on the tongue, but cannot cure bad breath because they do not remove the source of the bad breath. In order to prevent the production of the sulfur-containing compounds mentioned above, the bacteria on the tongue must be removed, as must the decaying food debris present on the rear areas of the tongue. Most people who clean their tongue use a tongue cleaner (tongue scraper), or a toothbrush.

Mouthwashes. There has not been a single documented medical case of successfully cured chronic halitosis using any of the currently available mouthwashes. However, a 2008 systematic review determined the efficacy of antibacterial mouthrinses for treating bad breath. Mouthwashes often contain antibacterial agents including cetylpyridinium chloride, chlorhexidine, zinc gluconate, essential oils, hydrogen peroxide, and chlorine dioxide. Cetylpyridinium chloride and chlorhexidine can temporarily stain teeth. Zinc and chlorhexidine provide strong synergistic effect. They may also contain alcohol, which is a drying agent.

Other solutions rely on odor eliminators, such as oxidizers, to eliminate existing bad breath on a short-term basis.

A new approach for home treatment of bad breath is the use of oil-containing mouthwashes and two-phase (oil:water) mouthwashes. Essential oils have been found effective in reducing halitosis, and are being used in several commercial mouthwashes.

PLAN OF HALITOSIS TREATMENT AND PREVENTION

- Motivation on halitosis occurrence risk factors.
- Prescription of IOHP and instruction in their using techniques.
- Professional oral hygiene.
- Therapy measures (restoration, endodontic, periodontal and mucosal treatment)
- Surgery measures
- Orthodontic and prosthetic treatment.
- Other medical specialist’s consultation (treatment) on dentist’s referral.
- Monitoring visits and maintenance therapy.

XVII. PREVENTION OF ORAL CANCER

Causes of oral mucosa diseases include:

• Genetic
• Environment/lifestyle
• Biological, physical, chemical factors
• Infections

Oral cancer is a type of head and neck cancer and is any cancerous tissue growth located in the oral cavity.

Head and neck cancers are the sixth most common form of cancer globally, and around 500,000 new cases of oral and oropharyngeal cancers are diagnosed annually, three-quarters of which occur in the developing world.

Main risk factors of oral cancer:

1. Tobacco, alcohol and the HPV virus induce such genetic alterations (including key disorders such as epidermal growth factor receptor, TP53, NOTCH1, Cyclin D1, etc.) that trigger transformation of stromal cells, immune suppression, and chronic inflammation. The combination of tobacco and/or alcohol risk factors with certain gene polymorphisms may increase oral cancer susceptibility.
2. Chronic trauma
3. Hereditary
4. Professional hazards

Up to 70% of oral cancers are receded by premalignant oral lesions, such as persistent red or white patches in the mouth.

Oral potentially malignant disorders (OPMD) and optionally malignant disorders.

Clinical signs of malignization:

1. Clinical features associated with an increased risk of malignant progression: lesion characteristics (larger size (>200 mm)), surface texture (smooth and indurated), inhomogeneous aspects (hyperkeratosic, thick), colour (red coloured or speckled, extent, unifocal, multifocal or diffuse pattern);
2. Lesion location in the mouth, i.e. tongue, floor of mouth;
3. Patient risk factor assessment and detailed medical or systemic illness/cancer history and lesion histopathology.

Primary preventive mesures:

Preventive check-ups

Elimination of risk factors

Secondary preventive mesures:

Biopsy

Oncologist consultation

Most conditions that affect oral mucosal health are acquired, through environmental or lifestyle factors, albeit the genetic constitution may well influence the result of an onslaught by some agent such as a micro-organism. Various biological, physical, and chemical factors may act singly or in concert to cause disease, some of which is preventable. A range of infections can affect the oral mucosa, but few are more devastating than HIV, which can result in oral and other fungal, viral, or other mucosal infections, or neoplasms. Mucosal integrity is central to protecting the mouth against infections, or insults from the environment and lifestyle. Adequate nutrition and intact immune and other defences are, in turn, central to mucosal integrity. This chapter deals with the major preventable threats to the oral mucosa in four sections. The first relates to preventing oral infections, particularly in vulnerable patients. The second deals with preventable threats from lifestyle. There then follow sections on iatrogenic disease, particularly preventing and managing mucositis (an area in which there is new evidence emerging), and on nutrition. The latter is a large and complex subject, but the role of good nutrition in preventing mucosal disease occurs at many levels.

Causes of oral mucosal diseases:

- Genetic
- Environment/lifestyle
- Biological, physical, chemical factors
- Infections

Infections

The prevention of oral mucosal infections in an immunocompetent person is a relatively minor problem mostly effectively achieved by avoiding contact. Only candidiasis is really open to preventive intervention, and in some cases this may be indicated. For immunocompromised patients though, the impact of both endogenous and exogenous infections can be considerable, and the range of organisms that can cause problematic infection is large. In such cases steps to prevent infections, or to limit them, may be indicated. The most common mucosal infections are candidiasis and herpesvirus.

Oral infections in the immunocompromised person

There are dramatic increases in the number of immunocompromised persons both as a consequence of the effects of infection by HIV and of treatment of organ transplant patients with immunosuppressive agents. Both are characterized by a predominantly T lymphocyte immune defect. T cells are essential to protection

against infection with fungi, viruses, and some bacteria—mainly mycobacteria: immunocompromised patients are thus liable to infection both with fungal, mycobacterial, and viral pathogens (if they come into contact with them) and with opportunistic organisms, particularly, candida and herpesviruses. Infections in immunocompromised persons tend to be recurrent or protracted, severe, and sometimes resistant to treatment. Occasionally they disseminate. In general, the spectrum of infections is wider, and their severity greater, the more profound the immune defect.

Lifestyle issues in preventing oral mucosal disease

Lifestyle, or changes in lifestyle, can have a major impact in preventing disease in many systems, including the oral mucosa, but it is in the area of oral epithelial dysplasia and cancer that lifestyle factors are most significant in preventing oral disease.

Trauma, Chemicals, or Burns

Trauma from appliances or prostheses may cause oral ulceration and, very rarely, neoplastic change. Oral mutilation may be seen in some psychiatrically disturbed patients or those with learning disability. In some Chinese and Hindu cultures the lips, cheeks, or tongue are ceremonially pierced by spears or other objects while the person is in a state of trance. In some East-African groups, the uvula is removed in children in the belief that health will be improved.

Adverse oral effects of dental amalgam may include allergy, lichenoid reactions, electro-galvanism, and amalgam tattoos. Amalgam ‘tattoos’ are the most common oral tattoos, but are not discussed here. In the developing world, a range of different types of tattoos can be seen, some deliberately induced, others accidental. Accidental tattooing can originate from use of the bark of a plant Juglans regia (Derum or Dendava) used as an oral hygiene aid. One particularly obvious tattoo is that of the labial and buccal maxillary gingiva created using soot. In Eritrea, females are tattooed on the anterior maxillary gingiva in childhood: males are tattooed only in the canine regions. In parts of North Africa, the lip may be tattooed and in some parts of West Africa, the skin at the commissure is tattooed. In the developed world, deliberate tattoos not uncommonly use tribal or personal names especially in the lower labial mucosa.

Chemically induced lesions often present as mucosal burns or white lesions. They can be caused by:

• Drugs

• analgesic tablets

• cocaine, snuff or smokeless tobacco deliberately rubbed into the gingivae or vestibule

• pancreatin can cause ulceration

• Mouthwashes
• Chlorhexidine
• Others, especially alcohol-containing washes
• Dental procedures
• acids (chromic, trichloracetic, phosphoric)
• self-curing resins, especially epoxy resins
• Natural products
• Tobacco products
• Areca nut
• the houseplant Dieffenbachia
• the enzyme bromelin in pineapple
• others

Diagnosis is from the history and clinical features.

Thermally induced lesions often present as mucosal burns or white lesions, and can be caused by hot foods or drinks, hot instruments dental handpiece or extraction forceps, for example), electrical burns, cryosurgery, or radiation. Those seen especially on the palate or tongue, for example, 'pizza-palate', present as white lesions, blisters, or ulcers. Diagnosis is from history and clinical features.

In some groups in developing countries such as in some Amazonian tribes and in the Surma tribe of Ethiopia, large plates are worn in the lower lip. Others wear lip plugs. Some African tribes such as the Toposa of Sudan wear a piece of wire, others such as the Dogon of Mali wear rings, in their lower lip. In the western countries, jewellery is usually applied to the lips (labret) but the practice of lingual piercing is a cause of some concern since oedema can be pronounced and may be hazardous to the airway.

Epithelial dysplasia and cancer

Oral epithelial dysplasia

Studies in Western populations confirm the associations of oral epithelial dysplasia (OED) with tobacco and alcohol use. Analysis of the effects of chewing or smoking tobacco, alcohol consumption, body mass index, and vegetable, fruit, and vitamin/iron intake on the risk of erythroplakia in Indian populations also showed that tobacco chewing and alcohol drinking are strong risk factors for erythroplakia.

The role of diet in preventing oral epithelial dysplasia is less clear. Fruit and vegetable intake are considered important variables in lowering the risk of oral cancer, but this may not necessarily always be the case with oral epithelial dysplasia. A study of female tobacco/betel chewers in South India suggested that a diet deficient in foods of animal origin was a more significant risk factor for oral premalignancy than is a diet deficient in fruits and vegetables.

Oral squamous cell carcinoma: risk factors

Sun exposure

Working outdoors increases the risk of lip cancer: fair complexion may be a cofactor.

Tobacco use

Tobacco contains nicotine and other alkaloids. N-nitrosamines are the compounds thought to be the major carcinogenic agents in tobacco. Volatile and other nitrosamines may also be contributors.

The excessive use of tobacco products has been associated with various lesions in the oral cavity. Tobacco smoking can have a range of adverse effects on oral health including predisposing to candida carriage, candidiasis, and leukoplakia. Other tobacco-associated lesions include tooth stains, abrasions, smoker's melanosis, acute necrotizing ulcerative gingivitis, and other periodontal conditions, burns and keratotic patches, black hairy tongue, nicotinic stomatitis, palatal erosions, epithelial dysplasia, and squamous-cell carcinoma.

Tobacco is smoked as cigarettes, cigars, or in a pipe and, in some instances may be treated in a variety of ways, or contain additives. Alcohol synergizes with tobacco as a risk factor for all upper aerodigestive tract squamous cell carcinomas. The effect of smoking falls off soon after smoking ceases.

Analytical studies strongly suggest that tobacco smoking of any type, but especially pipe smoking significantly increases the risk of lip cancer. Details of tar yield of cigarettes, and type of cigarette used for the longest period can be used as the basis of a classification to examine the effects of different types of cigarette. Cigarettes can be classified as low or medium if the tar yield is below 22 mg, and as high if tar yield is above 22 mg. Compared with non-smokers, the risk of oral cancer for smokers using low to medium cigarettes is 8.5 and for high tar cigarettes is 16.4.

As regards intra-oral carcinoma, the sites of tongue, mouth, oropharynx, and hypopharynx are so often grouped together in analytical studies, or grouped in a variety of different combinations, that it is difficult to discuss these tumours individually.

Smokeless tobacco

Smokeless tobacco contains a number of carcinogens and its use is to be deprecated. There is clear concern about the possible carcinogenicity and other adverse effects of the snuff sold in small 'teabag' pouches. There is some limited evidence for an association between the use of such smokeless tobacco and oral cancer: there is no doubt, however, that smokeless tobacco can induce oral keratosis and gingival recession. The fact that this form of smokeless tobacco is held in the mouth for very long periods, and is popular with children and

adolescents is a cause for concern. Lifestyle issues in preventing oral mucosal disease.

Alcohol

A study of alcohol misusers from south London showed a high incidence of tooth wear and trauma to the dentition, and a small minority had oral mucosal lesions, including two previously treated carcinomas (Harris et al. 1997). Alcoholics have demonstrable cytological abnormalities on oral smears, though whether these are due to a direct effect of alcohol or an effect secondary to associated malnutrition is unclear.

• The prevention of oral cancer is best achieved by reducing risk factors known to cause cancer in the mouth and elsewhere in the body. Tobacco, excessive alcohol consumption, betel use, and prolonged exposure to sunlight, are four of the most important risk factors implicated in the aetiology of oral cancer.

• The same risk factors are also implicated in the development of potentially malignant lesions.

• In addition, improving the diet by increasing intake of fruit and vegetables, treating possible infections, such as candidosis or syphilis, and improving oral hygiene, may reduce the prevalence of pre-malignant lesions.

• Denture-induced stomatitis can be prevented if dentures are not worn at night, plaque is removed by brushing, and the dentures disinfected.

• Apart from high standards of hygiene, and the avoidance of contact with those with communicable diseases, or their tissues, little can be done to prevent primary infections with viruses that can cause mucosal lesions.

• Oral infections in the immunocompromised person may be prevented by prophylactic therapy, particularly with antifungal and antiviral agents.

• Chlorhexidine gluconate aqueous mouth rinses may have an effect in the management of RAS, possibly by reducing secondary infection.

• Proven allergic reactions in the mouth can be prevented by identifying and avoiding the cause.

XVIII. PREVENTION OF MALOCCLUSION

The occlusion is characterized by **three main positions**:

I. The jaws spatial interposition

II. The shape of the dental arches

III. The position of individual teeth

I. Occlusion (jaws spatial interposition) is examined in fixed central position of the jaws (central occlusion). Firstly, key opposing teeth position is determined in the three projections:

- sagittal
- vertical
- horizontal

A) sagittal projection

1) mesio-buccal cusp (MBC) of the upper jaw first molar (primary or permanent) projects on the buccal groove of the low jaw first molar ("occlusion key " by Angle);

2) the upper jaw canine median line o projects between the low jaw canine and the first molar in the primary dentition and the canine and first premolar in permanent dentition;

3) the upper and the lower jaw incisors contacts with each other.

B) vertical projection

1) the upper and lower jaws teeth – antagonists (molars and premolars) have complete “fissure-cusp” contact with each other;

2) incisal overlap is 1/3 crown height of lower incisors;

C) horizontal projection:

1) buccal cusps of the lower molars and premolars are located in upper molar antagonists fissures;

2) the line between central incisors projection of upper and lower jaws are matched;

3) buccal cusps of the lower molars and premolars are located outwards from buccal cusps of the upper teeth.

II. The shape of the dental arches

Dental arches are examined with open jaws. In the primary dentition the dental arches have the shape of a semicircle, in the permanent dentition upper dental arch is shaped like a semi-ellipse, lower dental arch is shaped like a parabola.

III. The position of individual teeth

The teeth should take the place of an appropriate group affiliation, have contact with the adjacent teeth, does not displace the adjacent teeth and cause crowding.

To assess the quality of permanent dentition WHO recommends **index DAI (Dental Aesthetic Index) (1997, WHO).**

DAI is used for patients from 12 years old on 10 positions characterizing the main orthodontic problems (lack of teeth, incisors anomaly position and etc.), using different codes (Tab.№1). DAI - is the sum of the codes and values (mm). The DAI evaluation reveals the level of patient's orthodontic needs in treatment.

Tab. №21 "The standard DAI registration equation"

DAI Components	Weight
1. Number of missing visible teeth (incisors, canines and premolars teeth in maxillary and mandibular arches).	6
2. Crowding in the incisal segment (0=no segment crowded; 1=1 segment crowded; 2=2 segments crowded).	1
3. Spacing in the incisal segment (0=no spacing; 1=1 segment spaced; 2=2 segment spaced).	1
4. Midline diastema in millimeters.	1
5. Largest anterior irregularity on the maxilla in millimeters.	1
6. Anterior maxillary overjet in millimeters.	2
7. Anterior mandibular overjet in millimeters.	4
8. Vertical anterior openbite in millimetrs.	4
9. Anterior-posterior molar relation (largest deviation from normal either left or right): 0=normal; 1=1/2cusp either mesial or distal; 2=one full cusp or more either mesial or distal.	3
10. Constant.	13
Total	**DAI sco**

WHO, 1997.

Etiology of malocclusion can be categorized as:

1. **Malocclusion which is genetically induced or acquired during developmental processes**, associated with deep disturbances in the embryonic period and various harmful influences during the primary and permanent dentition.(short frenulum of tongue and lips, the small oral vestibulum, palate split, etc.) It includes anomalies of teeth, jaws positions or system anomalies in the maxillofacial region.

The dentist should diagnose the risk factors either during postnatal occlusion formation or even during mother's pregnancy.

2. Malocclusion caused by premature tooth loss due to caries or trauma

Primary partial or total adentia

Secondary partial adentia (early teeth extraction)

Prevention measures are prosthetics and appliances saving space in the dental arch

3. Malocclusion resulting from deleterious oral habits in a child.

Posture

Causes:

- the use of pillows for a under one year child
- premature baby sitting
- the wrong choice of furniture for your child

The role of the dentist:

- Direction to the orthopedist
- Direction to the orthodontist
- Elimination of harmful habits
- Motivation on effects of incorrect posture on occlusion

Sucking reflex and bad habits associated with it. Sucking reflex formation begins from the 18th week of prenatal development. And it disappears to the end of the 1st year of life.

Causes:

- prolonged breastfeeding
- psychological characteristics of the child
- thumb-sucking
- prolonged baby's dummy sucking
- feeding from a bottle after 1 year of life

The role of the dentist:

- ✓ Direction to the psychologist
- ✓ Direction to the orthodontist
- ✓ Elimination of harmful habits
- ✓ Motivation on effects of sucking reflex on occlusion
- ✓ Diet correction

Respiratory dysfunction.

Causes:

- ENT pathology (adenoid hyperplasia, atrophic rhinitis, deviation of nasal septum, antritis etc.)
- hypotonus of the oral circular muscles
- habits

Clinical symptoms:

- ❖ cheilitis
- ❖ wide and short bridge of nose
- ❖ slit-like entrances to the vestibule of the nose
- ❖ open mouth
- ❖ “gothic” palate

The role of the dentist:

- Direction to the ENT specialist (in according to needs)

- Direction to the orthodontist
- Oral myotherapy
- Elimination of harmful habits

Chewing dysfunction.

Causes:

- ✓ prolonged breastfeeding
- ✓ the habit to drink main dishes
- ✓ psychological characteristics of the child
- ✓ tmj dysfunction
- ✓ soft food diet

Clinical symptoms:

- lack of wearing cusps of primary teeth after 5 years
- the absence of tremas and diastemas
- *The role of the dentist:*
- Direction to the orthodontist
- Oral myotherapy
- Elimination of harmful habits
- Diet correction

Swallowing dysfunction.

- *Causes:*
- hypotonus of tongue muscles
- ENT pathology
- psychological characteristics of the child
- prolonged breastfeeding

Clinical symptoms:

- Open bite
- Protrusion of incisors
- “Thimble” sympthom

The role of the dentist:

- Direction to the ENT specialist (in according to needs)
- Direction to the orthodontist
- Oral myotherapy

- Elimination of harmful habits

The dysfunction of pronunciation

Causes:

- ✓ incorrect position of the tongue
- ✓ soft or hard tissues anomalies
- ✓ psychological characteristics of the child
- ✓ malocclusion

The role of the dentist:

- Direction to the logopedist
- Direction to the orthodontist
- Direction to the oral surgeon
- Oral myotherapy
- Ellimination of harmful habits

Malocclusion results in:

- Irregular /protruded teeth that interfere with function, esthetics & increase likelihood of injury to dento-facial complex.
- Functional disturbances involving speech, mastication, swallowing and TMJ function
- Impaired esthetics affecting child's psychological development.

Strategy for Prevention of Malocclusion:

1. Early identification of local and environmental factors that can induce malocclusion

2. Coordination with medical professionals to identify congenital anomalies.

3. Diagnose potential malocclusions in the deciduous dentition at the age of five or six.

4. Correct malocclusion like cross-bites, distal occlusion, space problems and deep bites in early-mixed dentition at the latest, without the need for fixed orthodontic treatment later.

5. Guide the growth of the jaws with myofunctional/orthopedic appliances in early or late mixed dentition.

6. Ensure the proposed preventive /interceptive treatment should have long term benefits

The clinical oral diagnosis includes:

- ➢ evaluation of facial proportions

- relation of teeth to the underlying bone structure
- tooth displacement
- functional analysis for the presence of any deleterious habits and or occlusal dysfunction
- identifying Oro-facial muscle imbalance
- caries risk assessment
- overall oral health

Diagnostic records: photographs, diagnostic casts, intra & extra-oral radiographs.

Primary Prevention

- Regular checkups for early intervention.
- Parent Teacher Counseling
- Facilitate speech therapy and genetic counseling if required
- Control of harmful oral habits
- Radiographic Assessment for malocclusion
- Preservation and restoration of primary and permanent dentition.

Secondary Prevention.

- ✓ Habit-breaking appliances.
- ✓ Correction of Oro-facial muscle imbalance with myofacial appliance
- ✓ Monitored Serial extractions for space maintenance.
- ✓ Space-maintainers/ regainers, and functional appliances to correct jaw relations.
- ✓ Simple appliances can be used to correct anterior cross-bites.

Tertiary prevention

The role of the general dentist at this stage is to refer the patient to an orthodontist as corrective orthodontic treatment includes the use of fixed and removal appliances and surgical orthodontics in cases of severe malocclusion that cannot be accomplished by a general dentist.

Recommendations for Preventive Orthodontic Program

- Clinical oral examinations
- Assess of oral growth and development
- Anticipatory Guidance
- Counseling for oral habits
- Radiographic Assessment

- Assessment/ treatment of developing Malocclusion
- Third Molar Assessment

Oral habits include:

1. Thumb/ Digit sucking: Non-nutritive sucking of thumb or finger.)

2. Bruxism : Habitual non-functional forceful contact between occlusal tooth surfaces

3. Self-injurious habits: Repetitive acts like lip biting, gingival mutilation resulting in damage.

4. Mouth breathing: Oral or combined pattern naso-oral breathing; habitual or due to airway obstruction.

5. Tongue thrusting. Abnormal tongue position & deviation from normal adult swallowing pattern.

Tab.№ 22 Etiology, Manifestations and Management of Oral habits

Habit	**Etiology**	**Systemic & Oral Manifestations**	**Management**
Thumb/Digit Sucking	Thumb sucking habit is a behavior of multi-factorial etiology. In fetuses, thumb sucking is first observed as early as 12 weeks gestational age. This habit has been related to sucking reflex (which remains until 12 months of age), and the rooting reflex (which remains until 7 months of age). Other suggested causes include emotional satisfaction which is often associated with this habit by	**A** - Child with Prolonged TS habit is usually emotional and an introvert and may display temper tantrums and show psychological problems. - Increased chances of accidental poisoning. Increased chance of*Enterobius-vermicularis* - Interference with selected aspects of speech production **B-** Digital malformations and inflammations, paronychia angular and rotational deformities. - Sublingual mucosal ulcerations. **C**- Oromyofunctional disorders: Mouth	Intervention before 4 to 4.5 years of age is not recommended Some forms of the habit should be eliminated before complications affect the child's health or functioning such as when habit is chronic or severe or is combined with another habit which needs to be stopped. If habit persists during the eruption of permanent incisors, the habit should be stopped due to dental considerations because of the

	serving as a comforter or tension reliever.	breathing. Forward resting posture of the tongue and Tongue trust **D**- Orofacial TMD-related pain in preschool children. - Minimal alterations of skeletal pattern. **Malocclusion** Depends on digit being sucked, intensity of the habit, position of mandible, cheek &lip pressure, facial morphology and frequency of the habit. More than 4-6 hours devoted for sucking may affect dentofacial anatomy. **Effects on dentition:** Flared and spaced maxillary incisors displaced forward. Lingually positioned lower incisors. Anterior open bite (asymmetrical).Narrow maxillary arch .Habit may also cause posterior crossbite and minor skeletal changes, If finger sucking stops at around age four, normal lips and cheek pressure will restore teeth to their original position.	increased tendency for permanent malocclusion. However, all interventions should not be forceful so as to not affect a child's well being. **Aversion taste treatment** Parents are instructed to coat their child's thumb (finger) nail three times a day with a commercially available substance **Ace bandage treatment** (elbow splint) This is used primarily for nocturnal habits. **Cotton glove** This seems to be the most effective measure of response prevention especially for unconscious (nocturnal) and non-meaningful habits. An advantage of this method is that it can be used at any time of the day. **Treatment** : - Child /Parent counseling - Fixed or removable habit

			breaking appliances.
Tongue Thrusting	Certain types of artificial nipples used in feeding infants. Thumb sucking, allergies, nasal congestion or obstructions contributing to mouth breathing causing the posture of the tongue to be very low in the mouth Large tonsils, adenoids, or many sore throats which cause difficulty in swallowing. An abnormally large tongue. Hereditary factors within the family, such as the angle of the jaw line Neurological, muscular, or other physiological abnormalities. Short lingual frenum.	Anterior open bite. Anterior thrust - upper incisors are extremely protruded and the lower incisors are pulled in by the lower lip. Unilateral thrust - the bite is characteristically open on either side. Bilateral thrust - the anterior bite is closed; however the posterior teeth from the first bicuspid to the molars may be open on both sides. Bilateral anterior open bite - the only teeth that touch are the molars with the bite completely open on both sides including the anterior teeth. Once again a large tongue is also noted. Closed bite thrust - typically shows a double protrusion meaning that both the upper and lower teeth are flared out and spread apart**.**	- **Interception:** Patient counseling, myofunctional exercises and habit breaking appliances such as cribs, rakes, overlay bite plates, interocclusal elastics, maxillary expanders and loose fitting orthodontic trainer appliances. Myofunctional therapy is indicated in severe situations as soon as the child is able to understand the problem and cooperate with the therapist. Dentists can instruct the child in the proper way of swallowing and this knowledge, coupled with changes in the relationships of teeth during orthodontic treatment, can correct the problem**.** **Treatment** should also include Surgical or non-surgical treatment

			of the underlying cause.
Self-injurious habits	Rare in normal child. Associated with Mental retardation Psychiatric disorders Developmental disabilities and some syndromes	Mutilation of oral tissues resulting in infective conditions of oral mucosa, lip and tongue. Early loss of teeth	Pharmacological management, behavioral modification and physical restraint. Dental treatment includes lip bumper, occlusal bite plane, protective padding and selective extractions.
Bruxism	Emotional stress, Parasomnias Brain injury Neurological disabilities Malocclusion T MJ dysfunction Improper muscle function	Dental attrition and TMJ dysfunction muscle soreness and associated occlusion problems	**AAPD** supports intervention for bruxism when the habit is of sufficient persistence, duration, or intensity to damage the permanent teeth or cause other complications which affect the child's well-being .Self limiting in Juvenile stage but needs Patient /Parent education. Treatment include Occlusal splints, Psychological Techniques & medication.

Oral miofunctional therapy

Oral miofunctional therapyis one of the leading methods of prevention and treatment of dentoalveolar anomalies due to dysfunction of muscles. The most appropriate age for understanding and proper execution is 4-7 years . Main oral

miofunctional therapy includes 2 groups of exercises, static and dynamic, and always starts from game and breathing exercises.

Pedagogical principles of miotherapy:

- visualization,
- apprehensibility (in understanding for child form),
- sequence,
- regularity.

Miotherapy for patients with mouth breathing includes breathing exerises and exercises for oral circular muscles tonus, using apparatus (Dass's activator, Shonher's plate, ekvilibrator) and without them.

Miotherapy for patients with incorrect swallowing includes exercises for muscules of tongue front-third ("clock", "horse", "hammer")and soft palate (rinsing mouth with water, yawn), rarely for posterior third of tongue (only after lingual frenum plastic, eliminating of harmful habits, sucking fingers and other, normalized diet implement).

Miotherapy for patients with inactive and sluggish mastication includes exercises for muscles lifting and pushing forward the lower jaw after eliminating the causes of dysfunction (tmj dysfunction, soft food diet, adentia).

Miotherapy for patients with speech dysfunction includes exercises for the overall tone of the tongue muscles and more specific exercises with logopedist

Oral miofunctional therapy procedures can be individual or group (10-12 children with similar dysfunctions). Lessons of therapeutic exercises can be conducted in the dental clinic, the office of physical therapy, the speech specialist office, at home. Miofunctional therapy consist of introductory, basic and final parts. Duration of miotherapy course for maximal efficiency shouldn't be less than 3 months.

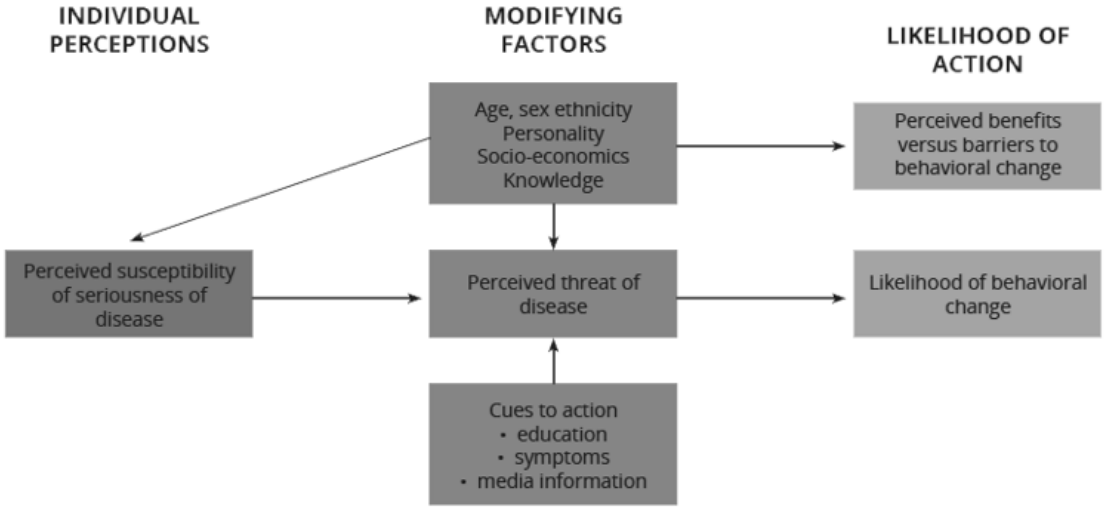
XIX. ORAL HEALTH EDUCATION AND PSYCHOLOGICAL ASPECTS AND STRATEGY FOR HEALTHY HABITS FORMATION

Health promotion - the process of enabling people to increase their awareness of, responsibility for, control over, and improvement of their health and well-being.

Education - the application of teaching and learning principles to facilitate the development of specific attitudes, knowledge, skills, and behaviours.

Motivation – psyhological concept of involving the person in his health problem; internal and external factors that stimulate desire and energy in people to keep health.

Health behavior is the activity undertaken by individuals for the purpose of maintaining or enhancing their health, preventing health problems, or achieving a positive body image. It is not limited to healthy people trying to stay healthy, but also includes the physically handicapped and persons with chronic diseases who seek to control, minimize, or contain their affliction through positive forms of health behavior, such as diet, exercise, and avoiding smoking.



Scheme № 4 «Model of health belief (U.S. Public Health Service, 1950)»

Steps in health education planning:

1. Identify needs and priorities.
2. Set aims and objectives.
3. Decide the best way of achieving the aims.
4. Identify resources.
5. Plan evaluation methods.
6. Set an action plan.
7. Evaluation.

WAYS OF PUBLIC EDUCATION:

- Dental public health professionals work with the schoolbased fluoride mouth rinse program.
- Suggestion and guidance on Community and school water fluoridation program.
- Classroom-based education for elementary school children on dental health, plaque awareness, and tobacco use prevention.
- Community oral health education and prevention initiatives in partnership with private dentists and other health care groups.
- Spit Tobacco Education Program activities.
- Community dental sealant, dental screening, early childhood caries, and baby bottle tooth decay education programs.
- Prevent abuse and neglect through Dental Awareness program.

Oral diseases are important public health problems. They are very prevalent and their impact on both society and the individual are significant. Pain, disability, and handicap from oral diseases are common, and the costs of treatment are a major burden to health care systems. The causes of dental diseases are known and the conditions are largely preventable. On the basis of those criteria, oral and dental diseases are a public health problem. Furthermore, inequalities in oral health are a problem; disadvantaged and socially excluded population groups suffer higher rates of disease than their more affluent contemporaries. The move towards an evidence-based approach to treatment and prevention has highlighted the limitations of conventional dental health education. Those limitations and the expansion of concepts on health promotion has lead to a wider recognition that there is a need to adopt a more progressive approach to prevention. In this chapter, oral health promotion will be defined and an outline given of the philosophy underlying the dominant approaches in health promotion, Health For All, and the intersectoral action. The epidemiological basis for strategy is explored and the application of an integrated approach to chronic disease control, the common risk/health approach examined. Case studies using food and periodontal health policies will illustrate the relevance of these approaches to oral health.

Limitations of conventional dental health education

Dental health education aims to promote oral health through educational means, principally the provision of information toimprove oral health knowledge and awareness. Through the acquisition of knowledge, a change in behaviour is then considered likely to occur. This rather simplistic and outdated approach has dominated dental health education practice for many years but fails to acknowledge

the complexities of human behaviour and the importance of the broader social, economic, and environmental factors determining behaviour change.

Definitions of health

The modern health promotion movement has emerged out of the need for a fundamental change in strategy to achieve and maintain health. It is based on a public health philosophy that should encompass the prevention of disease at a primary level, and secondly, the promotion of health (Milio 1988). These two concepts, when applied to developing environments which promote healthier choices for people in coping with their lives, need to be adopted in a manner that encourages those choices to be the easiest choices (Milio 1986). Health promotion can be considered 'as the combination of educational and environmental supports for actions and conditions of living conducive to health' (Green and Kreuter 1990). Strategies to change 'the range of options available to people and to make health-promoting choices easier and/or to diminish health damaging options by making them more difficult to choose' (Milio 1986). Another definition is 'health promotion is the process of enabling individuals and communities to increase control over the determinants of health and thereby improve their health. Health promotion represents a mediating strategy between people and their environment, combining personal choice and social responsibility for health to create a healthier future' (WHO 1984). It is directed to the underlying determinants as well as the immediate causes of health. The causes of the causes.

General principles of oral health promotion

Oral health promotion has emerged in line with developments in general health promotion. Oral health promotion strategiesshould be based upon the following guiding principles:

• Base action upon a comprehensive needs assessment using both normative and lay measures of need

• Develop a range of clearly stated and challenging goals

• Preventive rather than curative approaches—promote public health measures to the public and public authorities, e.g. fluoride in water.

• Be based upon contemporary theories of individual and organizational change

• A re-orientation from prescription to supportive health promotion methods—redress the balance of influence and make healthier choices easier. Promote self-esteem and facilitate decision-making skills rather than be prescriptive.

• Combat the influence of those interests which produce and profit from ill health. This involves controls on industrysponsored educational materials in schools, advertising, and campaigns to reduce barriers to good oral health.

• Public health rather than individually focussed programmes.

• Focus on the social causes of ill health rather than a victimblaming approach—acknowledging the limited real choices available to any individual.

• Address the underlying determinants of oral health.

• Tackle causes that are common to a number of chronic diseases.

• Supportive rather than authoritarian styles of action.

• A commitment to distribute success equitably

• Ensure actions are evidence based.

• Community participation rather than professionally dominated activities.

• Working in partnership with key groups and agencies.

Selecting a strategy is influenced by these criteria and philosophical, professional, and political perspectives. The epidemiological basis for strategy selection for oral health promotion is the Common Risk Factor Approach and the Whole Population Strategy (Rose 1992).

The role of dentists in oral health promotion

Most dental public health officer involvement will be as health advocates. Health advocacy is the actions of health professionals and others with perceived authority in health, to influence the decisions and actions of individuals, communities, and government which influence health. Health advocacy involves educating senior government and community leaders and journalists— decision-makers in general, about specific issues, and setting the agenda to obtain political decisions that improve health of the population. To increase effectiveness, advocates work within the dominant philosophy in public health, namely, building partnerships with the community, other professional groups, and other sectors. They place their skills at the disposal of the community. Being on tap not on top.

Dentists must become team members in advocacy and education working with other organizations, government sectors, and with community organizations. The role of individual practitioners in prevention, is limited. Public health dentists should work as health advocates and co-ordinate local health promotion initiatives by first establishing a local Oral Health Promotion Group (OHPG) to develop an action plan, using goals and strategies as guidelines. They and other health promoters should work with industry to improve key products (such as low sugars and sugarfree snacks and drinks). Other interventions require government action, most notably developing policies on sugar production and promotion, safer environments to enhance social cohesion and reduce violence and accidents.

Within the health service oral health promoters should be active in the training of other primary health care workers (including dental) and care workers outside the health services. Support should be given to carers in the youth

education and welfare services. This should include the promotion of oral health of individual workers, as they are unlikely to accept responsibility to promote good oral health habits in their clients unless they are supported in their efforts to achieve good oral health for themselves.

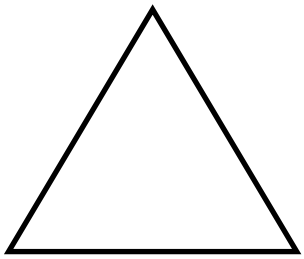
Dentist must organize working with children in such way that the treatment is the most effective and the child has positive attitude about dentistry. Pediatric dental appointment unites child's parents and staff of dental clinic to the "triangle of Pediatric Dentistry".

The role of patient's parents in the pediatric dental appointment:

1) parent is responsible person for the child his dental health, 2) family, personal qualities of the parents, their relation to dentistry in general and in particular to the doctor, parents' behavior before and during the appointment are important factors in determining the child's behavior in the dental office.

Thus there is formed a "triangle of Pediatric Dentistry"(Fig. 1).

Patient-child



Parents (family) *Dentist (dental office stuff)*

Fig. 1. Triangle of Pediatric Dentistry

Children learn the basic aspects of everyday life from their parents, this process is termed socialization and is an ongoing and gradual process. By the age of 4 years children know many of the conventions current in their culture, such as male and female roles. The process of transmitting cultural information early in life is called primary socialization. In industrialized countries, obtaining information on many aspects of life is gained formally in schools and colleges rather than from the family.

This is termed secondary socialization. Interestingly, primary socialization can have a profound and lasting effect. For example, fear of dental treatment and when we first begin to clean our teeth can often be traced back to family influence. So parents can shape a child's expectations and attitudes about oral health; thus, every attempt should be made to involve them when attempting to offer dental care or change a child's health habits.

Involving parents means that the dentist must look to positive reinforcement rather than 'victim blaming'. Parents who are accused of oral neglect may well feel

aggrieved or threatened. All too often children's oral health is compromised by a lack of parental knowledge so programmes have to be carefully designed to reduce any chances of making people feel guilty. Guilt often results in parents spending more time in seeking excuses for problems than trying to implement solutions. Parents who are convinced that their child has an oral health problem which can be solved tend to react in a positive way, both to their dental advisor and the preventive programme itself. It is especially helpful if the preventive strategy can include a system of positive reinforcement for the child . Features such as brushing charts, diet sheets, gold stars for brushing well, extra pocket money for curtailing thumb-sucking are all useful tips to help parents maintain a child's enthusiasm for a particular dental project.

It must be emphasized that preventive programmes must be carefully planned to include only one major goal at a time. Parents will be unable to cope if too much is expected of them at any one time. Programmes that involve families have much higher success rates than those which concentrate solely on the patient. Interestingly, families also have a profound influence on levels of dental anxiety among their children. Dentally anxious mothers have children who exhibit negative behaviour at the dentist.

The results of proper behavior management:

1. To prevent the development of anxiety it is more important to maintain trust than concentrate on finishing a clinical task.

2. The reduction in dental caries means that children with special psychological, medical, and physical needs can be offered the oral health care they require.

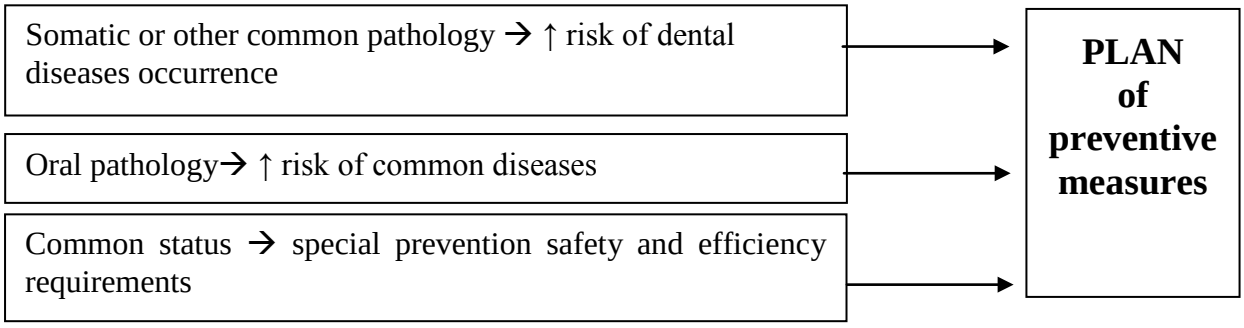
3. The care of children who are very anxious can be improved by using the techniques described in this chapter.

4. Preventing dental disease should always be given the same status as clinical intervention. However, it is important to ensure that preventive care is appropriate and relevant.

XX. INDIVIDUAL PROGRAMS FOR DENTAL DISEASES PREVENTION.

Physiological, mental, physical and oral patient's status influence on the occurrence and clinical course of dental diseases and hence on their prevention scheme. That's why a dentist should analyze a patient's status of several positions, which are described in Tab. №1

Tab.22 «Dentist's preventive tactics for patients with common pathology»



Tab. №23 **PREVENTIVE DENTAL PROCEDURES DURING AND AFTER PREGNANCY**

CHARACHTERISTICS	RISK FACTORS AND CONSECUENCES	PREVENTIVE MEASURES
Common pathological triggers	**Nausea and vomiting** →erosion of oral tooth surfaces.	Mouth guard, alkaline rinse, local fluoride-containing supplement application.
	Saliva properties change → ↓ pH and ↓stimulated salivary flow rate decrease remineralization opportunities.	Proper oral hygiene, healthy diet, local fluoride-containing supplement application.
	Increased level of estrogen → epithelium and GPRS proliferation and desquamation growth and ↑ currency of gingival fluid → ↑ ↑ bacterial growth → risk of caries and gingivitis.	Proper oral hygiene, healthy diet, local fluoride-containing supplement application.
	Increased levels of estrogen increase the permeability of capillaries →proliferative inflammation of	Proper oral hygiene.

	gingivitis gums→hyperplasia, pyogenic granuloma	
	Frequent snacks → increase in cariogenic plaque quantity	Proper oral hygiene, healthy diet.
Dental diseases influence on pregnancy	**Periodontal diseases**→risk of preterm birth, risk of low-weight baby birth.	Proper oral hygiene.
Oral hygiene specialties	**Nausea** → difficulties with the morning brushing.	Choice of individual oral hygiene products with suitable perfumes and toothbrush with small size head.
Maternal pathological status as risk factor for child oral diseases	**Pathology of pregnancy**→ malformations and / or low caries resistance of children teeth.	Dental health education, prophylactic medical examination
	Postpartum depression →poor oral care →increase in cariogenic flora → ↑ risk of child's contamination.	Dental health education, prophylactic medical examination.
Maternal oral diseases influence on child oral pathology	Chronic dental pathology → stress, infection, intoxication, drug effects → malformation and / or ↓ baby caries resistance.	Secondary prevention of oral diseases.
	Poor oral hygiene →increase in cariogenic microflora →risk of early childhood caries	Dental treatment, using of antiseptics, education in baby oral care.

PREVENTION OF ORAL DISEASE IN CHILDREN

Individual oral hygiene products are prescribed by dentist according to psychophysical, somatic and dental status of the patient, his age, severity of risk factors or actual dental pathology.

From birth to 1 year. Before tooth eruption the oral hygiene procedures can include mucosa wiping with impregnated gauze swabs or special hygiene wipes. Child's regular brushing (cleaning) procedures must begin from the moment of first tooth eruption with silicone-fingertip or parents' toothbrush (possessing the

proper form and size properties). The proper age to accustom the baby to his own toothbrush is 1year in average.

It is necessary to use of calcium/phosphate- and fluoride-containing (***500 p.p.m***) toothpastes alternately (for 2 weeks each cycle). But presently it is controversially to use fluoride-containing pastes in children under 3 years because of their unformed swallowing reflex and spitting possibility.

At the age of 1-3 years of brushing is provided by parents with narrow little head and long handle toothbrush. It is recommended to use calcium/phosphate- and fluoride-containing (***500 p.p.m***) toothpastes alternately (for 2 weeks each cycle).

Parents should teach the child to spit out the toothpaste, rinse his mouth after brushing, and help to improve the ***KAI method*** motions. All motions are demonstrated taking the child's hand in parents' one.

At the age of 4-6 years children continue to improve KAI method, parents control the quality of teeth brushing and help to complete the procedure. It is recommended to use soft bristle toothbrush (in the case of Priestley plaque medium hardness), "pea"-sized dose (0,2-0,3g) calcium/phosphate- and fluoride-containing (***500 p.p.m***) toothpastes alternately (for 2 weeks each cycle) for tooth brushing. Parents can begin to use interdental cleaning from 4-5 years.

In the age of 7-10 years (primary school) it is recommended to use medium bristle toothbrushes, "pea"-sized dose (0,2-0,3g) calcium/phosphate- and fluoride-containing (7-8 years ***800-900 p.p.m***., over 9 years **– *1000-1500 p.p.m.***) toothpastes alternately (for 2 weeks each cycle) for tooth brushing. This age children are able to learn flossing in the anterior region. But main part of procedure is provided by parents. Children older than 10 years have physical and mental ability to carry out hygienic procedures completely independently. ***Liquid oral hygiene products*** are used for a hygienic (mouthwash, oral deodorants) and special (balms and herbal solutions) purposes. Liquid hygiene products can be recommended only for patients, controlling swallowing, i.e. children over 6 years. Rinses are applied usually before and after tooth brushing, after meals.

In the age of 10-14 years children should to use Martaller's brushing method. But they need the supervision of their parents. It is recommended medium bristle toothbrushes and toothpastes with "adult" concentration of fluoride (1000-1500 p.p.m.).

In the age of 15-18 years the role of parents in children's oral hygiene is reduced. Parents take part in motivation and monitoring of their children oral hygiene.

PREVENTION OF ORAL DISEASES IN AGED PEOPLE

- Dental health education of the patient and his family.
- Individual oral hygiene fluoride-containing products.
- IOHP with antiseptics.
- Topical fluoride-containing supplements application.
- Correction of hyposalivation and xerostomia treatment.
- Using of additional IOHP (dental brushes, brushes for dentures, irrigators, electric tooth brushes, interdental stimulators, etc.).
- Regular professional oral hygiene with careful root cement cleaning.
- Correction of restorations, fillings, dentures and appliances.
- Dental treatment and prosthetic.
- Preventive clinical examination.

PREVENTION OF ORAL DISEASES IN PATIENTS UNDERGOING IMPLANTS TREATMENT

- Individual oral hygiene fluoride-containing products.
- IOHP with antiseptics.
- Topical fluoride-containing supplements application.
- Using of additional IOHP (dental brushes, brushes for appliances, moonbeam brushes, superflosses, dental tapes, irrigators, «Ortho» tooth brushes, i, etc.).
- Professional oral hygiene.
- Contraindications for ultrasonic scalers and brushes**.**

PREVENTION OF ORAL DISEASES IN PATIENTS WITH ORTHODONTIC APPLIANCES

- Proper dental home.
- IOHP with fluoride.
- IOHP with antiseptics.
- Topical fluoride-containing supplements application.
- Use of fluoride-emitting adhesives and fixing materials.
- Fissure sealing.
- Using of additional IOHP (dental brushes, brushes for appliances, moonbeam brushes, superflosses, dental tapes, irrigators, «Ortho» tooth brushes, i, etc.).
- Professional oral hygiene.
- No use of ultrasonic scalers and brushes.

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учебно-методическое пособие
на английском языке

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