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PREVENTIVE DENTISTRY

Part 1

Minsk BSMU 2015

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

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ПРОФИЛАКТИКА СТОМАТОЛОГИЧЕСКИХ ЗАБОЛЕВАНИЙ

PREVENTIVE DENTISTRY

Учебно-методическое пособие
В 2-х частях

Часть 1

Минск БГМУ 2015

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Посвящено вопросам профилактики стоматологических заболеваний как части предмета медицинской профилактики. Изложена современная концепция этиопатогенеза кариеса зубов и рассмотрены основные направления профилактики данной патологии. Также описаны особенности клинического обследования пациента на стоматологическом приеме и способы выявления управляемых факторов риска развития основных стоматологических заболеваний.

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

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WORKSHOP № 1
TEST CONTROL OF BASIC KNOWLEDGE. WORKPLACE FOR PREVENTIVE ACTIVITIES

Test Questions:

1. The concept of health and disease. Medical prevention. The objectives and stages of prevention in dentistry (primary, secondary, and tertiary).

2. Scientific rationale for primary prevention of dental diseases. Principles of organizing primary prevention activities.

3. WHO's global goals and objectives with respect to oral health improvement.

4. Sanitary standards of the workplace/working premises. Concept of minimum and optimum equipment standards for the preventive activities workplace.

5. Measures to ensure technical and chemical safety during dental procedures.

6. Measures to prevent cross-infection exchange between staff and patient. Methods and tools to disinfect and sterilize used dental materials/equipment.

Health is defined in the World Health Organization's Constitution as «a state of complete physical, social and mental wellbeing, and not merely the absence of disease or infirmity».

Health is complex and involves the interaction of various factors. Public Health Agency of Canada and the World Health Organization have identified 12 determinants of health:

– income and social status;

– employment;

– education;

– social environments;

– physical environments;

– healthy child development;

– personal health practices and coping skills;

– health services;

– social support networks;

– biology and genetic endowment;

– gender;

– culture.

Disease may be defined as a disorder of structure or function in a human that produces specific symptoms or that affects a specific location and is not simply a direct result of physical injury.

The theories of diseases causation are the next.

Germ theory: disease is caused by transmissible agents. A specific agent is responsible for one disease only (one-to-onerelationship).

Epidemiological triad: exposure to an agent does not necessarily lead to disease. Disease is the result of an interaction between agent, host and environment. Disease can be prevented by modifying the factors that influence the exposure and susceptibility.

Web of causation: disease is a result of complex interaction of many risk factors. Any risk factor can be concerned in more than one disease. Disease can be prevented by modifying these risk factors.

General susceptibility: some social groups have higher mortality and morbidity rates from all causes. It is an imperfectly understood general susceptibility to health problems. This is probably because of complex interaction of the environment, behavior and life-styles.

Socio-environmental approach: health is strongly influenced by social and physical environment. Risk conditions produced by such an environment affect health directly and through the physiological, behavioral and psychosocial risk

factor that they create. Improving health requires modification of these environments.

Prevention can be defined as «the action of keeping from happening, or of rendering impossible, an anticipated event or act». Prevention in health care means action to stop ill health before it begins.

The levels of medical prevention are:

– primordial prevention;
– primary prevention;
– secondary prevention;
– tertiary prevention.

Primordial prevention. It is the prevention of emergence or development of risk factors in countries or population group in which they have not yet appeared. Individual and mass education is the main intervention method in primordial prevention.

Primary prevention. It is defined as «action taken prior to the onset of the disease, which removes the possibility that a disease will even occur». It is carried out on healthy populations. Information and/or public health measure to the whole population may be sufficient to maintain a disease free environment. It may be accomplished by measures designed to promote general health and well being or by specific protective measures.

Secondary prevention. It can be defined as «actions which halts the progress of a disease at its incipient stage and prevents complications». It is carried out on targeted population identified by their being exposed to, or indulgence, in factors that place them «at risk». The individual or the population is required to change, either to take some new action, or to cease an established action, or both, in order to lower the levels of risk.

Tertiary prevention. It provides a cure at an early stage in disease process, containing the disease or its effects on a long term basis and seeks to prevent a recurrence of the disease. It can be defined as «all measures available to reduce or limit impairments and disabilities, minimizing suffering caused by existing departures from good health and to promote the patients adjustment to irremediable conditions». The individual or population is aware of the disease, can see its effects and requires rehabilitation.

A fundamental principle for all preventive action is that the positive effect is greatest where the risk of disease development is greatest.

Primary preventive dental services are those that prevent the initiation of oral disease. There are two approaches in primary prevention: *health promotion* and *specific protection*.

Health promotion is a 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.

• Lifestyle and behavioural changes which favor health.

Specific protection is the activities designed to protect against disease agents by decreasing the susceptibility of the host or by establishing barrier against agents in the environment. Methods include immunization, use of specific nutrition, avoidance of allergens, protection from carcinogens, ingestion of optimally fluoridated water and application of pit and fissure sealants.

Global Oral Health Goals, Objectives and Targets for the Year 2020

(Adopted by the FDI General Assembly: 18th September 2003 Sydney, Australia)

Goals. To promote oral health and to minimise the impact of diseases of oral and craniofacial origin on general health and psychosocial development, giving emphasis to promoting oral health in populations with the greatest burden of such conditions and diseases.

To minimise the impact of oral and craniofacial manifestations of general diseases on individuals and society, and to use these manifestations for early diagnosis, prevention and effective management of systemic diseases.

Objectives:

• To reduce mortality from oral and craniofacial diseases.

• To reduce morbidity from oral and craniofacial diseases and thereby increase the quality of life.

• To promote sustainable, priority-driven, policies and programmes in oral health systems that have been derived from systematic reviews of best practices (i. e. the policies are evidence-based).

• To develop accessible cost-effective oral health systems for the prevention and control of oral and craniofacial diseases using the common risk factor approach.

• To integrate oral health promotion and care with other sectors that influence health.

• To develop oral health programmes to improve general health.

• To strengthen systems and methods for oral health surveillance, both processes and outcomes.

• To promote social responsibility and ethical practices of care givers.

• To reduce disparities in oral health between different socio-economic groups within countries and inequalities in oral health across countries.

• To increase the number of health care providers who are trained in accurate epidemiological surveillance of oral diseases and disorders.

Targets. The targets should be selected to match predetermined oral health priorities at a national or local level. Consideration should be given to the following areas when selecting targets, based on local priorities: pain, functional disorders, infectious diseases, oro-pharyngeal cancer, oral manifestations of HIV-infection, noma, trauma, cranio-facial anomalies, dental caries, developmental anomalies of teeth, periodontal diseases, oral mucosal diseases, salivary gland disorders, tooth loss, health care services, health care information systems.

Sanitary standards of the workplace are different in various countries. According to concept of optimum equipment standards for the preventive activities workplace has to have:

– special room in a good natural and artificial lighting;

– central water;

– dental unit with an air gun, micromotor and turbine handpieces;

– saliva ejector and vacuum ejector;

– special chairs for the dentist and assistant;

– dental table;

– sterile table tools;

– cabinet for preventive drugs;

– desk for paper work.

If the dentist is working in minimum equipment standards for the preventive activities his workplace has only:

– ordinary chairs for doctor and patient;

– tray for tools;

– a portable light source and a reflector;

– container with antiseptic.

There are many various hazards in the work of dentist: mechanical, chemical, infectious.

Previous Centers for Disease Control and Prevention (CDC) recommendations regarding infection control for dentistry focused primarily on the risk of transmission of blood borne pathogens among dental health care personnel (DHCP) and patients and use of universal precautions to reduce that risk. Standard precautions integrate and expand the elements of universal precautions into a standard of care designed to protect DHCP and patients from pathogens that can be spread by blood or any other body fluid, excretion, or secretion.

Standard precautions apply to contact with 1) blood; 2) all body fluids, secretions, and excretions (except sweat), regardless of whether they contain blood; 3) nonintact skin; 4) mucous membranes.

The components of inflectional control are the following:

1. Immunization.

2. Patient screening.

3. Hand hygiene.

4. Barrier techniques.

5. Needle and sharp instrument safety.

6. Instrument sterilization and disinfection.

7. Surface disinfection and general operatory asepsis.

8. Radiographic asepsis.

9. Laboratory asepsis.

10. Disposal of contaminated wastes.

Personal protective equipment (PPE) is designed to protect the skin and the mucous membranes of the eyes, nose, and mouth of dental health care provider from exposure to blood. Primary PPE used in health care settings includes gloves, surgical masks, protective eyewear, face shields, and protective clothing (e. g. long sleeved gowns, jackets). Shoe and head covers are less frequently used types of PPE, but should be considered if contamination is likely.

Dental instruments are generally *sterilized* by one of the following methods:

1. Steam under pressure — gravity displacement or prevacuum sterilizer.

2. Dry heat — static air or forced air.

3. Unsaturated chemical vapor.

Single-use disposable instruments (e. g. prophylaxis angles; prophylaxis cups and brushes; tips for high speed air evacuators, saliva ejectors, and air/water syringes) should be used for one patient only and discarded appropriately. These items are neither designed nor intended to be cleaned, disinfected, or sterilized for reuse.

Postexposure Protocol for Occupational Exposure to Blood/Body Fluids:

1. Stop the procedure immediately.

2. Inform patient.

3. Remove gloves and apply first aid as required.

4. Injuries to the skin should be washed well with soap and running water and bleeding at the injured site can be encouraged while washing. Antiseptic treatment is also needed.

5. First aid measures should be applied to stop bleeding if required.

6. Mucous membranes should be flushed well with water. When splashes have occurred to the eye, the eyewash station is to be used to thoroughly flush the eyes. antiseptic treatment is also needed.

7. Clinical support staff should be notified to arrange for the completion of the treatment. The record in a special register should be made.

8. The clinical support staff should ask the patient to submit blood for the appropriate blood tests; (microbiology and serology) for HIV, etc.

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WORKSHOP № 2
ORAL EXAMINATION

Test Questions:

1. The purpose of oral examination during a preventive dental visit. Facilities and workplace organization for dental examination. Methods of oral examination.

2. Interaction with the patients, asking questions, evaluation of patient general condition.

3. External examination of maxillofacial area.

4. Examination of maxillofacial area functions.

5. Methods of objective examination of the oral cavity. The sequence of objective examination of oral cavity status. Rules for using hand instruments.

6. Assessment of oral soft tissues.

7. Bite assessment.

8. Teeth examination. Rules for dental status registration.

Case-history. The factors which influence the exposure of response include:

– age;

– sex;

– family size;

– marital status;

– religion;

– occupation;

– intercurrent disease;

– ethnic or racial factors;

– habits and customs;

– inherent immunity or non specific immunity;

– immunity — passive immunity, active immunity.

Extraoral examination. Clinical examination of the head and neck is an integral part of oral examination and provides valuable information on the overall assessment of possible oral diseases. The examination for cervical lymph glands is carried out by standing behind the individual and slightly flexing and bending the neck to the side so that the sternocleidomastoid muscle becomes relaxed and palpation and identification of any enlarged nodes will be easier. The presence of neck masses is not an uncommon finding, especially in subjects with oral infections. The submandibular, submental and upper deep cervical lymphnodes are commonly involved, although other regional lymph nodes may be enlarged as well. Lymphadenopathy secondary to infection will be tender and mobile, while metastatic lymph nodes are often asymptomatic, hard in consistency, and may be fixed to the underlying structures.

Examination should be carried out with adequate lighting from an external source such as fixed or head-mounted examination lights or hand-held flashlights, supplemented by room lighting. The procedure should be explained to the person and every effort should be taken to ensure that the subject is relaxed and not anxious. Anxiety during examination may cause a temporary dryness of the mouth. Palpation should be carried out wearing gloves.

The extraoral examination should be performed in the following sequence:

a) General overview of exposed skin areas (head, neck, limbs).

b) Perioral skin areas (nose, cheeks, chin).

c) Lymph nodes (head, neck).

d) Cutaneous parts of upper and lower lips.

e) Vermilion border and commissures.

f) Temporomandibular joint (TMJ) and parotid gland region.

Face. The extraoral assessment includes inspection of the face, head, and neck. The face, ears, and neck are observed, noting any asymmetry or changes on the skin such as crusts, fissuring, growths, and/or color change.

The regional lymph node areas are bilaterally palpated to detect any enlarged nodes. If enlargement is detected, the examiner should determine the mobility and consistency of the nodes. A recommended order of

examination includes the preauricular, submandibular, anterior cervical, posterior
auricular and posterior cervical regions.

Lips. Begin examination by observing the lips with the patient's mouth both closed and open. Note the color, texture and any surface abnormalities of the upper and lower vermilion borders.

Temporomandibular Joint Assessment. Temporomandibular joint pain and dysfunction, as characterized by the presence of crepitation, clicking and popping of the joints, may be detected by placing the tip of the little finger in the external auditory canal and having the person open and close the mouth and by moving the mandible laterally from side to side.

Clicking of one or both temporomandibular joints. Clicking is evaluated directly by an audible sharp sound or by palpation of the temporomandibular joints.

Tenderness (on palpation) of the anterior temporalis and/or masseter muscles on one or both sides. The tenderness should be evaluated by unilateral palpation with the firm pressure of two fingers, exerted twice on the most voluminous part of the muscle. Tenderness is recorded only if the palpation spontaneously provokes an avoidance reflex.

Reduced jaw mobility — opening of means < 30 mm, taken as the distance between the incisal tips of the central maxillary and mandibular incisors. As a general guide, in an adult jaw, mobility is considered to be reduced if the subject is unable to open his or her jaw to the width of two fingers.

INTRAORAL SOFT TISSUE EXAMINATION

Oral Mucosa. An examination of the oral mucosa and soft tissues in and around the mouth should be made on every subject. The examination should be thorough and systematic and be performed in the following sequence:

a) Labial mucosa and labial sulci (upper and lower).

b) Labial part of the commissures and buccal mucosa (right and left).

c) Tongue (dorsal and ventral surfaces, margins).

d) Floor of the mouth.

e) Hard and soft palate.

f) Alveolar ridges/gingiva (upper and lower).

Labial Mucosa. It is done with the patient's mouth partially open. Visually examine the labial mucosa and sulcus of the maxillary vestibule and frenum and the mandibular vestibule. Observe the color, texture, and any swelling or other abnormalities of the vestibular mucosa and gingiva.

Oral examination commences with the visual examination of the lips and the vermilion border. The lip is usually smooth and pliable. Maceration and cracking of the corners of the lips indicate angular cheilitis. Evert the lips and carefully inspect the labial mucosa. It should be smooth, soft and well-lubricated by minor salivary glands that can be palpated. One may observe a mucocele in the lower lip resulting from trauma to the minor salivary gland ducts, as the lower lip is frequently prone to injury, particularly from accidental biting.

Buccal Mucosa. Retract the buccal mucosa. Examine first the right then the left buccal mucosa extending from the labial commissure and back to the anterior tonsillar pillar. Note any change in pigmentation, color, texture, mobility, and other abnormalities of the mucosa, making sure that the commissures are examined carefully and are not covered by the retractors during
the retraction of the cheek.

Oral mucosa is generally pink in colour. Highly keratinized, firm, stippled and pale masticatory mucosa cover the hard palate, dorsal surface of tongue, and gingiva. Thin, less keratinized and more pinkish non-masticatory mucosa cover the remaining intra-oral structures. The examiner should be alert during the entire procedure to identify any change in colour and/or texture of the mucous membrane, inflammatory areas, erythema, hyperpigmentation, macules, papules, vesiculobullous lesions, white lesions, grayish white lesions, red
lesions, induration, ulceration, swellings and growth in the oral mucosa.

The opening of the parotid salivary gland duct, the Stensen duct, may be observed as a small papillary or punctate soft tissue mass on the buccal mucosa adjacent to the maxillary second molar tooth. Milking of the parotid gland may expel saliva at the duct opening. Ectopic sebaceous glands may be observed on

the buccal or labial mucosa as whitish-yellow, pinpoint papules; this developmental anomaly is termed as Fordyce conditions or granules. Minor salivary glands and Fordyce granules may lead to a granular feel on palpation of the buccal mucosa.

Gingiva. First, examine the buccal and labial aspects of the gingiva and alveolar ridges (processes) by starting with the right maxillary posterior gingiva and alveolar ridge and then move around the arch to the left posterior area. Drop to the left mandibular posterior gingiva and alveolar ridge and move around the arch to the right posterior area. Second, examine the palatal and lingual aspects as had been done on the facial side, from right to left on the palatal (maxilla) and left to right on the lingual (mandible).

Tongue. With the patient's tongue at rest, and mouth partially open, inspect the dorsum of the tongue for any swelling, ulceration, coating, or variation in size, color, or texture. Also note any change in the pattern of the papillae covering the surface of the tongue and examine the tip of the tongue. The patient should then protrude the tongue, and the examiner should note any abnormality of mobility or positioning, with the aid of mouth mirrors, inspect the right and left lateral margins of the tongue. Grasping the tip of the tongue with a piece of gauze will assist full protrusion and will aid examination of the more posterior aspects of the tongue's lateral borders. Then examine the ventral surface. Palpate the tongue to detect growth.

Floor. It is done with the tongue still elevated. Inspect the floor of the mouth for changes in color, texture, swellings, or other surface abnormalities.

Palate. With the mouth wide open and the patient's head tilted back, gently depress the base of the tongue with a mouth mirror. First inspect the hard and then the soft palate. Examine all soft palate and oropharyngeal tissues.

Bimanually palpate the floor of the mouth for any abnormalities. All mucosal or facial tissues that seem to be abnormal should be palpated.

Occlusion assessement:

– Relationship of the dental arches or dentoalveolar segments.

– Form of dental arches.

– Individual tooth positions.

Dentition Status. The examination for dental caries should be conducted with a plane mouth mirror. Radiography for detection of approximal caries is not recommended because of the impracticability of using the equipment in all situations. A tooth should be considered present in the mouth when any part of it is visible. If a permanent and primary tooth occupy the same tooth space, the status of only the permanent tooth should be recorded.

Care should be taken to diagnose tooth-colored fillings, which may be extremely difficult to detect. Codes for the dentition status of primary and permanent teeth (crowns and roots) are given in the table 1.

Table 1

Codes given by WHO

Codes		**Condition/status**
Primary tooth	**Permanent tooth**	
A	0	Sound
B	1	Decayed
C	2	Filled, with decay
D	3	Filled, no decay
E	4	Missing, as a result of caries
–	5	Missing, any other reason
F	6	Sealant, varnish
G	7	Bridge abutment, special crown or veneer/implant
–	8	Unerupted tooth
–	9	Excluded tooth
T	10	Trauma (fracture)

To assess dental caries in a population, a DMFT index is used. During a systematic examination with a mirror and CPI Probe that includes the crown and exposed root of every primary and permanent tooth, each crown and root are assigned a number based on the result of that exam. The numbers are recorded in boxes corresponding to each tooth to provide a DMFT chart.

It is recommended, that care should be taken to record all tooth-colored fillings, which may be difficult to detect.

Numbers are assigned as follows:

– 0: A zero indicates a sound crown or root, showing no evidence of either treated or untreated caries. A crown may have defects and still be recorded as 0. Defects that can be disregarded include white or chalky spots; discolored or rough spots that are not soft; stained enamel pits or fissures; dark, shiny, hard, pitted areas of moderate to severe fluorosis; or abraded areas.

– 1: One indicates a tooth with caries. A tooth or root with a definite cavity, undermined enamel, or detectably softened or leathery area of enamel or cementum can be designated as 1. A tooth with a temporary filling, and teeth that are sealed but decayed, are also termed 1. Score 1 is not assigned to any tooth in which caries is only suspected. In cases where the crown of a tooth is entirely decayed, leaving only the root, score 1 is assigned to both crown and root. Where only the root is decayed, only the root is termed as 1. In cases, where both the crown and root are involved with decay, whichever site is judged the site of origin is recorded as 1. These criteria apply to all numbers.

– 2: Filled teeth, with additional decay, are termed 2. No distinction is made between primary caries which is not associated with a previous filling, and secondary caries, adjacent to an existing restoration.

– 3: It indicates a filled tooth with no decay. If a tooth has been crowned because of previous decay, that tooth is judged 3. When a tooth has been crowned for another reason such as aesthetics or for use as a bridge abutment, 7 is used.

– 4: It indicates a tooth that is missing as a result of caries. Only crowns are given 4 status. Roots of teeth that have been scored as 4 are recorded as 7 or 9. When primary teeth are missing, the score should be used only if the tooth is missing prematurely. Primary teeth missing because of normal exfoliation needs no recording.

– 5: A permanent tooth missing for any other reason than decay is given as 5. Examples are teeth extracted for orthodontia or because of periodontal disease, teeth that are congenitally missing, or teeth missing because of trauma. The 5 is assigned to the crown, the root is given a 7 or 9. Knowledge of tooth

eruption patterns is helpful to determine whether teeth are missing or not yet erupted. Clues to help in the determination include appearance of the alveolar ridge in the area in question, and caries status of other teeth in the mouth.

– 6: A 6 is assigned to teeth on which sealants have been placed. Teeth on which the occlusal fissure has been enlarged and a composite material placed should also be termed 6.

– 7: A 7 is used to indicate that the tooth is part of a fixed bridge. When a tooth has been crowned for a reason other than decay, this code is also used. Teeth that have veneers or laminates covering the facial surface are also termed 7 when there is no evidence of caries or restoration. A 7 is also used to indicate a root replaced by an implant. Teeth that have been replaced by bridge pontics are scored 4 or 5; their roots are scored 9.

– 8: This code is used for a space with an unerupted permanent tooth, where no primary tooth is present. The category does not include missing teeth. Code 8 teeth are excluded from calculations of caries. When applied to a root, an 8 indicates the root surface is not visible in the mouth.

– 9: Erupted teeth that cannot be examined because of orthodontic bands, e.g. are coded a 9. When applied to a root, a 9 indicates the tooth has been extracted. The crown of that tooth would be scored a 4 or 5.

– T: Indicating trauma, a T is used when a crown is fractured, with some of its surface missing but with no evidence of decay.

The «D» of DMFT refers to all teeth with codes 1 and 2. The «M» applies to teeth scored 4 in subjects under age 30, and teeth scored 4 or 5 in subjects over age 30. The «F» refers to teeth with code 3. Those teeth coded 6, 7, 8, 9, or T are not included in DMFT calculations. To arrive at a DMFT score for an individual patient’s mouth, three values must be determined: the number of teeth with carious lesions, the number of extracted teeth, and the number of teeth with fillings or crowns. A patient who has two areas of decay, six missing teeth and 11 filled or crowned teeth, e. g. has a DMFT score of 19. Teeth that include both decay and fillings or crowns, are only given one point, a D. Thirteen teeth (based on a full dentition of 32) remain intact. It is also possible to determine more detailed DMFS (decayed, missing, or filled surface) scores. As anterior teeth have four surfaces and posterior teeth have five, a full

dentition of 32 teeth includes 148 surfaces. A patient with seven decayed surfaces, 20 surfaces from which teeth are missing, and 42 surfaces either filled or included in a crown, the DMFS score is 69. 79 surfaces are intact.

For primary dentition, scoring is referred to as «deft» or «defs» (decayed, extracted, or filled).

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WORKSHOP № 3
EVALUATION OF DENTAL CARIES INCIDENCE

Test Questions:

1. The objectives and methods of morbidity evaluation. Characteristics of dental caries epidemiology. Units of measurement in morbidity evaluation (person, tooth, surface, carious lesion).

2. Prevalence of dental caries.

3. Incidence of dental caries (tooth, surfaces, and lesion levels):

a) clinical records levels of caries D1-D4 (by Axelsson);

b) epidemiological method using index ICDAS;

c) assessment of epidemiological tooth and tooth surfaces caries intensity level by WHO indices (DMFT, deft, DMFS, defs, DMFT + deft, DMFS + defs); significant intensity index of caries (SiC);

d) IS index.

4. Caries process dynamics (increment, reduction).

5. The concepts of caries activity («compensated, sub-and decompensated caries forms» and «the level of caries intensity»).

6. Level of dental care (USP).

INTRODUCTION TO EPIDEMIOLOGY

Health and disease can be studied in 3 basic ways, (i) observation of effects on individuals (ii) laboratory experiments (iii) measuring their distribution in population (epidemiology).

The origin of the word epidemiology is from the Greek word «epi» meaning upon, «demos» meaning people and logos meaning «doctrine»; the literal translation would be «the doctrine of what is upon the people».

The international epidemiological association defines epidemiology as «the study of the *distribution* and *determinants of* health-related states and events in the populations and the application of this study to control health problems». The primary unit of concern is groups of persons not individuals.

Scope of Epidemiology. Epidemiology covers all major health problems in the community including:

– communicable diseases;

– chronic degenerative, metabolic, neoplastic diseases;

– nutritional deficiencies;

– occupational health and injuries;

– mental and behavioral disorders;

– population issues and demographic trends.

Dental caries is one of the most important problems in dentistry. The disease is manifested by lesion of dental hard tissues with the formation of cavities. Lesions can occur in any period of human life. Cavities are manifested in different forms and may have a different activity.

Dental caries has been an object of epidemiological studies during the last 50–60 years. Epidemiology studies public health, as well as the distribution and characteristics of the diseases, the impact on their environment and way of life.

Epidemiology should answer some questions. The first task is to assess the prevalence and intensity of major dental diseases among the population.

The second one is to compare the incidence of dental diseases in different regions, countries, etc. The third task is to identify the needs of treatment and prevention. The fourth task is to generate the results of statistics of dental diseases into the organization of dental services: in country, in region, in city, etc.

There are some stages of an epidemiological study.

The first is preparatory stage. It is carried out to standardize the assessment of morbidity. The World Health Organization (WHO) has developed
certain criteria. Thus, the most informative for the assessment of the state of the teeth and periodontal tissues are groups of 6, 12, 15 years old children. Oral diseases intensity among adult population is assessed in age group from 35 to 44 years of age and older — from 65 to 74 years, respectively. In group of 6 year olds we examine first permanent molars. In group of 12 year olds we register the state of all permanent teeth. In group of 15 year old adolescents we assess the condition of the periodontum. In adult groups the state of the teeth and periodontal tissues is registered.

The next step is an oral examination. There are more than 60 epidemiological indicators to assess the incidence of dental caries. But we will talk about the most commonly used. Thus, the object of study may be: a man, a tooth, the tooth surface, carious lesion.

The first object of statistical accounting of dental caries incidence is the person (individual).

The prevalence of the disease. The prevalence is the proportion of individuals who have a disease at the time of the study. Examined groups should be comparable with regard to age, sex, geographical and socio-economic conditions to obtain statistically reliable data. All the persons with decayed, filled
or missed due to caries teeth should be calculated. Then this sum is divided by the number of all examined persons. The indicator is expressed as a percentage.

$$\text{Prevalence} = \frac{\text{Number of existing cases of dental caries at a point of time}}{\text{Total population at that point of time}} \times 100\ \%.$$

Disadvantages of the index of prevalence of dental caries:

– do not take into account the number of decayed teeth;

– do not register the size and depth of the lesion;

– do not take into account the aggressiveness (chronic or acute) of the disease.

Decayed, Missing and Filled Teeth (DMFT) Index. This index was developed by Henry Klein, Carrole E. Palmer and J. W. Knutson in 1938. This index was based on the fact that the dental hard tissues are not self healing and established caries leaves a scar. The tooth either remains decayed and if treated may be extracted or filled. It is an irreversible index. DMFT describes the amount (the prevalence) of dental caries in an individual. DMFT numerically expresses the caries prevalence and is obtained by calculating the number of teeth (T) which are:

– Decayed (D);

– Missing (M);

– Filled (F).

It is thus used to get an estimation illustrating how much the dentition until the day of examination has become affected by dental caries. Thus:

• How many teeth have caries lesions (incipient caries not included)?

• How many teeth have been extracted?

• How many teeth have fillings or crowns?

Selection of Teeth. All 28 teeth are examined (based on 28 teeth).

Teeth not included are:

• Third molars.

• Unerupted teeth (a tooth is considered as erupted when the occlusal surface or incisal edge is totally exposed).

• Supernumerary and congenitally missing teeth.

• Teeth removed for reasons other than dental caries such as for orthodontic reasons and impactions.

• Teeth restored for reasons other than dental caries, such as trauma, use as a bridge abutment and cosmetic purposes.

• Retained primary tooth when the successor permanent is present. The permanent tooth is considered.

Procedure. Each tooth is examined using a mouth mirror, an explorer and adequate light. The teeth should be observed by visual means as much as possible and only questionable small lesions should be checked by using an explorer.

Rules for Scoring DMFT:

• No tooth should be counted more than once.

• Decayed (D), Missing (M) and Filled (F) teeth should be recorded separately.

• Tooth lost or filled due to reasons other than caries are not included.

• Deciduous teeth are not considered in DMFT index.

• A tooth with several filling is counted as one tooth.

Criteria for Recording

Decayed (D) recording:

• When dental caries and a restoration are present on the same tooth, the tooth is recorded as D.

• When a crown is broken due to caries, it is recorded as D.

• Tooth with temporary restoration are recorded as decayed.

Missing (M) recording:

• When a tooth has been extracted because of dental caries.

• When a tooth is carious, cannot be restored and is indicated for extraction.

Filled (F) recording: permanent restorations are recorded as F.

Criteria for Identification of Dental Caries:

• Lesion is clinically visible and obvious.

• Discoloration or loss of translucency typical of undermined or demineralized enamel.

• Definite catch and the explorer tip can penetrate into soft yielding material.

DMFT Scores. The sum of the three figures forms the DMFT value. For example: DMFT of 4 + 3 + 9 = 16 means that 4 teeth are decayed, 3 teeth are missing and 9 teeth have fillings. It also means that 12 teeth are intact.

Individual DMFT

Total each component separately ie total D, total M, total F.

Total D + M + F = DMF SCORE.

Group Average:

• Total the D, M and F for each individual.

• Divide the total DMF by the number of individuals examined.

Average DMF = Total DMF/Total Number of Individuals Examined

Treatment Needs. Percentage needing restorations (%) = Total Number of D Tooth/Total Number Examined × 100.

LIMITATIONS OF DMFT INDEX

DMF values are not related to the number of teeth at risk. A DMF score does not directly gives an indication of the intensity of attack in any one individual, e. g. a child of 8-year-old may have DMF score of 3 with only nine permanent teeth in mouth (one-third of teeth have been already affected by caries), whereas an adult may have a DMF score of 8 (more than the child score) out of 32 teeth (only one fourth of the teeth have been affected).

The DMF index is invalid when teeth have been removed or lost due to other reasons, e. g. periodontal reasons.

The index gives equal weight to all the three components, i. e. missing decayed and well-restored teeth.

Does not tell about the treatment needs of a person.

The DMF index can overestimate caries experience in cases having teeth with preventive restorations.

Cannot be used for root caries.

Decayed, Missing, Filled Surface Index (DMFS). DMFS index assesses the total no of tooth surfaces affected rather than the tooth. It is a more detailed index in which DMF is calculated per tooth surface. DMFS Rules, method and criteria are same as that of DMFT index except that all tooth surfaces are examined in DMFS index.

Surfaces Examined. Anterior teeth: four surfaces are examined; Facial, Lingual, Mesial and Distal.

Posterior teeth: five surfaces are examined; Facial, Lingual, Mesial, Distal and Occulusial.

Maximum value for DMFS comes to 128 for 28 teeth.

Posterior teeth: 16 with 5 surface, each: $16 \times 5 = 80$.

Anterior teeth: 12 with 4 surface, each: $12 \times 4 = 48$.

Total = 128 surfaces.

Calculating the DMFS

Individual.

Total number of decayed surfaces = D.

Total number of missing surfaces = M.

Total number of filled surfaces = F.

Total DMFS score for an individual = D + M + F (surfaces).

It is a more precise index but takes a longer time to perform.

WHO MODIFICATION OF DMF INDEX

1. Third molars are included.

2. Teeth with temporary restorations are considered as decayed (D).

3. Initial caries is not regarded as decayed.

DENTAL CARIES INDEX FOR DECIDUOUS TEETH (DMFT AND DMFS)

This index for primary teeth was given by Grubbel in 1944. It is used for the primary dentition, consisting of maximum 20 teeth.

Designations are «deft» or «defs», where d = decayed primary teeth; e = extracted tooth/indicated for extraction (due to caries); f = filled teeth/surfaces.

Selection of Teeth or Surfaces

dmft: 20 teeth are evaluated (all the primary teeth are included).

For Surfaces

dmfs: 88 surfaces are evaluated.

Posterior teeth: 8 teeth × 5 surfaces = 40 surfaces.

Anterior teeth: 12 teeth × 4 surfaces = 48 surfaces.

Teeth not Counted.

• Missing teeth, including unerupted and congenitally missing teeth.

• Teeth restored for reasons other than dental caries are not counted as f.

• Supernumerary teeth.

Procedure and Criteria: same as for DMFT.

Calculating the def.

Total def score = d + e + f.

Total defs score = d + e + f surfaces.

MIXED DENTITION

In mixed dentition, DMFT or DMFS and a deft and defs index are done separately and never added together. Separate index is done for each child for permanent teeth and primary teeth starting with permanent teeth first.

WHO INDEX FOR DENTAL CARIES

To assess dental caries in a population, a DMFT index is used. During a systematic examination with a mirror and CPI probe that includes the crown and exposed root of every primary and permanent tooth, each crown and root are assigned a number based on the result of that exam. The numbers are recorded in boxes corresponding to each tooth to provide a DMFT chart. It is recommended, that care should be taken to record all tooth-colored fillings, which may be difficult to detect.

Numbers are assigned as follows:

• **0:** A zero indicates a sound crown or root, showing no evidence of either treated or untreated caries. A crown may have defects and still be recorded as 0. Defects that can be disregarded include white or chalky spots; discolored or rough spots that are not soft; stained enamel pits or fissures; dark, shiny, hard, pitted areas of moderate to severe fluorosis; or abraded areas.

• **1:** One indicates a tooth with caries. A tooth or root with a definite cavity, undermined enamel, or detectably softened or leathery area of enamel or cementum can be designated as 1. A tooth with a temporary filling, and teeth that are sealed but decayed, are also termed 1. Score 1 is not assigned to any tooth in which caries is only suspected. In cases where the crown of a tooth is entirely decayed, leaving only the root, score 1 is assigned to both crown and root. Where only the root is decayed, only the root is termed as 1. In cases, where both the crown and root are involved with decay, whichever site is judged the site of origin is recorded as 1. These criteria apply to all numbers.

• **2:** Filled teeth, with additional decay, are termed 2. No distinction is made between primary caries which is not associated with a previous filling, and secondary caries, adjacent to an existing restoration.

• **3:** It indicates a filled tooth with no decay. If a tooth has been crowned because of previous decay, that tooth is judged 3. When a tooth has been crowned for another reason such as aesthetics or for use as a bridge abutment, 7 is used.

• **4:** It indicates a tooth that is missing as a result of caries. Only crowns are given 4 status. Roots of teeth that have been scored as 4 are recorded as 7 or 9. When primary teeth are missing, the score should be used only if the tooth is missing prematurely. Primary teeth missing because of normal exfoliation needs no recording.

• **5:** A permanent tooth missing for any other reason than decay is given as 5. Examples are teeth extracted for orthodontia or because of periodontal disease, teeth that are congenitally missing, or teeth missing because of trauma. The 5 is assigned to the crown, the root is given a 7 or 9. Knowledge of tooth eruption patterns is helpful to determine whether teeth are missing or not yet erupted. Clues to help in the determination include appearance of the alveolar ridge in the area in question, and caries status of other teeth in the mouth.

• **6:** A 6 is assigned to teeth on which sealants have been placed. Teeth on which the occlusal fissure has been enlarged and a composite material placed should also be termed 6.

• **7**: A 7 is used to indicate that the tooth is part of a fixed bridge. When a tooth has been crowned for a reason other than decay, this code is also used. Teeth that have veneers or laminates covering the facial surface are also termed 7 when there is no evidence of caries or restoration. A 7 is also used to indicate a root replaced by an implant. Teeth that have been replaced by bridge pontics are scored 4 or 5; their roots are scored 9.

• **8:** This code is used for a space with an unerupted permanent tooth, where no primary tooth is present. The category does not include missing teeth. Code 8 teeth are excluded from calculations of caries. When applied to a root, an 8 indicates the root surface is not visible in the mouth.

• **9:** Erupted teeth that cannot be examined because of orthodontic bands, e. g. are coded a 9. When applied to a root, a 9 indicates the tooth has been extracted. The crown of that tooth would be scored a 4 or 5.

• **T:** Indicating trauma, a T is used when a crown is fractured, with some of its surface missing but with no evidence of decay.

The «D» of DMFT refers to all teeth with codes 1 and 2.

The «M» applies to teeth scored 4 in subjects under age 30, and teeth scored 4 or 5 in subjects over age 30. The «F» refers to teeth with code 3. Those teeth coded 6, 7, 8, 9, or T are not included in DMFT calculations.

To arrive at a DMFT score for an individual patient's mouth, three values must be determined: the number of teeth with carious lesions, the number of extracted teeth, and the number of teeth with fillings or crowns. A patient who has two areas of decay, six missing teeth and 11 filled or crowned teeth, e. g. has a DMFT score of 19. Teeth that include both decay and fillings or crowns, are only given one point, a D. Thirteen teeth (based on a full dentition of 32) remain intact.

It is also possible to determine more detailed DMFS (decayed, missing, or filled surface) scores. As anterior teeth have four surfaces and posterior teeth have five, a full dentition of 32 teeth includes 148 surfaces. A patient with seven decayed surfaces, 20 surfaces from which teeth are missing, and 42 surfaces either filled or included in a crown, the DMFS score is 69. 79 surfaces are intact. For primary dentition, scoring is referred to as «deft» or «defs» (decayed, extracted, or filled).

SIGNIFICANT CARIES INDEX

In 2000, the World Health Organization developed the significant caries index (SiC) to be used when studying DMFT scores on a global basis. A single population may include a number of individuals with low DMFT scores, as well as those with high scores. A mean DMFT value would not accurately reflect the status of the population. The SiC Index isolates and highlights those individuals with the highest caries values in a particular population.

To calculate a SiC Index, individuals are sorted according to DMFT values. The third of the population with highest caries scores is isolated, and a mean DMFT for this subgroup is calculated. The resulting value is the SiC Index. DMFT score show that there is a skewed distribution of caries prevalence. Clearly, the mean DMFT value does not accurately reflect this skewed distribution leading to incorrect conclusion that the caries situation for the whole population is controlled while in reality several individuals still have caries.

A new index called the «Significant Caries Index» (SiC) was proposed by Bratthall D. in the year 2000, in order to bring attention to those individuals

with the highest caries scores in each population. The SiC Index is the Mean DMFT of the one third of the study group with the highest caries score. The index is used as a complement to the mean DMFT value.

Steps to calculate Significant Caries Index:

• Sort the individuals according to their DMFT.

• Select the one third of the population with the highest caries values.

• Calculate the Mean DMFT for this subgroup, suppose the DMFT of 15 students was calculated as e.g. 0, 1, 5, 0, 0, 2, 1, 7, 0, 9, 0, 4, 1, 0, 5.

Arranged in increasing order the DMFT Scores are 0, 0, 0, 0, 0, 0, 1, 1, 1, 2, 4, 5, 5, 7, 9.

The highest 1/3rd scores are 0, 0, 0, 0, 0, 0, 1, 1, 1, 2, 4, 5, 5, 7, 9.

Thus, the SiC Index is 4 + 5 + 5 + 7 + 9 / 5 = 30/ 5 = 6.0.

DMFT — 0 + 0+ 0 + 0 + 0 + 0 + 1 + 1 + 1 + 2 + 4 + 5 + 5 + 7 + 9 / 15 = = 35 / 15 = 2.33.

Alternative indices for assessment of dental caries. In 2000, Dr. Axelsson proposed index of clinical records levels of caries (D1-D4; d1-d4).

In 2005, the WHO team of experts proposed index «International Caries Detection and Assessment System» — ICDAS. This index can be used not only in the clinical diagnosis of caries, but also in epidemiological surveys. Vision for ICDAS is: a detection & assessment system classifying stages of the caries process. You can find the software tools for ICDAS in the internet.

Increase of the intensity of dental caries (ΔDMFT) — a change in the number of carious teeth for a certain period of time (at least one year). For the calculation of the index is necessary to determine the difference between the values of the index DMFT (DMFT + deft, deft) at the time of the study and in the past. If the indicator is positive, it means that the carious process spreads. The Increasing of the intensity of dental caries can be determined individually and in groups of people.

Reduction, ie «Delay». Indicator measures the change in the number of carious teeth over time. Used for a qualitative assessment of the incidence of

dental caries. The indicator shows the share prevent tooth decay in people of the highest possible. The calculation uses the data rate (ΔKPU) index CPU (CPU + CPU, CPU). The higher the percentage (%), the prevention program conducted in this group effectively. Disadvantages:

– this requires a comparison group;

– the duration of the observations.

The level of dental caries intensity (LCI) — P. Leous. This index is a figure which is calculated from the ratio of the individual values of the DMFT (DMFT + defs, defs) and age. The author proposed to estimate the activity of caries in four levels: low, moderate, high and very high (table 2).

Table 2

The level of dental caries intensity

Age	Formula	Low level	Moderate level	High level	Very high level
1–8 years	deft/n*	≤0,4	0,5–0,8	0,9–1,2	≥ 1,3
9וו19 years	DMFT/n-5**	≤0,3	0,4–0,6	0,7–0,9	≥ 1,0
≥ 20 years	DMFT/n*	≤0,155	0,16–0,3	0,31–0,6	≥ 0,6

Note: * n — the number of years; **5 — age of the beginning of the first molars eruption.

LITERATURE

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WORKSHOP № 4
CAUSES AND CONDITIONS FOR THE DEVELOPMENT OF DENTAL CARIES AS OBJECTS OF PRIMARY PREVENTION

Test Questions:

1. Key theories of the origin and development of dental caries.

2. Caries management strategies: history.

3. Modern concept of dental caries etiology:

a) microorganisms factor;

b) carbohydrates factor;

c) «host» factor;

d) time factor.

4. Modern concepts of the pathogenesis of carious destruction of dental hard tissues.

5. Prevailing directions of causal and pathogenetic caries prevention.

Dental caries is a destructive process causing decalcification of the tooth enamel and leading to continued destruction of enamel and dentin, and cavitation of the tooth (Dorland's Medical Dictionary for Health Consumers. © 2007 by Saunders, an imprint of Elsevier).

THEORIES OF DENTAL CARIES

The Legend of the Worm. Ancient Sumerian text known as «The legend of the worm» gives reference of the tooth decay and tooth pain. It was obtained from the Mesopotamian areas which date back to about 5000 BC. According to the legend, toothache was caused by a worm that drank the blood of teeth and fed on the root of the jaws.

Endogenous Theories. *Humoral Theory.* The ancient Greek believed that a person's physical and mental constitution was determined by four elemental humors of the body: blood, phlegm, black bile and yellow bile. An imbalance in these humors is the cause of all diseases including dental caries.

According to *Galen*, the ancient greek physician and philosopher, «dental caries is produced by internal action of acrid and corroding humors». Hippocrates referred to accumulated debris around teeth and to their corroding action. He also stated that stagnation of juices in the teeth was the cause of tooth ache.

Vital Theory (Proposed during 18th Century). According to this theory, the tooth decay originated like bone gangrene, from within the tooth itself.

Exogenous Theories. *Chemical Theory.* Parmly (1819) proposed that an unidentified «chemical agent» was responsible for caries. According to this theory, teeth are destroyed by the acids formed in the oral cavity by the putrefaction of protein which produced ammonia and was subsequently oxidized to nitric acid. Robertson (1895) proposed that dental decay was caused by acids formed by fermentation of food particles around teeth.

Parasitic or Septic Theory. Dr Miles and Underwood proposed the so-called «septic theory». They claimed that dental caries is caused by direct action of microorganisms that penetrate the dental tubules and destroy the organic component of the dentine leaving the inorganic parts to be broken down and washed away in fluids of the mouth.

Chemoparasitic Theory (W. D. Miller). It is a blend of chemical and parasitic theory, because it states that caries is caused by acids produced by microorganisms of the mouth. According to this theory, microorganisms of the mouth, by secretion of enzymes or by their own metabolism, degrade fermentable carbohydrate food materials to form acids which demineralize the enamel and the disintegrated enamel is subsequently mechanically removed by force of mastication. Miller summarized his theory as follows. Dental decay is a chemoparasitic process consisting of two stages — decalcification or softening of the tissue and dissolution of the softened residue.

Proteolytic Theory (Gottileb, 1947). According to this theory, microorganisms invade the organic pathways (lamellae) of the enamel and initiate caries by proteolytic action. Subsequently, the inorganic salts are dissolved by acidogenic bacteria. Pincus (1950) stated that initial caries process in dental caries was due to the proteolytic breakdown of the dental cuticle.

Proteolysis Chelation Theory. This theory proposed by Shalz et al implies a simultaneous microbial degradation of the organic components (proteolysis) and the dissolution of the minerals of the tooth by the process of chelation. According to this theory, dental caries results from an initial bacterial and enzymatic proteolytic action on the organic matter of enamel without preliminary demineralization. This causes the release of a variety of complexing agents, such as amino acids, polyphosphates and organic acids which then dissolves the crystalline apatite.

FACTORS AFFECTING THE EPIDEMIOLOGY OF DENTAL CARIES

In the 1960s, the caries theory was depicted as three circles representing the three prerequisites for dental caries (Keyes Triad). Three indispensable factors for development of caries were: 1) carbohydrate (diet); 2) bacteria (dental plaque); 3) susceptible teeth (the host) (Keyes and Jordan, 1963).

Since then, many modifying factors have been recognized, resulting in a more complex model that includes saliva, the immune system, time, socioeconomic status, level of education, lifestyle behaviors, and the use of fluorides. An important breakthrough in the understanding of dental caries was the recognition of the remineralization process as a result of plaque fluid and saliva at pH levels above a critical value being highly saturated with calcium and phosphates. The caries process can be described as loss of mineral (demineralization) when the pH of plaque drops below the critical pH value of 5.5; the critical value for enamel dissolution is 5 to 6, and an average pH of 5.5 is the generally accepted value. Redeposition of mineral (remineralization) occurs when the pH of plaque rises. The presence of fluoride reduces the critical pH by 0.5 pH units, thus exerting its protective effect. It is now established that dental caries is a multifactorial disease and results from a combination of four principal factors (Newbrun) (fig. 1): 1) host and teeth factors; 2) microorganism in dental plaque; 3) substrate (diet); 4) time.

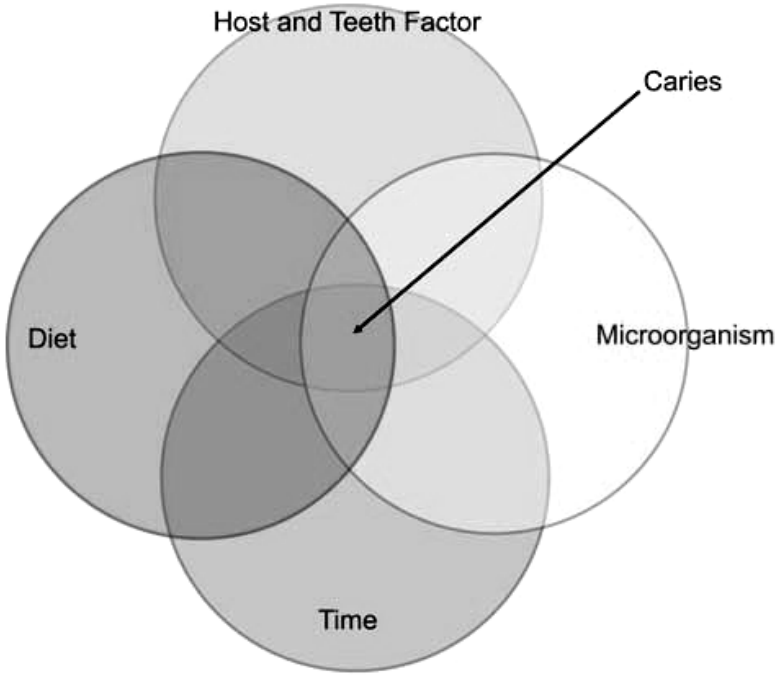

Fig. 1. Four principal factors in dental caries (Adapted from Newbrun)

FACTORS AFFECTING DEVELOPMENT OF DENTAL CARIES

Host and Teeth Factors:

A. *Tooth*:

– composition;

– morphology;

– position.

B. *Saliva*:

– composition;

– buffering capacity of saliva;

– quantity.

C. *Sex.*

D. *Age.*

E. *Race and ethnicity.*

F. *Socioeconomic status.*

G. *Heredity.*

H. *Emotional disturbances.*

Agent Factors:

A. *Microorganism.*

B. *Plaque.*

Environmental Factors:

A. *Diet*:

– total consumption of carbohydrate;

– frequency and form of carbohydrate.

B. *Geographic variation.*

C. *Climate.*

D. *Oral hygiene.*

E. *Soil.*

F. *Fluoride.*

AGENT FACTORS

Microorganisms. The mouth has a diverse resident microbial flora. The normal inhabitants become established early in life. There have been a few epidemiological studies to investigate the link between oral flora and dental caries. *Streptococcus mutans* was first identified in 1924 by Clarke and subsequently *Lactobacillus acidophilus* by Bunting (1930). These acid producing bacteria were found to be associated with the formation of dental caries. *Streptococcus mutans* is of interest because it has the ability to form an extracellular polymer of glucose (Glucan) from sucrose, which aids the microorganism in adhering to the enamel surface and in establishing a stable relationship there. Another extracellular polymer (levan) is synthesized from fructose. It plays the role of extracellular microbial energy reserve.

L. acidophillus and other acidogenic microorganism in plaque and carious lesion may be capable of producing caries by themselves, or they may be able to act synergistically with *Streptococcus mutans* in caries initiation. *Actinomyces* are Gram-positive pleomorphic rods (GPPR) which form a large proportion of the oral microflora of all mammals. *Actinomyces* are also among the earliest colonizers of dental surfaces and may constitute up to 27 % of the pioneer bacteria. They have been implicated in root caries, although their role in dental caries initiation and progression is not wellunderstood.

Dental Plaque. Bacterial plaque is a dense non-mineralized, highly organized mass of bacterial colonies in a gel-like intermicrobial, enclosed matrix or slime layer. It is a transparent film that can be *supragingival*, coronal to the gingival margin on the clinical crown of the tooth and *subsgingival*, apical to the margin of the gingiva.

HOST AND TEETH FACTOR

Tooth. *Composition.* Number of studies on the relation of caries to the chemical composition have shown that there was no difference found in the calcium, phosphorus, magnesium and carbonate content of enamel from sound and carious teeth. But there was a significant difference in fluoride content of teeth, i. e. more in sound teeth. It was also noted that surface enamel is more resistant to caries than subsurface enamel. Surface enamel is more highly mineralized and tends to accumulate greater quantities of fluoride, zinc, lead and iron than the underlying enamel. The surface is lower in carbon dioxide, dissolves at a slower rate in acids and has more organic material than subsurface enamel. These factors contribute to caries resistance.

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

Position. Malaligned, out of position, rotated teeth are difficult to clean, favoring the accumulation of food and debris. This may predispose to the development of caries.

Saliva. It can be considered as an environmental factor also as teeth are constantly bathed by it. This influences the process of dental caries. Saliva has a flushing action on teeth.

Sex. In young people caries has been seen to higher in the females but some studies show no significant difference between the sexes. Root caries is seen more in males. Girls may be more prone to caries due to early eruption of teeth and hormonal changes (puberty and pregnancy).

Age. Although present in all ages, it was believed that dental caries was disease of childhood. WHO global data bank has shown a decline in DMFT values in 12-year-old children. Some studies indicate greatest intensity of dental caries occurs in 15 to 25 years of age. Root caries is seen in over 60 years age group people, mainly due to denuded root surface because of gingival recession.

Race and Ethinicity. A number of studies indicate that blacks (Negroes) of comparable age and sex have a lower caries scores than Caucasians. Chinese population has shown to have a lower caries rate than corresponding white population. These differences are probably more due to environmental factors.

Socioeconomic Status. There is an inverse relationship between socioeconomic status and dental caries experience in primary dentition. The relation has not been established in adults, though some studies suggest so.

ENVIRONMENTAL FACTORS

Diet. According to acidogenic or chemoparasitic theory, dental caries occurs when acid is produced by bacteria in dental plaque when refined carbohydrates are eaten. The presence of refined carbohydrate as sugar is essential for the majority of caries development and sucrose is the most cariogenic of all sugars. In human consumption, sucrose accounts for 60 percent of all sugars eaten.

Geographic Variation. It is well documented that dental caries experience has been decreasing in children in developed western (1973–1983) countries. But this decrease is beginning to level out. Gradual increase in caries in 5 years old have been found in some areas.

Climate. Sunshine and high temperature areas seems to have lower dental caries (inverse relationship). Whereas areas with more relative humidity and rainfall have shown increase dental caries.

Oral Hygiene. Inverse relationship has been seen between oral hygiene and dental caries. Poor oral hygiene increases the rate of dental caries.

Soil. Trace elements in soil have shown a relation with caries. An increase in dental caries is seen in areas where selenium is present in soil, whereas molybdenum and vanadium are said to decrease dental caries.

Fluoride. Fluoride in water and soil decreases incidence of dental caries.

THE CARIES PROCESS (PATHOGENESIS)

Bacterial Plaque and Acid Production. The mechanism of dental caries formation is essentially straightforward. Plaque on the surface of the tooth consists of a bacterial film that produces acids as a byproduct of its metabolism. To be specific, certain bacteria within the plaque are acidogenic — that is, they

produce acids when they metabolize fermentable carbohydrates. These acids can dissolve the calcium phosphate mineral of the tooth enamel or dentine in a process known as demineralization. If this process is not halted or reversed via remineralization (the redeposition of mineral via saliva) it eventually becomes a frank cavity.

Dental caries of the enamel typically is first observed clinically as a so-called «white-spot lesion». This is a small area of subsurface demineralization beneath the dental plaque. The body of the subsurface lesion may have lost as much as 50 percent of its original mineral content and often is covered by an «apparently intact surface layer». The surface layer forms by remineralization. The process of demineralization continues each time there is carbohydrate taken into the mouth that is metabolized by the bacteria. The saliva has numerous roles, including buffering (neutralizing) the acid and remineralization by providing minerals that can replace those dissolved from the tooth during demineralization.

The critical pH value for demineralization varies among individuals, but it is in the approximate range of 5.2 to 5.5. Conversely, tooth remineralization can occur if the pH of the environment adjacent to the tooth is high due to: 1) lack of substrate for bacterial metabolism; 2) low percentage of cariogenic bacteria in the plaque; 3) elevated secretion rate of saliva; 4) strong buffering capacity of saliva; 5) presence of inorganic ions in saliva; 6) fluoride; 7) rapid food clearance times. Whether dental caries progresses, stops, or reverses is dependent on a balance between demineralization and remineralization. However, if demineralization overtime exceeds remineralization, an initial carious lesion (the so-called «white spot» lesion) can develop and may further progress to a frank cavity.

Demineralization can be reversed in its early stages through uptake of calcium, phosphate, and fluoride. Fluoride acts as a catalyst for the diffusion of calcium and phosphate into the tooth, which remineralizes the crystalline structures in the lesion. The rebuilt crystalline surfaces, composed of fluoridated hydroxyapatite and fluorapatite, are much more resistant to acid attack than is

the original structure. Bacterial enzymes can also be involved in the development of caries.

The cause of dental caries is the consumption of fermentable carbohydrates (sugars). There is a dose-response relationship between the quantity
of the sugar consumed and the development of dental caries. It is suggested, at levels below 10 kg/person per year dental caries will not develop. (15 kg/person per year in fluoridated areas).

LITERATURE

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WORKSHOP № 5
THE ROLE OF OPTIMAL CONDITIONS FOR THE FORMATION OF NATURAL TOOTH ENAMEL RESISTANCE TO DECAY DURING ODONTOGENESIS (TISSUE DEVELOPMENT, MINERALIZATION). NUTRITION ASSESSMENT AND CORRECTION. PREERUPTIVE CARIES PREVENTION

Test Questions:

1. Preeruptive factors determining enamel resistance to decay.

2. Critical periods in the teeth development and their resistance to decay.

3. Food as a factor in the preeruptive formation of resistance to decay. Evaluation and correction of dietary intake during odontogenesis .

4. Calcium and phosphate in dental caries prevention mechanisms of influence, sources of and normal consumption rates, systemic supplements with calcium and vitamin D.

5. Body's resistance to decay. Vitamins, adaptogens, immunostimulants and anabolic steroids as means of increasing body's resistance to decay: mechanisms of action, indications, dosage, effectiveness.

It is well known that the anlage and the formation of organs and tissues of maxillofacial area occurs in prenatal period and after birth. If these stages passed well and completely the teeth are resistant to unfavorable external and internal environment.

According to WHO, a significant influence on the body formation has nutrition. Gross violations of nutritional norms cause of weakened immunity and low caries resistance. Deficiency of essential nutritional components suppresses the immune response and increases pathogenicity of microbes.

Food. Currently, 2 million people suffer from diseases due to lack of vitamins A and Group B, iodine, iron, zinc and fluoride. Vitamin A deficiency causes a decrease in production of mucin. Malnutrition affects the formation and secretory function of the salivary glands, reduces the resistance of teeth hard tissues to dental caries, disturbs the structure and development of the teeth. Consequence of disruption of Ca and P homeostasis is hypoplasia, developing as a result of hypocalcemia, reduced function of parathyroid glands, neonatal tetanus, premature childbirth and diarrhea.

Vitamin D deficiency during tooth development leads to delayed teeth eruption and disruption of calcification of teeth and skeleton.

Malnutrition causes tooth caries in 3 ways:

1. Development of hypoplasia reducing caries resistance.
2. Atrophy of salivary glands.
3. Delay of tooth loss.

In industrialized countries assortment of food is in abundance. Food has become a health risk. It favors the overweight, cardiovascular disease and — last but not least — caries (Kohlmeier et al., 1993). Although food is a partial

factor of dental disease, nutrition is available to influence preventive efforts of dentist.

We should pay attention to those foods which have «hidden» sugar, as their original taste isn't sweet (e. g., ketchup, canned meat, pickles).

Unlimited use of fatty food leads to overweight. Eating in front of the TV or computer promotes excessive weight due to the small energy consumption and simultaneous mealtime.

Food Pyramid. The nutrition will be full of value when products are selected in the right amount from all 7 groups with preference in the lower 5 groups of the pyramid. Also attention should be paid to food diversity.

Fewer products should be chosen from both top groups. Within the group of «fish, meat, eggs» products should be changed consistently, but sea fish should be more preferable (DCE, 1997).

Improvement of the population oral health is possible in case of consumption of lean meat with lots of fatty acids. Low-fat milk is recommended.

«5 times a day» — fruits and vegetables.

People include lots of carbohydrates into the diet. Oral bacteria use this carbohydrates for feeding, constructing matrix of dental plaque. This final product of this process is organic acid causing demineralization of enamel.

Promising ways of reducing carbohydrates cariogenicity are:

– reduction the consumption of carbohydrates;

– reduction in the frequency of consumption of carbohydrates;

– replacement of carbohydrates metabolizable in the mouth to non-metabolizable;

– reduction in the residence time of carbohydrates in oral cavity;

– excretion of free carbohydrates from the mouth by their enzymatic cleavage or restriction the ability to engage in metabolism.

We can not completely eliminate sugar from the diet. That's why sucrose substitutes were found, especially for those foods and drinks that are used often in between meals. Sweeteners can not be used by microbial plaque to form acids. They stimulate salivation. It provides their anticaries effect.

Change of easy metabolizable carbohydrate to non-metabolizable is a progressive trend in prevention of dental caries. Indeed, a number of carbohydrates, mainly edible polyhydric alcohols (xylitol, sorbitol, mannitol) have
a sweet taste, and are harmless to the oral cavity.

This direction does not yet have wide practical application. In this way, most widely used sugar substitutes «Aspartame», «Slastilin», etc. are added to food instead of sugar, especially in liquid food (tea, coffee, etc.).

Mouthrinsing after each meal can reduce the cariogenic effect of carbohydrates by evacuating sweet cariogenic food or by stimulating the function
of salivary glands and increasing the volume and rate of salivation. The same effect is produced by chewing gum chewing.

Evacuation of carbohydrates leads to the elimination of metabolism substrate for microorganisms, and therefore decreases its cariogenic effects.

Food in addition to its primary function acts as a factor of self-purification and training of the oral cavity. Self-cleaning of the oral cavity is a natural process exemption from food debris detritus. It is carried out in the act of swallowing movements of the lips, tongue, cheeks, jaw and under the influence of current saliva.

Self-cleaning of oral cavity is difficult in modern man because of the reduction of dental system and its connection with the presence of a large number of anomalies, caries and periodontal disease. Some kinds of food have cleansing properties.

There are hard fruits and vegetables — apples, radish, carrot, cucumber. Good training of dental system occurs with hard and dry foods requiring salivation and prolonged intensive chewing (rump bread, crackers, meat piece, dry sausage and fish). In such cases, the self-cleaning mechanism is associated with two factors — direct influence of food on the teeth and gums (due to

food's density and hardness during chewing, biting, crushing and its movement along the tooth and cleansing of corresponding surfaces) and purification (due to intensive salivation food is washed out from the mouth). Food affects the tooth by changing acidity of dental plaque.

Duration of saving pH level depends on:

– product properties;
– properties of saliva;
– rate of arrival of new portions of food.

Factors of foodstuff:

– aggressive;
– protective.

Factors of oral cavity:

– depth of fissures;
– active chewing;
– the buffer capacity and mineralizing potential of saliva.

Factors of human behavior:

– frequency of meal intake;
– type of foods products;
– level of oral hygiene.

Person should get during the day:

1–2 g per kg of body weight proteins (including 50–60 % of animal origin);

1–2 g/kg fats (including not less than 30 % of the plant origin);

4–6 g/kg carbohydrates (including no more than 30% refined sugars);

1–2 g of calcium;

0.02–0.05 mg/kg fluoride.

Studies of food hygiene show that diet features of population are protein deficiency, excess animal fat and carbohydrates (sugars), deficiency of calcium, phosphorus, fluorine, vitamins A and D.

Dentists and scientists actively develop problems of systemic prevention of dental caries involving the intake of active substances affecting the preeruptive enamel cariesresistance formation.

Mineral supplements are widely used.

In high risk caries cases vitamins, adaptogens, immunostimulants, anabolic steroids are recommended.

Preeruptive prevention. Calcium is an essential macronutrient, a majority of which is concentrated in mineralized tissues in the form of phosphate (apatite, phosphates). However, it is of vital importance to maintain a normal level of calcium in the blood, as it is required for hemostasis, nerve impulses, muscle contraction and stability of cell membranes, etc.

The process of growth and skeletal development is in a constant state of self-renewal, remodeling, which is the result of two parallel processes: resorption and modeling (formation).

The skeleton in children and adolescents is completely updated within 1–2 years during the period of intensive growth. Among the factors that have a decisive influence on the processes of bone formation and growth of the skeleton and attainment of optimal, genetically determined peak mass, nutrition and reliable supply of growing body with all the essential minerals and vitamins play an important role. The absorption of calcium in the intestines depends on presence of vitamins.

Vitamin D3 transformed in kidneys to hormone calcitriol which is necessary for the normal process of absorption of calcium in the intestines. Vitamin C is involved in the process of maturation of collagen, plays an important role in the formation of a transport form of vitamin D3, facilitates the absorption of iron and is involved in many of the redox reactions in the body.

Vitamin B6 is involved in the metabolism of carbohydrates, fats and proteins; plays an important role in nervous system functioning. It is a part of enzyme lizoloksidaze ensuring the formation of crosslinks between the adjacent protein chains of collagen fibers that imparts a particular strength.

Vitamin A is responsible for the development and differentiation of intestinal epithelial cells carrying out the absorption of calcium and other nutrients essential for the formation of epithelial structures and synthesis of visual pigment.

Zinc is a component of various enzymes (200) which synthesize proteins and nucleic acids. Also it provides gene expression, cell growth and regeneration. It has positive influence on the activity of alkaline phosphatase enzyme.

Manganese promotes the synthesis of substances that are components of bone and cartilage tissue (glycosaminoglycans). It potentiates the ability of vitamin D to hold on Ca.

Copper is involved in the formation of elastin, collagen. It helps to stop the phenomenon of demineralization of bone structures.

Bor normalizes the activity of parathyroid hormone which is involved in the metabolism of calcium, cholecalciferol, phosphorus, magnesium. It effects on mineral metabolism of parathyroid hormone depend on vitamin D3.

Average need for calcium supply with food is 1500 mg Ca/day in combination with intake of vitamin D of 400 IU/day.

About 20–30 % of Ca are absorbed from dairy products and more than 50 % from plant products.

Conditions when needs of Ca increase to 2000 mg/day.

• In the second half of pregnancy due to fetal tissue mineralization (during the third trimester of pregnancy, the fetus accumulates in the skeleton 25 g of calcium).

• During lactation (breast feeding requires 0.3 g of calcium per day).

• During periods of rapid growth in children (adult skeleton contains about 1 kg of calcium).

• In old age (due to loss of calcium due to hormonal changes).

Disturbances in the assimilation and distribution of Ca:

• Pathology of the gastrointestinal tract;

• Lack of phosphorus in the diet.

Deficit of active forms of vitamin D (calcitriol), required for calcium absorption in the intestine, may be due to several causes:

– shortage of products containing Vitamin D (vegetable oil, chicken egg yolk, liver);

– a limited form of calcitriol in the skin by photolysis dehydrocholesterol under the influence of ultraviolet rays (this is a problem with low insolation under adverse climatic and environmental conditions);

– disturbances in the synthesis of the active form of vitamin D in the pathology of the liver and kidneys;

– impairment in the hormonal regulation of calcium metabolism (imbalance between calcitonin, calcium retention in the depot, and parathyroid hormone, calcium, deducing from the depot, estrogens, progestins, etc.).

Frequent vomiting in pregnancy toxemia.

Preeruptive prevention:

– calcium supplements;

– calcium chloride;

– calcium lactate;

– calcium gluconate;

– calcium glycerophosphate;

– calcium carbonate;

– Calcinova;

– Calcium D3 Nycomed, Calcemin.

Drugs are prescribed by a doctor — a pediatrician (general practitioner) in age dosages and periodically repeated courses for 2–4 weeks. In the winter time it is recommend to combine calcium supplement drugs with Vitamin D.

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WORKSHOP № 6
DENTAL BIOFILM AS THE MAIN CARIOGENIC FACTOR: DIAGNOSIS, ASSESSMENT OF CARIOGENIC POTENTIAL

Test Questions:

1. Classification of dental deposits.

2. Pellicle: composition, properties, formation mechanism, and detection methods.

3. Dental plaque as a biofilm: composition, structure, formation mechanisms, role in dental caries occurrence.

4. Materia alba, food debris: composition and properties.

5. Methods of detection and removal of dental deposits.

6. Evaluation of dental plaque cariogenic activity:

a) bacteriological methods of oral fluid analysis;

b) bacteriological methods of dental plaque analysis;

c) biochemical research methods of dental plaque analysis.

Dental plaque is a complex biofilm growing on teeth which is the etiological agent of caries and periodontal disease.

The movement of saliva around the mouth, mainly as a result of tongue action creates a shear force which limits plaque accumulation to sheltered sites.

Plaque is initiated by the colonization and subsequent outgrowth of organisms competent to adhere to the tooth pellicle.

The major «pioneer species» are Streptococcus mutans, Streptococcus mitis and Streptococcus sanguis. Others include Neisseria and Actinomyces.

During and following outgrowth, other species colonize the forming biofilm by adhering to the pioneer species. This process of coaggregation can be highly specific.

As secondary colonization progresses the species diversity of the biofilm increases.

Eventually, a combination of secondary colonization and cell proliferation, moderated by detachment of organisms from the biofilm by shear force, produces a climax community.

The climax community is stable and complex yet responsive to changes in the environment.

Plaque encourages caries formation by:

– enabling bacteria to stick to the teeth;

– allowing acids to accumulate around the teeth;

– preventing the saliva from reaching the teeth surface, so stopping it from washing them and neutralizing the acid;

– providing the cariogenic bacteria with a reserve energy supply, i. e. the polysaccharides which can be used in the absence of sugars.

Classification of dental plaque.

A. Localization:

– supragingival;

– subgingival.

B. Structure:

– cuticle;

– pellicle;

– plaque;

– calculus.

Cuticle has an embryonic nature as produced in the last stage of enamel formation.

Ameloblasts secrete a small amount of protein substance after finishing enamel secretion. This substance is located in the subsurface layer of enamel and on its surface. Ameloblasts attach to the enamel surface using gemidesmosoms.

Cuticle can be detected only on the surface of newly erupted tooth. It rapidly destructs under the influence of external factors and hasn't any clinical significance.

Pellicle is closest to the enamel thin (1 micron) transparent layer of acquired dental plaque.

The pellicle is a thin coating of salivary proteins that attaches to the tooth surface within minutes after cleaning.

Composition of the pellicle repeats mineral and organic composition of saliva differing only in the high concentration of proteins (particularly immunoglobulins G and M).

Pellicle is easily and quickly formed on any solid surface placed in the saliva. First layers found in 20 minutes. Pellicle thickness reaches 50–1000 nanometers per day.

Glycoproteins of the first (subsurface) pellicle layer are chemically bonded to enamel. Acid groups are connect with calcium, the alcaline groups — with phosphate hydroxyapatite.

The second (surface) layer is formed as a result of further precipitation of saliva components.

The third (oversurface) layer of the pellicle is formed by the same components. It has a rough surface which facilitates connection to the pellicle following layers of deposits.

Dental plaque (also called as microbial plaque, dental plaque biofilm) is a dense, nonmineralized, highly organized complex mass of bacterial colonies in a gel-like intermicrobial matrix.

The matrix protects the bacteria from the defensive cells of the body (neutrophils, macrophages, and lymphocytes).

It adheres firmly to the acquired pellicle and also to the teeth, calculus, and restorations.

Acquired pellicle is an amorphous layer that forms over exposed tooth surfaces, as well as over restorations and dental calculus.

It begins to form within minutes after all external material has been removed from the tooth surfaces with an abrasive.

It is composed primarily of glycoproteins from the saliva that are selectively adsorbed by the hydroxyapatite of the tooth surface.

Although pellicle performs a protective function, acting as a barrier to the acids, it also serves the initial site of attachment to the bacteria and begins the first stage of biofilm development.

A biofilm community comprises bacterial microcolonies, an extracellular slime layer, fluid channels, and a primitive communication system. As the bacteria attach to a surface and to each other, they cluster together to form sessile, mushroom-shaped microcolonies that are attached to the surface at a narrow base.

Each microcolony is a tiny, independent community containing thousands of compatible bacteria. Different microcolonies may contain different combinations of bacterial species. Bacteria in the center of a microcolony may live in a strict anaerobic environment, while other bacteria at the edges of the fluid channels may live in an aerobic environment.

Thus, the biofilm structure provides a range of customized living environments (with differing pHs, nutrient availability, and oxygen concentrations) within which bacteria with different physiological needs can survive.

The extracellular slime layer is a protective barrier that surrounds the mushroom shaped bacterial microcolonies.

The slime layer protects the bacterial microcolonies from antibiotics, antimicrobials, and host defense mechanisms.

A series of fluid channels penetrates the extracellular slime layer. These fluid channels provide nutrients and oxygen for the bacterial microcolonies and facilitate movement of bacterial metabolites, waste products, and enzymes within the biofilm structure.

Each bacterial microcolony uses chemical signals to create a primitive communication system used to communicate with other bacterial microcolonies.

FORMATION OF DENTAL PLAQUE BIOFILMS

Stage I: the electrostatic interaction between the pellicle surface and polar charged microbial cell ensures their convergence.

Stage II: the mechanical attachment of microbial cells to the surface layer of the pellicle by means of special cell membrane protrusions (pili); Str. mutans is attaching more successfully.

Stage III: the creation of irreversible chemical bonds between proteins specific microbial cell surface adhesins (mostly talking about Str. mutans) and complementary groups of the surface of the pellicle.

Pellicle Formation. The initial attachment of bacteria begins with pellicle formation. The pellicle is a thin coating of salivary proteins that attaches to the tooth surface within minutes after cleaning. This layer is thin, smooth, colorless and translucent and is called as acquired salivary pellicle. Initially pellicle is bacteria free.

The function of salivary pellicle is mainly protective.

Salivary glycoproteins and salivary calcium and phosphate ions are absorbed on to the enamel surface and this process may compensate for tooth loss due to abrasion and erosion.

Pellicle also restricts the diffusion of acid products of sugar breakdown. It can bind other inorganic ions such as fluoride which promotes remineralization.

The pellicle acts like doublesided adhesive tape, adhering to the tooth surface on one side and on the other side, providing a sticky surface facilitating bacterial attachment to the tooth surface.

Following pellicle formation, bacteria begin to attach to the outer surface of the pellicle. Accumulation is greatest in sites which are protected from functional friction and tongue movement.

The interdental region below the contact area is the site for greatest plaque accumulation.

Bacteria are connected to the pellicle and each other with hundreds of hair-like structures called fimbriae.

Once they stick, the bacteria begin producing substances that stimulate other free floating bacteria to join the community.

Within the first two days in which no further cleaning is undertaken, the tooth's surface is colonized predominantly by gram-positive facultative cocci, which are primarily streptococci species.

It appears that the act of attaching to a solid surface stimulates the bacteria to excrete an extracellular slime layer that helps to anchor them to the surface and provides protection for the attached bacteria.

Within the first few hours species of Streptococcus and a little later Actinomyces attach to the pellicle and these are the initial colonizers.

Dental plaque is translucent unmineralized soft substance adjacent to the pellicle.

Formation of Microcolonies. Microcolony formation begins once the surface of the tooth has been covered with attached bacteria. The biofilm grows primarily through cell division of the adherent bacteria, rather than through the attachment of new bacteria. Next, the proliferating bacteria begin to grow away from the tooth.

Plaque doubling times are rapid in early development and slower in more mature biofilms.

Bacterial blooms are periods when specific species or groups of species grow at rapidly accelerated rates.

A second wave of bacterial colonizers adheres to bacteria that are already attached to the pellicle.

Coaggregation is the ability of new bacterial colonizers to adhere to the previously attached cells.

The bacteria cluster together to form sessile, mushroom-shaped micro-colonies that are attached to the tooth surface at a narrow base.

The result of coaggregation is the formation of a complex array of different bacteria linked to one another.

Supragingival plaque formation is also pioneered by bacteria with an ability to form extracellular polysaccharides which allow them to adhere to the tooth and each other and these include Streptococcus mitior, S. sanguis, Actinomyces viscosus and A. naeslundii.

Plaque grows by both internal multiplication and surface deposition. Internal multiplication slows considerably as the plaque matures.

The gram-negative cocci such as Neisseria and Veillonella species occupy any remaining interstitial space formed by the bacterial interactions in initial colonization phase.

Maturation. Following a few days of undisturbed plaque formation, the gingival margin becomes inflamed and swollen. These inflammatory changes result in the creation of a deepened gingival sulcus.

The biofilm extends into this subgingival region and flourishes in this protected environment, resulting in the formation of a mature subgingival plaque biofilm.

Gingival inflammation does not appear until the biofilm changes from one composed largely of gram-positive bacteria to one containing gram-negative anaerobes.

A subgingival bacterial microcolony, predominantly composed of gram-negative anaerobic bacteria, becomes established in the gingival sulcus between 3 and 12 weeks after the beginning of supragingival plaque formation.

Most bacterial species currently suspected of being periodontal pathogens are anaerobic, gram-negative bacteria.

Structure and Composition. Dental plaque can be broadly classified as supragingival or subgingival.

Supragingival plaque is found at or above the gingival margin and may be in direct contact with the gingival margin.

Subgingival plaque is found below the gingival margins, between the tooth and the gingival sulcular tissue.

Approximately 70 to 80 percent of plaque is microbial and the rest represents extracellular matrix.

The intracellular matrix which accounts for about 20 percent of plaque mass consists of organic and inorganic materials derived from saliva, gingival crevicular fluid and bacterial products.

Organic constituents of the matrix include polysaccharides, proteins, glycoproteins, and lipids.

The most common carbohydrate produced by bacteria is dextran.

The principal inorganic components are calcium, phosphorus, sodium, potassium, fluoride and some traces of magnesium.

Calcium ions may aid adhesion between bacteria and between bacteria and the pellicle.

The source of both the organic and inorganic components is primarily saliva and as the mineral content increases, the plaque may be calcified to form calculus.

SUPRA AND SUBGINGIVAL PLAQUE

It can be defined as the community of microorganisms that develops on the tooth surface coronal to the gingival margin (at or above the gingival margin).

When it is in direct contact with the gingival margin it is termed as the marginal plaque.

It typically demonstrates a stratified organization of a multilayered accumulation of bacterial morphocytes.

The first cellular material adhering to the pellicle in it consists of the coccoid bacteria with numbers of epithelial cells and polymorphonuclear leukocytes.

Gram-positive cocci and short rods predominate at the tooth surface, whereas gram-negative rods and filaments as well as spirochetes, predominate in the outer surface of the mature plaque mass.

The material present between the bacteria in the plaque is called the intermicrobial matrix and accounts for approximately 25 percent of plaque volume.

Fructans (levans) and glucans are the well characterized polysaccharides present in the plaque matrix.

Presence of the small amounts of lipids in it is also documented.

Subgingival Plaque. It can be defined as the community of microorganisms that develops on tooth surfaces apical to the gingival margin (found below the gingival margin, between the tooth and the gingival pocket epithelium).

Generally, the subgingival microbiota differs in composition from supragingival plaque mainly because of the local availability of blood products and low redox potential which characterizes the anaerobic environment.

The apical part of subgingival plaque is predominated by spirochetes, cocci and rods, whereas in the coronal part, more filaments are observed.

Between subgingival plaque and the tooth an electron dense organic material is interposed called as cuticle.

A characteristic feature of subgingival plaque is the presence of leukocytes interposed between the surface of the bacterial deposit and gingival sulcular epithelium.

Plaque Retention Factors. These are conditions that favor plaque accumulation and hinder plaque removal by the patient and the dental professional. Examples of these are:

– orthodontic appliances;

– partial dentures;

– malocclusions;

– faulty restorations;

– calculus;

– deep pockets;

– mouth breathing;

– tobacco use;

– certain medications.

SIGNIFICANCE OF DENTAL PLAQUE

The role of dental plaque in the initiation of dental caries and periodontal infections is now well documented. Dental caries and periodontal disease result from the bacterial products of the plaque flora.

Calculus and its Relationship with Plaque:

• Calculus is formed by the deposition of calcium and phosphate salts in bacterial plaque. These salts are present in salivary and crevicular fluids.

• Calculus is porous and can act as a reservoir or nidus of bacteria and endotoxin related to the disease process.

• Calculus formation has been observed in germ-free laboratory animals but is far more abundant in similar germ-infected laboratory animals. The role of the bacteria in relation to calculus formation is not completely understood.

METHODS OF PLAQUE VIZUALIZATION

Clinically, plaque presents as a transparent film and therefore, is difficult to visualize.

It can be detected with an explorer by passing the explorer over the tooth surface near the gingival margin to collect plaque, which makes it easier to see.

Plaque disclosing solutions that stains the invisible plaque is used for easy detection of plaque. It stains the plaque and makes it visible to the eyes. These solutions disclose the extent and location of the plaque.

DISCLOSING AGENTS

Disclosing agents are materials used to make the presence of plaque biofilm visible.

Since dental plaque is white, it cannot easily be identified particularly if it is not thick enough. A disclosing agent stains dental plaque and thus makes it fully evident to the individual. They are available in liquid or tablet form.

Solutions can be applied as a concentrate with a cotton swab or diluted with water in a cup to be used as an oral rinse.

Tablets are chewed after using disclosing agent and swished around in the mouth. Pellicle, plaque, debris and calculus absorb the disclosing agent.

Ideal requirement of a disclosing agent:

• It should distinctly stain only the plaque.

• It should have a pleasant taste.

• It should be biocompatible.

• Preferably it should stain new and old plaque in different colors for identification.

• The color should remain in the plaque for about 15–30 minutes (instruction period) and should not be removed by saliva.

• It should have antiseptic and astringent properties.

Chemicals Used as Disclosing Agents:

– iodine solutions;

– erythrosine;

– basic fuchsin;

– fast green — FD&C Green No-2.5 percent or 3.5 percent;

– Bismark brown;

– mercurchrome preparations;

– mercurochrome solution (5 %);

– flavored mercurochrome disclosing solution;

– merbromin;

– fluorescein FD&C Yellow No 8;

– 1–3 Tetrazolium compound with methylene blue;

– 2-Tone;

– FD&C Green No 3 and FD&C Red No 3.

2-Tone disclosing solution stains:

• Red — newly formed biofilm usually supragingival and is thin.

• Blue — older biofilm which is thicker and more tenacious usually seen at and just below the gingival margin, specially on proximal surfaces. It may be associated with calculus deposit.

Uses:

– patient instruction and motivation;

– self evaluation;

– estimation of plaque index;

– evaluation the effectiveness of oral hygiene maintenance;

– in research, evaluation the effectiveness of plaque control devices like dentifrices, toothbrushes, etc.

Contraindications:

– should not be used on teeth with glass ionomer and resin filling to prevent the staining of these filling;

– should not be used in patients with known allergy to any type of disclosing agent.

Interpretation:

• Clean tooth surfaces do not absorb any colouring agent.

• When pellicle and dental biofilm are present they absorb the disclosing agent and become stained.

• Pellicle stains as a thin relatively clear layer and dental biofilm appears darker, thicker and more opaque.

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WORKSHOP № 7
DIET AS CARIES-PRODUCING AND CARIES-PROTECTIVE FACTOR

Test Questions:

1. Mechanisms of diet influence on the state of dental plaque, saliva and dental hard tissues (food products and processes related to food intake). Foodstuffs acidogenes and cariogenicity.

2. Biochemical properties and dental effects of products containing carbohydrates:

a) the structure and biochemical characterization of carbohydrate groups;

b) the cariogenic potential of products containing both simple or complex carbohydrates;

c) dental effects of fruits and vegetables;

d) the effects of dental dairy and starches.

3. Caries-protective effects of protein and fat.

4. Diet and nutrition modification.

5. Oral care after eating carbohydrate food.

6. Intense and bulk sweeteners.

7. Chewing gum.

Dental caries is a highly prevalent chronic disease and its consequences cause a lot of pain and suffering. A dynamic relation exists between sugars and oral health. Diet affects the integrity of the teeth; quantity, pH, and composition of the saliva; and plaque pH. Sugars and other fermentable carbohydrates, after being hydrolyzed by salivary amylase, provide substrate for the actions of oral bacteria, which in turn lower plaque and salivary pH. The resultant action is the beginning of tooth demineralization.

Dental caries occurs due to demineralization of enamel and dentine by organic acids formed by bacteria in dental plaque through the anaerobic metabolism of sugars derived from the diet. Despite improved trends in levels of dental caries in developed countries, dental caries remains prevalent and is increasing in developing countries undergoing nutrition transition.

Sucrose and starches are the predominant dietary carbohydrates in modern societies. While the causal relationship between sucrose and dental caries development is indisputable, the relationship between food starch and dental caries continues to be debated.

Role of diet. There is abundant epidemiological evidence that dietary sugars are the major dietary factor affecting dental caries prevalence and progression.

Sugars, particularly sucrose, are the most important dietary etiological cause of caries. Both the frequency of consumption and total amount of sugars is important in the etiology of caries. Caries is associated with increase in the proportions of acidogenic and acid tolerant bacteria, especially Streptococcus mutans. These bacteria are able to rapidly metabolize dietary sugars to acid, creating locally a low pH. They grow and metabolize optimally at low pH and under such conditions become more competitive, whereas most bacterial species associated with enamel health are sensitive to acidic conditions. It is indisputable that mutans streptococci's role in caries development is conditional upon a frequent intake of fermentable sugars. (Fermentable carbohydrate: any carbohydrate that can be hydrolyzed by salivary amylase in the initial stage of carbohydrate digestion and subsequently fermented by bacteria).

Streptococcal mutans are able to rapidly metabolize dietary sugars to acid, creating locally a low pH. High frequency of fermentable carbohydrate intake with regular pH drops favors the proliferation of mutans streptococci and there is a direct effect of sucrose on the numbers of these micro-organisms in the mouth. As the pH falls, calcium and phosphate ions are lost from the enamel. The drop in pH below 5.4, the critical pH, is sufficient to decalcify enamel and dentine.

Sucrose appears to be the most cariogenic sugar, (Cariogenic: foods and drinks containing fermentable carbohydrates that can cause a decrease in plaque pH to < 5.5 and demineralization of underlying tooth surfaces) not only

because its metabolism produces acid, but also because MS (Mutans streptococci) utilize this sugar to produce the extracellular polysaccharide glucan. Glucan polymers are believed to enable MS to both adhere firmly to teeth and to inhibit diffusion properties of plaque.

The critical pH value for demineralization varies amongst individuals, but is in the approximate range of 5.2 to 5.5. Conversely, tooth remineralization can occur if the pH of the environment adjacent to the tooth is high due to: 1) lack of substrate for bacterial metabolism; 2) low percentage of cariogenic bacteria in the plaque; 3) elevated secretion rate of saliva; 4) strong buffering capacity of saliva; 5) presence of inorganic ions in saliva; 6) fluoride; 7) rapid food clearance time. However, if demineralization over time exceeds remineralization, an initial carious lesion (the so-called «white spot» lesion) can develop and may further progress to a frank cavity.

The basic Stephan Curve. The Stephan Curve describes the change in dental plaque pH in response to a challenge. The type of challenge does not matter but it is usually some element of the diet. On the other hand the challenge could be an inert substance placed in the mouth with the aim of determining its effect on plaque pH. For example: mechanical stimulation of the salivary glands caused by masticating chewing-gum base. This would be of interest in an investigation into the effect of saliva flow on the pH, while studying the changes happening in plaque after a challenge by a cariogenic food.

Characteristically the Stephan Curve reveals a rapid drop in plaque pH, followed by a slower rise until the resting pH is attained. The time course varies between individuals and the nature of the challenge.

The initial drop is usually rapid with the lowest pH being attained within a very few minutes. However, pH recovery can take anything between 15 and 40 minutes depending to a large extent on the acid-neutralizing properties of the individual’s saliva.

The initial rapid drop in pH: it is due to the speed with which plaque microbes are able to metabolize sucrose. Larger carbohydrates, such as starch, would diffuse into plaque more slowly and would need to be broken down before assimilation by the microbes. In the case of starch, salivary amylase would produce a mixture of glucose and maltose together with incompletely digested material comprising the branch points of the starch molecule (limit

dextrins). The glucose and maltose would then be taken up by plaque bacteria and metabolized. The rate of starch breakdown slows up glycolysis and, therefore, acid production producing a less steep drop in pH.

The value of the Stephan Curve is that it provides a means by which the cariogenic challenge to a tooth may be measured (fig. 2). Actually, it only measures the potential cariogenic challenge because the critical pH value varies between individuals.

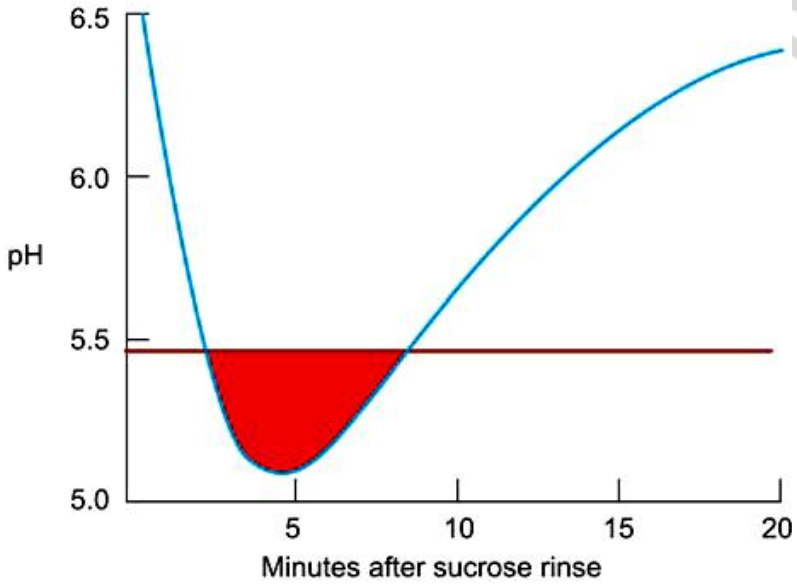

Fig. 2. Stephan curve

Human studies as opposed to animal experiments have shown a very strong correlation between frequency of intake of sugar and weight of sugar consumed. As one rises the other rises too and vice-versa. Hence both the frequency of consumption and total amount of sugars are important in the etiology of caries because frequency of intake and amount of sugars ingested are closely related. An increase in frequency of sugary intakes of more than 4 per day increased the risk of caries.

Food Form. The form of the fermentable carbohydrate directly influences the duration of exposure and retention of the food on the teeth. Prolonged oral retention of cariogenic components of food may lead to extended periods of acid production and demineralization and to shortened periods of remineralization.

Duration may also be influenced by the frequency and amount of fermentable carbohydrate consumed. Liquid sugars, such as those found in beverages and milk drinks, pass through the oral cavity fairly quickly with limited contact time or adherence to tooth surfaces. However, fluid intake patterns can influence the caries risk of the beverages. Holding sugar containing beverages in the oral cavity for a prolonged time or constant sipping of
a sugared beverage increases the risk of caries. Long-lasting sources of sugars, such as chocolates, hard candies and lollipops, have extended exposure time in the oral cavity because the sugars are gradually released during consumption.

Oral Clearance. Oral clearance properties vary by individual person and depend on metabolism by microorganisms, adsorption onto oral surfaces, degradation by plaque and salivary enzymes, saliva flow and swallowing. Most carbohydrates will be cleared by these simultaneous mechanisms. Retentiveness of foods is not the same as stickiness. A caramel or jellybean may be sticky, but its retentive properties are fairly low and they are cleared from
the oral cavity faster than are retentive foods such as cookies or chips. Studies have shown that, the longer the foods are retained in the oral cavity, the greater the potential the starch has to break down into sugars and contribute to the caries process. The initial content of sugars was not the culprit; rather, it was the type of starch and extent of starch retention time in the oral cavity that determined the relative cariogenic risk of the food.

Frequency. The frequency of consumption seems to be a significant contributor to the cariogenicity of the diet, although Bowen et al concluded that it is not the frequency of ingestion per second that is related to the development of caries but the time that sugars are available to microorganisms in the mouth. The importance of frequency is clear when caries is regarded as the outcome of the alternation of demineralization and remineralization.

DIETARY FACTOR AND DENTAL CARIES

Frequency and Amount of Ingestion of Dietary Sugars and Dental Caries. Data from animal studies have shown that dental caries experience increases with increasing frequency of intake of sugars, even when the absolute intake of sugar was the same. Since the pH of dental plaque falls each time sugars are ingested, so more times in a day sugar is consumed, the more times

the plaque pH will be depressed to a level at which dental enamel may dissolve. Higher frequency means more demineralization and less remineralization.

Cariogenicity of sugars. Earlier studies investigating the relationship between dental caries and sugar focused mainly on sucrose. Animal studies show that there is not much difference between cariogenicity of mono and disaccharides except that of lactose. Plaque pH studies have shown that less acid is produced by plaque bacteria from lactose, compared to other sugars. The cause of dental caries is the consumption of fermentable carbohydrates (sugars). There is a dose — response relationship between the quantity of the sugar consumed and the development of dental caries. It is suggested, at levels below 10 kg/person per year dental caries will not develop. (15 kg/person per year in fluoridated areas).

Sugars are classified into *Intrinsic* and *Extrinsic* based on physical location of sugars. *Intrinsic Sugars* located within the cellular structure of food, make them unavailable for metabolism by the oral bacteria, and hence are harmful to teeth. *Extrinsic sugars* are located outside the cellular structure of the food and include «Milk Sugar» (lactose present naturally in milk is not harmful to teeth) and «Non-Milk Extrinsic Sugars» (NMES). These added sugars, added by the manufacturer, cook or consumer, are better called non-milk extrinsic sugars — nicely shortened to NME or «enemy» are harmful to teeth. The World Health Organization (WHO) calls these sugars «free» sugars and WHO recommends that, first, there is no nutritional need for these NME or free sugars and, second, that consumption should be less than 10 % of total food energy intake.

The current dose-response relationship between caries and extrinsic sugars suggests that the sugars level above 60 g/person/day for teenagers and adults increases the rate of caries. For pre-school and young children the intake should be proportional to those for teenagers; about 30 g/person/day for pre-school children. There is no evidence that sugars naturally incorporated in the cellular structure of foods (intrinsic sugars) or lactose in milk or milk products (milk sugars) have adverse effects on health. Foods rich in starch, without the addition of sugars, play a small role in coronal dental caries.

CARBOHYDRATES AND DENTAL CARIES

Dietary Carbohydrates

A. Sugars.

Dietary carbohydrates can be divided into simple sugars and complex carbohydrates, e. g., starches.

B. Starches.

Starches are also a major component of the human diet. Cereal grains are often a primary source of calories, proteins, minerals, and vitamins.

Classifications. There are a variety of interrelated classification schemes. The most useful classification scheme divides the carbohydrates into groups according to the number of individual simple sugar units (table 3). Monosaccharide contain a single unit; disaccharides contain two sugar units; and polysaccharides contain many sugar units as in polymers — most contain glucose as the monosaccharide unit.

Table 3

Classification of carbohydrates

Monosaccharides	Disaccharides	Polysaccharides
Glucose	Sucrose	Starch
Galactose	Maltose	Glycogen
Fructose	Lactose	Cellulose
Ribose		
Glyceraldehyde		

Sugars are a form of fermentable carbohydrate. Fermentable carbohydrates are carbohydrates (sugars and starch) that begin digestion in the oral
cavity via salivary amylase. Sugars enter the diet in 2 forms: those found naturally in foods (e. g., fruit, honey, and dairy products) and those that are added to foods during processing to alter the flavor, taste, or texture of the food. Starches are subsequently digested by salivary amylase to oligosaccharides, which may be fermented by the oral microflora. According to Lingstrom et al, only

the gelatinized starches are susceptible to breakdown by salivary amylase into maltose, maltotriose, and dextrins.

Starch constitutes a heterogeneous food group. It may be highly refined or consumed in its natural state. It may also be consumed raw (fruits and vegetables) but is mainly consumed in a cooked form. Animal studies have shown that raw starch is of low cariogenicity. Cooked starch causes caries but only about half the amount caused by sucrose. Enamel slab experiments in humans have shown that raw starch does not cause remineralization. Epidemiological studies have shown that starch is of low risk for causing dental caries. People who consume high-starch/low-sugar diet generally have low caries experience than people who consume low-starch/high-sugar diets.

After an extensive review on the relationship between starchy foods and caries Rugg-Gunn and Rugg-Gunn and Nunn concluded that:

• Cooked staple starchy foods such as rice, potatoes, and bread are of low cariogenicity in humans.

• The cariogenicity of uncooked starch is very low but, since this is seldom eaten by humans, this finding is of little relevance.

• Finely ground and heat-treated starch can cause dental caries, but the amount of caries is less than caused by sugars.

• The addition of sugar increases the cariogenicity of cooked starchy foods. Foods containing cooked starch plus substantial amounts of sucrose, appear to be as cariogenic as a similar quantity of sucrose.

Starch is converted by salivary amylase to maltose and glucose. This is a slow process and hence dietary starch alone is much less important than dietary sugars as a cause of dental caries.

FRUITS AND DENTAL CARIES

There is little evidence to show fruit to be an important factor in the development of dental caries when it is consumed as a part of the mixed human diet. A number of plaque pH studies have found fruits to be acidogenic, although less than sucrose. The extent of this varies according to texture and

sugar content of the fruit. However, plaque pH studies measure acidogenicity and not cariogenicity and do not take into account the protective factors in fruits. Animal studies have shown that all fruits cause less caries than sucrose. Epidemiological studies have also shown that fruits which are habitually consumed are of low cariogenicity.

Dried fruits may potentially be more cariogenic since the drying process breaks down the cellular structure of the fruit, releasing free sugars; also the dried fruits tend to have a longer oral clearance. Increasing consumption of fresh fruits in order to replace non-milk extrinsic sugars in the diet is likely to decrease the level of dental caries.

PROTECTIVE FACTORS AND CARIES

Some constituents of diet help to protect teeth against dental caries.

Cheese. Studies have shown that several types of cheese are not only non-cariogenic but also have anti-cariogenic properties. Cheese stimulates salivary secretion and increases plaque calcium concentration, and causes adsorption of protein (casein) on to the enamel surface thereby physically slowing the caries process.

Milk. Cow's milk and human milk contain lactose which may be classified as cariogenic. But lactose is least cariogenic of the dietary sugars and milk also contains calcium, phosphorus and casein which inhibit caries. Evidence from animal experiments show that cow's milk is not only non-cariogenic, but also has an anti-cariogenic effect.

Fibrous Foods. Fibrous foods protect the teeth because they mechanically stimulate salivary flow. Other foods that are good gustatory and/or mechanical stimulants to salivary flow are peanuts, hard cheese.

Black Tea. Black tea contains fluoride, polyphenols and flavanoids. Black tea extract increases plaque fluoride concentration and reduces the cariogenicity of a sugar rich diet.

Sugars are a form of fermentable carbohydrate.

Fermentable carbohydrates are carbohydrates (sugars and starch) that begin digestion in the oral cavity via salivary amylase. Sugars enter the diet in 2 forms: those found naturally in foods (e. g. fruit, honey, and dairy products) and those that are added to foods during processing to alter the flavor, taste, or texture of the food.

EFFECT OF FLUORIDE ON SUGAR-CARIES RELATIONSHIP

Fluoride alters the resistance of the teeth to demineralization and the speed of remineralization of the enamel surface after an acid attack. Post eruptive effect of fluoride includes:

• Reduces and inhibits demineralization.

• Remineralization of enamel

• Affects plaque by inhibiting bacterial metabolism of sugars. It has been shown that without any dietary modification topical fluorides reduces caries in children by 20 to 40 %, but does not eliminate dental caries.

Widespread use of fluoride largely accounts for the decline in dental caries that has been observed in developed countries over the past three decades. Where there is a good exposure to fluorides, sugar consumption is a moderate risk factor for dental caries in most people. Sugar consumption is a major indicator for risk of caries in people who are not exposed regularly to fluoride. With widespread use of fluoride, sugar consumption still has a role to play in the prevention of caries but this role is not as strong as it is without exposure to fluorides.

NON-SUGAR SWEETENERS AND DENTAL

Caries. These can be classified into two: 1) intense sweeteners; 2) bulk sweeteners.

A large numbers of sweet compounds are known but a few are permitted to be used on foods in and these vary from country to country.

Intense Sweeteners. These are not metabolized to acids by oral micro-organisms and hence cannot cause dental caries. These are not chemically

related to sugars. They are hundred to thousand times sweeter than sucrose but have a negligible energy value.

Saccharine has a bitter taste in concentration over 0.1 percent. It is used as table-top sweetener and in sweetening tablets.

Acesulfame Potassium is chemically synthesized sweetener. It is stable in aqueous solutions of wide ranging pH and does not break down on heating. Hence a useful sweetener in reduced energy soft drinks, boiled sweets, confectionary, chewing gum and other foods.

Aspartame is moderately stable in solutions and breaks down on prolonged heating. It is used extensively in soft drinks, chewing gum, frozen foods. It is banned in children less than 12 years of age as it causes neurotoxicity.

Thaumatin is a naturally occurring intense sweetener, which is extracted from a plant found in West Africa. It is mainly used in pharmaceutical products.

Limitations of use:

– poor taste quality;

– instability;

– lack of bulk.

Use. Used in food products like soft drinks, beer, confectionary, desserts, ice-cream and jams.

Bulk sweeteners. These are chemically similar to sugars, and they add volume and sweetness to a product. They are 0.5 to 1.0 times as sweet as sucrose. The various bulk sweeteners are sorbitol, mannitol, isomalt, xylitol, lactitol and hydrogenated glucose syrup. Bulk sweeteners have negligible cariogenicity as compared to sugars.

Xylitol is a sugar-substitute with sweetness equal to that of table sugar (sucrose), but with 40 % fewer calories. It is a member of the sugar alcohol or polyol family, which includes other common dietary sweeteners such as sorbitol, mannitol, and maltitol. Xylitol is produced commercially from birch trees and other hardwoods containing xylan. More recently, to reduce production cost, commercial xylitol is being produced from corn cobs and the waste of sugarcane or other fibers using biotechnology. Xylitol can be found in

small quantities in fruits and vegetables and is produced as part of human metabolic processes.

Xylitol is widely used in sugar-free products such as chewing gums, candies, and toothpastes and are frequently combined together with small amounts of high intensity artificial sweeteners such as saccharin or aspartame to improve the flavor and sweetness of products.

Microorganisms do not readily metabolize xylitol into energy sources, and its consumption has a minimal effect on plaque pH. Xylitol has been shown to have a protective effect and to reduce tooth decay in part by reducing the levels of Strep-tococcus mutans in plaque and saliva and by reducing the level of lactic acid produced by these bacteria.

Studies have shown that a range of 6 to10 g divided into at least 3 consumption periods per day is necessary for xylitol to be effective with chewing gum as the delivery system. Xylitol has been approved by the FDA since
the 1960s and is safe for use with children. Polyols are absorbed slowly by the human gastrointestinal tract. The main side effect associated with most polyol consumption is osmotic diarrhea which, for xylitol, only occurs when it is consumed in large quantities, 4 to 5 times that needed for the prevention of dental caries.

The available evidence indicates that the level of dental caries is low in countries where the consumption of free sugar is below 15 to 20 kg/person/ year. This is equivalent to a daily intake of 40 to 55 gm and the value equates to 6 to 10 % of energy intake.

A number of countries have adopted policies for free sugars (non-milk extrinsic or refined) intake. The 1990 WHO report «Diet, Nutrition and the Prevention of Chronic Diseases» also recommended that free sugars should contribute to no more than 10 % to energy intake. Individuals should be advised to reduce the frequency of consumption of foods containing free sugars to four times a day thereby limiting the amount of free sugars consumed. Food manufacturers can contribute to products rich in free sugars, including drinks by producing low sugar/sugar-free alternatives.

Chewing gum. Chewing gum has been employed to deliver a variety of agent for oral health benefits. There appears to be significant benefit to dental health through the use of sugar-free chewing gum. Chewing gums alone appear to have little benefits in plaque control, but when used as an adjunct to normal tooth brushing reduction in plaque levels has been seen. The use of sugar-free chewing gum as a mechanical salivary stimulant after eating can accelerate the clearance of dietary substances and microorganisms, promote buffers to neutralize plaque acids and provide antibacterial substances. Chewing sugar-free gum for about 20 minutes after eating reduces the fall in plaque pH. This action reduces the time for demineralization and enhances the potential for remineralization of early carious lesions. The saliva flow rate is stimulated three to ten folds above the resting level and may be prolonged for over 30 minutes. This approach may enhance saliva function in those with low flow rates such as elderly sufferers of xerostomia or provide symptomatic relief from dry mouth. However, effects on gingivitis or calculus formation have not been demonstrated. The popularity of sugar-free chewing gums offers convenient delivery for chemotherapeutic agents. Gums promote salivation and require hydration to release the agent, which can then be effective for longer periods of time than rinses or dentifrices. Clinical studies with CHX acetate (Chlorhexidine) or CHX-xylitol chewing gums used twice daily showed CHX is released after chewing and can reduce plaque and gingivitis. However, the widespread clinical value of these formulations for the prevention of gingivitis awaits

epidemiological studies. A popular noncariogenic sugar substitute, the sugar alcohol xylitol, also has antibacterial action, reducing plaque acidogenicity following a sucrose rinse and reducing mutans streptococci levels. Gum containing sodium fluoride (0.25 mg fluoride) can release fluoride and promote salivation, complementing fluoride dentifrices and rinses in patients with xerostomia or rampant caries. The fluoride released tends to accumulate on the chewing side rather than distributing around the oral cavity.

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WORKSHOP № 8
METHODS OF ORAL HYGIENIC CONDITION ASSESSMENT. METHODS AND TOOLS FOR SOFT DENTAL DEPOSITS REMOVAL (SELF-CLEANING, WIPING, RINSING)

Test Questions:

1. Definition of «hygiene index». Characteristics of ideal index. Concepts of epidemiological and clinical indices.

2. Methods to assess oral hygiene:

a) protocol hygiene O'Leary;

b) Tureski index of oral hygiene;

c) PLI (plaque index Silness-Low);

d) OHI-S;

e) index PHP.

3. Oral hygiene: definition, purpose, strategy, and hygiene products.

4. Self-cleaning, wiping, rinsing as methods of removing dental deposits.

5. Liquid oral hygiene products: components, safety requirements, indications for use.

PLAQUE CONTROL RECORD

It was given by O'Leary, T. J. Drake and J. E. Naylor (1972). This system measures plaque present, rather than plaque not present, but no attempt is made to differentiate the quantity of plaque seen on each surface.

Selection of Teeth and Surfaces:

• All teeth are examined.

• Missing teeth are indicated on the record form as a single thick horizontal line.

• Four surfaces are examined: facial, lingual, mesial and distal.

• The number of surfaces examined may be increased from four to six. When using six surfaces, they are facial (or buccal), mesiofacial, mesiolingual, lingual, distolingual, and distofacial.

Procedure. Plaque is disclosed by either applying disclosing agent or the patient is asked to chew disclosing tablet and swish and rub the solution over the tooth surfaces with the tongue before rinsing. Each tooth surface is examined for plaque at the gingival margin and recording is done.

Scoring. For individual: the number of surfaces with plaque is multiplied by 100, and divided by the number of tooth surfaces examined.

$$\text{Percent with plaque} = \frac{\text{The number of surfaces with plaque}}{\text{Number of tooth surfaces examined}} \times 100\ \%.$$

For example, if an individual has 26 teeth, that equals 104 surfaces.

If eight surfaces are found to have plaque, then 800 are divided by 104, leaving a plaque control index of 7.6 percent.

A score under 10 % is considered good.

MODIFIED PLAQUE SCORING SYSTEM OF TURESKY ET AL.

Criteria Score:

No plaque — 0.

Separate flecks of plaque at the cervical margin of the tooth — 1.

A thin continuous band of plaque (up to 1 mm) at the cervical margin of the tooth — 2.

A band of plaque wider than 1 mm coercing less than one-third of the crown of the tooth — 3.

Plaque covering at least one-third but less than two thirds of the crown of the tooth — 4.

Plaque covering two-thirds or more of the crown of the tooth — 5.

Total score = Sum (scores for all facial and lingual surfaces).

Index = (total score) / (number of surfaces examined).

Interpretation:

A score of 0 or 1 is considered low.

A score of 2 or more is considered high.

Plaque Index (PI). The PI as developed by Silness and Loe (1964) assesses the thickness of plaque at the cervical margin of the tooth (closest to the gum). Four areas, distal, facial or buccal, mesial, and lingual, are examined.

• Each tooth is dried and examined visually using a mirror, an explorer, and adequate light. The explorer is passed over the cervical third to test for the presence of plaque. A disclosing agent may be used to assist evaluation.

• Missing teeth are not substituted.

• Four different scores are possible.

• Each of the four surfaces of the teeth (buccal, lingual, mesial and distal) is given a score from 0 to 3.

PI Score for Tooth. The scores from the four areas of the tooth are added and divided by four in order to give the plaque index for the tooth with the following scores and criteria.

Scoring Criteria: the Plaque Index. *Scores Criteria*:

0 — no plaque.

1 — a film of plaque adhering to the free gingival margin and adjacent area of the tooth. The plaque may be seen in situ only after application of disclosing solution or by using the probe on the tooth surface.

2 — moderate accumulation of soft deposits within the gingival pocket, or the tooth and gingival margin which can be seen with the naked eye.

3 — abundance of soft matter within the gingival pocket and/or on the tooth and gingival margin. The indices for the following six teeth may be

grouped to designate the index for the group of teeth: 16, 12, 24, 36, 32, and 44.

PI for an Individual. The index for the patient is obtained by summing the indices for all six teeth and dividing by six.

Interpretation for PI Scores. Four ratings may be assigned:

0 = Excellent oral hygiene.

0.1–0.9 = Good oral hygiene.

1.0–1.9 = Fair oral hygiene.

2.0–3.0 = Poor oral hygiene.

The Simplified Oral Hygiene Index (OHI-S). This index was given by John C. Greene and Jack R. Vermillion in 1964. It offers a more rapid method for evaluation of oral cleanliness of population groups, but lacks in degree of sensitivity in comparison to the original OHI index.

It differs from the original index in:

• Number of tooth surfaces scored (6 rather than 12).

• The method of selecting the tooth surfaces to be scored.

• The scores which can be obtained.

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

• In the posterior teeth, the first fully erupted tooth distal to the second bicuspid, usually the first molar but sometimes the second or third molar, is examined on each side of each arch.

• In the anterior portion of the mouth upper right central incisor and lower left central incisor are scored.

• In the absence of either of these anterior teeth, the central incisor on the opposite side of the midline is substituted.

• Only fully erupted permanent teeth are scored. A tooth is considered to be fully erupted when the occlusal or incisal surface has reached the occlusal plane.

• Natural teeth with full crown restorations and surfaces reduced in heights by caries or trauma are not scored.

Instead an alternate tooth is examined.

Surfaces to be Seen. Six surfaces are examined (from four posterior teeth and two anterior teeth).

Upper molars [6 | 6]: the buccal surfaces of selected teeth is inspected.

Lower molars [6 | 6]: the lingual surfaces of the selected teeth are checked.

Upper and Lower Central incisor 1: labial surface is scored.

Examination Method. To obtain the scores for debris and calculus, each of the six selected tooth surfaces are examined for debris and then calculus. The surface area covered by debris is estimated by running the side of a № 5 explorer (Shepherd's Crook) along the tooth surfaces being examined (Explorer is moved from incisal/occlusal to gingival margin). The occlusal or incisal extent of the debris is noted as it is removed. Same № 5 explorer is used to estimate the surface area covered by the supragingival and subgingival calculus.

1. Spray water into patient/client's mouth and instruct patient/client to swish.

2. Insert saliva ejector into patient/client's mouth.

3. Select teeth for examination by choosing six specific teeth with one in each sextant.

4. Evaluate teeth:

a) start evaluation with maxillary posterior sextant and work way around maxillary arch;

b) drop down to mandibular left lingual posterior sextant and work way around to other side of mouth.

5. Evaluate teeth for soft debris by recording six debris scores on appropriate recording form(s).

6. Evaluate teeth for calculus by recording six calculus scores.

7. Calculate debris score by totalling debris scores and dividing by number of teeth scored.

8. Calculate calculus score by totalling calculus scores and dividing by number of teeth scored.

9. Calculate OHI-S score by adding debris score to calculus score that equals OHI-S score.

10. Record OHI-S score in patient/client's chart or on appropriate recording form(s).

Scoring Criteria (Debris):

0 — no debris or stain present.

1 — soft debris covering not more than one third of the tooth surface being examined or presence of extrinsic stains without 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.

Scoring Criteria (Calculus):

0 — no calculus present.

1 — supragingival calculus covering not more than onethird of the exposed tooth surface being examined.

2 — supragingival calculus covering more than one-third but not more than two thirds of the exposed tooth surface and/or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth.

3 — supragingival calculus covering more than two third of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth.

In the simplified OHI, the worst score possible is 6.

Interpretation. Individually DI-S and CI-S is scored as follows:

0.0 to 0.6 = Good oral hygiene.

0.7 to 1.8 = Fair oral hygiene.

1.9 to 3.0 = Poor oral hygiene.

An OHI-S is scored as follows:

0.0–1.2 = Good oral hygiene.

1.3–3.0 = Fair oral hygiene.

3.1–6.0 = Poor oral hygiene.

PATIENT HYGIENE PERFORMANCE INDEX (PHP INDEX)

It was developed by A. G. Podshadley and J. V. Haley (1968) to assess the extent of plaque and debris over a tooth surface as an indication of oral cleanliness. Debris for PHP was defined as the soft foreign material consisting of bacterial plaque, material alba and food debris that is loosely attached to tooth surfaces. It is most useful for individual patients who have significant plaque accumulation.

Teeth and Surfaces Examined. Tooth Numbers in FDI System:

16 — upper right first molar;

11 — upper right central incisor;

26 — upper left molar;

36 — lower left first molar;

31 — lower left central incisor;

46 — lower right first molar.

Surfaces:

Facial surfaces: incisors and maxillary molars.

Lingual surfaces: mandibular molars.

Substitutions for Missing Teeth. The second molar is used if the 1st molar:

– is missing;

– less than three–fourth erupted;

– has a full crown;

– is broken down:

• the third molar is used when the second molar is missing;

• the adjacent incisor of the opposite side is used, when the central incisor is missing.

Procedure:

• Disclosing solution is applied.

• Patient is asked to swish for 30 seconds and expectorate but not rinse.

• Examination is made using a mouth mirror.

• Each tooth surface to be evaluated is subdivided into five sections as follows.

Vertically: three divisions mesial, middle and distal.

Horizontally: the middle third is subdivided into gingival, middle and occlusal or incisal thirds.

• Each area with plaque is scored a point so each tooth score can range from 1 to 5 points.

Scoring

Debris scores for individual tooth: add the scores for each of the five subdivisions. The scores range from 0 to 5.

PHP for an individual: total the scores for the individual teeth and divide by the number of the teeth examined. The PHP value ranges from 0 to 5.

PHP Index for a group: to obtain the average PHP score for a group or a population, total the individual score and divide by the number of people examined.

Interpretation. Nominal scale for evaluation of scores. Rating scores:

1. Excellent = 0 (no debris).

2. Good = 0.1–1.7.

3. Fair = 1.8–3.4.

4. Poor = 3.5–5.0.

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WORKSHOP № 9
METHODS AND TOOLS FOR SOFT DENTAL DEPOSITS REMOVAL (TOOTHBRUSHES, DENTAL FLOSS, TOOTHPICKS CHARACTERISTICS AND APPLICATION)

Test Questions:

1. Manual, powered and special toothbrushes: characteristics, structural elements, maintenance requirements, indications for replacement.

2. Mechanical interdental hygiene: rationale for interdental aids choice and use:

a) flossing: types of dental floss and tapes, their characteristics, methods of use;

b) toothpicks: types, indications, technique of application.

Mechanical Plaque Control. *Toothbrushes*: although different cleaning devices have been used in different cultures (toothbrush, chewing stick, etc.) the conventional toothbrush is the instrument most frequently used to remove dental plaque. The efficacy of brushing with regards to plaque removal depends upon three main factors:

1. The design of the brush.

2. The skill of the individual using the brush.

3. The frequency and duration of use.

If a properly designed brush is used with an effective technique and for a sufficient duration of time, plaque control can be achieved on a long-term basis.

Objectives of Toothbrushing:

1. To clean teeth of food, stains and debris.

2. To disturb and remove plaque formation.

3. To stimulate and massage the gingival tissue.

4. To apply fluoride dentifrice.

5. Cleaning of tongue.

MANUAL TOOTHBRUSHES

It is believed that the first brush made of hog's hair was mentioned in the early Chinese literature. By the early nineteenth century, craftsmen in various European countries constructed handles of gold and ivory in which replaceable brush heads could be fitted. The first patent for toothbrush in USA was issued to HN. Wadsworth in the middle of nineteenth century. Nylon came into use in toothbrush construction in 1938. World War II prevented Chinese export of wild boar bristles and synthetic materials were substituted for natural bristles.

Ideal requisites of a toothbrush:

1. Handle size appropriate to user age and dexterity.

2. Head size appropriate to the size of the patient's mouth.

3. Use of end-rounded nylon or polyester filaments not larger than 0.009 inches in diameter.

4. Use of soft bristle configuration as defined by the international industry standards.

5. Bristle pattern which enhances plaque removal in the approximal spaces and along the gum line.

6. Causing minimum damage to soft and hard dental tissue.

7. The brush should be easy to keep and clean.

8. Should be nontoxic.

9. Having a reasonable lifespan.

Parts of a Toothbrush. Total brush length is about 15 to 19 cm (6 to 7.5 inches). The brushes for children may be shorter in size.

The Head. It is the working end which consists of tufts of bristles or filaments and the stock where the tufts are secured. The brush head should be small enough to be manipulated effectively everywhere in the mouth. A length of about 2.5 cm for an adult and 1.5 cm for a child is satisfactory. A brush head may be 5–12 tufts in 3–4 rows wide. Tufts may be flat or may vary (rippled, dome, multilevel).

The Handle. It is the part which is grasped in hand during tooth brushing. It is made of a variety of materials such as acrylic and polypropylene. Its flexibility, size and shape must be convenient for manual use in the mouth. The handle must be comfortable and rest securely in the hand. It should be thick enough to allow a firm grip and good control.

Shank. It is the part that connects the head and the handle.

Tufts. Bristles when bunched together are known as tufts.

The Filaments (Bristles). Nowadays bristles are made of either polyester or nylon. These are polymers with good chemical resistance and are inert so that, they will pass through the body unchanged if swallowed. Nylon is said to wear less rapidly than polyester, and is more hygienic due to its antistatic

properties. The bristles can be classified into three depending upon the diameter of the filaments:

1. Soft 0.15–0.18 mm (0.006″–0.007″).

2. Medium 0.18–0.23 mm (0.007″–0.009″).

3. Hard/Extra hard 0.23–0.28 mm (0.009″–0.11″).

Bristle stiffness also depends upon the length of the filament, its elasticity, whether the brush is used dry or wet and the temperature of the water. Nylon looses approximately 30 percent of its stiffness when wet.

The bristles of children's brushes should always be soft (0.1–0.15 mm). Hard brushes can lacerate the gingival, encourage gingival recession and cause tooth abrasion and their bristle diameter is too large to reach gingival crevice, hence should never be recommended. Bristles should be of even length so that, they can function simultaneously. Bristles in an adult tooth brush are usually about 10 to 11 mm long. To be as nonabrasive as possible the end of the bristles should be round. The bristles of toothbrushes are usually arranged in about 40 tufts in three or four rows.

POWERED TOOTHBRUSHES

Types of power toothbrushes. The electrical toothbrush designs are categorized by the type of the brush head's shape and movement:

• The first generation of power toothbrushes had a head looking like the one of manual toothbrushes, and moving back and forth to simulate manual brushing. Only few low cost power toothbrushes use this not efficient mode today.

• Rotary toothbrushes: The next generation moved to a design with a circular head that is rotating in one direction.

• Counter-rotational toothbrushes with different tufts of bristles rotating in opposite directions.

• Rotating-oscillating toothbrushes in which a circular head spins back and forth in quick bursts.

• Oscillating-pulsating toothbrushes have in addition a pulsating motion to enhance the cleaning action.

They are also called as automatic, mechanical or electric toothbrushes. These were introduced to the market more than 50 years ago. They are now a well accepted part of the homecare regimen. They are potentially faster than manual tooth brushes at cleaning tooth surfaces. In contrast with old electric brushes, using a combination of horizontal and vertical movements, the new ones apply rotary and oscillating pulsating movements with bristles moving at high frequencies. Rotating-oscillating-pulsating toothbrushes have a small round head with stationary tufts that move in a 60-degree counter-rotational motion with approximately 7600 strokes per minute. There are brushes that have three dimensional movements that add a pulsating action of 20,000 to 40,000 movements per minute. Rotating-oscillating and rotating-oscillating-pulsating powered toothbrushes are distinctive in that the brush head is meant to be moved from tooth to tooth instead of using it in manner like a manual toothbrush.

Brush heads of powered toothbrushes tend to be more compact than manual toothbrushes. The bundle of bristles is arranged either in circular pattern or in rows which are mounted in a round head. The bristles are arranged as more compact single tuft, facilitating interproximal cleaning and brushing in less accessible areas of mouth. Studies have shown that if properly used both automatic and manual toothbrushes can remove plaque effectively.

Indications:

1. Those with physical or learning disability.

2. Fixed orthodontic appliances.

3. Young children.

4. Aged persons.

5. Institutionalized patients who depend upon care providers for brushing.

6. Arthritic patients.

7. Individuals with poor dexterity.

8. Poorly motivated patients.

Generally, the handles of electric toothbrushes are more ergonomic and comfortable for a firm grasp.

SONIC AND ULTRASONIC TOOTHBRUSHES

The cleaning action generated by a sonic toothbrush is actually based on two separate mechanisms. One of these is conventional and is similar in nature to that mechanism employed by all other types of toothbrushes. The second cleaning action is based on a new technology that is entirely unique to sonic toothbrushes.

1. The primary mode of cleaning that a sonic toothbrush can provide is by the scrubbing action of its brush head's bristles on the surface of the user's teeth. This method of cleaning teeth is not new. All toothbrushes, both electric and manual, rely on this same principle for removing dental plaque.

2. Sonic toothbrushes are also capable of producing a secondary cleaning action, one based upon a new technology developed by the brush's creators. This cleaning action is founded on the intense speed at which the bristles of the sonic toothbrush vibrate. This vibratory motion is able to impart energy to the fluids that surround teeth (such as saliva). The motion of these agitated fluids is capable of dislodging dental plaque, even beyond where the bristles of the toothbrush actually touch. Only a sonic toothbrush can make this claim. The brush head of sonic toothbrush has been designed to vibrate at more than 30,000 brush strokes per minute. This high speed brushing action in turn creates turbulent fluid dynamics near the tips of its bristles. The result is the creation of waves of pressure and shear forces in the liquids that surround your teeth, and also the creation of minute bubbles that are propelled forcefully against surfaces where plaque resides. The combination of these various fluid dynamics results in forces that are capable of dislodging dental plaque in those hard to reach areas such as between teeth and below the gum line. The cleaning effect of these fluid forces has been measured to occur at distances of up to 4 millimeters (slightly more than $1/8^{th}$ of an inch) beyond where the bristles of sonic toothbrush actually touch.

IONIC TOOTHBRUSHES

Principle:

1. The bonding between the pellicles and bacteria is mediated by Ca2+ bridge formation. The anions, supplied by the lithium battery inhibit the bonding between the bacteria and Ca2+ and prevent the bacteria from absorbing to the pellicles. Hence, the plaque accumulation is reduced because the above mentioned anions continuously supplied from the tips of the bristles of the ionic toothbrushes prevent the mild electrostatic bonding between the bacteria per se.

2. Instead of using friction or sound (sonic) waves to try to «blast apart» this bond, ionic technology changes the polarity of tooth surfaces from –ve to +ve. As brushing is done, plaque material is actively repelled by teeth and drawn to the negatively charged bristles, even in hard-to-reach areas of the mouth.

BIONIC TOOTHBRUSH — SOLADEY

Soladey was invented in Japan by Dr Yoshinori Nakagawa. The name originates from the words «Solar» and «Dental».

Mechanism of Action. There is a light-activated titanium rod (semiconductor) inside the handle. When exposed to any good light source the photosensitive titanium rod inside Soladey converts light into negatively-charged ions (electrons) which attract the positive ions in the acids in dental plaque. The rod releases these ions, which blend with saliva to attract positive (hydrogen) ions from the acid in the dental plaque on teeth. These ions permeate the saliva to effectively breakdown plaque on a molecular level. The polarity of the tooth surface is temporarily reversed by the ionic action which helps to prevent new plaque from forming. This ionic action disintegrates plaque in places one cannot reach with bristles, rinse, toothpaste or interproximal devices.

INTERDENTAL ORAL HYGIENE AIDS

As the interdental region is the most common site of plaque retention and the most inaccessible to the toothbrush, special methods of cleaning are needed. Removal of plaque without injuring the soft tissue should be the aim.

To remove the existing confusion with respect to terms such as approximal, interproximal, interdental and proximal sites, The European Workshop

on mechanical plaque control in 1999 proposed the following definition, «*Approximal (*proximal) areas are the visible spaces between teeth that are not under the contact area».

Interproximal and *interdental* may be used interchangeably and refer to the area under and related to the contact point. Tooth brushing alone cannot effectively control interproximal plaque, and the adjunctive methods of cleaning are to remove from these hard to reach sites. A number of interdental cleaning methods have been used for this purpose, ranging from floss to the recently introduced electrically powered cleaning aids. However, not all interdental cleaning devices suit all patients or all types of dentition.

Factors Effecting Selection of Interdental Aids:

1. Contour and consistency of gingival tissue.

2. Probing depth.

3. The size of the interproximal embrasure.

4. Tooth position and alignment.

5. Ability and motivation of the patient.

6. Condition and type of restorative work present.

7. Susceptibility of the person to disease.

DENTAL FLOSS

Although flossing requires more digital skills and is more time consuming than toothpick, there appears to be no alternative for using a floss or tape (a type of broader dental floss) in cleaning proximal surfaces when a normal healthy papilla fills the interdental space.

Uses of Dental Floss:

1. Removes plaque and debris adherent to the teeth, restorations, orthodontic appliances, fixed prosthesis and gingival in the interproximal embrasure.

2. Polishes the surfaces as it removes the plaque.

3. Massages the interdental papilla.

4. Helps to identify the presence of subgingival calculus deposits, overhanging restorations and interproximal carious lesions.

5. Maintains general oral hygiene and controls halitosis.

6. Reduces gingival bleeding.

Disadvantages of Dental Floss:

1. Not easy to perform especially in posterior areas.

2. Requires good manual dexterity.

3. Time consuming.

4. Risk of frequent shredding when passing through contact point.

5. Risk of tissue damage if improperly used.

Types of Dental Floss:

1. Twisted or nontwisted.

2. Bonded or nonbonded.

3. Waxed or unwaxed.

4. Thin or thick.

Unwaxed versus Waxed Floss. Studies have shown no difference in the effectiveness of unwaxed versus waxed dental floss. *Unwaxed dental floss* is usually recommended for individuals with normal tooth contacts because it slides through the contact areas easily. *Waxed dental floss* is recommended for individuals with tight proximal tooth contacts, moderate to heavy calculus deposits, crowded teeth or defective and overhanging restorations. It is preferred because of its ability to slide through tight contacts and resist fraying.

Dental tape or ribbon is a waxed dental floss that is wider and flatter than conventional dental floss. The flat-sided surface of dental tape is preferred by some, particularly when the surface area to be flossed is large.

Floss Threader. It is another device designed to assist individuals with flossing. In case of fixed partial dentures, floss cannot be passed through the interdental contacts because this is closed. A floss threader assists in

introducing floss into an area such as between an abutment tooth used for support of a fixed bridge and a pontic end of the floss threader is passed under the pontic or fixed partial denture connector from the facial aspect.

Super Floss. It is a type of floss that incorporates a rigid plastic portion that can be introduced under fixed bridge. Distal to rigid plastic portion is a spongy region that is ideal for plaque removal. The terminal portion of superfloss is similar to standard dental floss. The rigid portion is passed into the embrasure space between the retainer and the pontic and pulled through to the lingual aspect.

The spongy region is then used in apico-coronal stroke along the interproximal surfaces of the abutment teeth and along the intaglio surface of the pontic.

Indications:

1. Patient with physical disabilities.
2. Patient lacking manual dexterity.
3. Individuals with large hands.
4. Individuals with strong gag reflex.
5. Caregivers.
6. Type I embrasures.

Floss holder is a flossing aid. The majority of floss holders consist of a device with a handle with two prongs in Y shape or C shape. The floss is tightly secured between the two prongs. The patient holds the handle of the device to guide it during use.

Disadvantages:

1. More time consuming.
2. Unable to maintain tension of floss.
3. Must be rethreaded when the floss becomes soiled or frayed.
4. Need to set fulcrum to avoid floss cuts.

TOOTHPICKS

Toothpicks are usually made of softwood and have a triangular, round or rectangular shape. Triangular are said to be superior to the rest as they are ineffective on lingual aspect of proximal surfaces.

Indications:

1. Type II and Type III embrasures from facial aspect only.

2. Accessible furcations.

3. Small root concavities.

4. Interproximal open spaces.

Contraindications:

1. Type I embrasures.

2. Healthy gingiva.

Disadvantages:

1. Wearing down of papilla and marginal tissues from incorrect usage.

2. Wood ends may cause tissue trauma/cuts or abrasion.

3. Enamel abrasion from incorrect use.

4. Can force bacteria or debris into gingival attachment if used improperly.

5. May cause opening of the embrasure.

Technique. Inserted interproximally and activated with short back and forth strokes in between the teeth.

Disadvantages:

1. Different types may be needed to fit different open interproximal spaces.

2. Trauma to tooth or gingiva from sharp wire center of some.

SINGLE TUFT BRUSHES

Single tuft or end tuft brushes are small brushes with nylon bristles that are attached to a plastic handle. There are variations in the shape of the tufts and the width and length of the handles. Single tuft brushes are indicated

in open embrasure areas where there is little or no papilla. They are used by placing the brush in the interdental area. The brush is then jiggled in a small circular motion and/or use a sweeping motion away from the gingiva.

Indications. The single tuft brush is for patients who prefer a longer handle.

Toothpick Holder. It is also called as periodontal aid. It is an instrument designed to increase patient's application of the traditional toothpick by holding it securely at the proper angle. Indications:

1. Plaque removal along the gingival margin.
2. Type II and Type III embrasures from facial or lingual aspect.
3. Accessible furcations.
4. Concave surfaces in interproximal areas.
5. Fixed prosthetic and orthodontic appliances.
6. Sulcular cleansing in areas of shallow pocketing.
7. Application of fluoride, antimicrobials and desensitizing agents.

INTERPROXIMAL BRUSHES

They are also called as interdental brushes, proxa brush. They are available in various sizes and shapes. The most common are conical or tapered and designed to be inserted into a plastic or metal reusable handle that is angled to facilitate interproximal adaptation. Studies have shown that they are equal to or more effective than floss for plaque removal and for reducing gingival inflammation in Type II and Type III embrasures and exposed furcations areas.

Indications:

1. Type II and Type III embrasures.
2. Diastemas.
3. Exposed root furcations.
4. Orthodontic and fixed appliances.
5. Application of fluoride, antimicrobial or desensitizing agents.

6. Patients who are not able to change the refills of the interproximal brush.

7. Patients who cannot control the interproximal brush due to manual dexterity or disabilities.

8. Distal of posterior most teeth in arch.

WEDGE STIMULATOR

Wooden and plastic sticks or wedge stimulators reduce bleeding and inflammation when used to reduce plaque. They are wooden or plastic oral hygiene devices designed for interdental cleansing and stimulation. These devices are made of balsa, bass, birch, or linden wood or plastic. Some of the wood sticks are treated with xylitol. They are recommended for use only from the facial aspect, where the proximal surfaces are exposed to avoid traumatizing gingival tissue. Wooden and plastic sticks or wedge stimulators reduce bleeding and inflammation when used to reduce plaque. Most wedge stimulators are triangular. They are inserted interdentally, with the base of the triangle resting on the gingival side, the pointing occlusally or incisally and the sides of the triangle against the adjacent tooth surfaces. Such a placement of base of triangle against the tissues prevents damage such as gingival cuts and clefts, to the interdental papilla and gingival margins.

One of the most commonly used tools, toothpicks differ from the wooden triangular sticks in their shape and size and may not be as effective in plaque removal in open embrasures.

Indications:

1. Type II and Type III embrasures from facial aspect only.

2. Accessible furcations.

3. Application of fluoride, antimicrobial or desensitizing agents.

Disadvantages:

1. Wearing down of papilla and marginal tissues from incorrect use.

2. Enamel abrasion from incorrect use.

3. Splaying of wood ends may cause tissue trauma or abrasions.

4. Improper use can force bacteria or debris into gingival attachment leading to abscess formation.

LITERATURE

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WORKSHOP № 10
METHODS FOR DENTAL DEPOSITS REMOVAL

Test Questions:

1. Basic methods of tooth brushing.

2. Additional methods of brushing.

3. Toothbrushing techniques for manual mechanical, ionic, and electric toothbrushes.

TOOTHBRUSHING TECHNIQUES

According to the direction of brushing stroke:

i. Vertical (Leonard's method).

ii. Horizontal.

iii. Vibrating technique (Charter's, Stillman, and Bass).

iv. Circular technique (Fones method).

v. Physiological technique (Smiths method).

Requirement of a Satisfactory Method of Tooth Brushing:

1. The technique should clean all tooth surfaces especially the area of gingival crevice and the interdental region.

2. The movement of brush should not injure the soft or hard tissues. Certain methods, e.g. vertical and horizontal scrubbing methods can produce gingival recession and tooth abrasion.

3. The technique should be simple to use and easy to learn.

4. The method must be well organized so that, each part of the dentition is brushed in turn and no area overlooked.

THE BASS METHOD: SULCULAR BRUSHING

It is widely accepted and particularly useful in removing plaque not only at the gingival margin, but also subgingivally.

Indications:

1. For plaque removal adjacent to and directly beneath the gingival margin in all individuals.

2. Particularly useful in open interproximal areas, cervical areas beneath the height of contour of the enamel and exposed root surfaces.

3. Postperiodontal surgery.

Procedure. The head of the brush is positioned in an oblique direction towards the apex, in order to introduce the bristles into the gingival sulcus. The bristles are about 45 degrees to the axis of the teeth. The brush is pressed towards the gingival and moved with a small circular motion so that the bristles go into the crevice. 20 strokes are completed in the same position, three teeth at a time. For occlusal surface cleaning, bristles are pressed firmly into the pits and fissures and activate the brush into 20 short back and forth strokes.

To reach the lingual surface of anterior teeth, the brush is inserted vertically. The heel of the brush is pressed into the gingival sulci and proximal surface at a 45 degree angle to the long axis of the tooth. The brush is then activated.

STILLMAN'S METHOD

Indications. As the bristle ends are not directed into sulcus, this method can be recommended for individuals with progressive gingival recession.

Technique. This method was originally developed to provide gingival stimulation. The brush is positioned with bristles inclined at a 45 degree angle to the long axis of the tooth, with the bristles placed partly on the gingiva and partly on the cervical portion of the tooth. The strokes are activated in a short back and forth (vibratory) motion, with slight pressure to stimulate the gingiva.

Approximately 5 to 10 strokes are completed in each region, and the brush is moved to the next area. Brush placement is vertical on the anterior lingual surfaces and the heel of the brush is used.

CHARTER'S METHOD

Purpose and Indications:

1. Massage and stimulate marginal and interdental gingiva.

2. Cleaning of orthodontic appliances.

3. Cleaning following periodontal surgery.

4. Fixed prosthetic appliances.

5. Person with exposed root surfaces.

6. Cases with receded interdental papillae.

Procedure. In this technique the bristles are pointed towards the crown of the tooth rather than apically. The bristles are placed at the gingival margin and directed towards the occlusal surface at a 45 degree angle to the long axis of the tooth. A short back and forth motion is used for activation. The process is repeated in a sequence around the mouth until all areas are cleaned.

Disadvantages:

1. As brush ends do not engage the gingival sulcus, it does not remove subgingival bacterial accumulation.

2. High digital dexterity required.

3. In some areas such as lingual surface, the correct brush placement is impossible or difficult, therefore, modifications are required adding to complexity of the procedure.

CIRCULAR: THE FONES METHOD

This method may be recommended as an easy to learn technique for young children.

Indication. Young children with primary teeth; otherwise not recommended.

Technique. The brush is placed inside the mouth. With the teeth closed and brush tips contacting the gingiva over the last maxillary molar, bristles are activated in circular motion that sweeps from the maxillary gingiva to the mandibular gingiva.

Disadvantage. Possible damage to gingiva.

VERTICAL: LEONARD METHOD

Technique. With the teeth edge-to-edge, place the brush with the filaments against the teeth at right angles to the long axis of the teeth. The brush
is activated with mostly up and down strokes on the tooth surface. The upper and lower teeth are not brushed in the same series of stroke. This technique is usually not recommended.

PHYSIOLOGIC: SMITH'S METHOD

It was described by Smith. It was based on the principle that the tooth brush should follow the same physiologic pathway that food follows when it traverses over the tissues in a natural masticating act.

Technique. The toothbrush bristles are positioned at the incisal or occlusal surfaces and are swept towards the gingiva. The direction of the brushing motion from the occlusal to the gingiva was like duplicating the nature's self cleansing mechanism.

Scrub Brush Method. It is probably the most commonly used toothbrushing method. The procedure consists of vigorously combined horizontal, vertical and circular strokes with some vibratory motions for certain areas.

Disadvantages:

1. Transition to other technique becomes difficult so not recommended.

2. Can cause gingival recession.

3. Can lead to tooth abrasion.

No one manual toothbrush design appears superior for plaque removal. Toothbrush designs continue to be modified by competing manufacturers. New modifications include nylon multi-tufted round-ended bristles for improved efficacy, smallsized head for better access, designs to favor interproximal access and longer to enable a firm grip. However, the latter may increase application force, resulting in gingival irritation and recession. Although brush stroke movements vary (for example, roll, circular, scrub) and should concentrate on the cervical and interproximal areas where plaque is most detrimental, the individual's dexterity and thoroughness are more critical than technique or design in determining efficacy of plaque removal.

Recent well controlled studies report the new electric toothbrushes to be superior in plaque removal to manual toothbrushes but significant improvements in gingival health are yet to be shown. For small children, toothbrushing should be performed by an adult until the child is about 6 years, when increasing dexterity and cognition may permit supervised brushing until the child is capable of independent brushing.

Methods of Using Dental Floss:

1. Spool method.

2. Loop or circle method.

Spool Method. It is recommended for teenagers and adults who have acquired the required the level of neuromuscular coordination and mental maturity to use floss correctly.

Method. A piece of floss approximately 18 inch long is taken. The bulk of the floss length is lightly wound around the middle finger. The rest of the floss is similarly wound around the same finger of opposite hand. Space should be left between wraps to avoid cutting off circulation to the fingers. The last three fingers are clenched and both hands are moved apart, pulling the floss taut. The thumb and index finger of each hand are free. The floss is then secured with the index finger and thumb of each hand by grasping a length about 1 inch in each hand. The finger of opposite hand can wind the floss as it becomes soiled or frayed to permit access to an unused portion.

Loop Method. This method is particularly suited for children as well as adults with less nimble hands or handicaps such as poor muscular coordination or arthritis.

Method. A piece of 18 inch long is made into a circle and tied securely with three knots. All fingers except the thumbs are placed within the loop, so that fingers or thumbs will be about 1 inch apart. The floss is guided with the two index fingers for mandibular teeth and with two thumbs or one index finger and one thumb for maxillary teeth. As teeth are flossed, the loop is rotated so that, each proximal area receives unused floss for proper cleaning.

To be effective the floss should be pulled around the tooth curvature so that, close contact with tooth surface is made. Patients with tight contact areas need thin unwaxed floss that can be slipped easily between the contact areas, whereas in patients with crowded teeth, heavy calculus deposits, or defective and overhanging restorations, a bonded unwaxed floss or waxed floss is the dental floss of choice because they do not fray as easily as unwaxed floss. Dental tape is recommended when there is considerable interdental space resulting from gingival recession and bone loss.

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WORKSHOP № 11
MEANS FOR ORAL HYGIENE (TOOTH POWDERS, PASTES, GELS). THE CHOICE OF METHODS AND MEANS FOR INDIVIDUAL PATIENT HYGIENE

Test Questions:

1. Means for oral hygiene: prescription, safe requirements.

2. Toothpowders: basic components, effectiveness, indications and contraindications for use.

3. Hygienic toothpastes, gels: components, effectiveness, indications and contraindications for use.

4. Therapeutic and prophylactic anticaries components in hygiene products.

5. The choice of oral hygiene methods and means in different age groups.

DENTIFRICES

A dentifrice is usually used in combination with toothbrushing with the purpose of facilitating plaque removal and applying preventive and therapeutic agents to the tooth surfaces. A toothpaste is defined as a semi-aqueous material for removing naturally occurring deposits from teeth and is supposed to be used simultaneous with a toothbrush.

It is a substance used in conjunction with a toothbrush or interdental cleaner to facilitate bacterial plaque biofilm removal, or as a vehicle for transporting therapeutic or cosmetic agents to the tooth and its environment. A dentifrice is usually used in combination with toothbrushing with the purpose of facilitating plaque removal and applying preventive and therapeutic agents to the tooth surfaces.

The dentifrice can either be:

Cosmetic which cleans and removes material alba, plaque biofilm, food debris and stains from tooth surfaces and polishes.

Therapeutic which transports the drug substance to the tooth surface or the oral tissue. It is suppose to reduce some disease process in the mouth. Therapeutic effect is to reduce caries incidence, gingivitis, calculus formation, or tooth sensitivity.

Ingredients. Dentifrices were originally used for their cosmetic effect, i. e. for removal of extrinsic stains (end products of bacterial metabolism) present on the surface of tooth.

Abrasives. It is the largest component of dentifrice. The characteristic of an abrasive agent used in a dentifrice depends upon:

• Inherent hardness of an abrasive.

• Shape and size of the abrasive particle.

The effect of polishing agent (abrasive potential) used during the use of a dentifrice is also influenced by the brushing force applied, brushing technique, type of bristles(hardness), properties of abrasive, surface being polished (enamel or dentine). The harder the abrasive and sharper the particle, the more the wear of the tooth surfaces. Calcium carbonate (chalk) and calcium phosphates were the most commonly used abrasives previously. But these agents specially calcium reacted adversely with fluoride and due to their interaction fluoride ions were not available to react with the tooth enamel. Most fluoride dentifrices no longer use calcium carbonate and instead silicon oxides, insoluble sodium metaphosphate are being used now days. Abrasives often dull the tooth luster and to compensate this polishing agents like small size particle of aluminum, calcium, tin, magnesium are added to the dentifrice.

Humectant. The primary function of humectant in a dentifrice is to prevent loss of water or retain moisture, thus prevents hardening of the paste when it is exposed to air. It helps to maintain the consistency of the paste. The most commonly used agents are glycerol, sorbitol and propylene glycol. Both glycerol and sorbitol have a sweet taste and hence may also work as a sweetening agent.

Water. Water is one of the important ingredients. Deionized or distilled water is used.

Binding agent. The function of a binding agent is to prevent the separation of liquid and solid phases of a dentifrice and provide a stable suspension specially during storage. All binders are hydrophilic colloids which disperse or swell or absorb water to form a viscous material. Gum Arabic, gum karaya and gum tragacanth, Irish moss extracts and alginates (seaweed colloid) are natural products and are used as binders. Synthetically, prepared water-dispersible
derivatives of cellulose such as methyl cellulose and carboxymethyl cellulose are increasingly being used for economic reasons.

Detergents. These are the agents which clean the tooth surface. They lower the surface tension, penetrate and loosen surface deposit and emulsify and suspend the debris which is then removed from the tooth surface by the dentifrice. Natural soaps used earlier have been replaced by newer synthetic compounds. These compounds /detergents are soluble in water, function in acid or alkaline solution and does not form precipitates in hard water or saliva.

These are compatible with many cleaning and polishing agents. Detergents cause the foaming action of a dentifrice, which gives a pleasant sensation. The commonly used detergents are sodium lauryl sulfate, sodium N-lauryl sarcosinate, sodium cocomonoglyceride sulfonate. Of these sodium lauryl sulfate is the most commonly used as it has an excellent detergent property and also possesses some antibacterial effect.

Flavoring agent. The acceptability of toothpaste by a consumer is influenced by its taste, smell and consistency. For taste acceptance the flavor must be pleasant, provide an immediate taste sensation and the flavor should be long lasting. The important factor to be considered is that the selected flavor ingredient must be compatible with the other ingredients of the toothpaste and must remain unchanged during manufacture and storage of the paste. The commonly used essential flavoring oils are peppermint, spearmint, wintergreen, cinnamon, anise, clove, caraway, pimento, menthol and eucalyptus.

Preservative. Humectants and certain organic binders in a paste can permit bacterial or mold growth. Therefore, preservatives such as sodium benzoate, dichlorophene, formaldehyde or paraben are added in toothpaste.

Therapeutic agent. These are the agents which are added in a dentifrice to produce a beneficial effect upon the oral tissue. The most commonly used agent is fluoride to prevent dental caries.

Sweetening agents. Earlier honey and sugar was used as sweetening agent in the dentifrices. Nowadays synthetic sweeteners such as saccharin are used. Sorbitol and mannitol are also used which serve the role of sweetening agent as well as humectants.

Composition of a typical dentifrice.

Ingredients Percent:

Abrasive — 40–50.

Humectant — 20–30.

Water — 20–30.

Binding agent — 1–2.

Foaming agent (Detergent) — 1–3.

Flavoring agent — 1–2.

Preservative — 0.05–0.5.

Therapeutic agent — 0.5–2.

Sweetening agent — 0.

THERAPEUTIC DENTIFRICES

A therapeutic dentifrice is one that contains a drug substance that has been incorporated into the formulation in an effort to produce a beneficial effect upon the oral tissues.

The beneficial effect may include:

• Reduction and prevention of dental caries.

• Prevention and reversal of gingival diseases.

• To reduce hypersensitivity.

Various therapeutic products tried in the past include chlorophyll and antibiotics such as penicillin. Penicillin dentifrices were used experimentally but were not accepted and major deterrent for their routine use possible allergic sensitization, development of resistant strains of microorganism and over growth of unwanted microorganisms.

ANTICARIES

Fluorides. Fluoride containing dentifrices have been approved by American Dental Association (ADA) which provides protection against dental caries. Commercially available dentifrices contain sodium fluoride (NaF) 0.22 %, stannous fluoride (SnF_2) — 0.4 % or sodium monofluorophosphate (MFP) — 0.76 %. All dentifrices currently marketed in the US are formulated to contain either 1000 or 1100 ppm F, mostly in the form of NaF and MFP. There is
evidence of an improved anticaries effect with higher F concentrations for both MFP and NaF.

Calcium/Phosphate. Calcium and phosphate supplementation in a dentifrice will increase the concentration of these ions in the oral cavity. This has been reported to improve remineralization and increase fluoride uptake.

Sodium Bicarbonate. Several studies have shown that bicarbonate is one of the salivary components that potentially modify the formation of caries. It increases the pH in saliva, and in this way creates an unsuitable environment for the growth of aciduric bacteria. Sodium bicarbonate can also prevent caries by reducing enamel solubility and increase remineralization of enamel.

ANTI-PLAQUE AGENTS

Sodium Lauryl Sulfate (SLS). It has been shown that the enzymes glucosyltransferase and fructosyltransferase are incorporated in an active form into the pellicle; and by synthesizing glucan *in situ* from sucrose, can provide a surface for colonization by *Streptococcus mutans*. These enzymes can be inhibited by SLS, and hence retard the regrowth of plaque.

Triclosan. Triclosan is a synthetic nonionic chlorinated phenolic agent with antiseptic qualities. Triclosan has a broad-spectrum efficacy on gram-positive and most gram-negative bacteria. It is also effective against mycobacterium and strictly anaerobic bacteria, and against the spores and fungi of
the Candida species. The mechanism of its antiseptic action is by acting on the microbial cytoplasmic membrane, inducing leakage of cellular constituents and thereby causing lysis of the microorganisms. Inspite of its activity *in vitro*, clinical plaque studies have revealed only moderate levels of antiplaque activity.

Evidence has accumulated to suggest that triclosan in itself does not produce optimal plaque inhibitory effects without the addition of other chemicals which increase its antibacterial effect. Most commonly used are copolymer PVM/MA and zinc citrate. They enhance surface retention of triclosan.

ANTICALCULUS

These dentifrices are formulated to inhibit the development of new calculus and which contains, among other ingredients, either pyrophosphate or zinc. It has no effect on existing calculus.

Pyrophosphate. «Antitartar» toothpastes contain soluble pyrophosphates which prevent calcification of plaque. The percentage reduction in supragingival (but not subgingival) calculus is between 32 percent and 45 percent.

Pyrophosphate has recently been introduced in dentifrices to inhibit the formation of supragingival dental calculus. Pyrophosphate is added as tetrasodium pyrophosphate, tetrapotassium pyrophosphate or disodium pyrophosphate. It has been shown that pyrophosphate has high affinity to hydroxyapatite (HA) surfaces, probably by an interaction with Ca^{2+} in the hydration layer.
By interacting with HA and the enamel surface, pyrophosphate reduces their protein-binding capacity. It also has the ability to inhibit calcium phosphate formation. It is therefore conceivable that pyrophosphate introduced in the oral cavity through dentifrices may affect pellicle formation.

ANTIHYPERSENSITIVITY

The condition is also referred to as «dentine hypersensitivity». The sensitivity of dentine is caused by fluid-filled tubules in communication with the pulp. It is a sensation of pain affecting many individuals resulting mainly from thermal and/or tactile stimuli on exposed dentine.

Potassium Salts. Potassium ions (potassium nitrate) are thought to act by blocking action potential generation in intradental nerves. It is claimed that potassium salts in dental preparations increase the concentration of potassium ions around the pulpal nerves, and thereby depolarizes the nerve. This can inhibit a nerve response from different stimuli.

Other Agents. Other commonly used agents include strontium chloride, sodium citrate, etc.

WHITENING AGENTS

Whitening toothpastes simply remove surface stains with abrasives or special chemical or polishing agents, or prevent stain formation.

Abrasives. Some whitening toothpastes contain coarse abrasives that can damage the dental tissue. An abrasive is required for the effective removal of a discolored pellicle. Abrasives provide a significant whitening benefit,

particularly on smooth surfaces, but are of limited use for areas along the gum line and interproximally.

Dimethicones. They cause a smooth surface on the tooth that prevents stain formation. Dimethicones are versatile substances that ranges from low molecular weight polydimethylsiloxane fluids to high molecular weight polymers that are gum-like in nature.

Papain. Papain is a sulfhydryl protease consisting of a single polypeptide chain, extracted from the *Carica papaya* plant. It is used in toothpastes as nonabrasive whitening agent.

Sodium Bicarbonate. It is claimed that dentifrices containing high concentrations of sodium bicarbonate are more effective in removing intrinsic tooth stain than those not containing sodium bicarbonate.

Factors Affecting Dentifrice Effectiveness. In addition to the inherent properties of a fluoride dentifrice product, biological and behavioral factors can modify its anticaries effectiveness. All of these factors interplay in what can be described as the «application» phase (the initial interaction of relatively high concentrations of fluoride with the tooth surface and plaque), and the «retention» phase (the fluoride remaining in the mouth after brushing that is retained in saliva, plaque and plaque fluid, the tooth surface, and oral soft tissue reservoirs). Behavioral factors include the frequency of dentifrice use, length of brushing, rinsing practices after brushing, the time of day that dentifrice is applied, and amount of dentifrice applied to the brush. It is well established that the frequency of use has a major influence on effectiveness. Bushing twice per day or more has a greater preventive effect than once per day. Length of the brushing time (application phase) determines how long the relatively high fluoride concentration in the dentifrice slurry stays in contact with the teeth and plaque, allowing fluoride uptake to take place. The higher the fluoride concentration, the greater the driving force for fluoride diffusion through plaque toward the tooth surface. Rinsing behaviors after toothbrushing affect the amount of fluoride retained in the mouth and have been reported to affect caries experience. Physiologic (biological) factors, mainly salivary flow rate during and after fluoride application influence the rate of fluoride clearance. Bedtime use of fluoride dentifrice results in longer fluoride retention than daytime application due to greatly decrease salivary flow during sleep. The amount of fluoride applied to the toothbrush (dose) is not as important as

the concentration of available fluoride in a dentifrice. Reduced fluoride concentration dentifrices are not as effective as regular concentration products. The fluoride dose is, however, important in regard to enamel fluorosis in children under six years of age because of dentifrice ingestion. For this reason, reducing the amount of fluoride applied is a better strategy than lowering the dose of products intended for use by children.

Mouthrinses (Mouthwash). A mouthwash is defined as a nonsterile aqueous solution used mostly for its deodorant, refreshing or antiseptic effect. Mouthwashes or rinses are designed to reduce oral bacteria, remove food particles, temporary reduce bad breathe and provide a pleasant taste. Many types of mouthrinse active ingredients have been evaluated for their plaque reducing effectiveness and ability to reduce mutans streptococci, including chlorhexidine, essential oils, triclosan, cetylpyridinium chloride, sanquinarin, sodium dodecyl sulphate, and various metal ions (tin, zinc, copper).

Mouthrinses are generally classified by FDA as either *cosmetic* or *therapeutic* or a *combination of the two*. The cosmetic mouthrinses are over-the-counter products that are mainly intended as mouth fresheners. Therapeutic rinses are available on prescription or over-the counter products that have an added active ingredient and are marketed as antiplaque/antigingivitis and anticaries drug products.

Cosmetic rinses are commercial over-the-counter products that help remove oral debris before or after brushing, temporary suppress bad breath, diminish bacteria in the mouth and refresh the mouth with a pleasant taste.

Therapeutic rinses often have the benefits of their cosmetic counterparts, but also contain an added active ingredient, for example. fluoride or chlorhexidine, that help protect against some oral diseases.

The amount of the different components in mouthwashes varies from product to product.

Some practically have the same composition as toothpastes, although they do not contain abrasives. Distinct from toothpastes most mouthrinses contain alcohol, as a preservative and a semiactive ingredient. The amount of alcohol is usually ranging from 18 to 26 percent.

Mouthrinse formulations are generally much simpler than dentifrices, and compatibility problems are not as large an issue as they are with dentifrice products.

The oldest and simplest used mouthrinse has been a dilute saline solution.

Ideal properties of oral rinses include the following:

– safety;

– access to bacteria even in difficult areas;

– palatability;

– low-cost;

– high solubility within the formulation;

– effective antibacterial impact;

– broad spectrum preferably;

– selectivity;

– adequate bioavailability (plaque penetration and reactivity);

– specificity with regard to oral bacteria;

– minimal side effects;

– ability to reach and provide adequate retention in sites of disease initiation;

– stability in storage.

Ingredients

Humectant: for example, sorbitol and glycerin to prevent drying.

Surfactant: helps to keep ingredients in solution.

Alcohol: to enhance antibacterial activity and taste. Also to help keep flavoring agents in solution.

Antibacterial agents: the most commonly used antimicrobial agent is the quaternary ammonium compounds such as cetylpyridinium chloride,

benzethonium chloride and povidineiodine, sodium lauryl sulphate, zinc citrate trihydrate, triclosan, metal salts.

Sweetening agents: saccharin.

Flavoring agents: Spearmint, peppermint, eucalyptus and menthol are often used as flavoring agents mouthwashes. The flavoring agents are solubilized and dispersed through liquid via the detergent.

Therapeutic Rinses. *Fluoride containing*: Sodium fluoride (NaF) mouthrinse has been used as 0.2 percent for weekly rinse and 0.05 percent for daily rinsing. It is the most widely used fluoride rinse because of its low cost, convenience in handling and pleasant taste.

Chlorhexidine Rinses. Chlorhexidine digluconate, useful in decreasing gingivitis and plaque buildup, is an active ingredient in certain ADA-approved commercial mouthrinses. It is one of two mouthrinse shown to reduce gingivitis in long-term clinical trials and appears to be the most effective antiplaque and antigingivitis agent known today.

But since the effect of chlorhexidine is influenced by anionic tensides such as sodium lauryl sulphate, when using a toothpaste containing sodium lauryl sulphate you should wait for at least 30 minutes between brushing and rinsing with a CHX mouthrinse.

CHX 0.2 percent is suitable as supportive measure during treatment of gingivitis and periodontitis, but it should not be used for longer than two weeks. After this, however, it is important to restore healthy oral flora.

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WORKSHOP № 12
SALIVA: CARIES PROTECTIVE PROPERTIES, THEIR DIAGNOSIS AND CORRECTION

Test Questions:

1. Whole saliva: composition, volume, distribution and movement in the oral cavity.

2. Mechanisms of production, composition and structure of the saliva. Characteristics of stimulated saliva and resting saliva.

3. Local functions of saliva (protection and regeneration of tissues, lubrication, digestion, infection control).

4. Local functions of saliva: clearance and neutralization of acids, (re) mineralization of enamel and cement.

5. Characteristics of saliva properties: salivation rate, viscosity, pH, buffer capacity (formation mechanisms, methods of assessment, evaluation of tests results).

6. Causes and mechanisms of hyposalivation. Principles of causal and pathogenetic treatment.

7. Symptomatic treatment of xerostomia and hypo salivation. Features of oral care and dental caries prevention organization.

8. Sialorrhea: causes, manifestations, care measures.

Saliva is secreted by three paired masses of cells — the submaxillary, sublingual, and parotid glands. Small accessory glands are also scattered over the oral mucous membranes. Each of these has its own duct.

The salivary glands are under the control of the autonomic (involuntary) nervous system, receiving fibers from both its parasympathetic and sympathetic divisions. Stimulation of either the parasympathetic (chorda tympani) fibers or the sympathetic fibers to the submaxillary or sublingual gland causes a secretion of saliva. The secretion resulting from parasympathetic stimulation is profuse and watery in most animals. Sympathetic stimulation, however, causes

a scanty secretion of a thick, mucinous juice. Stimulation of the parasympathetic fibers to the parotid gland causes a profuse, watery secretion, but stimulation of the sympathetic fibers causes no secretion.

Composition: varies from person to person. Saliva is dilute fluid; over 99 percent being made up of water.

1. *Proteins*: they include enzymes, immunoglobins and other antibacterial factors, mucous glycoproteins and certain polypeptides.

2. *Enzymes*: α-Amylase.

3. Immunoglobulins — secretary IgA.

4. Antibacterial proteins — Lysozyme, Lactoferrin, Sialoperoxidase.

5. Glycoproteins.

6. Polypeptides — Statherin, Sialin (helps to regulate pH of plaque).

7. Other Organic Compounds:

– Free Amino Acids;

– Urea (it is hydrolysed by many bacteria with release of Ammonia, leading to rise in pH);

– Glucose.

8. *In Organic Constituents*:

– Major Ions (Sodium, Potassium, and Chloride and Bicarbonate) contribute to osmolarity of saliva.

– *Bicarbonates:* Principal buffer in saliva.

– *Thiocyanate:* Has antibacterial action.

– *Fluoride:* Has anticaries action.

Saliva: it has a critical role to play in the development of caries or its prevention. Saliva provides calcium, phosphate, proteins, lipids and antibacterial substances and buffers. Saliva buffering can reverse the low pH in plaque.

Buffering and neutralization: pH of saliva depends on the bicarbonate concentration. Saliva is alkaline and is an effective buffer system. These properties protect the oral tissues against acids and plaque. After eating a sugary food if saliva is stimulated by chewing substances such as wax or sugar free chewing gum, the drop in pH in plaque which would have occurred is reduced or even eliminated. This salivary neutralization and buffering effect markedly reduces the cariogenic potential of foods.

Quantity: rate of flow of saliva may be an additional factor which helps contribute to caries susceptibility or caries resistance. Mild increase or decrease in flow may be of little significance, near total reduction in salivary flow adversely affects dental caries. There is an inverse relation between salivary flow and dental caries.

Many features of saliva affect the risk of dental caries like:

• Low buffering capacity (as acids are not neutralized).

• Low quantity.

• High S. mutans and Lactobacillus count.

• Xerostomia is a known predisposing factor.

XEROSTOMIA

Xerostomia is defined as a subjective complaint of dry mouth that may result from a decrease in the production of saliva. To assess the risk for caries involvement due to xerostomia, the clinical evidence of hyposalivation must be identified. Dry lips, dryness of buccal mucosa, absence of saliva in response to gland palpation, and a high number of decayed, missing, or filled teeth have been cited as an easily assessed set of clinical parameters for identifying most patients with salivary gland dysfunction. A reduction in the salivary flow may be temporary or permanent. When the quantity is only moderately reduced, the oral structures may appear normal. A pronounced reduction or complete absence of saliva, however, results in an acidic environment with rampant caries. In addition to the rapid destruction of the teeth, there may be dryness and cracking of the lips, with assuring at the corners of the mouth, burning and soreness of the mucous membranes, crusting of the tongue and palate, and sometimes paresthesia of the tongue or mucous membrane.

XEROSTOMIA AND DENTAL CARIES

A major complication of xerostomia is the promotion of dental caries (fig. 2). This process is accelerated owing to a reduction in oral irrigation and an inability to clear foods from the oral cavity rapidly, particularly if proteins and electrolytes that inhibit cariogenic microorganisms and buffer oral acids, respectively, are diminished. The development of rampant caries, particularly at the cervical area, has been observed within a few weeks after radiation therapy to the head and neck.

CAUSES OF XEROSTOMIA

– Primary Sjögren's syndrome.

– Secondary Sjögren's syndrome.

– Surgical removal of glands due to neoplasm.

– **Connective tissue disease:**

• Rheumatoid arthritis.

• Systemic lupus erythematosus.

• Systemic sclerosis.

• Mixed connective tissue disease.

– **Other conditions:**

• Radiation therapy.

• Primary biliary cirrhosis.

• Vasculitis.

• Chronic active hepatitis.

• HIV.

• AIDS.

• Bone marrow transplantation.

• Graft-vs-Host disease.

• Renal dialysis.

• Anxiety or depression.

– **Drugs that decrease salivary flow:**

• Anticholinergics.

• Antihistamines.

• Antianxiety.

• Diuretics.

• Antidepressants.

• Anticonvulsants.

• Narcotics.

Determination of Salivary Flow. If a patient has no known existing conditions that may cause hyposalivation and if the clinician notices a small pool of saliva in the floor of the mouth during oral examination, it is not unreasonable to assume that the patient has adequate salivary quantity and flow — Little information is available about salivary flow rate in children, but Crossner reported that in children 5 to 15 years of age, the rate of mixed whole stimulated saliva increases with age, and boys have consistently higher rates than girls. If inadequate salivary flow is known to exist or is suspected, measurement of salivary flow can provide a baseline useful for comparing with later measurements after implementation of adjunctive therapy.

To evaluate the adequacy of salivary flow, Zunt recommends establishing the unstimulated salivary flow (USF rate. The USF rate is measured after a period of 1 hour without eating, drinking, chewing gum, or brushing the teeth. Sitting in the «coachman» position, on the edge or the dental chair, the patient passively drools into a funnel inserted into a graduated cylinder for 5 minutes. The eyes should remain open except for blinking during the 5-minute collection period. The head and neck should be bent, and the arms should rest comfortably on the thighs or knees. The volume of saliva collected in the cylinder after 5 minutes is divided by 5 to determine the USF. A USF rate of less than 0.1 mL per minute is diagnostic of salivary gland hypofunction. If the USF rate is less

than 0.1 mL per minute, the next step is to measure the stimulated salivary flow (SSF). The patient should chew unflavored paraffin for 45 chews or 1 minute and expectorate into a funnel inserted into a graduated cylinder. The SSF rate should be 1 to 2 mL per minute; less than 0.5 mL per minute is scored as an abnormal rate. A convenient alternative method for measuring USF is the modified Schirmer technique, which uses a calibrated paper test strip to collect saliva in the floor of the mouth.

In patients who are known or suspected to have salivary deficiency, it is not unusual to find a salivary flow ranging from slightly below normal to practically dry mouth. If there is a deficiency of saliva or a dry mouth, the cause should be sought. Sometimes the cause is readily determinable; sometimes it is obscure. An emotional disturbance should not be overlooked as a cause in a patient of any age. Psychotherapy may be helpful in these cases. If the cause cannot readily be determined, perhaps it should be assumed that the sparse flow is related to inadequacies in the diet, particularly a vitamin deficiency or excessive sugar consumption to die exclusion of needed foods. Monthly quantitative analyses of the saliva should be performed to determine whether dietary improvement is accompanied by an increased flow.

If the salivary glands have not undergone degenerate or metaplastic change and if the nerve pathways between the central nervous system and the salivary glands are still intact, salivary stimulants may be recommended. If dryness of the mouth is attributable to dehydration, increased fluid intake should be recommended.

The use of gustatory stimulants (sugar-free candy) or masticatory stimulants (xylitol gum) has been suggested as an adjunct to encourage salivation. Prescription sialagogue medications, also known as secretagogues, such as pilocarpine and cevimeline may be of benefit in improving the salivary flow rate in patients with Sjogren syndrome or with radiation damage to salivary glands. The use sialagogue medications has not been studied in pediatric populations, but these agents are considered safe for most adult patients and have been used successfully in older children. The use of salivary substitutes been suggested by Shannon and colleagues as helpful in preventing soft tissue

problems associated with dry mouth. Saliva substitutes, as well as fluoride and chlorhexidine rinses, are also reported to enhance reminalization and promote resistance to demineralization tooth surfaces, and may help prevent radiation-induced caries.

Viscosity of Saliva. It has long been suggested that the viscosity of saliva is related to the rate of dental decay. Both thick, ropy saliva thin, watery saliva have been blamed for rampant dental caries. Previous work has shown a statistically significant direct relationship between the viscosity of saliva the number of decayed, missing, and filled teeth. This relationship held true for all members of the observation group, regardless of age. Patients with thick, ropy saliva
invariably had poor oral hygiene. The teeth were covered with stain or plaque, and the rate of dental caries ranged from greater than average to rampant. No evidence exists that viscosity changes with age under normal conditions. This property of the saliva is governed not only by the particular set of glands stimulated but also by the type of nervous stimulation and the amount of mucin (glycoprotein) present. Children who consume excessive amounts of carbohydrates often have not only a sparse flow but also viscous saliva. Even minimal doses of some antihistaminic drugs result in a greatly increased viscosity of
saliva in some persons.

There are apparently only a limited number of ways to alter the viscosity of saliva. Reduction of refined sugar take may be effective in some patients. Although relatively little information specific to salivary function, flow, and viscosity in children is available, an excellent review article by Leone and Oppenheim provides much additional information regarding the relationship between saliva and dental caries (fig. 3).

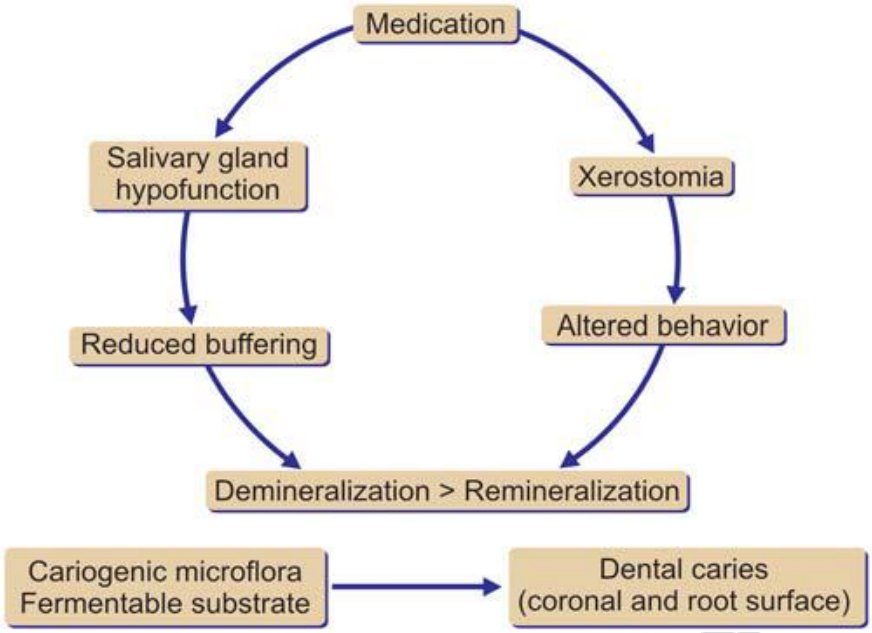

Fig. 3. Theoretical model of the relationship between medication, salivary gland hypofunction, xerostomia and dental caries

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WORKSHOP № 13
SYSTEMIC USE OF FLUORIDE FOR DENTAL CARIES PREVENTION

Test Questions:

1. Sources of fluoride exposure. Exchange of fluoride in the body.
2. Dose-dependent biological effects of fluoride. Systemic use of fluoride: mechanisms of influence on caries initiation and development.
3. The concept of «optimal» level of fluoride exposure.
4. Markers of the fluoride exposure. Control methods.
5. Fluoridation of communal water: compound of fluoride, a fluoridation technology, safety, and efficiency, limitations.

6. Programs with fluoridated milk
7. Programs with fluoridated salt.
8. Prescription of sodium fluoride supplements in tablets.

In biology, fluoride is usually classified as a trace element and belongs to the halogen group (fluorine, chlorine, iodine, and bromine). In biologic materials, the concentration of fluoride is generally as low as a few parts per million (ppm). However, fluorides occur in the environment at far higher concentrations than do so-called trace elements. Fluorine is the world's 13th most abundant element and constitutes 0.08 % of the Earth crust. It has the highest electronegativity of all elements.

Fluoride enters the atmosphere by volcanic action. It is returned to the earth's surface by deposition as dust, rain, snow, or fog. Fluoride enters the hydrosphere by leaching from soil and minerals into groundwater and by entry with surface water. Fluoride enters vegetation by processes such as uptake from soil and water and absorption of gaseous fluorides from air. It returns to the soil by plant waste or may enter the food chain and be returned as animal or human waste. Directly or indirectly, fluoride will also enter these pathways via different industrial processes and products.

Because of the small radius of the fluorine atom, its effective surface change is greater than that of any other element. As a consequence, fluorine is the most electronegative and reactive of all the elements. Because it reacts promptly with its environment, it rarely occurs in the free or elemental state in nature and occurs most frequently in the form of inorganic fluoride compounds. Fluorides reach their highest concentration in siliceous rocks, alkaline rocks, geothermal waters, hot springs, and volcanic fumaroles and gases.

There are about 150 known fluoride-containing minerals, of which fluorspar (fluorite (CaF) — 49 % F), fluorapatite ($Ca_{10}F_2(PO_4)_6$; 6.3 % F), and cryolite (N_3AlF_6; 54 % F) are the most important Fluorspar and fluorapatite (FA) are widespread in many countries.

Concentrations of fluoride in groundwater are influenced by such factors as availability and solubility of fluoride-containing minerals, porosity of the rocks or soils through which water passes pH, temperature, and the presence of other elements such as calcium, aluminum, and iron that may complex with fluoride. Normally the fluoride concentration in groundwater is

limited to 0.2 to 2.0 ppm, but in the United States, for example, fluoride concentrations greater than 60 ppm have been reported.

By contrast, most surface water contains less than 0.1 ppm of fluoride. In rivers, the concentration may range from 0.1 to 1.0 ppm. Seawater contains 1.2 to 1.4 ppm of fluoride.

Intake of fluoride is mainly derived from drinking water and beverages. It is estimated that about 60 % to 65 % comes from such sources in regions with fluoride levels of less than 0.3 mg/L in the drinking water and about 75 % to 80 % in regions with higher fluoride concentrations. Researchers have estimated the average fluoride intake by adults from the following dietary sources:

1. Water and nondairy beverages: 60 % to 80 %.

2. Grain and cereal products: 6 % to 8 %.

3. Meat, fish, and poultry: 5 % to 7 %.

4. All other foods: 10 % to 14 %.

Mineral water may contain 1.8 to 5.8 mg of fluoride per liter. Tea leaves are a particularly rich source of fluoride, most of which is rapidly released into tea infusions, within 5 to 10 minutes. Fluoride concentrations of brewed tea commonly range from 0.5 to 4.0 ppm (Duckworth and Duckworth, 1978). As would be anticipated, fluoride concentrations in tea made with fluoridated water are somewhat greater than are those found in tea brewed from water with low fluoride content.

Although the fluoride content of most meat, fish, and poultry products is quite modest, it is extremely high in a few items; in canned sardines, the content may be as high as 16 mg/kg. The fluoride content of mechanically deboned meat products is high because of the presence of bone particles.

Use of fluoridated water in commercial food preparation slightly increases the fluoride content of canned fruits, vegetables, soups, and stews, but overall such foods do not contribute large amounts of fluoride to the diet.

Fluoride goes from the air through the lungs, and from food and water — to the gastrointestinal tract and then into the blood and to all organs and tissues.

Part of the incoming fluoride (about 50 % in children, 45 % in adolescents 16–19 years old and 40 % in adults) remains in the body and selectively accumulates in the mineralized (bones and teeth) and cover (skin, hair, nails) tissues.

Fluoride excretion from the body is partly organized by the salivary and sweat glands (up to 1 %), but the main job of the kidneys excretion perform fluoride appears in the urine within a few minutes after receiving per os, the period of its half-life is 5 hours, by the end of the day fluoride excretion in the urine practically complete.

Accordingly, the vital markers of fluoride ingestion can be saliva, blood and urine, in the previous weeks — nails, in recent weeks and months — hair, in a more or less distant past — teeth.

Fluoride intake from diet (including drinking water and beverages with fluoride levels of less than 6 mg/L) and recommended use of fluoride — containing dental products such as toothpastes, mouthrinses, lozenges, and chewing gums will normally have no adverse effect on general health in young adults and adults. However, in children up to the age of 6 years, a high intake of fluoride will result in visible fluorosis of the teeth. The maturation phase of the maxillary incisors, when susceptibility to fluorosis is greatest, occurs when an individual is 22 to 26 months of age (Evans and Stam, 1991). To prevent the development of visible and esthetically disturbing fluorosis, fluoride intake in infants and preschool children should be limited and controlled.

The intake of fluoride associated with development of enamel fluorosis of the permanent teeth has been estimated to range from 40 to 100 μg/kg per day.

Professor T. Marthaler from Switzerland suggested how to calculate the upper and lower levels of fluoride ingestion. The calculation is based on data of the daily intake of fluoride by the average inhabitant living in the «optimal» region — an adult male which weight is 65 kg and energy costs 12,6 MJ/per day.

Recommendations of World Health Organization: 1,5 ÷ 4,0 mgF/per day. In recent years are recommended more «conservative» levels of fluoride intake: from 1,45 mgF/daily (corresponds to 0,6 mg/L in drinking water) till 2,90 mgF/daily (corresponds to 2,0 mg/L in drinking water).

The detection of fluoride consumption can be carried out by asking the information about ingestion of fluoride or by indicators of accumulation and excretion of fluoride. Currently the specialists determine the daily intake of fluoride by its urine excretion.

The only demonstrated positive impact of fluoride on human health is its contribution to prevention of dental caries (infection of teeth enamel). Hydroxyapatite in teeth enamel is made up of calcium, magnesium, and phosphate compounds and is susceptible to decay induced by acid-producing bacteria. Fluoride interacts with hydroxyapatite to form fluoroapatite, which is less susceptible to erosion by acid-producing oral bacteria. About 50 % of ingested fluoride is absorbed in the bones and teeth while the rest is excreted in urine. Most of the ingested fluorides reach the teeth via saliva, whose fluoride content varies from less than 0.01 to 0.05 ppm. Fluoride absorption in bones and teeth decreases with increasing age. It is widely accepted that fluoride only helps prevent dental decay by topical means — by direct action on the tooth enamel predominantly after eruption and dental plaque. However, it is important
to note that while fluoride contributes to the remineralisation process in the enamel of the tooth surface this is not dependent on fluoride, and that fluoride's anticaries effect is critically dependent on calcium and magnesium content of teeth enamel. Among young individuals with low calcium and magnesium in teeth enamel (usually due to undernutrition), fluoride ingestion and contact with teeth present histologically as hypo-calcification and/or hypoplasia, which may paradoxically make such individuals more vulnerable to dental caries. Fluoride has also been shown to inhibit cariogenic bacteria. This is postulated to occur mainly through inhibition of enzyme-mediated glycolysis in cariogenic microorganisms such as Streptococcus mutans. Fluoride is thought to adversely affect polysaccharide metabolism in bacterial cells, reduce the ability of such cells to maintain pH homeostasis, and inhibit encholase as well as other ATPase enzyme systems.

PREERUPTIVE EFFECTS OF FLUORIDE

Positive effects. As discussed earlier, the caries-inhibiting effect of fluoride is predominantly (almost totally) posteruptive. Preeruptive effects are very limited. However, some positive effects can be described. These would be most beneficial during eruption of the molars, a critical period for the initiation of fissure caries (Carvalho et al, 1989). A pre-eruptive effect of fluoride exists in reducing caries levels in pit and fissure surfaces of permanent teeth and fluoride concentrated in plaque and saliva inhibits the demineralisation of sound enamel and enhances the remineralisation of demineralised enamel.

In teeth, as in all the mineralized tissues, fluoride levels tend to be greatest at the surface because this region is closest to the tissue fluid that supplies the fluoride. Therefore, preeruptive fluoride accumulation is highest in the pulpal aspect of the dentin and the outer surface of the enamel. A much higher total fluoride concentration is found in the dentin because of an endogenous fluoride supply obtained from the vessels of the pulp. The outer surface
of the enamel will receive a “topical” supply of fluoride from the surrounding follicular fluid, explaining why fluoride concentrations decrease from the inner surface of the dentin and the outer surface of the enamel.

The concentration of fluoride is also higher in those parts of the enamel that are the first to develop and mature, ie, the incisal edges and the occlusal surfaces of the molars and premolars. These preemptive effects of fluoride may reduce susceptibility to the initiation of caries in the molar fissures during eruption and possibly around the approximal contact surfaces before the second maturation is completed after eruption.

In the enamel, the dentin, and the root cementum, some fluoride is incorporated within the interior of the mineral crystallites as an integral part of the crystal lattice. However, fluoride may also be more superficially located, perhaps absorbed on crystal surfaces or loosely entrapped in the hydration shells of the mineral crystallites. Most of the fluoride ions that enter the apatite lattice probably replace an OH~ ion or at least occupy an equivalent space. It has also been suggested that fluoride may replace the larger PO_4^{3-} ions and even substitute for CO_3^{2-} or HCO_3^{-} ions present in the mineral. Most of the fluoride buried within the crystallites will have been acquired during the period of crystal growth, a process sometimes known as accretion.

Being built into the crystal as it forms, such fluoride is quickly buried and will remain locked in the lattice interior for as long as the crystal exists. The incorporation of fluoride can significantly alter the properties of mineralized tissues because the inclusion of any extraneous element in a crystalline lattice will alter its reactivity. If it occupies a position normally occupied by a hydroxyl group, fluoride can greatly increase stability of the lattice. Fluoridated apatite lattices are more crystalline, more stable, and, therefore, less soluble in acid.

A decrease in carbonate content is generally found in highly fluoridated tooth mineral. This is probably due to direct substitution of carbonate by fluoride: The enamel will be more acid resistant because enamel with relatively high carbonate content is much more soluble. Fluoride will also reduce the citrate content of the enamel, which may increase initial acid resistance.

On the other hand, more superficially located fluoride may have relatively little effect on the behavior of the crystalline lattice. However, it can dramatically affect fluid-crystal equilibrium, which involves the interaction between ions at crystal surfaces and those in solution.

Negative effects: fluorosis. By far the best-known preeruptive effect of excessive fluoride intake is fluorosis, first described by Black and McKay (1916) as «mottled enamel». They suggested that it could be related to the water supply in the endemic areas. When it was subsequently shown in humans as well as in experimental animals that mottled enamel was an effect of fluoride on enamel formation and maturation, the condition was termed enamel fluorosis.

Characteristics of acute fluoride toxicity. Toxic signs are alarmingly rapid after ingestion of large amounts of fluoride. In nearly all cases of fluoride poisoning, the victims experience nausea, vomiting, and abdominal pain within minutes of ingestion. There may or may not be a variety of nonspecific symptoms, such as excessive salivation; tearing; mucous discharges from the nose and mouth; diarrhea; headache; cold, wet skin; or convulsions. As the episode progresses, generalized weakness, carpopedal spasms, or spasm of the extremities and tetany often develop.

These myopathologic signs are accompanied by declining plasma calcium concentrations, which may fall to extraordinarily low values and rising plasma potassium levels, which indicate a generalized toxic effect on cell membrane function. The pulse may be thready or not detectable. Blood pressure often falls precipitously to dangerously low levels. Respiratory acidosis, which diminishes the pH gradient across most cell membranes and results in a net migration of fluoride from extracellular fluid into the intracellular fluids, develops as the respiratory center is depressed. Cardiac arrhythmias may develop in association with the hypocalcemia and hyperkalemia. Extreme disorientation or coma usually precedes death, which may occur within the first few hours of fluoride ingestion.

The immediate treatment of acute fluoride toxicity should be aimed at reducing the amount of fluoride available for absorption from the gastrointestinal tract: Vomiting should be induced by administration of an emetic, such as ipecac. This should be followed by the oral administration of 1 % calcium chloride or calcium gluconate. If these solutions are not available, then as much milk as can be ingested should be given.

The hospital emergency department should be informed that a case of fluoride poisoning is in progress while these procedures are being carried out. The patient should be transported to the hospital at the earliest possible time. Vomiting should not be induced if the victim has no gag reflex or while the patient is unconscious or experiencing convulsions because of the danger of aspiration. In these cases, a cuffed endotracheal tube should be inserted and gastric lavage should be performed with a solution containing calcium or activated charcoal. At the hospital, specific routines should be available for medical treatment, depending on the severity of the signs and the symptoms (for details on fluoride toxicology, see Whitford, 1996).

It is essential that the fluoride concentrations in dental products be known to the persons who use them. It is even more important to know the amounts of fluoride that are contained in the unit packages (bottles or tubes) as well as the amounts involved during routine usage and how these amounts relate to the probable toxic dose (PTD). The fluoride concentrations in toothpastes are approximately 1,000 to 1,500 ppm. In some European countries, toothpastes for

young children contain lower fluoride levels (because of concerns about dental fluorosis); in others, fluoride levels in toothpaste range up to 2,500 ppm.

The vehicle of fluoride may be drinking or mineral water, drops or tablets, salt or milk.

Water fluoridation was the first experience of preeruptive fluoride prevention. It began in 1945 in the United States.

The recommended water fluoride concentration in temperate climates such as the United States is 0.7 to 1.2 mg/L; in warm to hot subtropical and tropical regions, only 0.5 to 0.7 mg/L is recommended to prevent the development of esthetically unacceptable fluorosis, in cold regions — 1.5 mg/L.Organization of the program at the community level is possible if there is public water system and a positive attitude towards the program of citizens and/or authorities.

Current estimates suggest that approximately 370 million people in 27 countries consume fluoridated water, with an additional 50 million consuming water in which fluoride is naturally occurring.

Positive effect of water fluoridation:

– technical availability (requires only one installation for water fluoridation at the municipal network);

– relative cheapness ($ 0.2/person/year);

– ability to «automatic» improvement of the dental health of the population, regardless of its level of preventive motivation.

Results from early studies of fluoridated water showed caries reductions of about 50 % in the permanent dentition and 40 % in the primary dentition, compared to control areas. Significant reductions in root caries were also observed.

Disadvantages of municipal water fluoridation:

– inability accurate dosing of fluoride due to individual differences in the amounts of consumption of tap water used for drinking and cooking;

– infringement of freedom of choice for the individual members of the population;

– undesirable introduction of fluoride in the processes, accelerate corrosion of metallic pipes, sewage pollution;

– negative impact on aquatic organisms (hydrobionts).

The dentist and pediatricians should remember about the content of fluoride in water for diluting of milk formula.

Based on the success of water fluoridation, a gynecologist started adding of fluoride to salt. The sale of fluoridated salt began in 1956 in the Swiss Canton of Zurich, and several other cantons followed suit. Studies initiated in the early seventies showed that fluoride, when added to salt, inhibits dental caries. The addition of fluoride to salt for human consumption was officially authorized in 1980-82. In Switzerland 85 % of domestic salt consumed is fluoridated and 67 % in Germany. Salt fluoridation schemes are reaching more than one hundred million in Mexico, Colombia, Peru and Cuba. The cost of salt fluoridation is very low, within 0.02 and 0.05 € per year and capita. Children and adults of the low socio-economic strata tend to have substantially more untreated caries than higher strata. Salt fluoridation is by far the cheapest method for improving oral health.

Salt fluoridation has cariostatic potential like water fluoridation (caries reductions up to 50 %). In Europe, meaningful percentages of users have been attained only in Germany (67 %) and Switzerland (85 %). In Latin America, there are more than 100 million users, and several countries have arrived at coverage of 90 to 99 %. Salt fluoridation is by far the cheapest method of caries prevention, and billions of people throughout the world could benefit from this method. Currently, sodium fluoridated salt containing 250 ± 100 mgF/kg is an important component of the preventive program for the population of Belarus.

The idea of milk fluoridation emerged, at about the same time, in Japan (1952), in Switzerland (1953) and the USA (1955). Early investigations showed that fluoride added to milk does not change its taste or other characteristics, is absorbed well, although slower than from fluoridated water. It was considered advantageous that fluoride is added to an important food for infants and small children and that consumption of fluoridated milk is not mandatory for everybody, only for those who need it most and agree to receive it. The caries

preventive effect of fluoride can even be enhanced by the milk vehicle, due to the cariostatic properties of the mineral, protein and fat content of milk.

The first clinical results were reported by Imamura in 1959, after a five-year study of Yokohama schoolchildren. Milk or soup, containing 2.0 to 2.5 mg sodium fluoride, was consumed at lunch-time, 150 to 180 days per year, by 167 children. Compared with the control group, 29 to 34 % caries reductions were observed in the permanent dentition. The amount of fluoride added to milk depends on background fluoride exposure and age of the children: commonly in the range 0.5 to 1.0 mg per day. An advantage of the method is that

a precise amount of fluoride can be delivered under controlled conditions. The cost of milk fluoridation programmes is low, about € 2 to 3 per child per year. Fluoridation of milk can be recommended as a caries preventive measure where the fluoride concentration in drinking water is suboptimal, caries experience in children is significant, and there is an existing school milk programme.

Tablets and drops containing sodium fluoride, appeared in 1940-50-ies on the proposal FAArnold and BGBibby as an alternative to water fluoridation

Tablets and drops are the most efficacy and safety supplementation of fluoride because they provide a personalized and targeted supplementation. Chewing of tablets creates the possibility of prolonged exposure of fluoride on tooth enamel, which manifests in such high concentrations of wonderful local preventive properties: mineralizing, antimicrobial. Tablets should be prescribed when concentration of fluoride in drinking water is less than 0.7 mg/l. The course is 250 days in a year. Dose — 0,0011 g (0,5 mg F) and 0,0022 g (1,0 mg F).

The disadvantages of the tablets include:

– the potential risk of poisoning;

– relatively high cost of drugs and related programs.

In high temperature zone the distribution of tablets is not carried out in the summer, since at this time the consumption of drinking water increases.

Medical efficacy of the fluoride tablets at the group level for several years is corresponding to 30–50 % reduction of dental caries.

An important rule of safety programs of preeruptive fluoride prevention is the prohibition of simultaneous free circulation of several supplements in the region. It should by only one vehicle of systemic fluoride.

LITERATURE

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WORKSHOP № 14
LOCAL FLUORIDATED DRUGS AND PRODUCTS FOR CARIES PREVENTION

Test Questions:

1. Factors determining the efficacy of mineralizing prevention.

2. Fluoride compounds in medicines and products for local caries prevention: advantages and disadvantages, indications.

3. Fluoride-containing solutions.

4. Fluoride-containing gels and foams.

5. Fluoride-containing toothpastes.

6. Other oral products that release fluoride (fluoride-containing depot, hygiene items, restoration materials, gums and pills).

7. Rationale for using calcium and phosphate compounds in the prevention of dental caries. Factors determining the efficacy of Ca-P-prevention.

8. Calcium and phosphate compounds in medications for local caries prevention: advantages and disadvantages, indications.

The term topical fluoride therapy refers to the use of systems containing relatively large concentrations of fluoride that are applied locally or topically, to the erupted tooth surface to prevent the formation of dental caries.

Rationale for the Use of Topical Fluorides. At the time of tooth eruption, the enamel is not yet completely calcified and undergoes a post eruptive period, approximately 2 years, during which enamel calcification continues (enamel maturation period). During this period fluoride deposition takes place in the superficial layer of enamel.

This fluoride is derived from saliva and also from exposure of teeth to fluoride-containing water and food. Thus most of fluoride incorporation into enamel occurs during the preeruptive period (by systemic fluoride) and the post eruptive period of enamel maturation. As immediately after eruption, the enamel is porous and immature it acquires fluoride rapidly. Application of topical fluorides immediately after eruption hastens fluoride uptake and makes enamel more resistant to dental caries (table 4).

Table 4

Range of therapeutic fluoride concentrations used to prevent caries

Method/vehicle	**Fluoride concentration, ppm F**
Water supplies	0.7–1.2
Fluoridated salt	200–250
0.05 % NaF Mouthrinse, **Once daily**	230
0.2 % NaF Mouthrinse, **Once weekly**	920
Dentifrices, children	500–1000
Dentifrices, adult **Twice daily**	1,000–1,500
1.1 % NaF gels **Once daily**	5,000
Professionally applied solutions (2 % NaF)	9,200
Professionally applied solutions, gels, foams (1.23 % APF)	12,300
Professionally applied solutions (8 % SnF2)	19,500
Professionally applied varnishes (5 % NaF)	22,600

ADVANTAGES AND DISADVANTAGES OF TOPICAL FLUORIDES

Advantages:

1. Does not cause fluorosis.

2. Preventive effect for people of all ages.

3. Available only to people who desire it.

4. Easy to use.

Disadvantages:

1. Person must remember to use.

2. Per capita cost is high compared to water fluoridation.

3. More concentrated professional use products can cause short-term side effects like nausea immediately after use.

Not all fluoride agents and treatments are equal. Different fluoride compounds, different vehicles, and vastly different concentrations of fluoride have been used with different frequency and duration of application. All of these variables influence the clinical outcome with respect to caries prevention. The efficacy of topical fluoride depends on:

a) the concentration of fluoride used;

b) the frequency with which it is applied and the duration of application;

c) the specific fluoride compound used.

MECHANISM OF ACTION

The presence of elevated concentration of fluoride in enamel surface makes tooth surface more resistant to development of dental caries. Fluoride ions when substituted into the hydroxyapatite crystals fit more perfectly than do hydroxyl ions. Also the greater bonding potential of fluoride makes the apatite crystals more compact and more stable, thereby more resistant to the acid dissolution. When concentrated topical fluoride agent reacts with enamel there is formation of calcium fluoride:

$$Ca_{10}[PO_4]_6[OH]_2 + 20F^- \leftrightarrow 10\ CaF_2 + 6[HPO_4^-]^3 + 2[OH]^-$$

Hydroxyapatite Calcium fluoride

Most topical fluoride agents have a fluoride ion concentration of between 10,000–20,000 ppm which leads to the formation of calcium fluoride and eventually Fluor hydroxyapatite. Commonly used topical fluoride agents

include Sodium fluoride, Sodium monofluorophosphate, Stannous fluoride and Amine fluoride.

CLASSIFICATION

Fluorides Applied by Dentist/Professionally Applied

A. Aqueous solutions:

• Sodium fluoride — 2 %;

• Stannous fluoride — 8%.

B. Fluoride Gels:

• Acidulated phosphate fluoride — 1.23 %.

C. Fluoride varnishes:

• Duraphat;

• Fluorprotector;

• Cavity Shield;

• MI-varnish.

D. Fluoride prophylactic paste.

E. Restorative materials containing fluoride.

F. Fluoride containing devices (slow release).

Self Applied:

• Fluoride dentifrices.

• Fluoride mouth rinses.

PROFESSIONALLY APPLIED FLUORIDES (PATF)

Dental personnel have been applying fluoride agents on teeth since 1940. It was seen that when fluoride was applied to teeth, it gets deposited in the outer enamel, making it more resistant to dissolution by acids. Although it is now known that frequency and availability of low concentration of fluoride is more important in caries prevention, but studies have shown to support the beneficial

effect of infrequent professional application of agents for prevention of dental caries. Topical fluoride applications are indicated for patients with active smooth surface caries and those patients in high caries risk groups. This includes special patient groups, such as those undergoing orthodontic treatment.

A. Aqueous Solutions

Sodium Fluoride: 2 % (Knutson's Technique) Available in both powder and liquid form. The compound is recommended for use in a 2 percent concentration.

Method of preparation. It can be prepared by dissolving 0.2 gm of powder in 10 ml (20 gm in 1 liter) of distilled water. The prepared solution has a basic pH and is stable if stored in plastic bottle. If stored in glass bottle, the fluoride ion of prepared solution can react with silica of glass forming SiF_2 (silicon fluoride), thus reducing the availability of free active fluoride. Hence reducing its anti caries action.

Indications for use of professionally applied topical fluorides:

• Patients who are at high risk for caries on smooth tooth surfaces.

• Patients who are at high risk for caries on root surfaces.

• To reduce tooth sensitivity.

• White spots.

• Active decay.

• Special patient groups, such as:

– orthodontic patients;

– patients undergoing head and neck irradiation;

– patients with decreased salivary flow.

• Children whose permanent molars should, but cannot be sealed.

• Additional protection if necessary for children in areas without fluoridated drinking water.

Recommended ages. It is recommended that a series of 4-weekly applications of 2 percent NaF be given at ages 3, 7, 11 and 13, coinciding with the eruption of different groups of primary and permanent teeth.

Method of application:

1. Cleaning and polishing of teeth is done.

2. Teeth are isolated with cotton rolls and dried with compressed air.

3. Teeth can be selected quadrant wise.

4. 2 percent aqueous NaF solution is applied with cotton applicator for 3 minutes.

5. Procedure is repeated for remaining quadrants until all of the teeth are treated.

6. Second, third and fourth applications are recommended at intervals of approximately 1 week and they are preceded by cleaning and polishing.

7. Patient is advised to avoid rinsing, drinking and eating for next half hour.

Mechanism of Action of Sodium Fluoride. When sodium fluoride solution is applied on the tooth surface it reacts with hydroxyl apatite crystals rapidly to form calcium fluoride. This initial rapid reaction is followed by drastic reduction in its rate and the phenomenon is called as «choking off». As a thick layer of calcium fluoride gets formed it interferes with the further diffusion of fluoride from aqueous solution to react with hydroxyl apatite. The calcium fluoride reacts with hydroxyapatite to form fluoridated hydroxyapatite. This increases the concentration of surface fluoride, making the tooth structure more stable, and surface more resistant to caries attack. It also helps in remineralization of the initial decalcified areas.

The chemical reaction involved is:

$$Ca_{10}(PO_4)_6(OH)_2 + 20\ F^- \leftrightarrow 10CaF^2 + 6PO_4^- + 2OH^-$$

$$CaF_2 + 2Ca_5(PO_4)_3\ OH \rightarrow 2Ca_5(PO_4)_3F + Ca(OH)_2$$

Advantages:

1. Relatively stable when stored in plastic containers.

2. Taste is acceptable.

3. Non-irritating to gingiva and does not cause discoloration of tooth structures.

Disadvantage. Patient has to make four visits in relatively short period of time.

B. Fluoride Gels.

Fluoride gels and foams contain a high concentration of fluoride, typically up to 12.3 mg/g fluoride.

Acidulated Phosphate Fluoride 1.23 percent (Brudevolds Solution). This is available as either as a solution or gel. Both are stable.

Solution. It is prepared by dissolving 20 gms of sodium fluoride in 1 liter of 0.1 M phosphoric acid. To this is added 50 percent hydrofluoric acid to maintain a pH of 3.0 and fluoride ion concentration at 1.23 percent.

Gel: for preparation of gel (APF), a gelling agent methylcellulose or hydroxyethyl cellulose is added to the solution and the pH is adjusted 4–5.

Another form of APF Thixotropic gels is available. Thixotropic denotes a solution that sets in a gel like state but is not a true gel. Upon the application if pressure, thixotropic gels behave like solutions.

Recommended frequency of APF application is twice a year topically.

Procedure for the Application of Fluoride. Solution:

a) Oral prophylaxis is done.

b) Teeth are isolated with cotton rolls and dried with compressed air.

c) Fluoride solution is then applied continuously with cotton applicator so as to keep teeth moist with fluoride solution for 4 minutes.

d) After all the teeth are treated patient is asked to expectorate and instructed not to rinse, drink or eat for next half hour.

Procedure for the Application of Fluoride Gel:

a) Mouth trays should be tried in the patient's mouth. It may be necessary to adapt or trim trays.

b) Patient should be seated upright and suction should be used during the procedure.

c) Teeth should be air-dried before gel application. For caries prevention, cleaning or prophylaxis is unnecessary prior to APF.

d) Enough gel, or foam, should be used to completely cover the teeth, but should be no more than 2–2.5 grams per tray or 40 percent of the tray's volume.

e) Upper and lower trays should be inserted separately.

f) Fluoride should be applied for 4 minutes, not 1 minute.

g) Patient should expectorate for 1–2 minutes after tray removal.

h) Patient should not rinse, eat, or drink for at least 30 minutes after the procedure

For patients with porcelain or resin restorations, neutral sodium fluoride is recommended to prevent etching of restorations.

CLINICAL APPLICATION

The frequency of gel application varies based on the caries risk level of the patient, and is usually provided at least every 6 months. Gel application is uncomplicated and can be performed by a dental auxiliary. The four-minute application of fluoride gel is recommended based on studies of enamel fluoride uptake. When contact time is reduced to one minute, enamel fluoride uptake is significantly less.

A considerable amount of fluoride may be retained after gel application, even if suction devices are used (on average 7.7 mg in children). The risk of fluoride ingestion with fluoride foam is reduced, compared with gel, because a smaller amount is needed for applications. The exposure to and retention of fluoride foam by the patient may be significantly less compared with APF gel application.

Mechanism of Action. APF when applied on teeth initially leads to dehydration and shrinkage in the volume of hydroxyapatite crystals. There is

further hydrolysis and formation of dicalcium phosphate dehydrate (DCPD), which is highly reactive. The fluoride ions start penetrating into the deeper crystalline structure of enamel and forms fluorapatite which is stronger to acid dissolution.

Advantages:

1. It is stable when stored in a plastic container.

2. No staining of teeth.

3. Gels can self applied.

Disadvantages:

1. Cannot be stored in glass container because it may remove minerals from the glass (etch).

2. Repeated exposure of porcelain or composite restorations to APF can lead to loss of material leading to surface roughening and cosmetic changes hence not advisable to use acidic topical fluoride agent in patients with these type of restorations.

3. It has an acidic taste.

4. Repeated application necessitates the use of suction, limiting its use in field programs.

Guidelines for the Application of Topical Gels. These are designed to minimize the amount of fluoride that may be swallowed.

1. Limit the amount of gel placed in each commercially available disposable mouth tray to no more than 2 ml or 40 percent of the tray capacity.

2. Limit the amount of gel placed in each custom fitted mouth tray to 5–10 drops.

3. Sit the patient in an upright position with the head inclined forward.

4. Use suction throughout the gel application procedure.

5. Instruct the patient to expectorate, or use a saliva ejector for 30 seconds after the gel application.

6. Keep the container out of reach of the patient.

7. Never leave the patient unattended.

C. Fluoride Varnishes.

A fluoride varnish is a professionally applied adherent material. It is not intended to be as permanent as a fissure sealant, the purpose is to hold fluoride in close contact with the tooth for a period of time. They permit the application of high fluoride concentrations in small amounts of material.

The use of fluoride varnish increases the fluoride concentration in saliva, which remains significantly higher 2 hours after its application than after the use of other fluoride agents.

The evidence also supports the view that varnish application can arrest existing lesions on the smooth surfaces of primary teeth and roots of permanent teeth. Much of the evidence of effectiveness is derived from studies which have used sodium fluoride 22,600 ppm varnish for application. Fluoride varnish for use as a topical treatment has a number of practical advantages. It is well accepted and considered to be safe.

Further, the application of fluoride varnish is simple and requires minimal training. While a thorough prophylaxis is not essential prior to application, removal of gross plaque is advised.

The use of Duraphat is contraindicated in patients with ulcerative gingivitis and stomatitis. There is a very small risk of allergy to one component of
Duraphat (colophony), so for children who have a history of allergic episodes requiring hospital admission, including asthma, varnish application is contraindicated.

There are two types of fluoride varnish: natural based and synthetic based.

1. *Duraphat (NaF).* It was first fluoride varnish to be tested. It contains 2.26 percent NaF or 22.6 mgF/ml. It is a viscous, resinous lacquer which should be applied to dry, clean tooth. Duraphat hardens into a yellowish brown coating in the presence of saliva. Majority of clinical trials conducted to see the efficacy of Duraphat during 1970 have reported the effectiveness between 30 to 45 percent.

2. *Fluor protector (Silane fluoride).* It was developed in 1970. It contains Silane fluoride 0.7 percent (7000 ppm fluoride) in polyurethane — based lacquer. Fluor protector leaves a clear transparent film on the teeth. Another varnish that has been tested in Norway called CAREX, contains a lower fluoride concentration (1.8 % fluoride). The caries preventive efficacy of this new varnish was found to be equivalent to that of Duraphat.

When varnish is painted on the tooth surface, it acts as a fluoride depot from which fluoride ions are continuously released. These ions react with hydroxyapatite over a longer period of time as varnish is not quickly washed away by saliva. This leads to deeper penetration and significant anticaries effect. Method of varnish application:

1. Oral prophylaxis is done.

2. Teeth are dried and but not isolated with cotton rolls as varnish sticks to cotton.

3. First lower arch is taken up for application and then upper arch as saliva collects rapidly on the lower arch.

4. Dispense a small amount of varnish (0.3 ml to 0.5 ml, or 2 drops, for the entire primary dentition) to the applicator dish or pad.

5. Application is done with single tufted brush starting with proximal surfaces (Dental floss can be used to ensure that the varnish reaches interproximal areas).

6. Since varnish sets rapidly when they come in contact with saliva, no drying is necessary.

7. After application, patient is made to sit with mouth open for 4 minutes.

8. Patient is instructed not to rinse or drink anything for 1 hour, and not to eat anything solid and avoid brushing till next morning. Patient is advised to take liquids or semisolids only, as contact between varnish and tooth surface is maintained for about 18 hours. It is for prolonged interaction between fluoride and enamel.

A total of 0.3–0.5 ml of varnish is required to cover the full dentition. 0.5 ml Duraphat contains 11.3 mgF and 0.5 ml of Fluor protector contains

3.1 mgF. It is seen that inspite of lower Fluoride content in Fluor protector (0.7 %) as compared to Duraphat (2.26 %), the fluoride deposited in enamel is twice as more and on the contrary its ability to inhibit caries is far less than Duraphat. Arands and Schuthof (1975) showed that silane fluoride of Fluor protector reacts with water to produce considerable amount of Hydrofluoric acid (HF) which penetrates into enamel more rapidly than F– suggesting a possible mechanism of greater fluoride deposition. The fact that Ca dissolution is reduced more with Duraphat than Fluor protector suggest that part of fluoride deposited after treatment with Fluor protector may be in some form other than the bound form, i.e. fluorapatite.

Fluor protector a high viscosity varnish penetrates the porosities of enamel forming tags 0.5–1 mm long which acts as a fluoride reservoir accounting for more fluoride deposition in enamel. On the other hand these tags further block the pathways for fluoride and do not let remineralization of initial lesion occur explaining the less caries inhibition. An additional factor may be a chemical alteration of enamel by the presence of silane agent.

Fluoride varnish has a high fluoride concentration, but its safety is acceptable. Varnish is fast setting, fluoride is slowly released, and a small amount is needed for the complete dentition. Measurements of fluoride after topical treatments with varnish show levels far below those considered toxic. Consequently, varnishes may be a better alternative to fluoride gels, especially for young children.

The only disadvantage of sodium fluoride varnishes is that they cause a temporary change in tooth color, which dental professionals need to inform their patients of it.

APF gel treatments are difficult, if not impossible, to do on many young children and there is considerable risk of over ingestion of fluoride. Fluoride varnish can be successfully applied in most young children and there is no risk of overingestion of fluoride. These advantages make it possible to apply fluoride varnish safely to the newly erupting teeth of high-risk infants and young children in an effort to control ECC.

The varnishes are more convenient than the bottles of gel and trays to take into field situations. Intraoral suction is not required for the application of

varnish. And there is no messy cleanup and disposal problem following a varnish application.

D. Fluoride Prophylactic Paste.

Prophylactic paste contains abrasive particles which abrade the deposits and debris from tooth surface. Studies date back to 1946 when NaF prophylactic paste was tried. Stannous fluoride prophylactic paste was developed in 1960. Nowadays APF-silicone dioxide paste and SnF2 — Zirconium silicate paste are also available.

Studies have shown that their use alone cannot be considered as an effective cariostatic method. Tooth cleaning with a fluoride prophylactic paste should not supplement topical fluoride application with fluoride solution or gels for children. A thorough polishing may remove a thin, but highly mineralized outer layer of enamel. If prophylaxis is required for periodontal reasons or cosmetic reasons then fluoride prophylactic paste is recommended, as it may help replenish the minerals that are abraded during polishing. They may have a modest cariostatic effect.

E. Restorative Materials Containing Fluoride.

Fluorides releasing dental restorative material are also available, that provide site specific protection. In general, the rate of fluoride release from such materials is not constant but exhibits a relatively rapid initial rate, which decreases with time. These materials may feature greater longevity, a reduced incidence of marginal failure, an elevated concentration of fluoride in contingent plaque, together with an antibacterial action when compared with non-fluoride releasing materials.

The purpose of adding fluoride to restorative material is to capture its anticariogenic property. A major reason for the failure of restorations is recurrent or secondary caries. Secondary caries has been reported as being the most common reason for replacement of restorations. However, incorporation of fluoride into restorations may be beneficial because of the observed cariostatic action of fluoride. The fluoride ions are slowly released from the materials.

One difficulty with these materials is controlling the rate of fluoride release. Fluoride has also been added to amalgam in an attempt to reduce the risk of recurrent caries at restoration margins.

Fluoride may be released from dental restorative materials as part of the setting reaction, or it may be added to the formulation with the specific intention of fluoride release.

Fluoride containing restorative materials includes glass ionomer cements, resin modified glass ionomer cements, polyacid modified resin composites (compomers), resin composites, fissure sealants and dental amalgam.

Fluoride Ingestion and Toxicity of Professionally Applied Topical Fluoride (PATF). Fluoride applications must be carefully monitored because the potential for over ingestion and toxicity does exist. Fluoride is rapidly absorbed in the gastrointestinal tract and young children are particularly vulnerable. Patients should not be left unattended during the application of PATF.

A considerable amount of fluoride may be retained after gel application, even if suction devices are used (on average 7.7 mg in children). The risk of fluoride ingestion with fluoride foam is reduced, compared with gel, because a smaller amount is needed for applications. The exposure to and retention of fluoride foam by the patient may be significantly less compared with APF gel application.

Fluoride varnish has a high fluoride concentration, but its safety is acceptable. Varnish is fast setting, fluoride is slowly released, and a small amount is needed for the complete dentition. Measurements of fluoride after topical treatments with varnish show levels far below those considered toxic. Consequently, varnishes may be a better alternative to fluoride gels, especially for young children. PATF is not a risk factor for dental fluorosis when used at 6 months intervals, and if precautions are taken to minimize ingestion.

FLUORIDE DENTIFRICES

Investigation into effectiveness of adding fluoride to tooth-paste has been carried out since 1945 and covers a wide range of active ingredients in various abrasive formulations. Fluoride compounds that have been tested for caries-inhibitory properties include sodium fluoride, acidulated phosphate fluoride,

stannous fluoride, sodium monofluorophosphate and amine fluoride. Most toothpaste nowadays contain sodium fluoride or sodium monofluorophosphate as active ingredient, usually in concentration of 1000–1100 mg F/g.

The results suggest that increased fluoride levels give a greater reduction in the incidence of dental caries. Increased benefit is of the order of 6 percent for each 500 ppm over 1000 ppm fluoride. In Europe an upper limit of 1500 is suggested for toothpaste sold over the counter without prescription

Fluoridated Toothpaste for Children. Concern about the fluorosis risk from children swallowing toothpaste has led to trial of lower-strength dentifrices. Findings from studies of 500–550 mgF/g products suggest efficacy equivalent to 1000 mgF/g toothpaste. Since children can swallow between 0.12 and 0.38 mg of toothpaste per brushing, lower fluoride toothpaste may reduce the risk of fluorosis while substantially retaining the caries preventive benefits. However, the production of candy like flavors and toothpaste containing fluoride at 1500 ppm or more should not be encouraged for use by children, as it may lead to an excessive ingestion of fluoride.

FLUORIDE MOUTHRINSES

Frequent use of low concentration of fluoride is more cariostatic than less frequent use of higher concentration of fluoride for topical application. In areas where water fluoridation is not possible or has not been implemented, the fluoride mouthrinses have been found to be an effective tool in prevention of dental caries. Over the past few decades fluoride mouthrinsing has become one of
the most widely used caries-preventive public health measure.

Sodium fluoride mouth rinse is now widely used in school based programs as well as by individuals at home. Other less extensively tested fluoride mouth rinses include those containing APF, stannous fluoride, ammonium fluoride and amine fluoride. For reasons of lowest expense, convenience in handling as well avoidance of unpleasant taste, NaF became the most widely used of these tested products in public health programs.

RECOMMENDATIONS

Mouth rinses designed to be rinsed and spit out, either prescribed by your dentist or an over-the-counter variety. 0.2 percent of NaF solutions are

recommended (900 ppm F–) for weekly/fortnightly use, while 0.05 percent (225 ppm F–) for daily use.

Mouth rinses designed to be rinsed and spit out. The ADA recommends the use of fluoride mouthrinses, but not for children under six years of age because they may swallow the rinse.

Over-the-counter daily fluoride mouthrinses generally contain 0.05 percent NaF (200–220 ppm F). A 10 mL volume should be swished around the mouth vigorously once each day for one minute (ideally just before bedtime) and then expectorated. Patients should not rinse afterwards for 30 minutes. Pharmacy-only «weekly» fluoride mouthrinses typically contain 0.2 percent NaF (900 ppm F). They are designed to be used under adult supervision, once each week for one minute.

Fluoride mouthrinse should be used at a time of day when toothpaste is not used, and it should not be a substitute for brushing with fluoridated toothpaste. After rinsing, mouthrinse should be spit out, not swallowed.

ADVANTAGES

1. Caries reduction by 30 percent.

2. Seemingly low cost resulting from supervision by teachers, volunteer mothers or inexpensive hourly workers.

3. A reasonable procedure to use in high risk population.

Use of casein phosphopeptide — Amorphous calcium phosphate (CPP–ACP), which is available as tooth mousse, helps to remineralize the soft initial carious, demineralized areas of the teeth.

Amorphous Calcium Phosphate (ACP) is an essential mineral phase formed in mineralized tissues and the first commercial product as artificial hydroxyapatite. ACP is unique among all forms of calcium phosphates in that it lacks long-range, periodic atomic scale order of crystalline calcium phosphates.

ACP may be a potential remineralizing agent in dental applications. Recently developed ACP-filled bioactive composites are believed to be effective anti-demineralizing/remineralizing agents for the preservation and repair of tooth structures.

Casein phosphopeptides (CPP) has a remarkable ability to stabilize clusters of ACP into CPP-ACP complexes, preventing their growth to the critical size required for nucleation, phase transformation and precipitation. This product is primarily used for abrasive prophylaxis pastes and secondarily used for the treatment of tooth sensitivity especially after in-office bleaching procedures, ultrasonic scaling, hand scaling or root planing. However, its use for remineralizing dentin and enamel and preventing dental caries is an off-label application. Outside the United States, this product is marketed as GC Tooth Mousse.

CPP-ACP binds well to plaque, providing a large calcium reservoir within plaque and slowing diffusion of free calcium. This is likely to restrict mineral loss during a cariogenic episode and provide a potential source of calcium for the inhibition of demineralization and assist in subsequent remineralization. CPP are a safe and novel carrier for calcium, phosphate and hydroxide (fluoride) ions to promote enamel remineralization with application in oral care products, dental professional products and foodstuffs.

3M ESPE Preventive Care announces the new formulation of Clinpro™ White Varnish, now with an innovative tri-calcium phosphate (TCP) technology that delivers more protection to the teeth, and helps relieve sensitivity. In clinical studies, Clinpro White Varnish has been shown to flow more than competing varnishes, meaning that it can reach more places and deliver more fluoride to the teeth. Plus, Clinpro White Varnish with TCP:

• Releases Calcium.

• Relieves Hypersensitivity.

• Takes just seconds to paint on.

• Sets rapidly in the presence of saliva.

• Flows better interproximally and across tooth surfaces than other brands tested.

• Contains 22,600 ppm Fluoride.

MI Varnish with RECALDENT™(CPP-ACP) (GC) enhances enamel acid resistance and boosts salivary fluoride levels. This product remains on the teeth longer than other fluoride varnishes and contains high levels of

fluoride and calcium released in the oral cavity. The difference is in the RECALDENT™(CPP-ACP), which makes MI Varnish a natural and unique choice for your patients. Casein phosphopeptides (CPP) naturally occurs in milk casein; Amorphous Calcium Phosphate (ACP), which is found in the RECALDENT™, is also the source of calcium and phosphate. In the oral cavity, CPP binds to oral surfaces such as teeth, dentin, oral mucosa and biofilm. Calcium and phosphate ions are the building blocks for healthy teeth and MI Varnish delivers bioavailable calcium and phosphate ions into the saliva.

LITERATURE

1. *Marya, C. M.* A Textbook in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2011. P. 348–361.

2. *Cameron, A. C.* Handbook of Pediatric Dentistry / A. C. Cameron, R. P. Widmer. Mosby, 2008. P. 59–69.

WORKSHOP № 15
HEALTH EDUCATION: PSYCHOLOGICAL BASES, OBJECTIVES, METHODS

Test Questions:

1. The role of behavior in maintaining dental health.

2. Mechanisms of formation of human behavior: motivation and needs.

3. Physician-patient communication in the process of health education. Stages of formation of new behavioral habits.

4. Structure and content of educational project on prevention of dental diseases.

5. Methods and forms of health education; educational project design choice for different age groups.

6. Health education and training of children. Content, structure, form and conditions of hygiene lessons for preschoolers.

The maintenance of oral health is largely a function of behavior.

The notion that education could improve dental health is based on the findings concerning the generally low levels of relevant knowledge in the general population. Many people are unaware that the occurrence of caries is most closely associated with the frequency of eating sugar, rather than the absolute amount. Similarly, many people do not realize the importance of reducing plaque for the maintenance of healthy gums. Theories of health behavior (Connor and Norman 1996) predict that patients' levels of knowledge will have an effect on what they do to maintain good health. According to these models, people will begin to make changes to their behavior — or at least move from one stage of preventive care to another — only when they appreciate the importance of regular dental care and have a correct interpretation of symptoms.

Abraham Maslow (1954) attempted to synthesize a large body of research related to human motivation. Prior to Maslow, researchers generally focused separately on such factors as biology, achievement, or power to explain what energizes, directs, and sustains human behavior. Maslow posited a hierarchy of human needs based on two groupings: deficiency needs and growth needs (fig. 4).

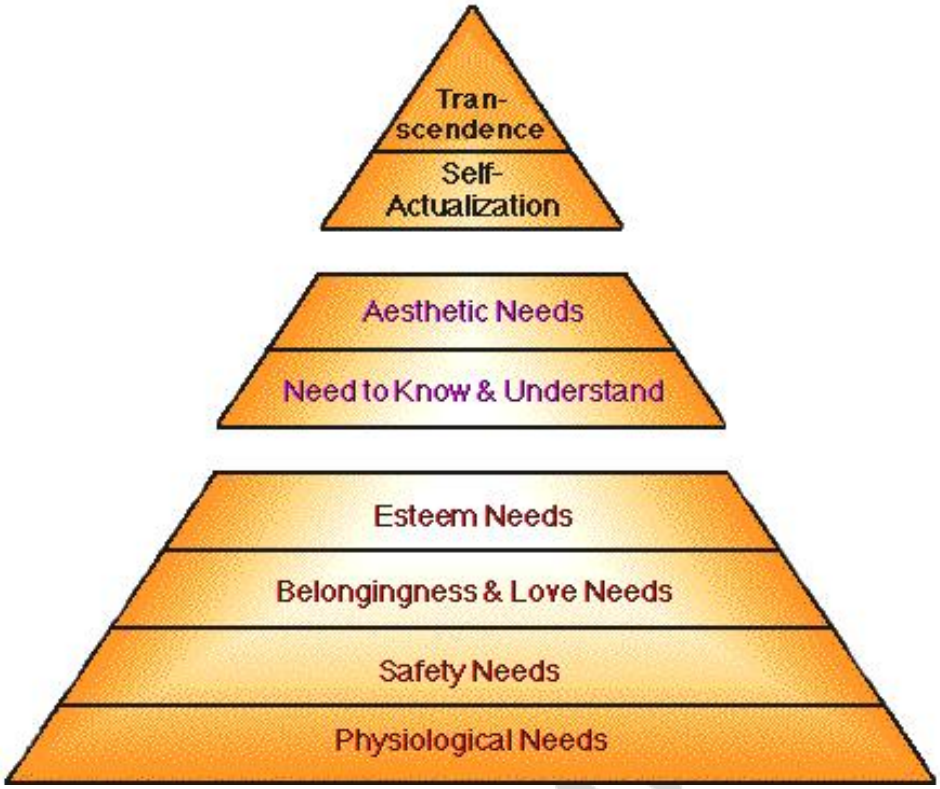

Fig. 4. Maslow's hierarchy of needs

The habit formation may be represented as a five-stage process (fig. 5).

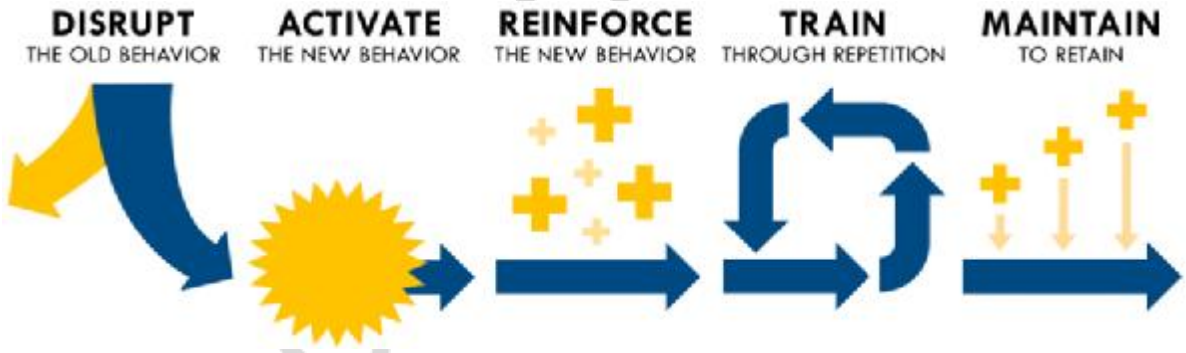

Fig. 5. Process of habit formation

Health education is defined as any educational activity which aims to achieve a health related goal. The three main objectives of health education are:

– informing people;

– motivating people;

– guiding into action.

There are three main domains of learning:

1. Cognitive: understanding factual knowledge (for example, knowledge that tobacco chewing is linked to development of oral cancer).

2. Affective: feelings, emotions and beliefs associated with health (belief that milk teeth are not important).

3. Behavioral: skills development (for example, skills required for effective brushing and flossing of teeth).

The content and methodology of health education are derived from the field of medicine and public health and from the physical, biological, social and behavioral sciences. There are TEN principles of health education based on the principles of learning:

1. Interest.

2. Participation.

3. Comprehension.

4. Known to unknown.

5. Reinforcement.

6. Learning by doing.

7. Motivation.

8. Good human relations.

9. Soil, seed and sower.

10. Social leaders.

There are four key elements in the process of physician-patient communication:

– communicator;

– audience;

– message;

– channels of communications.

Methods in health education can be broadly divided into three groups:

1. Health Education for Individual and Family.
2. One to one supervision.
3. Interactive computer software.

Health Education for the Group:

– lectures;

– symposium;

– group discussions;

– panel discussion;

– workshop;

– demonstration;

– role playing;

– institute;

– conference/seminars;

– simulation exercises;

– Health Education for General Public/Mass Approach (through the media).

A dentist can play a role in community or field based preventive programs like:

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.

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.

Requirements to motivational guide for health education with preschoolers:

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

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

3) in plain language, the method for cleaning preschooler teeth is described;

4) the amount of text is 2 printed pages;

5) 5 pictures, the size of each of at least half of the page.

The goal of the dental hygiene lesson Education Program for preschoolers is to help children learn about proper oral hygiene and develop good dental health habits that will last a lifetime.

The materials of the lesson will help reinforce the three simple steps to good oral hygiene and healthy mouth:

1. When and why it's important to brush, and how to brush correctly.

2. How eating nutritious foods and limiting sugary snacks help keep teeth healthy.

3. Why regular dental checkups are important.

LITERATURE

Marya, C. M. A Textbook in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2011. P. 87–97, 156–164, 224–228, 410–412.

CONTENT

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PREVENTIVE DENTISTRY

Учебно-методическое пособие

В 2-х частях

На английском языке

Часть 1

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