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КАФЕДРА СТОМАТОЛОГИИ ДЕТСКОГО ВОЗРАСТА

# ПЕРВИЧНАЯ ПРОФИЛАКТИКА КАРИЕСА ЗУБОВ

# PRIMARY PREVENTION OF DENTAL CARIES

Допущено Министерством образования Республики Беларусь
в качестве учебного пособия для иностранных студентов
учреждений высшего образования по специальности «Стоматология»

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Изложены особенности организации рабочего места и обследования пациентов на профилактическом приеме. Подробно освещены современные взгляды на этиологию и патогенез кариеса зубов, подходы к первичной профилактике данной патологии, описаны индивидуальные и профессиональные методы удаления зубных отложений и используемые для этого средства и предметы, особенности местного и системного использования фторид-, кальций- и фосфатсодержащих препаратов в профилактике кариеса зубов. Рассмотрены особенности развития и профилактики кариеса ямок и фиссур зубов.

Предназначено для иностранных студентов стоматологических специальностей, учащихся, магистрантов, клинических ординаторов, аспирантов, практикующих врачей-стоматологов.

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**ПЕРВИЧНАЯ ПРОФИЛАКТИКА КАРИЕСА ЗУБОВ**

**PRIMARY PREVENTION OF DENTAL CARIES**

Учебное пособие

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# INTRODUCTION

Dental caries is one of the oldest and most common diseases found in humans. With the recent shift from the surgical model, which emphasized restorative treatment, to a medical model of disease management, newer strategies emphasize disease prevention and conservation of tooth structure.

Preventive dentistry has its roots from the Latin terms “praevenire,” which means “to anticipate” and “dens,” which is the word for tooth. Dentists and their members strive every working day to “anticipate” what could happen to their patients’ teeth and supporting structures.

Textbook “Primary prevention of dental caries” has been prepared for the students who study Dentistry in Medical University in English. This book provides a framework for students to learn the subject and bring into focus some of the newer concepts in Preventive Dentistry.

“Preventive Dentistry” — the academic discipline of the module “Medical Prevention in Dentistry,” which contains systematized scientific knowledge about primary prevention of dental diseases in the population.

The aim of the discipline “Preventive Dentistry” is the formation of specialized competencies for maintaining the oral health of the population.

The objectives of the discipline “Preventive Dentistry” are to form students’ scientific knowledge about:

– the basic concepts of primary prevention in dentistry;

– methods and means of diagnosis and treatment of main oral diseases in the population;

– principles of diagnosis of individual risk level of main dental diseases;

– principles of planning and implementation of prevention programs.

This textbook has been prepared for the dental students who aspire to enter the rewarding profession of dentistry. It is also meant to be a resource for dental educators. It will be useful for understanding the role of prevention in Dentistry, the modern conception of dental caries development and methods of its prevention.

Each chapter discusses the current state of basic knowledge, with discussions as to which products have been successfully used in preventive dentistry.

# Chapter 1
# WORKPLACE ORGANIZATION FOR PREVENTION ACTIVITIES

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 for tools and instruments;
– cabinet for preventive drugs;
– desk and chair for paper work.

*Minimal equipment standards* for the preventive activities include:

– 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.

## INFECTION CONTROL

**Infection control** in dentistry is an ever-growing perturbation. Dental patients are high-risk patients relative to their potential to transmit as well as acquire an infectious disease. An equal concern has been exhibited for cross-contamination and disease transmission from patient to patient.

When addressing these problems, there are two identifiable considerations:

1) how the dentist and his staff can be safeguarded from disease acquisition and disease transmission to patients;

2) what steps should be taken to help minimize cross-contamination with instrumentation.

The constant dangers of cross-contamination in dental practice among patients, dentists, and ancillary staff have been pointed out by Murray and Slack; they reported the possibility of absorbent cotton pledgets, air syringes, glass slabs, and hand towels acting as sources of contamination.

Iniatially the dentistry was routinely done without protective gears but after 1991 dental personnel were required to wear gloves, masks, gown, and protective eyewear. Dentistry is one of the most exposed professions to respiratory diseases e.g. Covid-19.

**Infections could be transmitted** in the dental operatory through several routes:

1) direct contact with blood, oral fluids, or other infected materials;

2) indirect contact with contaminated objects, such as instruments, environmental surfaces, or equipment;

3) contact of conjunctival, nasal, or oral mucosa with droplets, such as spatter, containing microorganisms from an infected person and propelled by coughing, sneezing, or talking;

4) inhalation of airborne microorganisms that can remain suspended in the air for long periods.

**Infection Control Procedures:**

– Patient Screening;
– Personal hygiene;
– Personal protection;
– Instrument processing;
– Surface asepsis;
– Patient treatment.

**Patient Screening.** Initial patient screening is accomplished by the dentist during history-taking interactions before entering the operatory. The dentist's review of the patient's medical history is mandatory at the onset of every clinical appointment. Multiple reviews give the dentist opportunities to establish baseline medical history data and to compare individual patient responses over an extended period of time as well as a brief review of any infectious disease the patient is suffering. Thermal monitoring at the screening stage has become mandatory due to Covid-19.

**Personal Hygiene:**

1. The dentist's personal hygiene is an absolute necessity. As patients become more aware of the potential danger to themselves from materials and instruments that are not disinfected or sterilized, their confidence and acceptance of dental treatment become directly proportional to the image the clinician presents. Specific notes of hygiene include.

2. Hair is cleared away from the face. If a clinician's hair falls in such a way that it may contact the patient or dental equipment, it is fixed at the back of the head, or a surgical cap is worn. Facial hair is covered by a face mask or shield.

3. Jewelry is removed from the hands, arms, or facial area during patient treatment.

4. Fingernails are kept clean and short to prevent perforation of gloves and accumulation of debris. Fingernail polish is not worn.

5. Thorough forearm and hand washing are mandatory before and after treatment.

**Personal Protection Equipment.** 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 personnel are required to have current immunizations against communicable diseases, including hepatitis B. Gloves, are worn at all times when treating patients. Masks are worn in the patient treatment area. The use of disposable plastic face shields or glasses is highly recommended. Sharps disposal protocol is followed, with particular emphasis on the use of a hemostat when handling blades.

**Instrument Processing:**

1. Presoaking and cleaning.
2. Packaging.
3. Sterilization.

*Common Methods of Sterilization:*

● Steam at 121 degrees C for 20 to 30 minutes or 134 degrees C for 2 to 10 minutes.

Advantages — good penetration.

Precautions — carbon steel corrodes, damage to plastic and rubber items, packs wet after the cycle, hard water spots instruments. Unsaturated chemical vapor — 20 minutes at 134 °C.

● Unsaturated chemical vapor — 20 minutes at 134 °C.

Advantages — no corrosion of carbon steel, packs are dry after cycle.

Precautions — may damage plastic and rubber items.

● Dry heat (Oven Type) — 1 to 2 hours at 160 °C.

Advantages — no corrosion of carbon steel, packs dry after the cycle.

Precautions — may damage plastic and rubber items, do not open door during the cycle.

● Dry heat (rapid heat transfer type) 6 to 12 minutes at 191 °C.

Advantages — short cycle.

Precautions — may damage plastic and rubber items.

● Glass bead sterilizers: Used in endodontics to sterilize reamers, files though their efficacy is debatable when compared with autoclaving.

**Surface Asepsis.** There are two general approaches to surface asepsis:

1. Clean and disinfect contaminated surfaces, and
2. Prevent surfaces from becoming contaminated by the use of surface covers. A combination of both may also be used.

According to Miller and Palenik in 1994, the following chemicals are suitable for surface and equipment asepsis:

Chlorine — e.g., sodium hypochlorite;

Phenolic compounds;

Water-based — Water with ortho-phenylphenol or tertiary amylphenol or O-benzyl–p-chlorophenol;

Alcohol-based — Ethyl or isopropyl alcohol with ortho-phenylphenol or tertiary amylphenol;

Iodophor — butoxy polypropoxy polyethoxy ethanol iodine complex.

**Patient Treatment.** The following procedures are required when treating patients in the clinic: before seating the patient the operatory and chair are cleaned and wiped with a disinfectant solution; the area is sprayed and left for a minimum 10 minutes.

Hands are washed with an antimicrobial cleanser before gloving. Once gloved, only the patient and barrier-covered areas or areas that have been cleaned and disinfected are touched. The patient chart is not touched with contaminated gloves. If an entry must be made in the chart, gloves must be removed or a clean glove is placed over the contaminated glove and removed after finishing the chart. Alternatively, an appropriate barrier must be used on the pen and over the portion of the record that is to be touched. The doctor should not leave the operatory without removing their gloves and outer barrier garments.

All items leaving the clinic after being used in direct patient care or touched during patient care procedures that cannot be subjected to sterilization procedures are disinfected or placed in the phenol disinfection solution within a sealed plastic bag before departure. New latex gloves are worn for the disinfection procedures. Items bagged in disinfection solution must remain in solution for 10 minutes.

**Post Exposure Prophylaxis.**

Step 1 — Assessment of exposure — description of exposure, local wound care, and personal protection worn at the time of injury.

Step 2 — Assessment of healthcare worker.

Step 3 — Source case information.

Step 4 — Serological testing.

Postexposure Protocol for Occupational Exposure to Blood/Body Fluids:

1. Stop the procedure immediately.

2. Inform patient.

3. Remove gloves inside out 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 shouldbe flushedwellwithwater. Whensplasheshave 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.

## ERGONOMICS

The International Ergonomics Association (IEA) defines **ergonomics** (or human factors) as "the scientific discipline concerned with the understanding of the interactions among humans and other elements of a system, and the profession that applies theoretical principles, data and methods to design, in order to optimize human well-being and overall system performance."

During a conventional dental treatment, the dentist often bends over the patient to achieve the most accurate treatment possible. However, bending often leads to an unnatural, harmful posture that negatively impacts the dentist's health (Fig. 1.1).

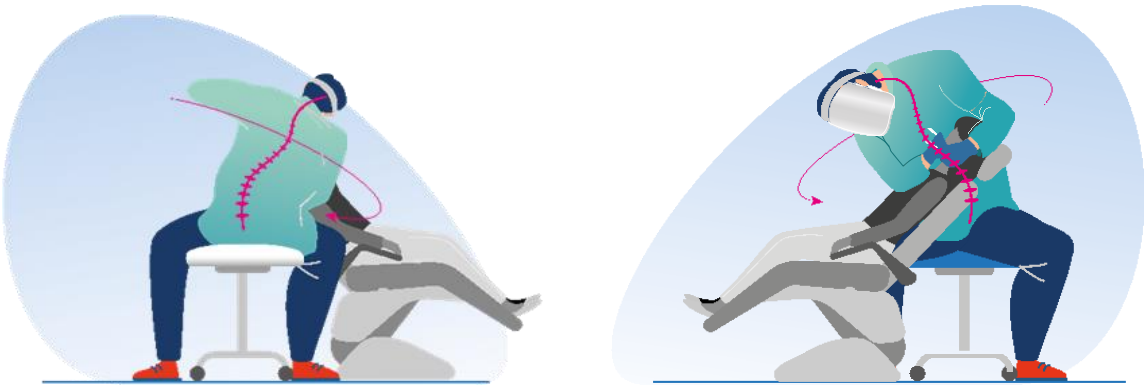

*Figure 1.1.* Unnatural harmful posture of dentist during work

Ergonomics in dentistry should strike a balance between keeping the dentist healthy and providing eficient treatment. Ergonomics in dentistry means preventing musculoskeletal problems by enabling the dentist to adopt a more natural and comfortable posture, achiev-ing patient-friendly treatment, improving treatment eficiency, and achieving treatment accuracy.

**Application of Ergonomics in Dentistry.**

**Work postures:**

– Dentist position;

– Patient position;

– Assistant position.

**Posture & vision:**

– Direct and indirect;

– Lighting and magnification.

**Instruments:**

– Examination and control instruments;

– Working instruments.

**Posture of Oral Health Professionals.** *What bad posture can cause.* The dark and narrow space in the oral cavity can cause oral health professionals (OHPs) to adopt an unnatural posture during dental treatment, which can lead to serious physical impact and repetitive strain injuries.

The potential to develop musculoskeletal disorders is higher when one disregards good ergonomic principles. In doing so, OHPs are at risk of compromising their technical expertise during procedures. This can lead to a limitation of certain procedures, potential career shortening, and, in the worst case, possible career-ending injuries.

The surveys show that the main complaints are neck and lower back pain. These issues develop over a number of years and worsen with a heavy workload. It has also been noted that female dentists appear to have a higher incidence of upper body com-plaints than their male counterparts.

**The Ideal Posture of the Oral Health Professional** (Fig. 1.2).

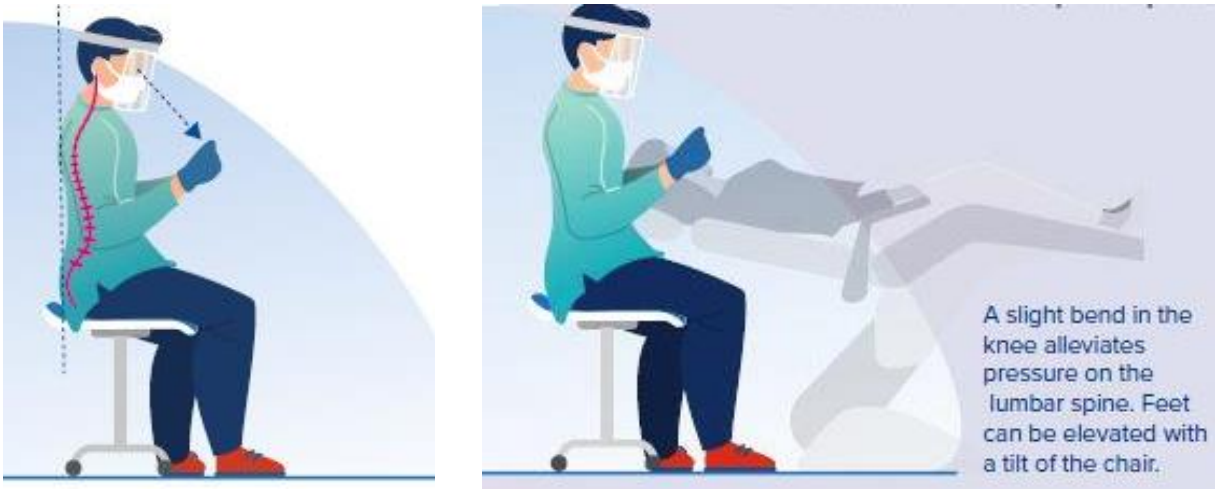


*Figure 1.2.* The ideal posture of the oral health professional

*The head.* To be inclined slightly forward, oriented over the shoulders. The interpupillary line is aligned horizontally not more than 15 to 20 degrees.

*Torso.* The longitudinal axis of the torso is upright. It promotes the natural curves of the spine — cervical lordosis, thoracic kyphosis, lumbar lordosis. If needed, the back rest of the chair can be positioned to provide lumbar support.

*Upper arms, elbows and shoulders.* Arms relaxed at one's side due to the force of gravity. The elbows do not stick out and the forearm is in front of the body. Shoulders are oriented over the hips.

*Wrists.* Should be kept in a neutral position with the wrists straight.

*The fingertips.* Should be held at the treatment point, at a height that is comfortable and affords a clear view of the procedure being performed.

*Seated posture.* Seating height at knee height; hips slightly higher than the knees; tilt the operator stool slightly downward.

*The feet.* To be flat on the floor. The lower legs are in a vertical position. Consider comfortable shoes and clothing to ease body movement.

*Rheostat positioning.* Place it close to the operator so that the knee is at about a 90 to 100 degree angle. If placed outside this zone, the dentist must shift weight to one side, leading to asymmetrical stresses on the back, hence low back pain. Consider alternating sides.

Movement throughout the day is key: staying too long in one position can cause fatigue and increase the risk of musculoskeletal problems.

**Position of the Patient.** A deliberate patient position should be determined according to the dentist's natural posture and his or her reference point, which allows the clinician to achieve optimal performance without any physical burden.

**Exceptional Cases.** *Treating patients in an upright position.* Occasionally, it may be necessary to treat a pa-tient while in an upright position, for example during certain procedures or when treating el-derly patients or those with complex medical histories (hypotension, vertigo). In this case the back rest should be vertical to provide lumbar support to the patient. OHPs may find it more comfortable to stand during these appointments. During pregnancy, a patient may experience postural hypotension, which can lead to fainting. Pregnant patients can be encouraged to lie on their side or be treated in a more upright position.

**Instrumentation.** *Preparation and placement of instruments without the four-handed system* (Fig. 1.3).

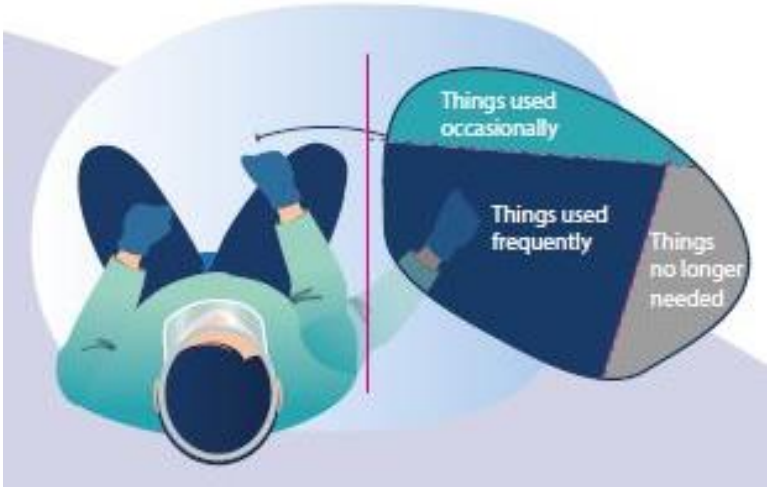


*Figure 1.3.* Preparation and placement of instruments without the four-handed system

There is a limit to the forearm's natural movement. Preparation and strategic placement of the instruments relieves the clinician's physical burden and improves concentration during treatment. Ideally, dentists should be able to pick up and return basic instruments, e.g., mirror, tweezers, explorer and excavator, without having to look away from the treatment area.

The basic principle is to differentiate foreseeable and unforeseeable tasks during treatment.

– Instruments and materials for which the use or timing is uncertain are prepared on the dentist's side.

– Instruments and materials that the dentist will need are prepared in the order and timing that they will be used on the assistant's side.

*Preparation and placement of instruments with the four-handed system* (Fig. 1.4).

*Figure 1.4.* Preparation and placement of instruments with the four-handed system

Place all of the necessary items for the patient and the procedure within the area of reach before the patient arrives.

**Direct and Indirect Visualization.** In order to maintain a properly balanced, upright posture, it is important to balance direct visualization with indirect visualization using the dental mirror.

In order to widen the view without compromising good posture, the dentist should ask the patient to place the head on the headrest and adjust it accordingly to allow easier access, depending on which surface they are working. The dentist may ask the patient to open the mouth wider or close it slightly when working buccally (Fig 1.5, 1.6).

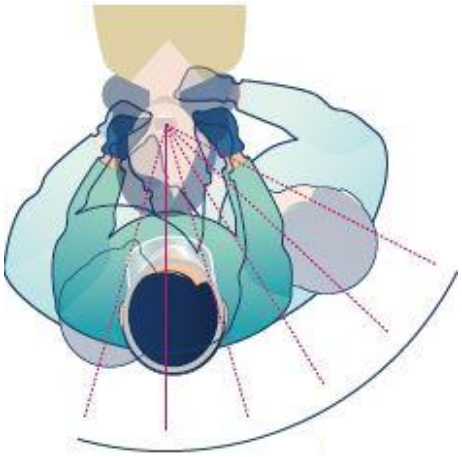

*Figure 1.5.* Dentist position

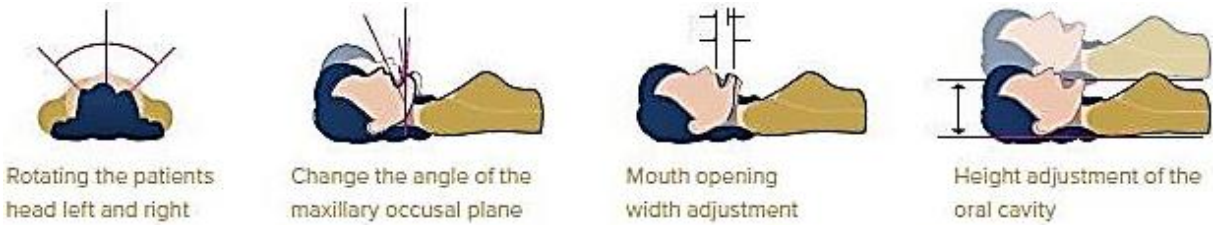


*Figure 1.6.* Five tips for direct views

**Choice of Instruments.** Sensations and perceptions are different for everyone. These general recommendations can help OHPs find the dental instruments that suit them best.

**Examination and Control Instruments.** The dental mirror and probe are light and thin. Their diameters typically range from 2 to 3 millimeters (mm). They are naturally held between the thumb and the index finger and do not usually lead to any muscle strain.

**Working Instruments.** Instruments with a large diameter (10 mm), textured handle, and a light weight (15 grams) require the least amount of muscle load and pinch force. Diameters larger than 10 mm have no additional benefit; instruments lighter than 15 g may require even less pinch force. Consider alternating tools with different diameter sizes to reduce the duration of prolonged pinch gripping.

Silicone instrument handles are further found to improve hand comfort, reduce hand fatigue, and improve grip and pinch strength.

Instruments should be held in a light, pen-like grip, using a fulcrum (finger rest) either intra-orally or extra-orally (Fig. 1.7). This plays an important role in stabilizing the clinician's hands during treatment, increasing the precise control needed and reducing muscle load and fatigue.

*Figure 1.7.* Position of instrument in fingers

**Gloves.** Favor gloves of proper size and fit and avoid ambidextrous or one-size-fits-all gloves. If they are too large and get wet, ill-fitting gloves require the practitioner to squeeze tighter to maintain the grip on the instrument. If they are too tight, the gloves may constrict the neurovascular structures of the fingers and hand.

# Chapter 2
# ORAL EXAMINATION

## EXTRAORAL EXAMINATION

**Patient Interview and Initial Examination.** Initial patient interviews and data gathering are vital to the initial exam. Initial data, medical history, dental history, and social history are commonly gathered through a patient interview. In this interview, there are a number of questions that should be answered, and the answers are written down to patient record (chart).

The dental record must include each of the following specific components:

– medical history;
– dental history;
– clinical assessment;
– radiographic or other images obtained, if any, and their interpretation;
– diagnosis or differential diagnosis;
– plan of preventive and treatment measures;
– parental consent;
– progress notes.

**Medical History.** An accurate, comprehensive, and up-to-date medical history is necessary for correct diagnosis, effective treatment planning, and patient safety. The practitioner, or staff under the supervision of the practitioner, must obtain a medical history from the parent (if the patient is under the age of 18) before commencing patient care. When the parent cannot provide adequate details regarding a patient's medical history, if the patient is medically compromised, or if the dentist providing care is unfamiliar with the patient's medical diagnosis, consultation with the medical health care provider may be indicated.

Documentation of the patient's medical history includes the following elements of information, with elaboration of positive findings:

– medical conditions and illnesses;
– name and, if available, telephone number of primary and specialty medical care providers;
– current therapies (e.g., physical, occupational, speech);
– hospitalizations/surgeries;
– anesthetic experiences;
– current medications;
– allergies/reactions to medications;
– other allergies/sensitivities;
– immunization status;
– review of systems;
– family history; and
– social history.

There should be areas on the form indicating the date of completion, the signature of the person providing the history (along with his relationship to

the patient), and the signature of the staff member reviewing the history with the parent. Records of patients with significant medical conditions should be marked ‘medical alert’ in a conspicuous yet confidential manner.

**Supplemental History for Infants/Toddlers.** The very young patient can present with unique developmental and social concerns that impact the health status of the oral cavity. Topics to be discussed may include a history of prematurity/perinatal complications, developmental considerations, feeding and dietary practices, timing of first tooth appearance, and tooth brushing initiation and timing as well as toothpaste use. Assessment of developmental milestones (e.g., gross/fine motor skills, language, social interactions) is crucial for early recognition of potential delays and appropriate referral to therapeutic services. Data gathered from this questionnaire will benefit the clinical examination, caries-risk assessment, preventive homecare plan, and anticipatory guidance counseling.

**Supplemental History for Adolescents.** The adolescent can present particular psychosocial characteristics that impact the health status of the oral cavity, care seeking, and compliance. Integrating positive youth development into the practice, the practitioner should obtain additional information confidentially from teenagers. Topics to be discussed may include nutritional and dietary considerations, eating disorders, alcohol and substance misuse, tobacco and electronic cigarette usage, over-the-counter medications and supplements, and body art (e.g., intra- and extra-oral piercings, tattoos), as well as the use of oral contraceptives and pregnancy for the female adolescent.

**Medical Update.** At each patient visit, the history should be consulted and updated. Recent medical attention for illness or injury, newly diagnosed medical conditions, allergy, and changes in medications should be documented. A written update should be obtained at each recall visit.

**Dental History.** A thorough dental history is essential to guide the practitioner’s clinical assessment, make an accurate diagnosis, and develop a comprehensive preventive and therapeutic program for each patient. The dental history should address the following:

– chief complaint;
– previous dental experience;
– date of last dental visit/radiographs;
– oral hygiene practices;
– fluoride use/exposure history;
– dietary habits (including breastfeeding, bottle/no-spill training cup use in young children);
– oral habits;
– sports activities;
– previous orofacial trauma;
– temporomandibular joint (TMJ) history;
– family history of caries;
– social development.

**Comprehensive Clinical Examination.** A visual examination should precede other diagnostic procedures. Components of a comprehensive clinical examination include:

– general health/growth assessment (e.g., height, weight, body mass index calculation, vital signs);
– pain assessment;
– extraoral soft tissue examination;
– TMJ assessment;
– intraoral soft tissue examination;
– oral hygiene and periodontal health assessment;
– assessment of the developing occlusion;
– intraoral hard tissue examination;
– radiographic assessment, if indicated;
– caries-risk assessment;
– assessment of cooperative potential/ behavior of child.

The dentist may employ additional diagnostic tools to complete the oral health assessment. Such diagnostic aids may include electric or thermal pulp testing, percussion, transillumination, caries detection devices, salivary tests, photographs, computed tomography (CBCT), laboratory tests, and study casts. Speech, in children who are able to talk, may be evaluated and provide additional diagnostic information. To enhance patient diagnosis and treatment documentation, the practitioner should consider including photographs of the child's oral condition in the dental record.

**Progress Notes.** An entry must be made in the patient's record that accurately and objectively summarizes each visit. The entry must minimally contain the following information:

• date of visit;
• reason for visit/chief complaint;
• radiographic exposures and interpretation, if any;
• preventive measures undertaken and materials used;
• treatment rendered including, but not limited to:
  – teeth restored and materials used;
  – the type and dosage of anesthetic agents;
  – medications, and/or nitrous oxide/oxygen;
  – type/duration of protective stabilization;
  – treatment complications; and
  – adverse outcomes; and
• post-operative instructions and prescriptions as needed.

A **visual examination** should be observed as the patient enters the office; take note of the patient's posture, mobility, facial asymmetries, scars, or lesions.

The examination of the soft tissues of the head and neck should be conducted to check for asymmetries, lymph node examination, and temporomandibular joint function.

If any enlargement, tenderness to palpation, ulceration, or abnormalities are present, this must be noted; the patient must be advised and treated accordingly.

Palpation of muscles of mastication for tenderness, swelling or asymmetry.

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. 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:

– general overview of exposed skin areas (head, neck, limbs);

– perioral skin areas (nose, cheeks, chin);

– lymph nodes (head, neck);

– cutaneous parts of upper and lower lips;

– Vermilion border and commissures;

– 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. Normally, the lymph nodes are painless on palpation, elastic, mobile, have a smooth surface.

A recommended order of examination includes the preauricular, submandibular, anterior cervical, posterior auricular and posterior cervical regions.

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 lymph nodes 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.

**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 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:

– labial mucosa and labial sulci (upper and lower);
– labial part of the commissures and buccal mucosa (right and left);
– tongue (dorsal and ventral surfaces, margins);
– floor of the mouth;
– hard and soft palate;
– 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. 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.

**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 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 (Fig. 2.1).

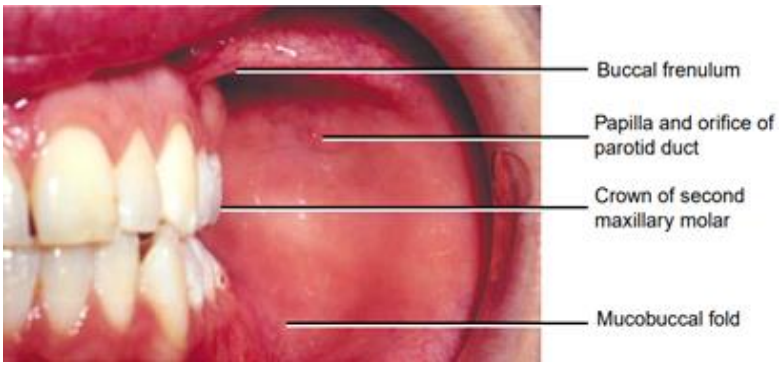


*Figure 2.1.* Buccal vestibulum and mucosa of the cheek

**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) (Fig. 2.2).

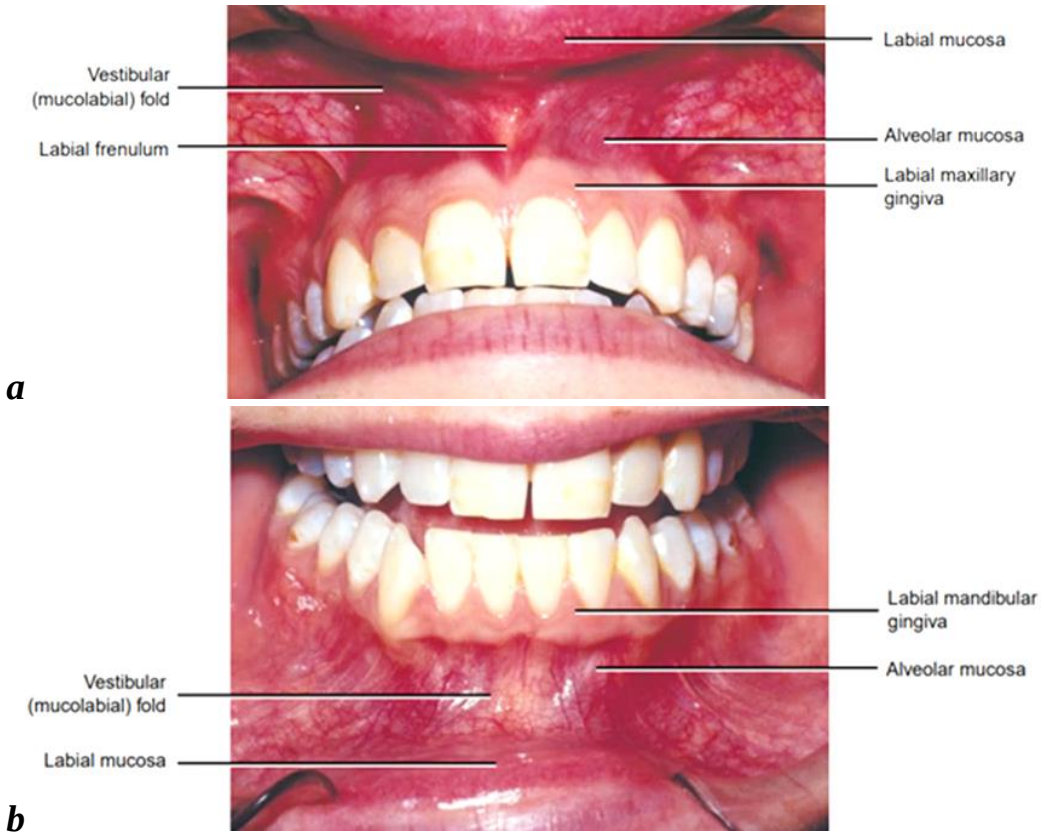


*Figure 2.2.* Vestibule and vestibular gingivae of the oral cavity:
*a* — maxilla; *b* — mandible

**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.

Normal occlusion occurs when the upper and lower arches interdigitate correctly, and is referred to as Class I occlusion. The following features are evident:

Centre lines of both arches are coincident.

Mesiobuccal cusp of the upper first molar lies in the buccal groove of the lower first molar.

Cusp of the upper canine lies in the point between the lower canine and first premolar.

Upper arch is slightly wider than the lower arch, so that the upper buccal cusps lie on the buccal sides of the lower teeth.

Horizontal overlap of the lower incisors by the upper incisors is between 2 and 4 mm — this is the overjet.

Vertical overlap of the lower incisors by the upper incisors is 50 % — this is the overbite.

Class I occlusion is illustrated in Fig. 2.3.

When the occlusion does not develop correctly, there is a degree of damage possible to individual teeth by the occlusal trauma produced when the arches bite out of synchrony with each other. Some teeth may experience heavy occlusal loading because of this, and may wear more easily, or fracture.

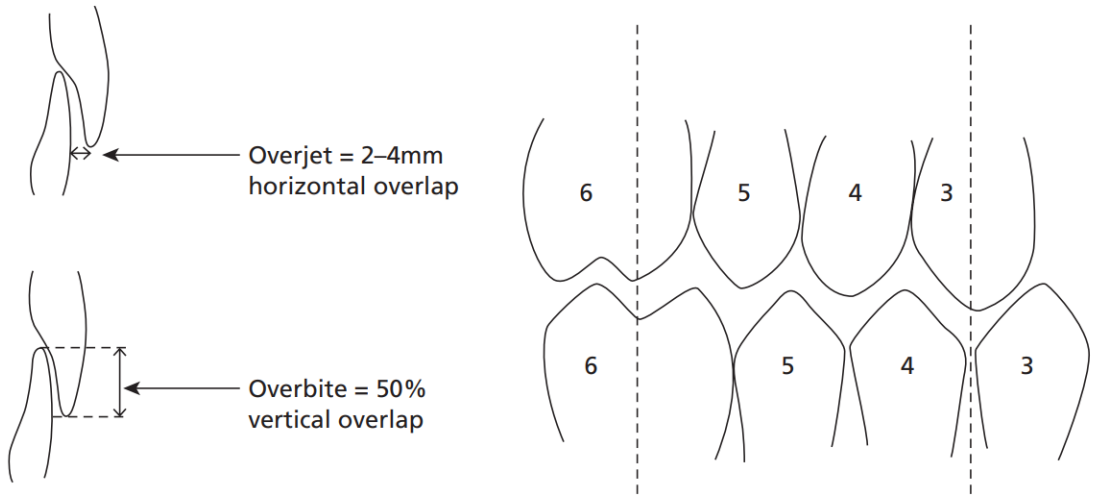


*Figure 2.3.* Class I occlusion

**Dentition Status.** Examine the teeth of the maxillary and mandibular arches. The complete permanent dentition contains 32 teeth; the child's dentition contains 20 primary teeth. By convention, the teeth are divided into four quadrants, and each quadrant is assigned a number (Fig. 2.4, Table 2.1).

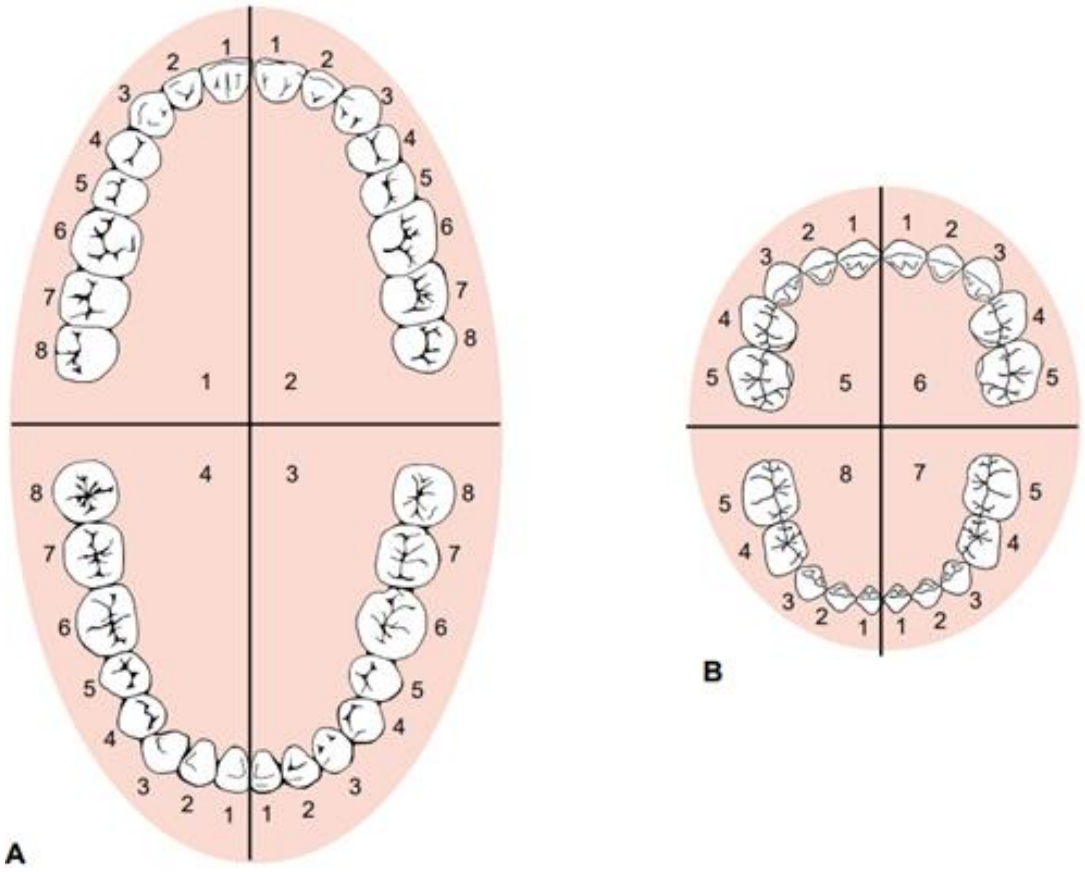


*Figure 2.4.* Numerical symbols for the dentition and quadrants:
*A* — adult; *B* — child

The permanent teeth in each quadrant are numbered from 1 to 8.
The primary teeth are numbered from 1 to 5 (Fig. 2.4, Table 2.2).

*Table 2.1*

**Numeration of quadrants in permanent and primary dentition**

| № of quadrant | Permanent dentition | № of quadrant | Primary dentition |
|---|---|---|---|
| 1 | upper right quadrant | 5 | upper right quadrant |
| 2 | upper left quadrant | 6 | upper left quadrant |
| 3 | lower left quadrant | 7 | lower left quadrant |
| 4 | lower right quadrant | 8 | lower right quadrant |

*Table 2.2*

**Numeration of teeth in permanent and primary dentition**

| № of tooth | Permanent teeth | № of tooth | Primary teeth |
|---|---|---|---|
| 1 | central incisor; | 1 | central incisor |
| 2 | lateral incisor; | 2 | lateral incisor |
| 3 | canine; | 3 | canine |
| 4 | first premolar; | 4 | first molar |
| 5 | second premolar; | 5 | second molar |
| 6 | first molar; | | |
| 7 | second molar; | | |
| 8 | third molar. | | |

Thus, the symbol for the lower right first molar is 4.6 (4 for lower right quadrant, 6 for first molar).

The examination for dental caries should be conducted with a plane mouth mirror. 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.

**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 2.3.

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.

*Table 2.3*

**Codes for the dentition status of primary and permanent teeth**

| 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) |

**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.
- 10: Indicating trauma, a 10 is used when a crown is fractured, with some of its surface missing but with no evidence of decay.

## Chapter 3
## THE ESTIMATION OF DENTAL CARIES INCIDENCE

The examination for dental caries should be conducted with a plane mouth mirror. 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.

To assess dental caries in a population, a DMFT/DMFS index is used. DMFT and DMFS describe the amount — the prevalence — of dental caries in an individual. DMFT and DMFS are means to numerically express the caries prevalence and are obtained by calculating the number of Decayed (D), Missing (M), Filled (F), teeth (T) or surfaces (S).

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 30 years and older, i.e. missing tooth due to caries or for any other reason. The "F" refers to teeth with code 3. Teeth coded 6 (fissure sealant) or 7 (fixed dental prosthesis/bridge abutment, special crown or veneer/implant) are not included in calculations of DMFT.

Individual DMFT value is the sum of the number of D (Decayed), M (Missing) due to caries, and F (Filled) teeth in the permanent teeth. 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.

Note: If a tooth has both a caries lesion and a filling it is calculated as D only. A DMFT of 28 (or 32, if "wisdom" teeth included) is maximum, meaning that all teeth are affected.

A more detailed index is DMF calculated per tooth surface, DMFS. Molars and premolars are considered having 5 surfaces, front teeth 4 surfaces. Again, a surface with both caries and filling is scored as D. Maximum value for DMFS comes to 128 for 28 teeth.

For the primary dentition, consisting of maximum 20 teeth, the corresponding designations are "deft" or "defs," where "e" indicates "extracted tooth."

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.

**Decayed, Missing and Filled Teeth (DMFT) Index.** 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).

DMFT is 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 teeth are examined. Teeth not included are:

– 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.

**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 i.e. total D, total M, total F. Total D + M + F = DMF SCORE

**Group Average DMFT.** Total the D, M and F for each individual. Divide the total DMF by the number of individuals examined.

$$\text{Average DMF} = \frac{\text{Total DMF}}{\text{Total Number Examined}}$$

**Treatment Needs**

$$\text{Percentage needing restorations} = \frac{\text{Total Number of D + M Teeth}}{\text{Total Number Examined}} \times 100$$

**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.

## DENTAL CARIES INDEX FOR PRIMARY TEETH (dmft and dmfs)

It is used for the primary dentition, consisting of maximum 20 teeth:

d = decayed primary teeth

m = 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 dmf
Total dmf score = d + e + f
Total dmfs score = d + e + f surfaces.

**Mixed Dentition.** In mixed dentition, DMFT or DMFS and a dmft and dmfs 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: DMFT + dmft.

**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.

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 "dmft" or "dmfs" (decayed, extracted, or filled).

**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 + dmft, dmft) 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.

**Dental Caries Reducing.** 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 of prevented dental caries in people of the highest possible. The calculation uses the rate (ΔDMFT) of index DMFT (DMFT + dmft, dmft). If reduction percentage is high, conducted preventive program in this group was effective.

Disadvantages:

– Requires a comparison group;

– The duration of the observations.

The Level of Dental Caries Intensity (LCI). This index was proposed by Prof. P. Leous. This index is calculated like the ratio between the individual value of the DMFT (DMFT + dmft, dmft) and the age. There are four levels of the dental caries activity: low, moderate, high and very high (Table 3.1).

*Table 3.1*

**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 |
| 9–19 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.

**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.

$$\textbf{Prevalence} = \frac{\text{Number of existing cases of dental caries at a point of time}}{\text{Total amount of examined persons at that point of time}} \times 100$$

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.

## Chapter 4
## PREVENTIVE DENTISTRY AS A PART OF MEDICINE. MODERN CONCEPT OF DENTAL CARIES PREVENTION

Preventive dentistry is the area of dentistry that focuses on those procedures and life practices that help people to prevent the beginning or progression of oral disease. It includes at-home dental care performed by patients, as well as dental care and education by professional dental staff in the office or clinic.

Preventive dentistry includes two aspects of dental care, both performed to help patients avoid dental disease or to catch it in its early, more treatable stages. In part, it is the oral hygiene care performed by the patient at home.

There are three levels of medical prevention: primary prevention, secondary prevention and tertiary prevention.

*Primary prevention* 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* involves identifying people in whom a disease process has already begun but who have not yet developed clinical signs and symptoms of the illness. 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* is slightly different than the first two categories because it involves reducing the long-term effects of a disease by helping patients manage their conditions and chronic symptoms. The goal is to achieve an improved quality of life and lengthen overall life expectancy by preventing complications in the future. Tertiary prevention is the most taxing on the health care system due to the substantial costs of surgery and lifelong management of chronic disease through medication and rehabilitation.

Most oral health conditions are largely preventable and can be treated in their early stages. Most cases are dental caries (tooth decay), periodontal diseases, tooth loss and oral cancers. Other oral conditions of public health importance are orofacial clefts, noma (severe gangrenous disease starting in the mouth mostly affecting children) and oro-dental trauma.

Dental caries is a destructive process causing demineralization of the tooth enamel and leading to continued destruction of enamel and dentin, and cavitation of the tooth.

**Etiological Theories of Dental Caries.** Different theories have been specified for the cause of dental caries which are as follows:

*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.

*Chemical theory.* It is often known as acid theory. This theory proposes that the putrefaction of protein leads to the production of acid formation in oral cavity producing ammonia and subsequently get oxidized to nitric acid which destroys the teeth.

*Parasitic theory.* This theory is also known as septic theory. It suggests that decomposition of the enamel and dentin are caused by filamentous microorganisms (denticolae) in the enamel cuticle and in carious lesions leads to tooth decay.

*Chemo-parasitic theory.* In this theory, W. D. Miller highlights that the fermentable carbohydrates are degraded to form acids by the metabolism and secretion of enzymes of different microbes present in the oral cavity. These acids demineralize the enamel and the disintegrated enamel is subsequently mechanically removed by force of mastication.

*Proteolytic theory.* Gottileb in 1947 provoked that microbes enter the organic pathways of the enamel and start caries by proteolytic action in this theory. Subsequently, the inorganic salts are dissolved by acidogenic bacteria.

*Proteolysis chelation theory.* This theory reveals that tooth decay initiates from an earliest bacterial and enzymatic proteolytic action on the organic matter of enamel without preliminary demineralization. This causes the emancipation of a several complexing agents such as aminoacids, polyphosphates and organic acids which soften the crystalline hydroxyapatite

**Modern Concept of Dental Caries Etiology.** 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 main features of the caries process are:

– fermentation of dietary carbohydrates by microorganisms in plaque to organic acids on the tooth surface;

– rapid lowering of the pH at the enamel surface to below the critical pH (5.5) at which enamel will dissolve;

– following plaque microbial metabolism the pH within plaque will rise due to the outward diffusion of acids and buffering so that remineralization of enamel occurs;

– demineralization and remineralization is an equilibrium so that dental caries progresses only when demineralization is greater than remineralization.

It is now established that dental caries is a multifactorial disease and results from a combination of four principal factors (Newbrun) (Fig. 4.1, Table 4.1): 1) host and teeth factors; 2) microorganism in dental plaque; 3) substrate (diet); 4) time.

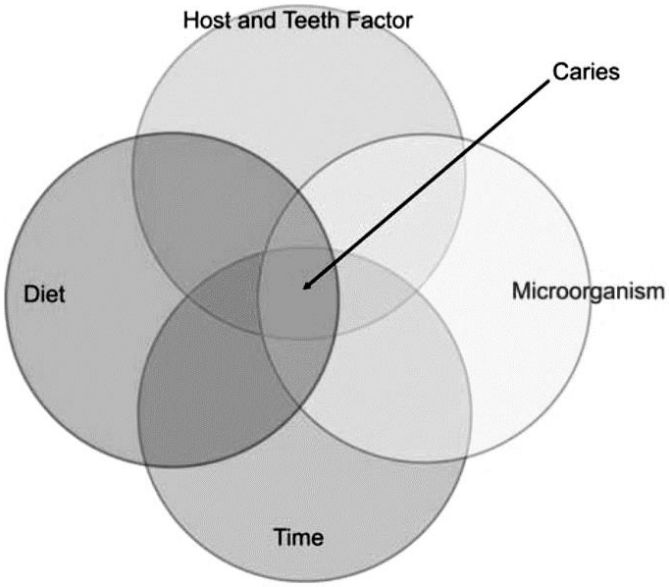


*Figure 4.1.* Four principal factors in dental caries (Adapted from Newbrun)

*Table 4.1*

**Factors affecting development of dental caries**

| **Host and Teeth Factors:** | **Agent Factors:** | **Environmental Factors:** |
|---|---|---|
| A. *Tooth*:<br>– composition;<br>– morphology;<br>– position | A. *Microorganism* | A. Diet:<br>– total consumption of carbohydrate;<br>– frequency and form of carbohydrate |
| B. *Saliva*:<br>– composition;<br>– buffering capacity of saliva;<br>– quantity | B. *Plaque* | B. *Geographic variation* |
| C. *Sex* | | C. *Climate* |
| D. *Age* | | D. *Oral hygiene* |
| E. *Race and ethnicity* | | E. *Soil* |
| F. *Socioeconomic status* | | F. *Fluoride* |
| G. *Heredity* | | |
| H. *Emotional disturbances* | | |

*Fermentable carbohydrate diet*. The formation of dental caries is associated with the carbohydrate component of the diet. Oral micro-organisms, especially S. mutans, utilize certain carbohydrates to form a sticky matrix that facilitate them to stick to the teeth. Organic acids are formed from the carbohydrates, which demineralize the teeth. The frequent consumption of soluble carbohydrate as well as their prolonged contact with tooth surfaces is highly significant risk factors.

*Microflora*. The microorganisms are responsible for dental caries. The early colonizers of these microorganisms are mainly mutans streptococci, produce large amounts of acid, especially lactic acid, which are potent in causing tooth demineralisation. Attachment of the S. mutans is now thought to be independent of sucrose and mediated by adhesions on the bacterial surfaces interacting directly with the salivary proteins, which form the pellicle on the tooth surface.

*Time.* Time is a significant factor in the progress of caries in relation to the frequency and amount of exposure of the liquid which will affect both the severity of the lesions and the number of teeth involved. The frequency of contact of the substrate has a major role in cariogenicity during a 24-h period.

*Susceptible tooth and host.* Implantation of S. mutans can occur only when teeth are present, because the teeth provide a non-shedding surface for colonisation of the micro-organisms. The amount of S. mutans depends on the number of erupted teeth present. Sometime after eruption, newly exposed enamel surfaces undergo the final stages of post-eruptive maturation and hardening. This period immediately after eruption and prior to final maturation, is when the tooth is most susceptible to caries. The presence of structural developmental defects in enamel may increase the caries risk and may manifest as partial or total loss of enamel (hypoplasia). Irregular surfaces such as pits and grooves lead to plaque retention, increased S. mutans and decreased elimination of carbohydrates. Dentin, when exposed, provides little resistance to acid attack.

*Saliva* can be considered as an environmental factor also as teeth are constantly bathed by it. This influences the process of dental caries. Saliva is supersaturated with calcium and phosphate which is important in determining the progression or arrest of caries.

*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.

## PREVAILING DIRECTIONS OF CAUSAL AND PATHOGENETIC CARIES PREVENTION

In dentistry there is no doubt that prevention is better than cure. Prevention of caries is so easy in theory but in practice involves many skills. There is no "magic bullet" that can be applied to teeth which will render them totally resistant to caries. The main reason for this is that control of the aetiological agents — plaque and fermentable carbohydrates — involve a change in behavior.

Prevention of dental caries underpins all dental care provided to children. All children require preventive input. The type of input depends on the child and their caries risk. Forming a comprehensive treatment strategy, tailored to the needs of each individual child, is an essential component of all paediatric treatment planning.

There are four practical pillars to the prevention of dental caries: plaque control/toothbrushing, diet, fluoride, and fissure sealing. Each of these will be considered in turn before being brought together in treatment planning and in relation to caries-risk.

The mainstay of preventive measures are:

– plaque control and regular toothbrushing with a fluoride toothpaste;

– sensible dietary advice;

– use of fluorides;

– fissure sealants;

– regular dental checks with appropriate radiographs.

All of these measures need to be coordinated and supervised by the dental team and reinforced with good patient and parental motivation.

Strategies that are consistent with the prevention of disease via the principles of the ecological plaque hypothesis include the following:

a) Inhibition of plaque acid production, e.g. by fluoride containing products or other metabolic inhibitors. Fluoride not only improves enamel chemistry but also inhibits several key enzymes, especially those involved in glycolysis and in maintaining intracellular pH. Fluoride can reduce the pH fall following sugar metabolism in plaque biofilms, and in so doing, prevent the establishment of conditions that favor growth of acid-tolerating cariogenic species. The value of fluoride is that it can be delivered in a variety of ways, some of which require minimal action by the patient.

b) Avoidance between main meals of foods and drinks containing fermentable sugars and/or the consumption of foods/drinks that contain non-fermentable sugar substitutes such as aspartame or polyols, thereby reducing repeated conditions of low pH in plaque.

c) The stimulation of saliva flow after main meals, e.g. by sugar-free gum. Saliva will introduce components of the host response, increase buffering capacity, remove fermentable substrates, promote re-mineralization, and more quickly return the pH of plaque to resting levels.

# Chapter 5
# THE ROLE OF MICROORGANISMS IN DENTAL CARIES DEVELOPMENT. DENTAL PLAQUE: STRUCTURE, PROPERTIES, ROLE IN DENTAL CARIES DEVELOPMENT

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.

The critical role of microorganisms was confirmed following the Second World War, initially when antibiotics were shown to prevent caries. Later, in a seminal study the development of germ-free (gnotobiotic) animal techniques allowed the conclusive demonstration of the essential role of dental microorganisms in the caries process. Germ-free rats were fed a highly cariogenic diet, developing no caries at all, whereas equivalent conventional animals developed numerous caries lesions. In the 1960s the classic experiments of Keyes and Fitzgerald shone the spotlight once again onto S. mutans, which was known to comprise a heterogeneous group with a range of serotypes. Four of these serotypes were subsequently elevated to species in their own right, S. sobrinus, S. rattus, S. ferus and S. cricetus — with S. mutans sensu stricto limited to the former serotypes c, e and f. Thereafter, this whole group of species have often been referred to collectively as ‘the mutans-streptococci’. Of these species, S. mutans and S. sobrinus were the only organisms found in moderate numbers or with regular frequency in humans.

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

**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. This layer is smooth, colorless and translucent and is called as acquired salivary pellicle. Acquired pellicle is an amorphous layer that forms over exposed tooth surfaces, as well as over restorations and dental calculus.

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

It is composed primarily of glycoproteins from the saliva that are selectively adsorbed by the hydroxyapatite of the tooth surface. Composition of the pellicle repeats mineral and organic composition of saliva differing only in the high concentration of proteins (particularly immunoglobulins G and M). Initially pellicle is bacteria free.

Glycoproteins of the first (subsurface) pellicle layer are chemically bonded to enamel. Acid groups are connecting 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.

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.

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. 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.

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

Dental plaque is the community of microorganisms found on a tooth surface as a biofilm, embedded in a matrix of polymers of host and bacterial origin. Plaque is natural and contributes (like the resident microflora of all other sites in the body) to the normal development of the physiology and defenses of the host.

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.

During and following outgrowth, other species colonize the forming biofilm by adhering to the pioneer species. Others include Neisseria and Actinomyces. 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 produces a climax community. The climax community is stable and complex yet responsive to changes in the environment.

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 matrix protects the bacteria from the defensive cells of the body (neutrophils, macrophages, and lymphocytes). 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 with in the biofilm structure.

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

**Steps of biofilm formation:**

a) Association — dental pellicle forms on the tooth & provides bacteria surface to attach.

b) Adhesion — within hours, bacteria loosely bind to the pellicle.

c) Proliferation — bacteria spreads throughout the mouth & begins to multiply.

d) Microcolonies — microcolonies are formed, streptococci secrete protective layer (slime layer).

e) Biofilm formation — microcolonies from complex groups with metabolic advantages.

f) Maturation — the biofilm develops a primitive circulatory system.

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

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.

The initial attachment of bacteria begins with pellicle formation. 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. 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.

Within the first few hours' species of Streptococcus and a little later Actinomyces attach to the pellicle and these are the initial colonizers. Bacteria are connected to the pellicle and each other with hundreds of hair-like structures called fimbriae.

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.

Once they stick, the bacteria begin producing substances that stimulate other free-floating bacteria to join the community. It appears that the act of attaching to a solid surface stimulates the bacteria to excrete an extracellular slime layer that helps to bond them to the surface and provides protection for the attached bacteria.

**Microorganisms of Dental Plaque.** From the 1960s onwards, a great range of clinical studies established a clear link between the presence of mutans-streptococci and susceptibility to and incidence of caries. In the same period, several studies were published connecting changes in the oral microflora to diet (specifically a high and frequent intake of fermentable sugar) and to dental caries. These studies established that S. mutans was obviously adapted for a glycolytic "lifestyle" and had a moderately higher rate of acid production from sugar than other oral streptococci.

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. An important factor in the colonization and establishment of S. mutans in the dental biofilm are mutacins (bacteriocins) produced by S. mutans.

The adhesion of S. mutans within dental plaque can be mediated via sucrose-independent as well as sucrose-dependent mechanism. Sucrose-independent adhesion, salivary components within the acquired enamel pellicle may initiate the attachment process whereas in sucrose-dependent adhesion,

sucrose can be principally accountable for developing colonization to the tooth surface (Fig. 5.1).

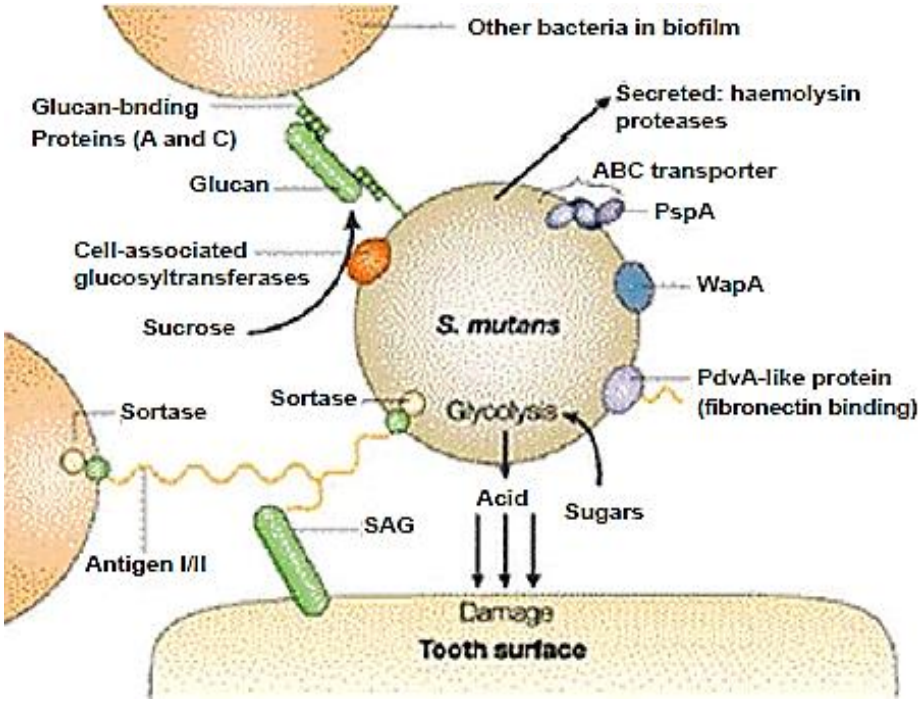


*Figure 5.1.* Adhesion

S. mutans contains glycolytic pathway which produce lactate, formate, acetate, and ethanol as fermentation products. The acidogenicity of S. mutans leads to ecological changes in the plaque flora that includes an elevation in the proportion of S. mutans and other acidogenic and acid-tolerant species.

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 well understood.

**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 microcolonies 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, Streptococcus sanguis, Actinomyces viscosus and Actinomyces 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.** Approximately 70 to 80 percent of plaque is microbial and the rest represents extracellular matrix.

The extracellular 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.

Dental plaque can be 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. 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). 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** is found below the gingival margins, between the tooth and the gingival sulcular tissue. 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.

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.

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.

**Progression of Dental Biofilm.** Dental plaque forms through a sequential event resulting in a structurally and functionally organized species rich microbial community. Once plaque forms, its species composition at a site is characterized by degree of stability among the component species. This stability is due to a balance imposed by numerous microbial interactions, such as conventional biochemical interactions where complex host glycoproteins catabolize to develop food chains and cell to cell signaling which leads to coordinated gene expression within the microbial community.

**Caries Transmission.** Cariogenic bacteria are not present in humans by birth. But, transmission of mutans streptococci is from caregivers, usually mothers, by mouth-to-mouth transmission via kissing or by sharing a spoon during feeding.

**Caries mechanism.** The bacterial flora and host defense systems are in the process of being established. Caries development is dependent on the following factors (Fig. 5.2):

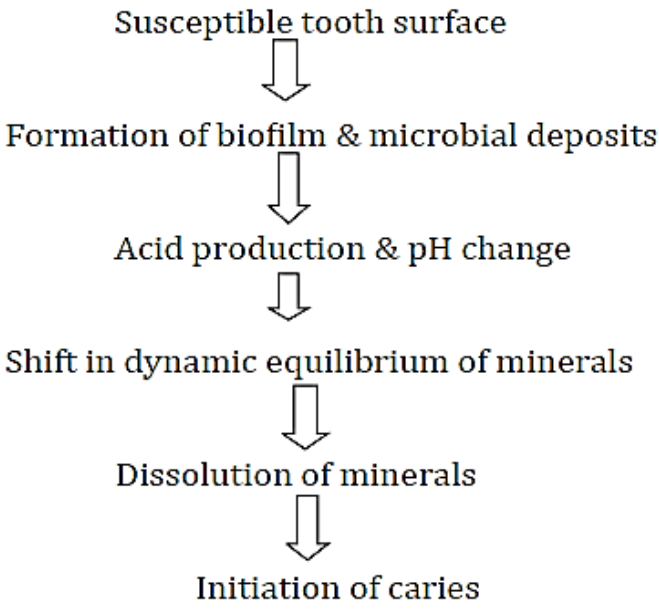


*Figure 5.2.* Illustrating caries mechanism

Dental caries is seen as a common and costly disease in the world that can greatly affect the health and quality of life. This review highlights significant role of the Streptococcus and Lactobacillus which are acidogenic and aciduric in the development of caries. Other Streptococci, Enterococci, Actinomycetes are also important etiological agents of dental caries. The population interactions are complex and apart from the commonly known mutualism, competitions etc. involve stress responses, adaptation, variation in gene expression, genetic variation and probably quorum sensing. The consumption of easily fermentable carbohydrates which stimulates the growth of oral microbes is the main cause of dental caries.

**Methods of Plaque Visualization.** 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.

Since dental plaque is white, it cannot easily be identified particularly if it is not thick enough. 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.

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;

– 1–3 Tetrazolium compound with methylene blue;

– 2-Tone.

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, especially on proximal surfaces. It may be associated with calculus deposit.

Indications:

– 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 coloring 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.

## MECHANISMS OF DEMINERALIZATION AND LESION PROGRESSION: PATHOGENESIS OF DENTAL CARIES

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 product 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. White spot lesions are also sometimes seen around orthodontic brackets when oral hygiene is not adequate and plaque has accumulated around the brackets.

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 subsurface body of the lesion and surface zone are usually seen in radiographs and histological sections of teeth. The surface layer forms by remineralization.

Progression of caries leads to weakening and breakdown of the surface layer producing a cavity with loss of both enamel and dentine.

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.

Periods of demineralization are followed by periods of remineralization and the balance of this equilibrium determines the outcome between mineral loss and gain. Healing of the carious lesion can only occur if the surface layer is unbroken but once a cavity has formed following surface breakdown a restoration is required. Early or "pre-cavitation" carious lesions are able to remineralize.

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 ("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).

## EVALUATION OF DENTAL PLAQUE CARIOGENIC ACTIVITY AND SALIVA

There is an association between types and numbers of bacteria in dental plaque and in saliva. Subjects with high number of cariogenic bacteria in saliva should be identified and treated before signs of clinical caries develop. The various kits that are available in the market for evaluation of dental plaque cariogenic activity:

– Strip Mutans test Dentocult® SM;
– Site-Specific Determination of Mutans Streptococci Levels;
– Lactobacilli test Dentocult® LB;
– CRT bacteria (Ivoclar Vivadent AG);
– and Saliva-Check Mutans (GC America, USA).

**Strip Mutans test Dentocult® SM** provides easy detection of Mutans Streptococci from a saliva sample and plaque. A chair-side method to estimate the number of mutans streptococci in saliva is available under the name of Dentocult® SM Strip Mutans. This test was developed in order to evaluate the level of Mutans streptococci in saliva.

The method is based on the use of a selective culture broth and the adherence and growth of Mutans Streptococci on the test strip. The results

reflect the number of Mutans Streptococci in saliva which colonize the tooth surfaces in the oral cavity. The mutans count is an important factor in making up the "Risk profile" of a patient.

Indications:

– for diagnosing S. mutans infected person in the family before eruption of a child's primary tooth;

– for demonstrating cariogenic infection;

– for evaluating the efficacy of Chemo-Therapeutic rinses.

The Strip Mutans test is available as a kit which includes:

– paraffin tablets for chewing to stimulate saliva secretion;

– the strip (a plastic strip with one side prepared to promote saliva and bacterial adhesion);

– selective broth for incubation;

– bacitracin tablets for making the broth selective;

– an incubator;

– an evaluation chart which shows numbers of mutans streptococci in four classes.

Eating, toothbrushing, smoking, taking snuff which can influence amount of Mutans Streptococci in saliva should be avoided before the test is performed.

The ***"Strip Mutans test"*** involves the following steps:

1. The broth is first activated by adding a bacitracin disc at least 15 minutes before use.

2. Bacitracin will inhibit growth of other bacteria than Mutans Streptococci.

3. The person to be sampled chews a piece of paraffin at one minute.

4. The paraffin is removed, and a plastic strip is rotated 10 times over the tongue to become contaminated by saliva.

5. The strip is removed through slightly closed lips to remove excess saliva, and inserted into the tube.

6. Incubate at 37 °C for 48 hours.

7. The strip is removed from the tube and dried at room temperature.

8. After 2 days, the numbers of adherent colonies are compared with a chart supplied by the manufacturer, and given a score between 0 and 3 to indicate low to high Mutans Streptococci levels.

9. For assessment of the level of Mutans Streptococci in saliva: **Mutans streptococci per ml saliva**.

Class 0–1 = < 100 000 CFU/ml saliva.

Class 2 = 100 000–1.000.000 CFU/ml saliva.

Class 3 = > 1 mill CFU/ml saliva.

Class 2 and 3 = high risk value.

**Site-Specific Determination of Mutans Streptococci Levels.** The method has similarities with the **"Strip Mutans test"** (Jensen and Bratthall 1989).

It consists of a plastic strip with four separate pads and a broth selective for mutans streptococci. After contamination with plaque bacteria, the strip is incubated at 37 °C in the broth. After about 48 hours, the number of colonies on

the strip is compared with a predetermined scale, and given a score from 0–3, where “0” indicates a very low yield and “3” a dense growth of mutans colonies on the strip.

How to take a sample (approximal surfaces):

– gentle air-blow the sites to be sampled;

– insert a toothpick until it reaches firm contact with the two approximal tooth surfaces;

– remove the toothpick and let it contaminate a pad on the plastic test strip. Move the toothpick in small circles over the pad with a gentle pressure, using one pad for each of the two sides of the toothpick;

– take further samples (if needed) from other surfaces of interest and use one pad for each surface sample until all four pads have become contaminated;

– incubate the strip in the broth at 37 °C (selective broth with bacitracin disc added in advance);

– remove after about 48 hours, dry, compare with chart (similar to the Strip Mutans test).

In a similar way, the method can be used for occlusal surfaces or for buccal/lingual surfaces — please observe that other sampling devices than toothpicks can be used, for example swabs.

***Lactobacilli test.*** A chair-side method to measure the level of lactobacilli in saliva is available under the name of **Dentocult® LB**.

The results reflect the number of Lactobacilli which colonize the tooth surfaces and the mucous membranes in the oral cavity. High numbers of Lactobacilli in saliva are usually related to high intake of carbohydrates and sugars. Also, retention sites such as fillings with over-hangs, bad margins etc. can increase the amount of Lactobacilli. Eating, tooth brushing, smoking, taking snuff which can influence amount of Lactobacilli in saliva should be avoided before the test is performed.

Dentocult® LB, a kit which includes:

– paraffin tablet for chewing to stimulate saliva secretion;

– dip-slide which on each side has a selective agar for Lactobacilli;

– an evaluation chart which shows numbers of Lactobacilli per ml saliva in four classes;

– a cup or tube;

– a funnel;

– an incubator.

The ***Lactobacilli test*** involves the following steps:

1. The person chews a piece of paraffin at least one minute.
2. The saliva is then spat out in a cup or tube.
3. The collected saliva is poured over both sides of the slide, the excess let to drip off.
4. Insert the slide into its plastic tube, and tighten it.
5. Incubate at 37 °C for 4 days.

6. After 4 days, the number of adherent colonies on the slide is compared with the chart. The result can differ on the two sides — the mean value will be used.

For assessment the level of lactobacilli: **Lactobacilli per ml saliva**

more than 100 000 ($10^5$) — high risk value,

less than 10 000 ($10^4$) — low.

**CRT bacteria** (Ivoclar Vivadent AG, Liechtenstein), enables the simultaneous determination of the Mutans Streptococci and Lactobacilli counts in saliva by means of selective agars.

The stimulated saliva is spit in the tube containing the media to which $NaHCO_3$ tablet is added at the bottom which produces $CO_2$ making conditions favorable for S. mutans and Lactobacilli growth. The blue mitis-salivarius-agar with bacitracin is used to detect Mutans Streptococci, while the light culture medium, Rogosa agar, is used to evaluate Lactobacilli.

Dentocult® SM Strip Mutans and CRT bacteria chair-side methods are highly specific and sensitive for S. mutans but takes 48 hours to test positive.

**Saliva-Check Mutans** (GC America, USA) detects S. mutans in saliva using a highly specific immunochromotography process. The S. mutans present in saliva react with colloidal gold labeled anti S. mutans monoclonal antibody which is contained in the test device. Thus, gold colloidal particles attach to the surface of S. mutans. This reacts with another S. mutans antibody to form the red line on the test window and to show positive results.

This kit test is highly specific and sensitive for S. mutans and gives results within 15 minutes, therefore it is considered as a powerful tool for rapid S. mutans detection.

# Chapter 6
# METHODS FOR REDUCING OF CARIOGENICITY OF DENTAL PLAQUE. THE ROLE OF DIET IN THE DEVELOPMENT OF DENTAL CARIES. PROBIOTICS

**Stages of Formation and Vital Activity of Dental Plaque Biofilm as a Target for Caries Prevention.** One of the main aims of caries prevention is the fight against etiological factors — cariogenic dental deposits on each step of their formation and manifestation (Fig. 6.1).

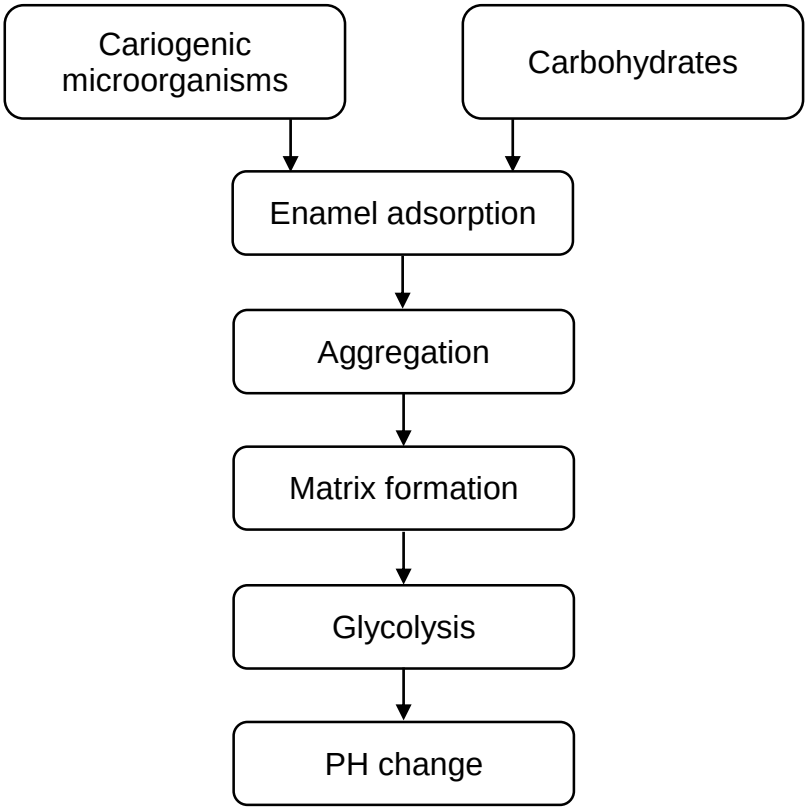


*Figure 6.1.* Stages of formation and activity of cariogenic dental deposits as objects of primary dental caries prevention

Targets for primary prevention of dental caries according WHO recommendations:

1) prevent the formation of cariogenic dental deposits by:

– reducing the content and the activity of substrates in the oral cavity, which can be used for dental plaque formation — cariogenic microflora and carbohydrates;

– preventing the plaque formation;

2) to prevent pH decreasing in the formed dental deposits by:

– decreasing of glycolytic activity (break the glycolysis);

– restore the pH balance using alkaline agents;

3) to prevent the maturation of dental deposits on all surfaces of the teeth, i.e. improve oral hygiene.

**Prevention of Colonization of the Oral Cavity by Cariogenic Microflora.**

*Prevention of early infection* of the baby's oral cavity with cariogenic microflora should be the earliest care of his/her family in relation to the prevention of dental caries. Child's teeth are colonized with streptococci in 90 % of cases from the mouth of a mother, grandmother or nanny — the caregivers. A significant problem is the licking of the comforter by the parent or sharing of utensils, which leads to parent-child bacterial transmission.

Streptococci are able to organize plaque as soon as a hard surface — the cutting edge of the first incisor — appears above the gum. If there is no cariogenic microbiota in the oral cavity at this time, the tooth surface will be colonized by a protective microbiota, which may further prevent colonization by S. mutans.

It is hardly possible to be free from cariogenic microflora throughout life, however, it is important to delay colonization for at least 1–2 years: primary teeth have time to strengthen during the posteruptive maturation, there are real opportunities for rationalizing the diet and its regime, for effective hygienic caring for a child's teeth (the dmft of preschoolers can be reduced by 2–3 times).

1) The prevention of caries in children should already start during pregnancy, this is called pre-prevention. The organism of the future mother undergoes numerous changes that increase the risk of oral cavity diseases through decreased saliva pH, worse self-cleaning of the oral cavity, as well as increased inflammatory response and weakened immune mechanisms.

The elimination of carious lesions in the mother and other people around the child reduces the risk of transmission of carious bacteria. Every pregnant woman should have at least one follow-up visit to the dentist, which should be used not only for dental treatment but also as an opportunity to provide the mother-to-be with information on caries prevention for her offspring.

2) It is necessary to exclude salivary contacts of the family with the child.

3) Human immunization against dental caries has been discussed since the early 1970s. Studies performed in numerous laboratories have demonstrated the feasibility of immunizing experimental rodents or primates with protein antigens derived from Streptococcus mutans or Streptococcus sobrinus against oral colonization by mutans streptococci and the development of dental caries. Protection has been attributed to salivary IgA antibodies which can inhibit sucrose-independent or sucrose-dependent mechanisms of streptococcal accumulation on tooth surfaces according to the choice of vaccine antigen. Strategies of mucosal immunization have been developed to induce high levels of salivary antibodies that can persist for prolonged periods and to establish immune memory. Studies in humans show that salivary antibodies to mutans streptococci can be induced by similar approaches, and that passively applied antibodies can also suppress oral re-colonization by mutans streptococci.

4) It is important to note the following: despite the fact that once introduced antibodies remain in the oral cavity for no more than 3 days, re-colonization of teeth with cariogenic microflora becomes possible only after a few months;

the trace effect is explained by a shift in the balance in the biocenosis, due to the action of antibodies. Several different antigen targets are used for active immunization. It has been proven that a one-and-a-half-month course of intranasal (or intraoral) administration of a suspension killed streptococci and lactobacilli to animals provides the production of IgA and IgG that block the adhesion of microorganisms, which is sufficient to reduce the two-year increase in caries by half.

A decrease in adhesive abilities is possible with active immunization to the fibrillar surface proteins of S. mutans or to the gene encoding the production of these proteins. When immunized to streptococcal glucosyltransferase (an enzyme that provides the synthesis of glucan from sucrose) or its components, the production of secretory IgA increases, which within 3 weeks provides a reduction in the number of streptococcus colonies on smooth surfaces of teeth by 50–85 %.

5) In dental prophylaxis, experience has been gained in the use of non-specific immunostimulants and adaptogens, which are usually prescribed to children and adults with high risk of dental caries.

6) WHO names the competitive colonization of the oral cavity with non-cariogenic microflora (replacement therapy, dental plaque design) among the promising areas of scientific development of caries prevention. Potential competitors include natural and genetically engineered microorganisms:

– related to S. mutans, and therefore capable to occupy their niche in the biocenosis of the oral cavity;

– more viable than S. mutans, and therefore able to displace them of the niche;

– having less strong ability for acid formation;

– genetically stable.

It has been proven that in cases of initial colonization of the tooth surface by S. sanguis, the titer of S. mutans does not rise till dangerous levels. The possibility of creating a hybrid of streptococcus and E. coli is being discussed. In an experiment with rats a reduction in dental caries was obtained when plaque was colonized by S. mutans strains in which the lactate dehydrogenase gene was replaced by the alcohol dehydrogenase gene.

The process of formation and maturation of dental plaque can be prevented by immunological, chemical, physical means and methods.

**Chemoprophylaxis and Chemotherapy of Soft Dental Deposits.** The formation of microbial plaque and its enzymatic activity can be influenced by various chemicals. It must be taken into account that the chemical control of plaque is carried out in specific conditions of the oral cavity:

– from the 400 species of oral microorganisms only 20–30 species are associated with pathology;

– an imbalance in the oral biocenosis leads to severe pathology (dysbacteriosis, candidiasis, etc.), so oral medications with antimicrobial properties should equally affect a wide range of opportunistic microflora in order to reduce its growth, but at the same time do not select resistant strains and do not break the balance overall;

– dental plaque microorganisms are protected from direct contact with medications by an adhesive, water-insoluble matrix;

– medications are quickly diluted with saliva in the mouth and swallowed, so their concentration quickly decreases. They get to the bloodstream and become systemic;

– oral medications should have pleasant taste;

– they can change the color of the oral tissues (mucous membrane, pellicle, etc.).

**Antiseptics.** Medications that negatively affect the vital processes of bacterial cells are highly toxic.

In 1962, Stralford proposed the following rule for calculating the effective dose of an antiseptic used during brushing: *the medication should suppress the reproduction of microorganisms for the next 6 hours; in the next 6 hours, the medication will not work, so the growth of microbial plaque will resume, but it will not have time to reach a pathogenic level by the time of the next cleaning with an antiseptic, which should be carried out 12 hours after the first.*

Over the past century, almost all classes of antiseptics have been used in dental plaque chemotherapy.

*Iodides* cannot be used daily for prophylactic purposes, since they bind irreversibly to the proteins of microbial membranes, damage them, thereby causing a bactericidal effect and leading to pathological changes in the oral microbiom.

*Chlorine-containing antiseptics* (chloramine, oxychlorozene) have an unpleasant taste, besides, chlorine ions in the oral cavity are quickly inactivated.

*Oxygen-containing antiseptics* (hydrogen peroxide 1–1.5 %, perborates) work effectively in the oral cavity for a short time, since the molecular oxygen they release is quickly blocked by enzymes (catalases) of the oral microflora.

*Zinc compounds (lactate)* have a bacteriostatic effect. It is known that the development of S. mutans is slowed down under the influence of zinc lactate. Zinc lactate inhibits the development of bacteria that produce volatile sulfur compounds, and also binds these compounds with the formation of insoluble derivatives, thereby eliminating bad breath. The substance retains in the oral cavity for 3–4 hours.

*Quaternary ammonium bases* (cetylpyridinium chloride 0.1–0.05 %, benzethonium chloride, defimen bromide, dodecyltriammonium chloride) have a bacteriostatic activity, but they have an unpleasant taste and relatively high toxicity.

*Benzene derivatives* — phenolic compounds — inhibit microbial enzymes and destroy the bacterial cell wall, therefore they have a wide spectrum of action. In preventive dentistry the use of thymol has the longest history. Being a component of mouth rinses, thymol provides a significant reduction in the mass of dental plaque. The negative effect of antiseptics of this class is the inhibition of gram-positive saprophytic microflora of the oral cavity.

The anticaries effect has been proven for two antiseptic from chlorhexidine and triclosan.

*Chlorhexidine* (CHX) is a cation-active bisguanide (Fig. 6.2).

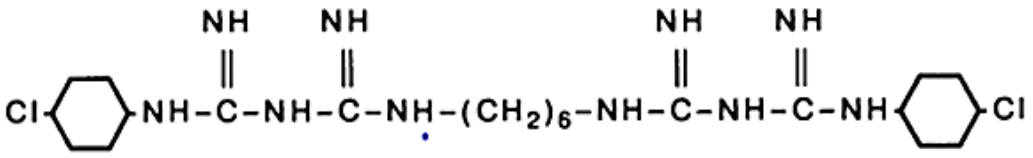

*Figure 6.2.* Chlorhexidine formula

Currently, CHX is recognized as the “gold standard” of chemical plaque control. CHX has a positive charge, therefore it is able to change the properties of the membranes of microorganisms that their integrity is affected: the cytoplasm starts to leak, precipitation of proteins inside the cell and its lysis begins. Therefore, CHX realizes a bactericidal effect for gram-positive and gram-negative microflora, as well as for fungi Candida. Due to the charge, CHX is absorbed by the tissues of the oral cavity and slowly released from them, and therefore has a prolonged effect.

In the 1980s–1990s, CHX was included in mouth rinses (0.05–0.2 %), gels (0.5–1.0 %), varnishes (10–20 %), and toothpastes. The modes of rinsing and applications of CHX are various:

1) 1 time per day for 2 weeks, repeat 3 times a year;

2) 3 times a day for 2 weeks, 3 courses per year;

3) 1 time per week for 1 month, 2 courses per year.

CHX gels are suggested to be used either daily for 1 week (4 courses per year), or 1 time per week for 1 month (2 courses per year).

Applications of CHX-varnishes are repeated monthly. It is recommended to apply varnish targeting the areas of greatest risk — the area of proximal contacts, on an erupting tooth, etc.

It has been shown that the systemic use of CHX medications for three years provides a 30 % reduction in DMFT, and the increase in caries of the proximal surfaces decreases by 50 times during this time.

However, CHX has serious side effects that limit its use. CHX is absorbed by the tissues in the oral cavity and colors them in a gray tint, has an unpleasant taste. It can cause burning of the oral mucosa. The CHX can be inactivated by stannous fluoride and sodium lauryl sulfate in toothpastes.

*Triclosan* is a bisphenol compound (2,2,4-trichloro-2-hydroxydiphenyl ether) with the chemical formula shown in the Fig. 6.3.

*Figure 6.3.* Triclosan formula

Triclosan is a low-toxic wide-spectrum antiseptic, active in low concentrations against gram-positive and gram-negative bacteria — prokaryotes and fungi, including the Candida genus.

Triclosan in bacteriostatic concentrations is used in the production of soaps, deodorants, rinses and toothpastes (0.2–0.3 %). Triclosan is well absorbed by plaque and remains active in it for 8 hours. The use of pastes with triclosan reduces the microbial number of plaque by 20 % and provides a reduction in caries by 25–30 %.

*Fluorides* have a bacteriostatic effect at concentrations of 0.01–0.1 % and a bactericidal effect at concentrations of 0.5–1 %.

The most important mechanisms of the antimicrobial effect of fluoride on S. mutans and Lactobacillus are:

– blockade of the mechanisms of glucose transport into the microbial cell;

– blockade of the ATP-dependent pump that removes H + through the microbial wall, which causes acidosis in the cell, a decrease in the activity of enzymes and acid production;

– in low pH environment fluorides penetrate into the cell as part of HF and can block enolase — the enzyme responsible for the transformation of 2-phosphoglycerate into phosphoenolpyruvate in the glycolysis cycle — energy and lactic acid production is disrupted in the cell;

– the inhibition of the metabolic processes in the microbial cell slows down the production of polysaccharides (levan and glucan), which are necessary for cell energy and for the formation of plaque;

– fluorides reduce bacterial production of lipoteichonic acid and reduce the adhesive ability of S. mutans.

As a result of the use of highly concentrated fluorides, plaque growth is reliably inhibited within 3–4 days, and the initial number of microorganisms is restored only by the 20th day.

*Stannum compounds* block the glycolysis. In addition, they are able to reduce the cohesion and adhesion of microbial cells in the process of plaque formation. This effect is used in oral products containing stannous fluoride $SnF_2$.

However, in an aqueous solution, hydrolysis of this compound and precipitation of its components occurs; in the presence of oxygen, the divalent tin ion is oxygenated and becomes tetravalent ($Sn^{4+}$). These chemical processes negatively affect the antimicrobial properties of fluoride, so tin fluoride is stabilized in pastes by combining it with aminofluoride.

*Lysozyme, lactoperoxidase, lactoferril* are synthetic analogues of the natural components of saliva with antiseptic properties. These products are included in oral hygiene products to reduce plaque formation and are often recommended for people with saliva deficiency.

**Antibiotics.** In monitoring people who take antibiotics (tetracycline, erythromycin, penicillin) for the treatment of chronic somatic diseases for a long time, it was found that these medications inhibit the development of caries. But the negative side effects of antibiotic therapy (dysbacteriosis, sensitization, the formation of resistant flora) hold back their use for the prevention of caries.

**Products for Modifying the Surface of the Tooth.** Some substances change the properties of the tooth surface and the pellicle in a way that

the formation of the first layers of plaque becomes impossible. These substances include, first of all, detergents (surfactants, surfactants) — aethonium, chlorhexidine, sodium lauryl sulfate, organic fluorides (aminofluorides) in the mouth rinses, toothpastes, gels.

Surfactants give the enamel hydrophobic properties and prevent the adhesion of plaque. The fixation of microorganisms on the tooth surface is difficult in the presence of some food substrates and their analogues that form hydrophobic coatings: essential oils (cinnamon, peppermint, spearmint, cloves) and creatinine (protein breakdown product).

Fluorides have a major role in the modification of the enamel surface, they can change the electrical potential of the surface and prevent attaching of bacteria.

Physical methods are proposed for changing the tooth surface's charge using an ionic toothbrush.

**Products for Controlling the Activity of the Formed Dental Plaque.** The next stages of the formation and vital processes of plaque depend on many conditions, one of which is the presence of food residues.

*Neutralization of the pH* in dental plaque is another chance to protect the enamel from acid attack. Neutralization is carried out by the natural buffer systems of plaque and saliva. Significant help is provided by salivation stimulators (xylitol).

Carbamide, i.e. urea, is a special additive in pastes and chewing gums. It dissolves into carbon dioxide and ammonia with alkaline properties and increases the pH level. Phosphate buffer compounds and baking soda in hygiene products serve the same purpose.

*The destruction* of the formed soft dental deposits can be done using chemical and physical mechanisms.

Chemical agents include surfactants that *loosen* the structure of plaque, as well as enzymes that destroy the organic matrix of plaque: lysozyme (including salivary), pancreatin, dextranase, ribonuclease. These medications are the components of many oral hygiene products.

## DIET AS CARIES-PRODUCING AND CARIES-PROTECTIVE FACTOR

Dental caries is a highly prevalent chronic disease and its consequences cause a lot of pain and suffering. Despite improved trends in levels of dental caries in developed countries, dental caries remains prevalent and is increasing in developing countries undergoing nutrition transition.

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).

There exists a biunique relationship between diet and oral health: a balanced diet is correlated to a state of oral health (periodontal tissue, dental elements, quality, and quantity of saliva). Vice versa an incorrect nutritional intake correlates to a state of oral disease.

Although food is a partial factor of dental disease, nutrition is available to influence preventive efforts of dentist.

Diet influences the health of the oral cavity, conditioning the onset of caries, the development of the enamel, the onset of dental erosion, the state of periodontal health, and of the oral mucous in general. Diet affects the integrity of the teeth: quantity, pH, and composition of the saliva; and plaque pH.

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.

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.

**The Structure and Biochemical Characterization of Carbohydrate Groups.** People include lots of carbohydrates into the diet. Oral bacteria use these carbohydrates for feeding, constructing matrix of dental plaque. This final product of this process is organic acid causing demineralization of enamel.

Dietary carbohydrates can be divided into simple sugars and complex carbohydrates, e.g., starches: A. Sugars; B. Starches.

**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 6.1). Monosaccharide contains 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 6.1*

**Classification of carbohydrates**

| Monosaccharides | Disaccharides | Polysaccharides |
|---|---|---|
| Glucose<br>Galactose<br>Fructose<br>Ribose<br>Glyceraldehyde | Sucrose<br>Maltose<br>Lactose | Starch<br>Glycogen<br>Cellulose |

Sucrose and starches are the predominant dietary carbohydrates in modern societies. 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.

**The Cariogenic Potential of Products Containing Both Simple or Complex Carbohydrates.** 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) 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. We should pay attention to those foods which have “hidden” sugar, as their original taste isn’t sweet (e.g., ketchup, canned meat, pickles).

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.

Starches are also a major component of the human diet. 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. 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 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.

**Dental Effects of Fruits and Vegetables.** 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 (Fig. 6.4).


*Fugure 6.4.* Food with 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.

**Dental Effects of Products Containing Carbohydrates.** 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[1] sugars.

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 microorganisms 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[2] sugar, 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.

[1] Fermentable carbohydrate: any carbohydrate that can be hydrolyzed by salivary amylase in the initial stage of carbohydrate digestion and subsequently fermented by bacteria.

[2] Cariogenic: foods and drinks containing fermentable carbohydrates that can cause a decrease in plaque pH to < 5.5 and demineralization of underlying tooth surfaces.

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. 6.5). Actually, it only measures the potential cariogenic challenge because the critical pH value varies between individuals.

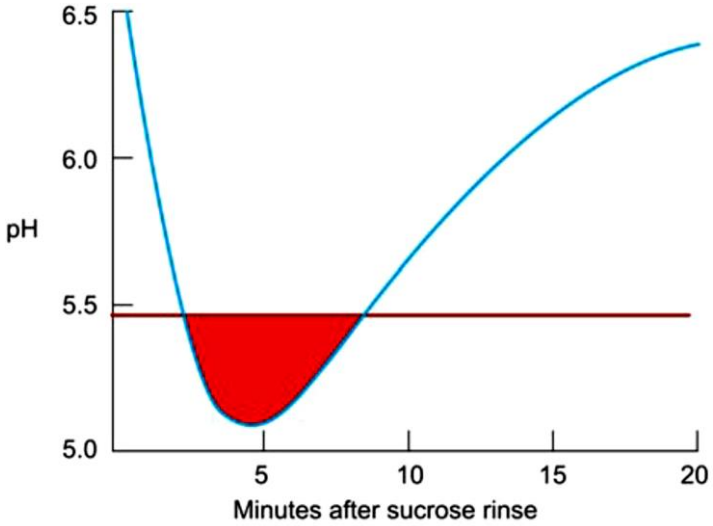


*Figure 6.5.* Stephan curve

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.

**Caries-Protective Effects of Protein and Fat.** 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.

*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.

**Ways of Reducing Carbohydrates Cariogenicity and Oral Care after Eating Carbohydrate Food.** 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.

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.

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. Evacuation of carbohydrates leads to the elimination of metabolism substrate for microorganisms, and therefore decreases its cariogenic effects. The same effect is produced by chewing gum chewing.

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).

*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.

There appears to be significant benefit to dental health through the use of sugar-free chewing gum. 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.

Chewing gum is employed to deliver a variety of agent for oral health benefits.

Clinical studies with CHX acetate (Chlorhexdine) or CHX-xylitol chewing gums used twice daily showed CHX is released after chewing and can reduce plaque and gingivitis. 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, omplementing 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.

***Dietary advice for prevention of dental caries:***

– Discourage prolonged on demand breast feeding (high lactose).

– Do not use sweetened drinks in a bottle or feeder cup.

– Recommend safer drinks (water, plain milk and tea) without added sugar.

– Recommend safer snacks (fruit, cheese, plain crisps, bread).

– Restrict sugary snacks to mealtimes or one day per week.

– Avoid chewy, sticky and boiled sweets.

– Be aware of hidden sugars (dried fruits like raisins, yoghurts, flavoured crisps and ketchup).

– Sugar-free chewing gums can help to stimulate salivary flow and buffer oral acidity.

***Dietary advice for prevention of dental erosion[3]:***

– Avoid acidic foods (citrus fruits, yoghurts, pickled food).

– Limit carbonated and fruit cordial drinks to once a day and preferably at mealtimes.

– Use a straw whenever possible.

– Do not hold or swish erosive drinks in the mouth.

– After an erosive challenge rinse with water or with an antierosive rinse.

– Eating cheese can raise the oral pH and limit the erosive attack

– Do not brush teeth after erosive challenges for at least 30 minutes.

[3] "Dental erosion is the progressive irreversible loss of dental tissue that is chemically corroded by extrinsic and intrinsic acids through a process that does not involve bacteria…"
Extrinsic Acids Derived from Diet — they citric, phosphoric, ascorbic, malic, tartaric, and carbonic acids that are found in fruit, in fruit juices, in drinks, and in vinegar. Intrinsic Acids — they are derived from serious gastroesophageal reflux.

## NON-SUGAR SWEETENERS (SUGAR-SUBSTITUTE)

***Sweeteners*** 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 (Non-caloric/Synthetic Sweeteners)** provide no energy but provide intense sweet taste. 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 tabletop 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. It is used in food products like soft drinks, beer, confectionary, desserts, ice-cream and jams.

*Stevioside* is a glycoside derived from the stevia plant, which can be used as a sweetener. Dried leaves, as well as aqueous extracts, from Stevia rebaudiana, a plant originating in South America, have been used for decades as a sweetener in many countries, notably in Latin America and Asia (Japan, China). The sweetening power of stevioside was estimated to be about 300 times stronger than cane sugar.

**Bulk Sweeteners (Caloric).** 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 Streptococcus mutans in plaque and saliva and by reducing the level of lactic acid produced by these bacteria.

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.

Thus, 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.

WHO recommends that free sugars (non-milk extrinsic or refined) 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.

## PROBIOTICS

There has been a paradigm shift away from treating dental diseases by targeting specific oral pathogens towards an ecological and microbial community-based approach to understand conditions, such as caries and periodontal diseases. These approaches recognise the importance of maintaining the natural balance of the resident oral microbiota and the need to carefully modulate host immune responses to the microflora at a site.

One approach that has gained interest over recent years is the use of probiotic bacteria for oral applications. Probiotics are defined as viable microorganisms that confer health benefit when administered in sufficient doses. The organisms that have been used as probiotics are primarily certain species of lactobacilli and bifidobacteria, and Saccharomyces spp., but some streptococci, enterococci and commensal Escherichia coli have also been claimed to have beneficial effects in certain situations. The diversity of conditions that may benefit from ingestion of probiotics illustrates the variety of mechanisms that may be involved in their actions and that some effects are systemic rather than only local. It is likely that these mechanisms vary according to the specific strain or combinations of strains used, the presence of prebiotics and the condition that is being treated, as well as the stage of the disease process in which the probiotic

is administered. There are common themes emerging in studies of the modes of action of probiotics and numerous mechanisms have been proposed including:

– prevention of adhesion of pathogens to host tissues;

– stimulation and modulation of the mucosal immune system;

– increasing production of anti-inflammatory cytokines;

– improvement of intestinal barrier integrity and upregulation of mucin production;

– killing or inhibition of growth of pathogens through production of bacteriocins or other products, such as acid or peroxide, which are antagonistic towards pathogenic bacteria.

Dairy products such as milk, yogurt, and cheese have been selected as delivery vehicles for the selected bacteria. Probiotics can also be delivered by lozenges, powder, gelatine, straw, or tablets; by chewing gum, toothpastes, gels; however, the best vehicle for probiotic delivery has yet to be identified.

In daily routines, the administration of probiotics to breastfed children and toddlers is difficult. The pacifier can be used as a vehicle to transport essential nutrients to the infant's body (Fig. 6.6), but the pacifier is also a risk for mother — child S. mutans transmission. The later the primary teeth are colonized by mutans streptococci, the less likely that dental caries will develop, if at all. Since up to 90 % of teething children develop a sucking habit pacifier were recently designed to release preventive agents into the oral cavity.

*Figure 6.6.* The pacifier with probiotics

Clinical studies have indicated that bacteria with established probiotic effects (lactobacilli and bifidobacteria) have some promise for prevention of caries. LGG ingested in dairy products (milk, cheese) reduced salivary mutans streptococcal counts in adults and protected against caries in children. Other lactobacilli have also been shown to reduce mutans streptococcal counts in saliva. Lactobacillus reuteri, when delivered by yoghurt, straw or tablet, by chewing gum or as a lozenge, significantly reduced the counts of mutans streptococci in saliva. The short-term consumption of yoghurt or ice cream containing Bifidobacterium spp. resulted in a significant reduction in salivary mutans streptococci but not in lactobacilli. There is increasing evidence that the use of existing probiotic strains can deliver oral health benefits.

# Chapter 7
# FACTORS DETERMINING SUSCEPTIBILITY TO CARIES. EVALUATION AND CORRECTION OF THE DIET

**Terms of Initiation, Mineralization and Eruption of Teeth in Children.** Teeth start to form during weeks 6–8 of embryonic life, and the process of tooth formation continues until the roots of the third permanent molars are completed at about 20 years of age. The stages of tooth formation are the same whether the tooth is of the primary or the permanent dentition, although obviously the teeth develop at different times. The tooth-germs develop from the dental lamina, a sheet of epithelial cells which itself develops from the primary epithelial band. The dental lamina forms a series of epithelial buds that grow outwards into the surrounding connective tissue. These buds represent the first stage in the development of the tooth-germs of the primary dentition.

Once dentine formation begins, the cells of the internal enamel epithelium differentiate into ameloblasts and commence the formation of enamel. Dentine and enamel formation initially occur in the region of the cusp tips and incisal edges of the teeth, and then continues towards the cervical margin of their crowns (Fig. 7.1). The epithelial cells — the root sheath of Hertwig — induce the formation of odontoblasts from the cells of the dental papilla and the production of dentine to form the roots of the teeth, a process which is not complete until 3–5 years after the eruption of the crown of the tooth (Table 7.1, 7.2).

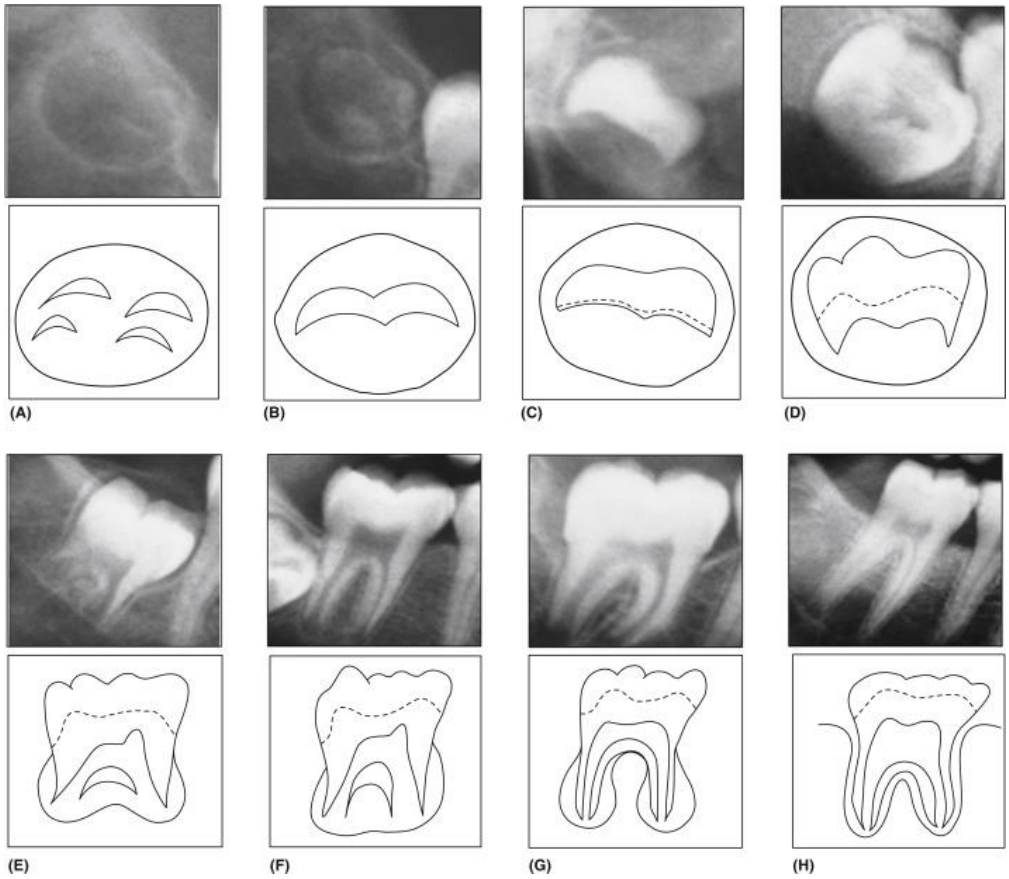


*Figure 7.1.* Stages of tooth development

*Table 7.1*

**The chronology of the development of the primary dentition**

| Stage of development | Central incisor | | Later incisor | | Canine | | First moral | | Second moral | | Time |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Max | Mand | Max | Mand | Max | Mand | Max | Mand | Max | Mand | |
| Hard tissue formation begins | 13–16 | 13–16 | 14.7–16.5 | 14.7–16.5 | 15–18 | 16–18 | 14.5–17 | 14.5–17 | 16–23.5 | 17–19.5 | Weeks after ovulation |
| Crown formation complete | 1.5 | 2.5 | 2.5 | 3 | 9 | 8–9 | 6 | 5–6 | 11 | 8–11 | Months after birth |
| Beginning of eruption | 8–12 | 6–10 | 9–13 | 10–16 | 16–22 | 17–23 | 13–19 | 14–18 | 25–33 | 23–31 | Months after birth |
| Completion of root formation | 33 | 33 | 33 | 30 | 43 | 43 | 37 | 34 | 47 | 42 | Months after birth |

*Table 7.2*

**The chronology of the development of the permanent dentition**

| Stage of development | Central incisor | | Later incisor | | Canine | | First premolar | | Second premolar | | Time |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Max | Mand | Max | Mand | Max | Mand | Max | Mand | Max | Mand | |
| Hard tissue formation begins (histology) | 3–4 | 3–4 | 10–12 | 3–4 | 4–5 | 4–5 | 18–24 | 18–24 | 24–30 | 24–30 | Months after birth |
| Hard tissue formation begins (radiology) | – | – | – | – | 6 | 6 | 19 | 19 | 36 | 36 | Months after birth |
| Crown formation complete | 3.3–4.1 | 3.4–5.4 | 4.4–4.9 | 3.1–5.9 | 4.5–5.8 | 4.0–4.7 | 6.3–7.0 | 5–6 | 6.6–7.2 | 6.1–7.1 | Years of age (decimal) |
| Beginning of eruption | 6.7–8.1 | 6.0–6.9 | 7.0–8.8 | 6.8–8.1 | 10.0–12.2 | 9.2–11.4 | 9.6–10.9 | 9.6–11.5 | 10.2–11.4 | 10.1–12.1 | Years of age (decimal) |
| Completion of root formation | 8.6–9.8 | 7.7–8.6 | 9.6–10.8 | 8.5–9.6 | 11.2–13.3 | 10.8–13.0 | 11.2–13.6 | 11.0–13.4 | 11.6–14.0 | 11.7–14.3 | Years of age (decimal) |

| Stage of development | First moral | | Second moral | | Third moral | | Time |
|---|---|---|---|---|---|---|---|
| | Max | Mand | Max | Mand | Max | Mand | |
| Hard tissue formation begins (histology) | 7–8ao | 7–8ao | 33–36mo | 33–36mo | 7–9yr | 8–10yr | Months after ovulation (ao) or months (mo) and years (yr) after birth |
| Hard tissue formation begins (radiology) | 2mo | 2mo | 36–48mo | 36–48mo | 9–10yr | 9–10yr | months (mo) and years (yr) after birth |
| Crown formation complete | 2.1–3.5 | 2.1–3.6 | 6.9–7.4 | 6.2–7.4 | 12.8–13.2 | 12.0–13.7 | Years of age with decimal fractions |
| Beginning of eruption | 6.1–6.7 | 5.9–6.9 | 11.9–12.8 | 11.2–12.2 | 17.0–19.0 | 17.0–19.0 | Years of age with decimal fractions |
| Completion of root formation | 9.3–10.8 | 7.8–9.8 | 12.9–16.2 | 11.0–15.7 | 19.5–19.6 | 20.0–20.8 | Years of age with decimal fractions |

The process of enamel formation is referred to as amelogenesis. Enamel development involves two major functional stages, secretory and maturation, with a brief transition between the two stages. During the secretory stage, ameloblasts are highly polarized cells. These cells synthesize and secrete a limited number of structural enamel matrix proteins, most notably amelogenin, ameloblastin and enamelin. They are all members of the secretory calcium-binding phosphoproteins.

Although crystal growth takes place during both the secretory and maturation stages, it is during the maturation stage that the crystals greatly expand in width and thickness, giving enamel its characteristic durability and hardness. During enamel maturation the transport of ions occurs.

**Crystal Structure of Apatite.** Calcium ($Ca^{2+}$) and phosphate ($PO_4^{3-}$) ions are only sparsely soluble in water and thus precipitate at rather low concentrations as a crystalline or amorphous solid. Under physiological conditions, apatite has the lowest solubility among the calcium phosphate minerals and is therefore the most chemically stable mineral phase.

In saturated aqueous calcium phosphate solutions with physiological range of pH (6.0 to 7.4), precipitation of stoichiometric hydroxyapatite (Hap) can occur according to the following reaction:

$$10Ca^{2+} + 6HPO_4^{2-} + 2H_2O \rightarrow Ca_{10}(PO_4)_6(OH)_2 + 8H^+$$

The unit cell (the simplest repeating unit) of Hap corresponds to the chemical formula $Ca_{10}(PO_4)_6(OH)_2$. There are two types of $Ca^{2+}$ positions in the Hap-lattice, of which Ca is unique as it forms channels that allow anions to move along the c-axis of the apatite crystal. Hydroxyl ions are able to diffuse and be replaced by other ions such as fluoride ($F^-$), carbonate ($CO_3^{2-}$), or chloride ($Cl^-$) from aqueous solutions. This makes apatite composition highly adaptable to its solution environment, which is critical to its properties in biological apatites. Carbonate ions ($CO_3^{2-}$) can also replace two hydroxyl groups along Ca channels. Carbonate substitutions for phosphate ($PO_4^{3-}$) or hydroxyl ions ($OH^-$) affect the ideal crystal structure of apatite and lower its symmetry, resulting in lower binding energies and ultimately increase the chemical solubility of the mineral phase. Carbonated apatite is therefore much more susceptible to acidic dissolution and dissolves at pH around 5, which is readily produced by cariogenic (caries-producing) bacteria. In contrast, $F^-$ fit perfectly between Ca triangles and stabilize the hexagonal symmetry and crystal lattice. Exchange of $CO_4^{3-}$ for $F^-$ therefore lowers the solubility by at least three orders of magnitude, and fluoroapatite can withstand a pH as low as 4 without dissolution. This partly explains the high benefit of fluoride supplements in toothpastes and drinking water for caries prevention and erosion reduction in teeth.

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.

Morphologic features which may predispose 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. 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.

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.

Diet influences the development of the oral cavity: depending on whether there is an early or late nutritional imbalance, the consequences are certainly different. In fact, an early nutritional imbalance influences malformations most.

Insufficient number of vitamins and minerals has negative effect on development of the fetus, maxillofacial area, dental organogenesis.

An insufficient supply of proteins can lead to the following:

– atrophy of the tongue papillae;
– degeneration of the connective tissue;
– violation in dentinogenesis;
– violation in cementogenesis;
– development violation of the maxilla;
– malocclusion;
– prenatal hypoplasia of the enamel.

An insufficient supply of fats can lead to the following:

– inflammatory and degenerative pathologies;
– parotid swelling — hyposalivation;
– degeneration of glandular parenchyma;
– violation of mucosal trophism.

An insufficient supply of carbohydrates can lead to the following:

– altered organogenesis;
– influence of the metabolism on the dental plaque;
– dental caries development;
– periodontal diseases.

**Disturbances of Enamel Development.** A lack of vitamins D and A and protein-energy malnutrition have been associated to hypoplasia of the enamel and atrophy of the salivary glands, conditions that determine a susceptibility to

caries. Hypoplasia and dimples of the enamel correlate to a lack of vitamin A (Fig. 7.2); a lack of vitamin D is associated to the more diffused hypoplastic defects (Fig. 7.3).

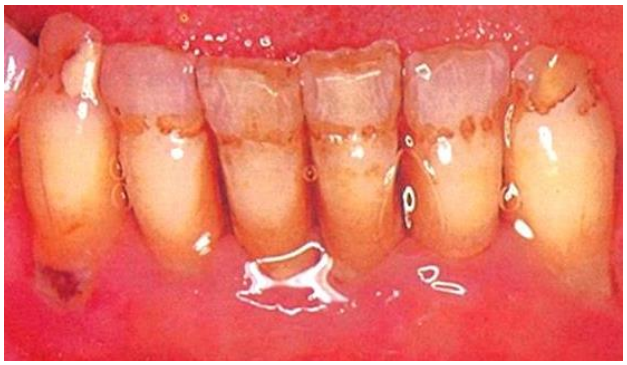

*Figure 7.2.* Hypoplasia and dimples on the enamel due to vitamin A deficiency

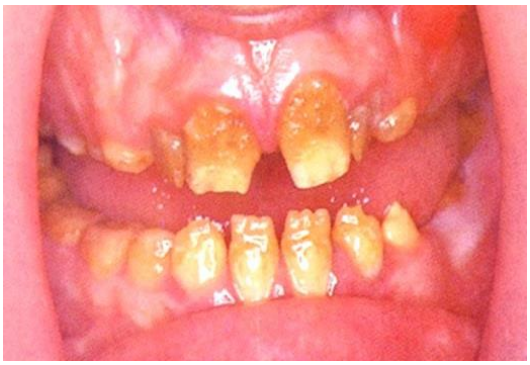

*Figure 7.3.* Hypoplasia of the enamel due to vitamin D deficiency

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.

Person should get during the day:

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

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

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

– calcium 1–2 g;

– fluorides 0.02–0.05 mg/kg.

Nowadays the features of the diet among population are protein deficiency, excess animal fat and carbohydrates (sugars), deficiency of calcium, phosphorus, fluorides, vitamins A and D. That's why the systemic prevention of dental caries is very important for the preeruptive development of caries resistant enamel. Vitamins, adaptogens, immunostimulants, anabolic steroids, mineral supplements are widely used for this purpose in patients with high risk of dental caries.

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 important 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. 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 recommended to combine calcium supplement drugs with vitamin D.

For evaluation and diet correction a food diary can be useful. The diary helps the patient and the doctor understand eating habits. It can help to realize what the patient consumes. It can help to make changes to the diet to improve it. The patient can keep track on paper. There are also many apps that can keep track of food and drink consumed during the day. After that the dentist should analyze the results (frequency, amount of proteins, fats, carbohydrates, minerals and vitamins) and make the corrections.

## Chapter 8
## SALIVA: COMPOSITION, FUNCTIONS, PROPERTIES. VIOLATION OF SALIVATION

Saliva is a secret of three pairs of major (названия) and hundreds of minor salivary glands scattered around the oral cavity (the mixed saliva) and number of components of not salivary origin (epithelium, microorganisms, blood cells, food remains).

**Composition of saliva:** varies from person to person. There are essential distinctions in structure and quality of secrets of various major and minor salivary glands, stimulated and nonstimulated saliva.

Approximately 1.0–1.5 litres of saliva is secreted by healthy persons each day.

Saliva is dilute fluid; contains 99.5 % of water and 0.5 % of inorganic (2/5) and organic (3/5) substances.

*Organic Components*

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

– Enzymes: α-Amylase.

– Immunoglobulins — secretary IgA.

– Antibacterial proteins — Lysozyme, Lactoferrin, Sialoperoxidase.

– Glycoproteins.

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

2. Other organic compounds:

– Free amino acids.

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

– Glucose.

*InOrganic Components:*

– 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).

**Production of Saliva.** Salivary glands activity is regulated by different departments of nervous system. The parasympathetic system supervises formation of an inorganic part of a saliva. Inorganic components such as water, macro- and microelements are arriving from blood. The sympathetic system is responsible for synthesis of organic substances in cells of glands. But there is a considerable cross-talk and synergism between the two regulatory pathways.

Salivary water and electrolyte secretion is an energy consuming active two-stage process. First the acini secrete isotonic primary saliva into the luminal terminal end pieces of the gland parenchyma. This saliva is then modified by electrolyte reabsorption to form a hypotonic secretion in the ductal systems. Salivary proteins are continuously synthesized, and stored in secretory granules

within the cell. Both electrolyte and protein secretion are highly regulated processes (Fig. 8.1).

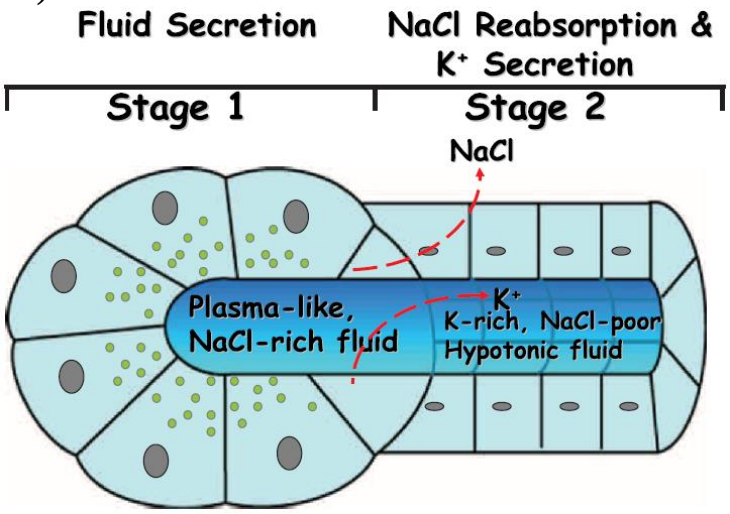


*Figure 8.1.* Two stage salivary gland secretion model

**Primary Saliva Secretion Model.** Acinar cells secrete fluid in a $Cl^-$-dependent manner. The coordinated activity of ion channels, water channels, pumps, cotransporters and exchangers result in the primary saliva formation. The molecular components of this physiological process and their gene names are shown. Individual acinar cells express all of the different transport mechanisms, but are spread out for clarity (Fig. 8.2).

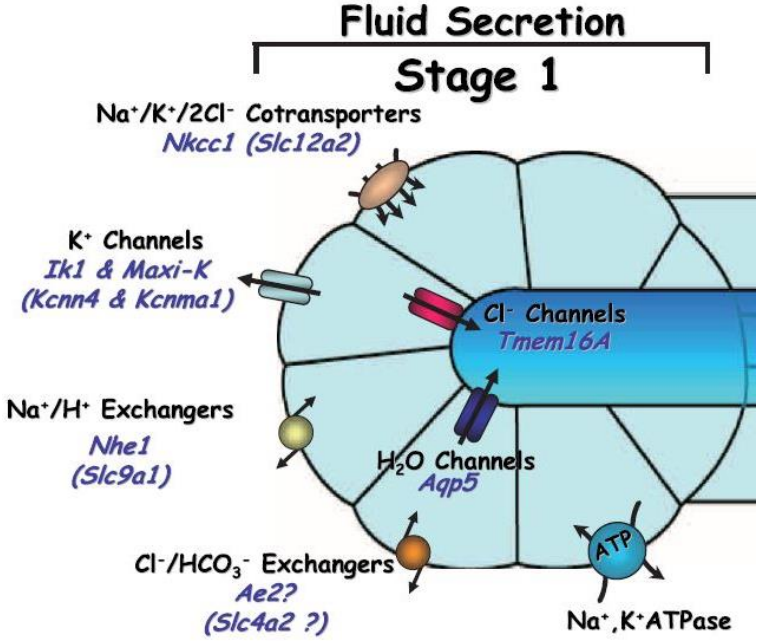


*Figure 8.2.* Primary saliva secretion model

**Ductal Function Model.** Salivary gland duct cells reabsorb NaCl and secrete KHCO3. NaCl reabsorption exceeds KHCO3 secretion resulting in a hypotonic final saliva. Ion channels, pumps and exchangers involved in ductal function are shown in Fig. 8.3.

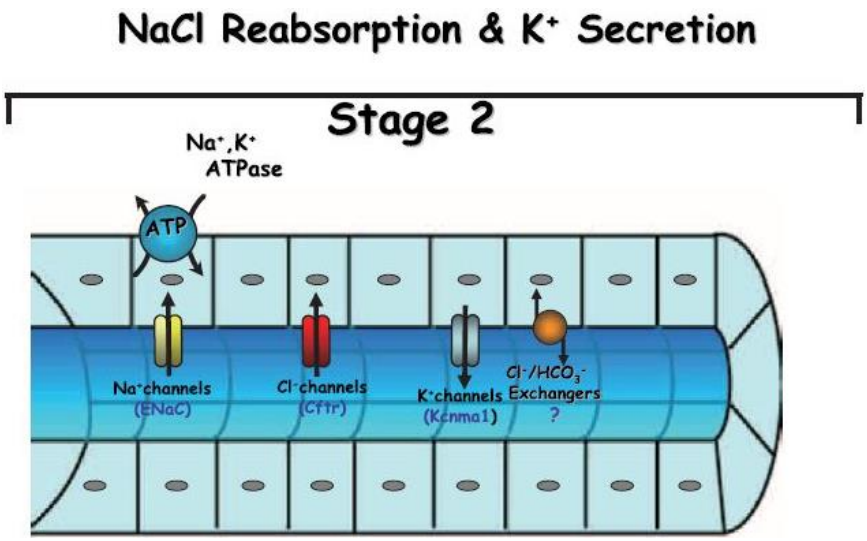


*Figure 8.3.* Ductal function model. Salivary gland duct cells reabsorb NaCl and secrete $KHCO_3$

In stage 1, acinar cell secrete a NaCl-rich fluid called primary saliva. In stage 2, the primary saliva is subsequently modified through its passage along the ductal tree mostly by reabsorbing NaCl and secreting $KHCO_3$. Because the ductal epithelium is poorly permeable to $H_2O$, the final saliva is hypotonic.

Acinar cells secrete fluid in a $Cl^-$-dependent manner. The coordinated activity of ion channels, water channels, pumps, cotransporters and exchangers result in primary saliva formation. The molecular components of this physiological process and their gene names are shown. Individual acinar cell expresses all of the different transport mechanisms, but are spread out for clarity.

NaCl reabsorption exceeds $KHCO_3$ resulting in a hypotonic final saliva. Ion channels, pumps and exchangers involved in ductal function are shown in this figure.

**Functions of Saliva.** Saliva carries out both *systemic* and *local* functions.

At the *systemic level* saliva participates in regulation of metabolism processes, blood pressure, adaptive reactions.

At the *local level (in oral cavity)* saliva carries out:

– protective function (antimicrobial and mechanical protection for soft and hard tissues of oral cavity);

– trophic function (stimulates epithelium regeneration, provides conditions and delivers materials for enamel and cement (re)mineralization);

– alimentary function (carries out fermentation of complex carbohydrates, ensures the effectiveness of the food taste recognition, facilitates mastication and swallowing);

– speech function.

Saliva has a critical role to play in the *development of caries* or *its prevention*. Among the main cariesprotective mechanisms are:

– *pellicle and biofilm formation* — basic component of pellicle and biofilm organic matrix is glycoprotein (salivary mucin);

– *antimicrobial activity* (provided by proline rich proteins, lysozyme, lactoferrin, sialoperoksidase, immunoglobulins, gistatin peptides);

– *clearance, buffering and neutralization* (diluting and taking out with the swallowed saliva) of food residues and microflora products (acids) and their neutralization by buffer systems (carbonate, phosphate, ammonium, protein). 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;

– *participation in posteruptive mineralization and (re)mineralization* of dental hard tissues (optimum saliva supersaturated by calcium and phosphate. Calcium and phosphate are the components of the apatites which hinders their dissolution and helps crystals to reprecipitate on the surface of damaged enamel structures. Ions of calcium and phosphate form multilayer structures (micelles) preventing deposition of ions in a neutral medium).

The viscosity of saliva largely depends on the content of mucin in it, a long glycoprotein polymer secreted by the acinar cells of the salivary glands. The most viscous is the saliva of the sublingual glands (13.4 poise), the saliva of the submandibular and small glands is medium-viscous (3–5 poise), and the most fluid is the saliva of the parotid glands (1.5 poise). The viscosity of saliva determines its surface properties and allows it to form protective films on the surface of the oral mucosa and on the enamel of the teeth (pellicle), but it makes it difficult for saliva to penetrate into narrow spaces — fissures and interproximal contact points, areas around elements of orthodontic systems fixed on the teeth, etc.

**Methods for Determining the Protective Properties of Saliva.** There are some *methods of assessment of salivary cariesprotective properties*. These methods can be used both in routine clinical practice and in scientific researches. These methods are including estimation of:

1. **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.

The normal rate of unstimulated salivation is considered to be 0.31–0.6 ml/min, hyposecretion is diagnosed at 0.03–0.3 ml/min, hypersecretion — at 0.61–2.40 ml/min.

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.

2. Viscosity of saliva. The relative viscosity of saliva with respect to water is measured using the "Ostwald's Viscometer."

3. Determination of saliva acidity. In practice, the indicator method is convenient: a standard paper indicator strip is impregnated with saliva, then its pH is determined using a comparative shade scale.

More accurate is the ionometric method, which requires an ionometer, a test tube, a microcuvette and a phosphate buffer solution (pH = 6.86). For research, 2–3 ml of unstimulated saliva is collected in a test tube. The instrument is calibrated before and after the study of saliva using a buffer solution placed in a microcuvette. After washing the electrodes, the cuvette is filled with 0.5 ml of saliva, the readings are read from the ion meter and decoded according to the calibration graphs. Normal consider the concentration of hydrogen ions in the pH range = 6.5–7.7.

4. Determination of the buffer capacity of saliva. In the dental clinic, it is convenient to use specially prepared test paper strips impregnated with acid and an acid-base indicator. A drop of saliva from a pipette is applied to such a strip, saliva to some extent neutralizes the acid impregnation of the strip, as evidenced by the change in the color of the indicator. Most often, the initial acidic level of pH = 4.5 corresponds to a yellow-brown color, slightly acidic pH = 4.5–5.5 — green, and neutral pH — blue. The closer to the neutral state the strip is under the influence of buffer forces, the better.

5. Method for determining the mineralizing potential of saliva (Leus P. A., 1977). The mineralizing potential of saliva (MPS) is indirectly assessed by whether crystals form when a drop of saliva dries slowly. To conduct the study, you need to have a pipette, a glass slide and a microscope. Unstimulated saliva is collected from the bottom of the oral cavity with a pipette and applied to a glass slide. Saliva dries in air at room temperature or in a thermostat. Dried drops are viewed under a microscope in reflected light at low magnification (2×6). The nature of the pattern on the glass is evaluated as follows:

1 point — a scattering of chaotically arranged structures of irregular shape;

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

3 points — individual crystals of irregular shape against the background of a grid and clumps;

4 points — tree-like crystals of medium size;

5 points — a clear, large, fern-like or parquet-like crystalline structure.

In this way, each drop of saliva out of three is evaluated and the average value of the MPS is calculated. MPS values from 0 to 1 are considered very low, from 1.1 to 2.0 — low, from 2.1 to 3.0 — satisfactory, from 3.1 to 4.0 — high, from 4.1 to 5. 0 — very high.

*Cariesprotective efficiency of saliva* depends on several factors:

– physiological properties of regional salivary gland (productivity potential, viscosity, pH, buffer and mineralizing properties) and the position of the tooth in oral cavity;

– the functional state of the salivary gland which is determined by the state of physical health, the level of water-salt metabolism (e.g. hydration) and fluoride intake, the health status of the salivary gland, as well as its activity phase (state of rest or stimulation);

– access capabilities of saliva to the tooth surface which depends on the quantity and quality of dental plaque, surface morphology (the availability decreases in the order smooth surface — contact surface — deep fissures and pits).

## VIOLATION OF SALIVATION

Objective decrease in the production of saliva is defined as *hyposalivation*, subjective complaint of dry mouth is defined as *xerostomia*.

Hyposalivation leads to:

– increase of the risk of dental caries and non-carious lesions (erosion, wedge-shaped defect);

– eating disorders due to a decrease in taste sensitivity (dysgeusia), difficulties in food chewing and swallowing;

– high risk of autotrauma and keratosis of the oral mucosa; glossitis;

– burning sensation in the mouth, candidiasis;

– social disadaptation due to speech difficulties, halitosis (bad smell of exhaled air); poor dentures fixation, etc.

Xerostomia may be due to two reasons:

1) low saliva production (hyposalivation);

2) high losses of saliva.

*Causes of hyposalivation:*

– Primary Sjögren's syndrome;

– Secondary Sjögren's syndrome;

– Surgical removal of glands due to neoplasm.

**Connective tissue diseases:**

– 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** (stimulate the sympathetic activity and inhibit the parasympathetic activity of salivary glands):

– Anticholinergics;
– Antihistamines;
– Antianxiety;
– Diuretics;
– Antidepressants;
– Anticonvulsants;
– Narcotics.

The dentist should help the patient to identify the causes of hyposalivation and to plan the treatment:

• to recommend measures of etiotropic treatment (treatment of general pathology as the cause of hyposalivation, correction drug therapy of general pathology, prevention and treatment of diseases of the salivary glands, salivary gland restoration using genetic engineering techniques, stem cells, etc.);

• to propose measures of pathogenetic treatment (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*, such as pilocarpine 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 have not been studied in pediatric patients, but these agents are considered safe for most adult patients and have been used successfully in older children.);

• to provide symptomatic relief:

– compensate the deficiency of saliva and its components (recommend constant hydration oral cavity by sips of water, ice cubes, salivary substitutes (containing water and vegetable colloids (cellulose, xanthan products, aloe) or animal mucus (from the pig's stomach, submandibular gland of cattle), mouth rinses with minerals, buffering compounds and antimicrobial agents (lysozyme, lactoperoxidase);

– behavioral therapy (increased fluid intake; food should be nondiuretic and nonirritating to the oral mucosa; avoid alcohol and tobacco);

– effective individual oral hygiene sparing hard tooth tissue (toothbrushing *before* meal and mouth rinsing *after* meal and at bedtime; the Bass method of toothbrushing with medium toothbrush; toothpaste and mouthwash should contain fluoride, calcium, phosphates, enzymes and antimicrobial agents but be without detergents and alcohol.

Loss of saliva may be associated with:

– mouth breathing (drying of saliva);
– drooling (sialorrhea) when lips are not tightly closed.

If losses associated with mouth breathing its recommended:

– ENT rehabilitation and

– normalization of muscular tone of orbicular muscle of mouth and muscles raising the lower jaw. If the problem occurs during sleeping the patient is recommended to sleep on side in a room with moistened air.

Sialorrhea is the norm for children under two years old and pathology after the age of four. Among the reasons of sialorrhea there is hypersalivation (during pregnancy, smoking, gastrointestinal pathology, cholinomimetic intake) and not closed lips and/or violation of swallowing (cerebral palsy, Alzheimer's disease, Parkinson's disease, stroke, multiple sclerosis). Sialorrhea treatment involves:

– measures to close the lips (myogymnastics, trainers, massage, head position control, etc.);

– in severe cases reduction of salivation (radiotherapy of salivary glands, resection of the sublingual glands, submandibular glands ducts movement, Botox, anticholinergics).

Thus the saliva is a natural factor in ensuring the health of the oral cavity tissues and particularly caries resistance. However saliva does not effectively protects teeth from caries if its quality and quantity decreases (hyposalivation, high viscosity, while sleeping, on the upper incisors, in the proximal surfaces and fissures) and/or the risk factors for dental caries s predominate over health factors (defects of tooth tissue formation, during and in the first time after tooth erutpion, poor nutrition, during long exposures of oral carbohydrates, poor oral hygiene and environmental shift in dental biofilm in favor of cariogenic microflora). In such cases special prevention efforts are necessary and dentist should be responsible for their organization.

# Chapter 9
# ORAL HYGIENE INDICES

An index is defined as a numerical value describing the relative status of a population on a graduated scale with definite upper and lower limits, which is designed to permit and facilitate comparison with other populations classified by the same criteria and methods (A. L. Russell).

Dental indices provide a quantitative method for measuring, scoring, and analyzing dental conditions in individuals and groups. It is an objective mathematical description of a disease or condition based on carefully determined criteria under specified circumstances.

Dental indices help researchers and clinicians in understanding trends and patient's needs.

**In epidemiological** oral health an index is used to demonstrate the prevalence and incidence of a particular condition, to provide baseline data, to assess the needs of a population, and to evaluate the effects and results of a community program.

**Researchers** use indices to determine baseline data and to measure the effectiveness of specific agents, interventions, and mechanical devices.

During dental appointment index scores are used to demonstrate, educate, and motivate the patient. By comparing scores from the initial exam during a follow-up exam, the patient can measure the effects of personal daily oral care.

Indices can be used for individual assessment, for clinical trials or epidemiological surveys (Table 9.1).

*Table 9.1*

**Levels of oral hygiene assessment**

| Type | Uses |
|---|---|
| Individual assessment | Evaluation and monitoring the progress and maintenance of oral health.<br>Measures effects of personalized disease control programs overtime.<br>Monitors progress of disease healing.<br>Patient education and motivation<br>Provides individual assessment to help patient to recognize an oral problem |
| Community health/ epidemiologic survey.<br>Survey for the study of disease characteristics of populations | Not designed for evaluation of an individual patient.<br>Measures the prevalence and incidence of an oral condition occurring within a population.<br>Provides baseline data to show existing dental health practices.<br>Compares the effects of a community program and evaluates the results.<br>Finds out the needs of a community |
| Clinical trial determines the effect an agent or procedure on the prevention, or control of a disease | Comparison of an experimental group with a control group.<br>Determines baseline data before the experimental factors are introduced.<br>Measures the effectiveness of specific agents used for prevention, control and treatment of oral conditions.<br>Measures the effectiveness of mechanical devices used for personal care, i.e. toothbrushes, interdental cleaning aids |

**Plaque Control Record.** It was offered by O'Leary, T. J. Drake and J. E. Naylor (1972). The plaque control record (O'Leary index) appears to be a commonly used oral hygiene index for assessing oral health skills. This index provides sufficient information for patient education; however, the time involved in data collection reduces its value. This index 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.**

*Selection of Teeth and Surfaces:*

– All teeth are examined.

– All facial and lingual surfaces are examined after using disclosing agent.

*Criteria Score:*

No plaque — 0.

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

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

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

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

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

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).

*Selection of Teeth and Surfaces:*

– 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.

– Four areas, distal, facial or buccal, mesial, and lingual, are examined.

– Missing teeth are not substituted.

*Procedure:*

– 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.

*Scoring*. 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.

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.

*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.

*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.

*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:

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

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

– Upper and Lower Central incisor: 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 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.

# Chapter 10
# TOOLS FOR INDIVIDUAL ORAL HYGIENE

**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:

– the design of the brush;

– the skill of the individual using the brush;

– 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 H. N. 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* (Fig. 10.1). Total brush length is about 15 to 19 cm (6 to 7.5 inches). The brushes for children may be shorter in size.

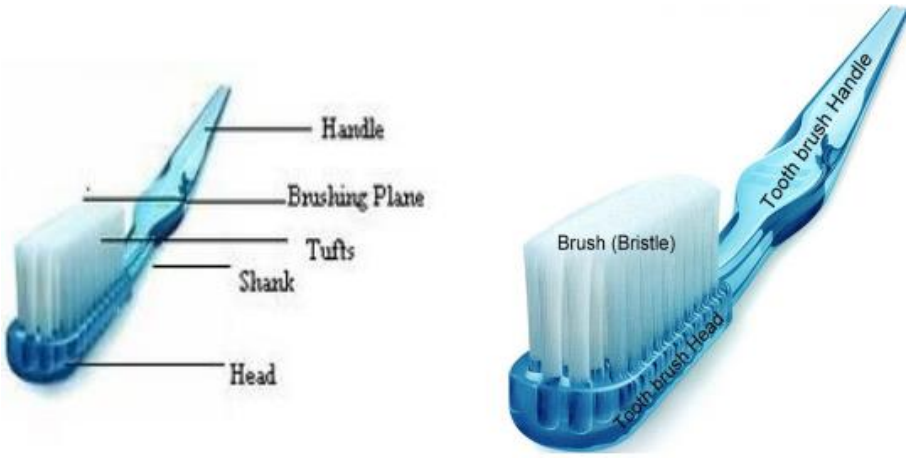


*Figure 10.1.* Structure of manual toothbrush

*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 loses 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.

Manufacturers of toothbrushes aim for innovations in the brush head design that will help to compensate for nonideal toothbrushing technique and time. The more basic designs include tooth brushes with standard (straight) bristles and more advanced models with angled bristles specially aiming at helping to remove plaque from teeth and along the gum line (Fig. 10.2). Theadvanced toothbrushes has the potential to remove greater amounts of plaque, especially from the gum lines and approximal surfaces than conventional tooth brushes incorporating straight bristles. On the other hand, there has been conflicting results whether which design is more capable of effective plaque control.

Standard, flat-trim bristles | CrissCross bristles; multi-section power tip bristles | Multi-level straight bristles, multi-section power tip bristles; outer gingival stimulators | CrissCross bristles; multi-section power tip bristles; outer gingival stimulators

*Figure 10.2.* Bristle design of manual toothbrushes

## POWERED (ELECTRIC) 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.

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.

*Technique of using.* 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.

*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. Studies have shown that if properly used both automatic and manual toothbrushes can remove plaque effectively.

## 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. 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

An ionic toothbrush is a type of electric toothbrush that uses high-frequency vibrations and ionic technology to clean teeth. They work by generating a stream of negatively charged ions that neutralize the positively charged plaque and bacteria on teeth. This helps to break down the plaque and bacteria, making it easier to remove during brushing.

*Principles of action.* 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 (Fig. 10.3).

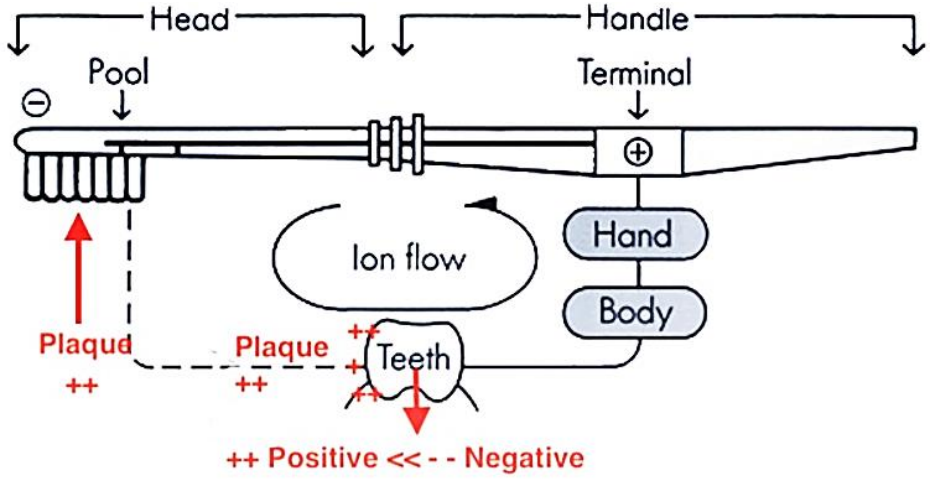


*Figure 10.3.* Mechanism of action of ionic toothbrush

The bonding between the pellicles and bacteria is mediated by $Ca^{2+}$ bridge formation. The anions, supplied by the lithium battery of ionic toothbrush inhibit the bonding between the bacteria and $Ca^{2+}$ 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.

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

*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 BIONIC TOOTHBRUSH 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:*

– Twisted or nontwisted.
– Bonded or nonbonded.
– Waxed or unwaxed.
– 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.

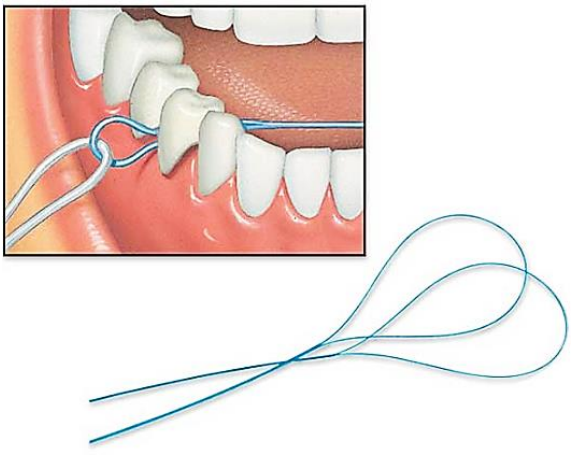

*Figure 10.4.* Floss Threader

**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 (Fig. 10.4).

**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.

6. Different types may be needed to fit different open interproximal spaces.
7. Trauma to tooth or gingiva from sharp wire center of some.

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

***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.

## SINGLE TUFT BRUSHES

*Figure 10.5.* End-tuft trim toothbrush

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 (Fig. 10.5).

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

## INTERPROXIMAL BRUSHES

*Figure 10.6.* Interproximal Brush

They are also called as interdental brushes, proxa brush. They are available in various sizes and shapes (Fig. 10.6). 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 (Fig. 10.7). 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.

*Figure 10.7.* Wedge gum stimulator

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.

# Chapter 11
# INDIVIDUAL AND PROFESSIONAL METHODS OF DENTAL PLAQUE REMOVING

## INDIVIDUAL METHODS OF DENTAL PLAQUE REMOVING

The main methods of removing dental plaque include self-cleaning, wiping, rinsing, brushing and flossing.

*Self-cleaning* of teeth occurs due to friction that occurs when chewing food on those parts of the surface of the teeth that come into contact with food. Self-cleaning effects are absolutely insufficient for the prevention of dental caries, since they practically do not affect the main areas for plaque retention — interproximal space, cervical zones, fissure area, etc.

*Wiping* — mechanical cleaning of teeth using soft objects that have a minimal abrasive effect. Wiping is the first oral procedure in a child's life, and can also serve as an alternative method of oral care in special situations (in the absence of a toothbrush, in the pathology of hemostasis, etc.) It should be remembered that the manual wiping cleans only convex surfaces of teeth.

*Rinsing* has a low cleansing effect, so it should be supplemented with the use of liquid oral agents.

Brushing is the main method of self removal of plaque and debris by an individual. Oldest tooth brushing method was described by Fones in 1913, although now only recommended only in children. Various brushing techniques have been proposed by various researchers since the early half of the twentieth century. However, each brushing technique has its own advantages and disadvantages respectively. It is the duty of the dentist or health care provider to aid and teach the patient about the various brushing techniques and recommend a particular technique that is suitable for that particular individual. It is important that the individual should perform the technique satisfactorily for adequate plaque control.

There are various methods of the toothbrushing that provide consistent, more or less thorough cleansing of all surfaces of all the teeth.

*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.
3. Certain methods, e.g. vertical and horizontal scrubbing methods can produce gingival recession and tooth abrasion.
4. The technique should be simple to use and easy to learn.
5. The method must be well organized so that, each part of the dentition is brushed in turn and no area overlooked.

Toothbrush design, brushing duration, parental involvement, and the brushing method, manipulative skill, and manual dexterity of the child are the most cited determinants of the effectiveness of toothbrushing. Many types of

brushing techniques have been recommended by various researchers over the past years.

The proper brushing technique is to:

Place your toothbrush at a 45-degree angle to the gums.

Gently move the brush back and forth in short (tooth-wide) strokes.

Brush the outer surfaces, the inner surfaces, and the chewing surfaces of the teeth.

To clean the inside surfaces of the front teeth, tilt the brush vertically and make several up-and-down strokes.

## BRUSHING TECHNIQUES

There are toothbrushing techniques according to the direction of brushing stroke:

– Vertical (Leonard's method; Roll Stroke Brushing Technique);

– Horizontal (Scrub Technique);

– Vibrating technique (Charter's, Stillman, and Bass);

– Circular technique (Fones method).

**Bass Method.** It is one of the most effective Brushing techniques because of its property to remove plaque or Bio Film and clean the Area directly beneath the Gingival margin which helps in controlling Periodontal infections.

Technique (Fig. 11.1):

– The head of the brush is kept parallel to the occlusal plane, with the brush head covering almost 3–4 teeth starting from the distal most teeth of the arch.

– The bristles are placed at the gingival margin at an angle of 45 degrees to the long axis of the tooth (up for Maxillary and Down for Mandibular) to long axis of tooth, mostly it is difficult to place at 45 degrees so placing parallel to teeth is also beneficial.

– Now press the bristles slightly so that they enter the Gingival Sulcus and Embrasures. Vibrate the Brush back and fort with Short Strokes for 10–15 strokes for each position and move to the next teeth. (Use gentle but firm vibratory strokes without removing bristle ends from sulcus).

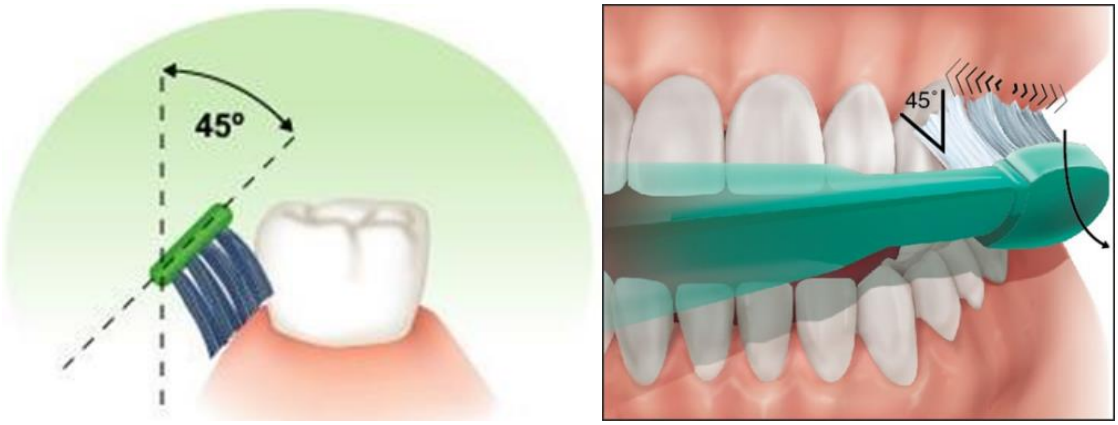


*Figure 11.1.* Bass's Brushing Technique

Indications of Bass/Sulcular technique: For Open proximal areas, Cleaning of Cervical areas Beneath the height of contour of Enamel, in case of teeth with exposed root surface.

**Stillman's Brushing Technique.** Similar to Bass technique with a slight modification to remove debris more effectively from the embrasures. Bristles are directed apically and angles same as bass, Bristles are placed partly over cervical part of tooth and partly on adjacent gingiva.

Method/Technique: this technique is similar to Bass technique with just the slight modification. The Stillman technique involves brushing using short horizontal strokes on all the tooth surfaces. Short back and forth strokes are used and brush head is moved occlusally with light pressure (Fig. 11.2).

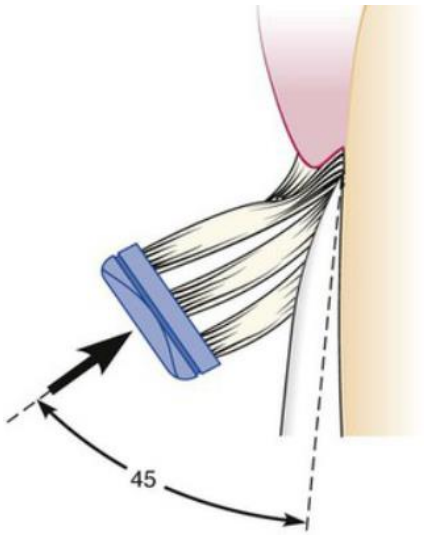


*Figure 11.2.* Stillman's Brushing Technique

Indications: in case of Gingival Recession, for Gingival stimulation, to clean from large embrasures, remove plaque and biofilm from cervical region.

**Charter's Brushing Technique.** It is a technique used mostly in presence of any appliances in the oral cavity, it effectively cleans the interproximal areas between teeth. It is the opposite of the Bass technique.

Method/Technique:

1) Place the head of your toothbrush alongside your teeth, with the bristle tips angled at 45 degrees against the gum-line. Let some bristles slide gently under the edge of the gum.

2) Move the head of the brush in small circles, very slowly, keeping the bristles stationary at the gum-line. Short back and forth strokes are given mostly like vibratory motion.

3) Count to 10 and brush away from the gum-line in a sweeping motion.

4) Using the small same circular strokes, brush the outer, inner and chewing surfaces of each tooth.

5) Brush the inside surfaces of the front teeth by tilting the brush vertically and making several gentle circular motions with the front part of the toothbrush (Fig. 11.3).

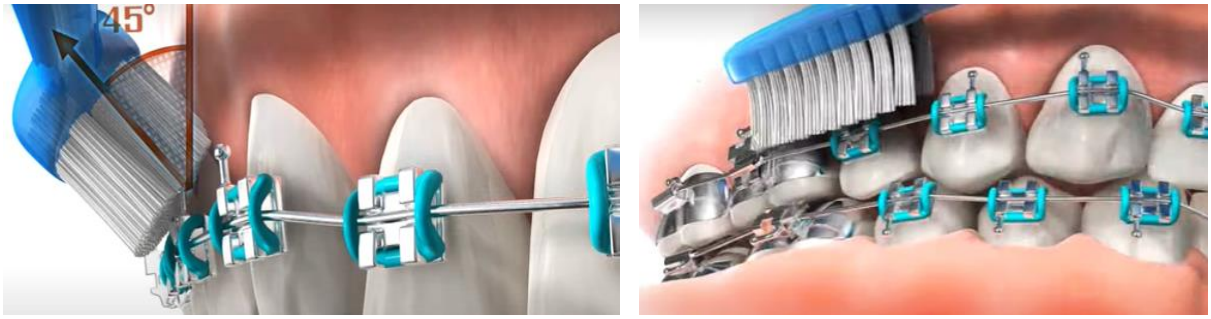


*Figure 11.3.* Charter's Brushing Technique

Indications: Charters Technique is used in patients with Orthodontic fixed prosthetic appliances for Effective cleaning of interproximal areas, is used in case of patient undergoing orthodontic treatment and patients who have just undergone periodontal surgery for temporary cleaning of surgical site.

**Modified Bass, Modified Stillman's and Modified Charter's.** These techniques are very simple to understand if you know the 3 main Tooth brushing techniques — Bass, Stillman and Charter's all you have to do is follow the technique and at the end Roll the Brush tufts Occlusaly towards the crown.

Method/Technique: follow the Techniques mentioned above about the 3 techniques Charter's, Stillman's or Bass and at the end of each technique Roll the brushes towards the occlusal surface of the teeth, roll tufts occlusal after cervical area is cleaned by prescribed method. This will help is clearing out the debris, biofilm out of the Embrasures (Fig. 11.4).

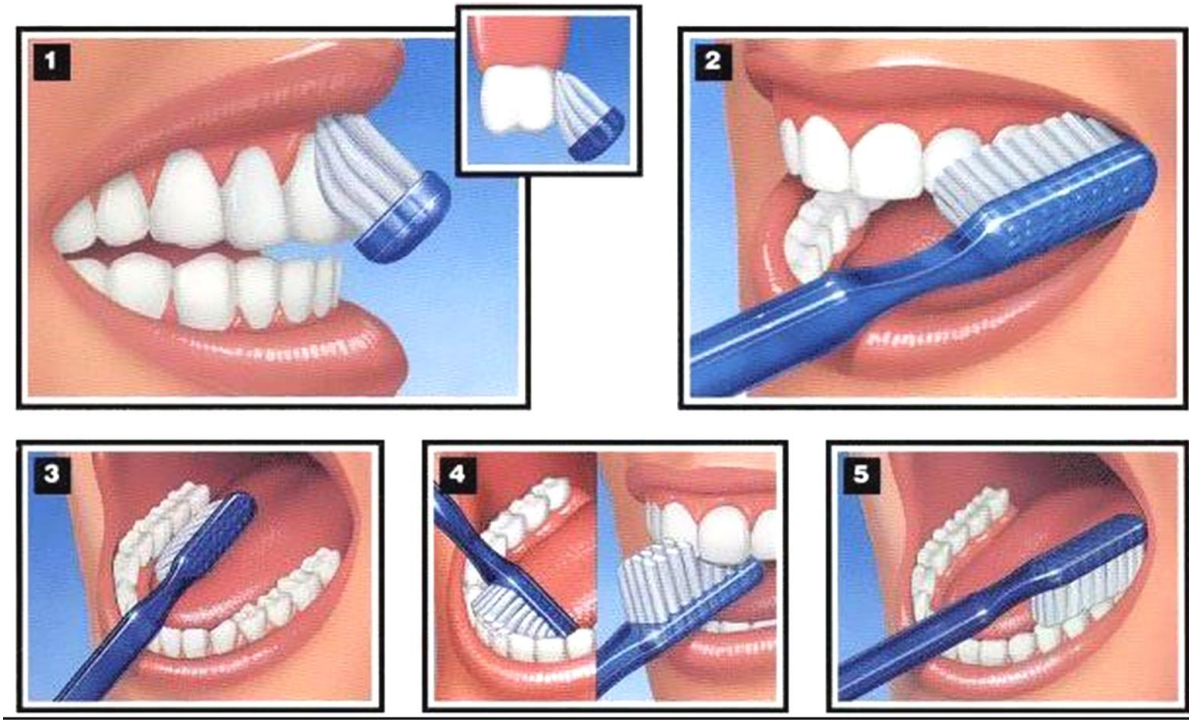


*Figure 11.4.* Modified Bass / Modified Stillman's Techniques

Indications: clean entire facial/buccal and lingual surfaces, rolling motion avoids damaging the base of the gingival sulcus.

**Circular Brushing Technique (Fones Technique).** It is the simplest of all methods of Brushing, all you have to do is move the Brush in a circular motion around a set of teeth and move on to the next set, it is useful in teaching children to brush. The technique is quite easy to learn and provides good gingival stimulation.

Method/Technique: The maxillary and mandibular teeth are kept in occluded position while performing this brushing technique. Place the Tooth Brush on a set of teeth, then activate the bristles by slightly pressing them against the teeth and give circular motion motion 4–5 times on each set of teeth and move on to the next set (Fig. 11.5).

*Figure 11.5.* Circular Brushing Technique

Indications: its mostly indicated for children but it can also be applied to adults. This method is usually recommended for young children with minimal manual dexterity. It is a simple technique but it has shown to be less effective then the three first methods.

***Leonard Technique.*** It is a vertical tooth brushing technique where the brush is moved up and down across the teeth (Fig. 11.6).

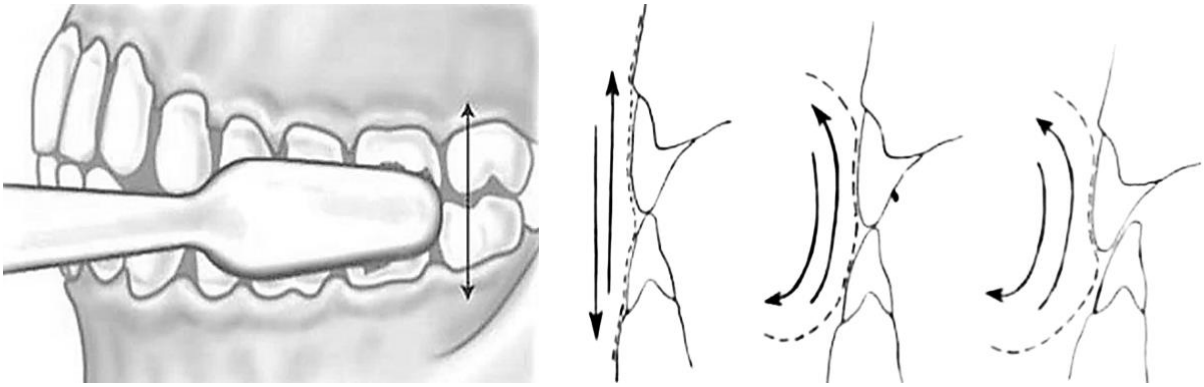

*Figure 11.6.* Shows Leonard's technique

Technique:

– Bristles are placed at 90 degree angle to the facial surface on the clenched anterior & posterior teeth.

– With the teeth edge to edge, place the brush with the filaments against the teeth at right angle to long axis of teeth.

– Brush vigorously without great pressure with up and down stroke on tooth surface with a slight rotation after striking the gingival margin with force. Pressure, enough to force the filaments into the interdental areas is applied, but soft tissue should not be injured by excessive pressure.

Advantages: most convenient; effective for small children with deciduous teeth.

Disadvantage: interdental areas are not cleaned properly.

**Horizontal Scrub Technique.** This is the most commonly used brushing technique due to ease of using. In this technique the bristles are placed at a right angle to the long axis of the teeth and gentle horizontal scrubbing movement of the brush is performed.

The bristles are placed at right angles to the long axis of the teeth and gentle horizontal scrubbing movement of the brush is performed (Fig. 11.7).

*Figure 11.7.* Horizontal Scrub Technique

The main advantage of this brushing technique is its simplicity.

However, a major disadvantage of this brushing technique is cervical abrasion of teeth, which is particularly seen in patients who do vigorous brushing and/or use hard tooth-brushes.More disadvantages: gingival recession and ineffective at plaque removal.

Horizontal scrub technique is a suitable way of tooth brushing in preschool children, considering the development of motor skills at that age and because of the inability to apply other tooth brushing methods.

***Roll Stroke Brushing Technique.*** This is another very simple brushing technique which dose not need much practicing, all you have to do is place the bristles on the Gingiva towards the root and give a upward stroke towards the occlusal surface.

Method/Technique: the Bristles are pointed apical towards the root and placed on the Gingival margin and then a stroke is given to roll the Brush occlusal cleaning the interproximal and the tooth surface in one stroke.

Indications: used mostly in addition to Bass, Stillman's and Charter's techniques where we give this roll stroke at the end of each such technique.

Following the proper tooth brushing techniques is useful in maintaining your oral hygiene, as your dentist for the preferred tooth brushing technique for

your oral cavity. The brushing techniques required for each patient is different and it is important to get the information from your dentist before you pick a particular brushing technique.

For all of these brushing methods, continue to brush the chewing surfaces, as well as the back of the bottom and front teeth, and the tongue by lightly scrubbing up and down, being careful not to damage any of the tissues. Also, remember to brush using a soft bristled toothbrush!

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.

## HOW TO USE YOUR ELECTRIC TOOTHBRUSH

Many electric toothbrushes come with a built-in two-minute timer, which can help you ensure that you are spending enough time brushes. Some models even include timers that chime every thirty seconds to prompt you to move on to the next section of your mouth. When brushing you should divide your mouth into four sections:

The front of your teeth;

The back of your teeth;

The chewing surfaces of your teeth and behind your back teeth;

Your tongue and the roof of your mouth.

When using an electric toothbrush, it is not necessary to press hard or scrub while brushing. Instead, gently guide the brush along as it scrubs. Some electric toothbrush models include pressure sensors, which will let you know if you are pressing too hard.

Depending on the size of your teeth and the size of the brush head your electric toothbrush should brush about one tooth at a time. Take your time while brushing so you can ensure you are being thorough.

1. Before you begin brushing make sure your electric toothbrush is charged. Most models are equipped with a charge indicator light to let you know if your brush is ready for use. You should also floss your teeth before you begin brushing. This helps to loosen any plaque or food particles that are sitting between your teeth and makes it easier for your toothbrush to sweep them away.

2. When you begin brushing hold your brush at a 45-degree angle, just like you would with a manual toothbrush. Starting with the outside surfaces of your teeth gently guide the brush head slowly from tooth to tooth. Make sure you hold the brush head in place for a few seconds against each tooth before you move on to the next one. While brushing, make sure you follow along with the shape of each tooth and the curve of your gums.

3. When you have finished the outer surfaces of your teeth repeat your actions on the inside surfaces, and then again on the chewing surfaces of your teeth. Don’t forget to brush behind your back teeth.

4. When you have finished brushing your teeth take a few seconds to direct the brush head along your gum line and on your gums. This will help remove any remaining plaque. Be careful not to press too hard when brushing your gums or you may irritate them.

5. Finally, use your brush to gently clean your tongue and the roof of your mouth. This will help remove any lingering food particles and help you freshen your breath.

**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.

Gently work the floss between the teeth toward the gums. Curve the floss around each tooth into a U-shape, and gently slide it under the gum line. Move the floss firmly up and down several times to scrape off the plaque. Popping the floss in and out between the teeth without scraping will not remove much plaque and can hurt your gums. 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.

Floss holders are very useful for people with difficulty flossing all their teeth. Disposable floss holders usually come with floss pre-prepared (Fig. 11.8, 11.9).

*Figure 11.8.* Floss holder (flosser)

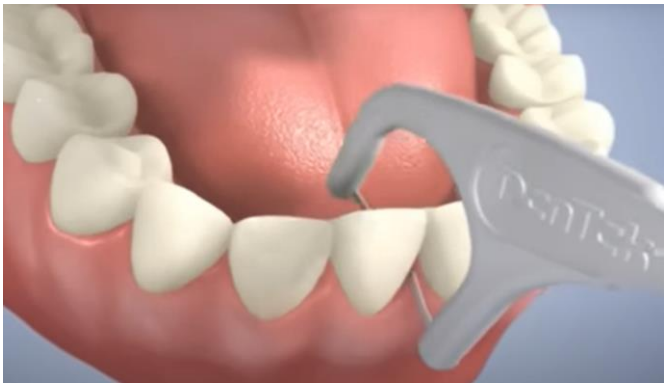

*Figure 11.9.* Using a floss holder

## PROFESSIONAL METHODS OF DENTAL PLAQUE REMOVING

**Professional mechanical toothcleaning (PMTC)** is a service provided by dental personnel (specially trained dental nurses, dental hygienists, and dentists) and is defined as the selective removal of plaque from all tooth surfaces, and particularly key-risk surfaces.

Gingival plaque biofilms located up to 1 to 3 mm subgingivally are removed with mechanically driven instruments and fluoride prophylaxis paste. Therefore, the procedure is more correctly described as gingival plaque control rather than supragingival plaque control. For most effectiveness, PMTC should target dental plaque, inflamed gingivae, diseased pockets, incipient caries lesions, and so on. In toothbrushing populations, the approximal surfaces of the posterior teeth are the main focus.

Traditional prophylaxis or polishing with a rotating rubber cup and prophylaxis paste on the buccal, lingual, and occlusal surfaces, i.e., the nonrisk surfaces, does not constitute PMTC.

If deep subgingival plaque biofilms are also removed, the procedure is referred to as debridement, and may be carried out only by dentists and dental hygienists. In patients with untreated diseased periodontal pockets, debridement may have to be supplemented with removal of calculus (scaling) and root planning.

Professional mechanical toothcleaning should not be confused with so-called prophylaxis or polishing, which involves the use of a rotating rubber cup and prophylaxis paste on the buccal, lingual, and occlusal surfaces, i.e., the nonrisk surfaces.

The following materials are required for Professional mechanical toothcleaning (PMTC):

– plaque-disclosing agents with erythrosin red or blue (Fig. 11.10);

– a contra-angle handpiece and reciprocating tips;

– prophylaxis contra-angle handpiece and rotating rubber cup and brush (Fig. 11.11);

– a fluoride-containing prophylaxis paste such as RDA 170 (medium abrasive);

– a syringe for injecting the paste interproximally (Fig. 11.12).

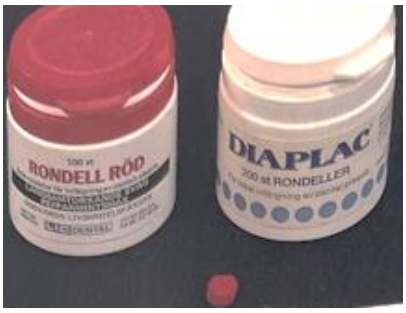


*Figure 11.10.* Plaque-disclosing agents

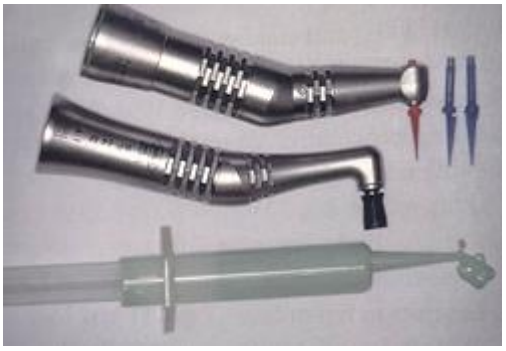

*Figure 11.11.* Contra-angle handpiece and reciprocating tips, prophylaxis contra-angle handpiece and rotating rubber cup; syringe for injecting prophylaxis paste interproximally

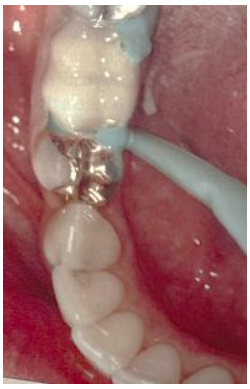

*Figure 11.12.* Syringed application of fluoride polishing paste

Needs-related PMTC should be carried out in the following sequence:

1. Dental plaque is detected with disclosing agents, supplemented by interdental probing of the posterior teeth.
2. Prophylaxis paste is syringed into the inter-proximal spaces of the posterior teeth.
3. The approximal surfaces of the mandibular posterior teeth are cleaned from the lingual side using a triangular-pointed tip in the Profin prophylaxis contra-angle handpiece. The procedure continues until all the approximal tooth surfaces are thoroughly cleaned.
4. Lingual, buccal, and occlusal surfaces are cleaned with prophylaxis paste in a rotating rubber cup in a prophylaxis contra-angle handpiece. This procedure starts on the lingual surfaces of the mandibular right posterior teeth in right-handed patients and on the opposite side in left-handed patients.
5. For quality control, plaque is redisclosed after PMTC, and the posterior interdental areas are probed.

**Plaque Disclosure.** Because PMTC must target the tooth surfaces normally neglected by the patient, disclosure of plaque is the first step. Application of a disclosing pellet takes less than 1 minute using the following procedure:

1. It is best to start where the plaque deposits are often heaviest, i.e., in the mandibular lingual embrasures, where abundant saliva makes disclosure difficult.
2. Plaque in the mandibular buccal embrasures is then disclosed by pressing the pellet lightly into each interproximal space.
3. Finally, the plaque on the maxillary palatal and buccal surfaces is disclosed.

Interproximally, the presence of plaque should be verified by probing; it is invariably present if continuous plaque is visible in the line angles or if the approximal surfaces are carious or restored and the interproximal space is completely occupied by the gingival papilla.

**Application of Prophylaxis Paste to Interproximal Areas.** Use of a disposable syringe facilitates the application of fluoride polishing paste to the interproximal areas (see Fig. 11.12). A rational procedure is to start from the lingual aspect of the mandibular teeth before the floor of the mouth is filled with saliva. The gingival papillae are pressed down with the point of the syringe before injecting the polishing paste. When paste is already applied to the surfaces requiring most attention, interproximal mechanical cleaning can be carried out very quickly.

**Interproximal PMTC.** The prophylaxis contra-angle handpiece and green medium, universal or the V-shaped, flexible (blue large and pink small) tips are used for interproximal PMTC (Fig. 11.13). An alternative is triangular-tipped wooden toothpick which comes with or without fluoride. The tips are self-steering and reciprocating with 1.0- to 1.5-mm strokes. For children with partially erupted teeth, a special dental tape holder is available for the contra-angle handpiece (Fig. 11.14). The double-tape design will clean the approximal surfaces subgingivally on both sides of the papilla at the same time.

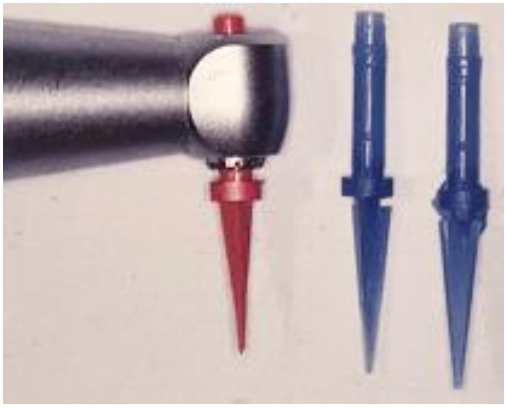

*Figure 11.13.* V-shaped, pointed, flexible plastic tips

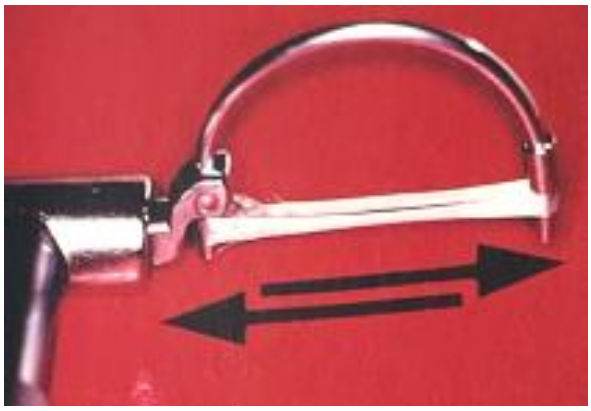

*Figure 11.14.* Holder with double-tape

Professional mechanical toothcleaning should start at the linguoapproximal surfaces of the posterior mandible because the average patient usually fails to clean this area properly. This also reinforces the principle that cleaning should always be started where it is most needed to improve motivation for both the patient and the operator. Entering the interproximal space, the tip will have a 10-degree coronal angle until the papilla is depressed. Because of the resilience of the papilla, a subgingival cleaning effect can be expected at least 2 to 3 mm subgingivally.

A suitable speed for the contra-angle handpiece is approximately 8.000 rpm (ie, 16 000 strokes per minute or 250 per second). At very low speeds (less than 5.000 rpm), vibration will cause discomfort to the patient. The direction of

the tip should continually be adjusted in both the vertical and horizontal directions to reach all the approximal surfaces. At the same time, the fluoride polishing paste is applied to all cleaned surfaces.

As described previously, PMTC should always commence from the lingual aspect of the mandibular molars. When the approximal surfaces have been carefully cleaned from the fairly easily accessible lingual side, they are then cleaned from the buccal direction. The maxillary interproximal surfaces are cleaned next in the same order-, lingual embrasures first and then the buccal embrasures.

**Lingual, Buccal, and Occlusal PMTC.** A regular prophylaxis contra-angle handpiece and rotating rubber cup are recommended for PMTC on the lingual and buccal surfaces. The same medium-abrasive prophylaxis paste used for approximal PMTC is used for these surfaces also. This step should also start on the surfaces most often neglected by the patient, i.e., the mandibular lingual surfaces. The rubber cup should clean the line angles and the subgingival surfaces also. It is possible to remove plaque to a depth of at least 1 to 2 mm subgingivally. To remove plaque from the fissures of erupting molars in particular, a rotating brush is used in the prophylaxis contra-angle handpiece (Fig. 11.15).

*Figure 11.15.* Prophylaxis contra-angle handpiece with rotating rubber cup or pointed brush (for fissures)

*Figure 11.16.* Airflow Air abrasion handpiece

After meticulous PMTC, plaque-disclosing pellets are used again to ensure that all tooth surfaces are plaque free.

The pellicle functions as a nonfrictional layer on the tooth surface. For example, in vivo studies established that it takes approximately 5 minutes to remove the pellicle completely from the tooth enamel using a rubber cup and pumice. With an average treatment time of 3 to 7 seconds for each surface, needs-related professional toothcleaning carries minimal risk of abrasive damage to the tooth surface. Resin composite restorations should not be polished with abrasive prophylaxis paste.

During the past 15 years, air polishing devices have been introduced for removal of supragingival plaque and stains (Fig. 11.16). The instruments are efficient, except on the key-risk surfaces, i.e., the approximal surfaces of

the molars and premolars, because the buccal and lingual papilla impede access of the abrasive powder. There is also a risk of substantial abrasion on exposed root surfaces and resin composite restorations. However, air abrasion is useful for removing extrinsic stains from enamel — particularly smoking- and chlorhexidine-induced stains (Fig. 11.17).

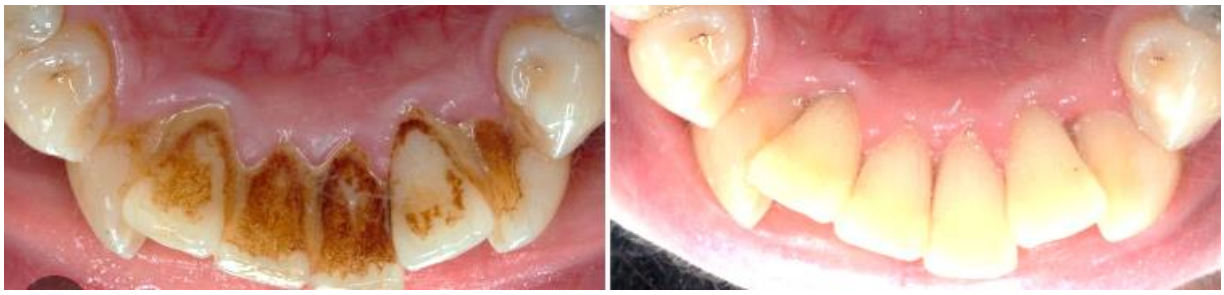

*Figure 11.17.* Staining on the lingual surfaces of the mandibula incisors and result after air abrasion

Hand instruments, such as curettes, may also supplement the reciprocating and rotating instruments for PMTC to remove partly mineralized plaque in the gingival sulcus or more deeply located subgingival plaque biofilms. This procedure is also known as *debridement.*

**Preventive Effects of PMTC.** Professional mechanical toothcleaning should completely remove supragingival plaque from all tooth surfaces and plaque to at least 1 to 3 mm subgingivally; i.e., it provides gingival plaque control.

After PMTC, re-formation of perceptible complex plaque in the dentogingival region is normally retarded for several days, compared to about 1 to 2 days after oral hygiene measures carried out by the patient.

Frequent PMTC also influences the composition of the subgingival microflora and reduces the number of periopathogens. After subgingival root surface instrumentation, frequent PMTC can prevent recolonization by subgingival microflora. Some effect could also be expected on caries-inducing pathogens, such as Streptococcus mutans, on the approximal surfaces, which are inaccessible to the tooth-brush.

Experimentally, it has been shown that after a single PMTC the volume of gingival exudate decreases continuously during the first 24 to 48 hours and does not regain the preexperimental week later. Three sessions of PMTC, at 2-day intervals, will induce healing of inflamed gingivae within 1 week. Indirectly, this results in a reducing in plaque formation rate. Studies have shown that reaccumulation rate of gingival plaque is directed correlated to the degree of gingival inflammation and the quantity of gingival exudate. Therefore, frequent initial PMTC, followed up at need-related intervals in the maintenance program, enhances the patient's own oral hygiene efforts by removal of mature, partially mineralized plaque and reduces the rate of formation of new plaque.

The fluoride ions in prophylaxis paste gain access to the cleaned approximal surfaces, even subgingivally, increasing the potential for remineralization of

enamel caries and root caries on these key-risk surfaces. This reduces the risk for future plaque-retentive factors such as secondary caries, restoration overhangs, and unfinished subgingival margins.

Professional mechanical toothcleaning may also be expected to have a strong patient-motivating effect if it is carried out in a needs-related fashion, similar to the oral hygiene procedures. The patient experiences PMTC as a positive treatment form and attempts to maintain the feeling of cleanliness with his or her own efforts.

PMTC needs-related sequence should not only result in optimal preventive effects, but also motivate the patient to apply the same sequence to self-care.

In new-risk and high-risk patients, an initial intensive period, with three to four visits in 7 to 10 days, is recommended.

Apart from comprehensive history taking, diagnosis, elimination of plaque-retentive factors (carious cavities, calculus, unfinished restorations, overhangs, and unplaned root surfaces), and education of the patient in self-care and self-diagnosis, PMTC should be carried out at every visit. These frequent sessions will result in:

1. Healing of inflamed gingivae within 7 to 10 days.
2. Elimination of gingival exudate and a dramatic reduction in plaque formation rate.
3. Reduction in the edema of the periodontal soft tissue and thus the probing depth. As a consequence, the subgingival environment will change from anaerobic conditions favoring prolific growth of the periopathogenic microflora toward an environment that favors the less harmful, facultatively anaerobic microflora.
4. Enhanced potential for arrest of active non-cavitated coronal enamel and dentin caries lesions, and arrest of active root caries lesions.

In addition, establishment of needs-related oral hygiene habits is significantly enhanced by frequent PMTC that is consistently performed in the same sequence.

Professional mechanical toothcleaning is usually scheduled more frequently initially andthen at gradually prolonged intervals as the patient becomes more efficient in controlling ei tal caries and periodontal disease by self-care.

The most efficient caries-preventive programs comprise needs-related combinations of PM IC, mechanical and chemical plaque control by self-care, and topical application of fluoride by the patient and by dental personnel.

If needs-related plaque control habits for self-care are established and supplemented by needs-related intervals of PMTC, very significant preventive effects will be achieved in high-risk individuals.

# Chapter 12
# MEANS FOR ORAL HYGIENE: TOOTH POWDERS, PASTES, GELS, FOAMS, RINSES

**Dentifrices** are an integral part of daily oral health maintenance. They not only help in cleaning and polishing the teeth, but also the fresh feeling that comes with the use of a dentifrice while tooth brushing is liked by most people.

These are available in paste, powder, gel or liquid form. Dentifrice is the French word for toothpaste.

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.

*History of dentifrices*. The history of dentifrices may be tracked back to the time of the Egyptians, Greeks, and Romans. Egyptians are known to make a tooth powder which consisted of ashes of ox hooves, myrrh, powdered eggshells, and pumice. Greeks improved it by adding abrasives such as crushed bones and oyster shells. The Chinese added herbs, salt, and ginseng to enhance the cleaning properties and taste of the powder. To prevent bad breath, the Romans also added powdered bark and charcoal to their teeth cleaner.

Although the prescriptions of toothpastes have been found in the Ebers Papyrus, an Egyptian medical reference book, but Hippocrates (460–377 BC) was the first to recommend the use of dentifrices. This dentifrice was made up of ashes from hares and mice, as it was believed that animals which had strong teeth may pass this attribute to humans in the form of ash. The Romans used dentifrice which was made up of the powder obtained from grinding horns and animal skulls mixed with goafs fat. The Chinese used a mixture of salt, musk, and urine, which they believed cleaned the teeth as well as improved gingival health.

Around 1000 A.D., the Persians realized the danger of overly abrasive tooth powders that cause tooth damage. So they created a milder substance using burnt snail shells, herbs, and honey.

In 1873, toothpaste was mass-produced for the first time. The major initiating factor for the development of new age dentifrices was the "chemo-parasitic theory of tooth decay" proposed by WD Miller in 1890. It created a boom in the toothpaste industry and since then toothpastes with a variety of compositions and properties have been introduced in the market.

By the early 19th century, two other important ingredients were added to common dental power: Borax for a foaming effect, and glycerin turning the powder into a paste. This made the product easier to use and added a more cleansing effect. Later on, toothpaste was put into collapsible tubes and contained fluoride to strengthen tooth enamel. Thus, toothpaste as we know it today was born. The first collapsible tube containing toothpaste was made by Dr. Washington Sheffield in 1892. It was named 'Dr. Sheffield's Creme Dentifrice.

**Toothpowder.** Oral hygiene seeking measures varies across the world owing to socioeconomic, traditional and religious backgrounds. Although the toothpaste-toothbrush method is the most popular oral hygiene regime. Other methods such as toothpowders are also popular in some parts of the world as a regular daily cleansing agent mainly due to their low cost.

Dental powder is mixture of a variety of ingredients to replace toothpaste as a cleaning substance. Toothpowders are essentially toothpastes without a liquid humectants system. It often contains baking soda, herbs like cloves, mint, or cinnamon, and an artificial sweetener for taste.

The key ingredients in dental powder are:

*Bentonite clay.* This clay naturally binds to toxins, helping eliminate heavy metal toxins from mercury fillings. Plus, it's rich in minerals that are nourishing to gums and teeth.

*Sea salt.* Unrefined sea salt is helpful for healing irritated gums and contains a plethora of tooth-benefitting minerals.

*Baking soda.* Baking soda is a long-time trusted tooth cleaner. It works as a gentle abrasive to gently polish teeth and clean and whiten your smile.

*Calcium carbonate* base contains high levels of enamel enriching Calcium.

*Xylitol.* Xylitol not only adds a sweeter taste to the powder, but also can benefit your oral health. It naturally stimulates saliva production, which helps protect and remineralize teeth.

*Stevia* is another good option if you wish to use a different sweetener.

*Sage.* For years, cultures around the world have used sage as a natural tooth-whitener.

*Cloves.* Cloves not only tastes great; it's also a natural anti-inflammatory ingredient to help sore gums.

*Essential oils.* Essential oils have an endless list of benefits. Peppermint oil is a great choice for dental powder because it is naturally antiseptic and antibacterial. On top of having pain-relieving properties, it also leaves your mouth feeling minty and fresh.

*Tooth powder advantages:*

– Studies have shown that tooth powder can be more effective at removing plaque than toothpaste;

– Most tooth powders are composed of natural and organic ingredients.

*Disadvantages:*

– powder is less convenient to clean with than a paste.

– herbs and clay can leave a funny texture in the mouth after use;

– powder can damage the enamel with daily use because it is more abrasive and harder than paste

– could not be introduced therapeutic agents like fluoride for prevention.

*How to use:* mix two parts powder to water in small container, or use wet brush to dip into dry powder and brush as normal. Can also be mixed with coconut oil for extended wet storage.

**Toothpaste.** 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.

Toothpastes are the most commonly used agents for the maintenance of oral health. Ideal toothpaste must have the following properties:

– It should cause a slight abrasion;

– It should produce froth;

– It should have acceptable taste;

– It should have a bleaching effect, and

– It should prevent plaque, calculus and caries development.

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. Therapeutic effect is to reduce caries incidence, gingivitis, calculus formation, or tooth sensitivity.

Toothpaste is a colloidal suspension of a mixture of ingredients that must be carefully balanced in order to provide an efficacious, safe, and consumer-friendly product. The primary ingredients of toothpaste are shown in Table 12.1, 12.3.

*Table 12.1*

**Composition of a typical dentifrice**

| Ingredients | Percent |
|---|---|
| Abrasive | 40–50 % |
| Humectant | 20–35 % |
| Water | 20–30 % |
| Binding agent | 1–2 % |
| Foaming agent (Detergent) | 1–3 % |
| Flavoring agent | 0–2 % |
| Therapeutic agent | 0–2 % |
| Preservative | 0.05–0.5 % |
| Sweetening agent | 0–2 % |

*Abrasives.* The primary function of abrasives is to remove plaque and stain from teeth. There are various abrasive agents used in toothpastes. It is the largest component of dentifrice — almost 50 % of toothpaste is made up of the abrasive agent. They are insoluble and help in plaque removal due to their abrasive action. These agents remove stains, polish the tooth surfaces and give a pleasing appearance to teeth.

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 is being used now days. Abrasives often dull the tooth luster and to compensate

these polishing agents like small size particle of aluminum, calcium, tin, magnesium are added to the dentifrice.

*Relative dentin abrasivity (RDA)* is a method of measuring of the erosive effect of abrasives in toothpaste on tooth dentin (Table 12.2). It involves using standardized abrasives compared against the test sample. It's essentially a scoring factor and indicates how much tooth structure is lost over a period of brushing due to the abrasive particles in a toothpaste. The determination of this value is done by determining the activity while cleaning worn dentin which is radioactively marked by mild neutron irradiation. The values obtained depend on the size, quantity and surface structure of abrasive used in toothpastes.

*Table 12.2*

**Toothpaste Abrasiveness Ranked by RDA (Relative Dentin Abrasion) Value**

| | The RDA Table |
|---|---|
| 0–70 | Low Abrasive |
| 71–100 | Medium Abrasive |
| 101–150 | Highly Abrasive |
| 151–250 | Regarded as Harmful Limit |

When RDA is in the range of 30–50, pasta is suitable for children, people with sensitive teeth, if you have problems with the gums, it is also used for cleaning dentures.

Toothpaste with the RDA 70–100 referred to moderately abrasive, they are suitable for continuous use in adulthood.

If RDA above 100, so called highly abrasive paste, it can be used only after consultation with your dentist and not more than once every 2 weeks. It may be recommended for smoking people.

*Humectant.* Toothpastes may become dry when they come in contact with air. 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.

Humectants are short-chained polyalcohols, added in the toothpastes to prevent loss of water and thus preventing drying of toothpaste. They also provide a creamy texture to the toothpaste. 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* is one of the important ingredients. Water acts as a solvent in toothpaste, allowing all the contents to dissolve and make a uniformly thick paste. Deionized or distilled water is used.

In preparations like mouth rinses, alcohol is used as a solvent which, along with acting as a solvent, also acts as a taste enhancer.

*Binding agent/Thickening agents.* 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.

These agents bind to water and control the viscosity of the toothpaste. They also prevent the toothpaste from drying out, also give the toothpaste a creamy consistency.

All binders are hydrophilic colloids which disperse or swell or absorb water to form a viscous material. These may be chemical compounds or natural plant or algae extracts. 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/Surfactants.* Surfactants or detergents are important components of toothpastes. These are the agents which clean the tooth surface. They produce foam and aid in the removal of plaque and debris due to their surface action. These reduce the surface tension of the liquid environment in the oral cavity, thus allowing easy contact between toothpaste and teeth. These also have the ability to penetrate and dissolve plaque, making tooth cleaning easy. The surfactant also helps in dispersing the flavoring agent throughout the mouth, thus proving a feeling of freshness.

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 this 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. These are dissolved into the toothpaste with the help of surfactants. Various flavoring agents are added to the toothpastes by different companies, according to the demand from the customers.

*Preservative.* Preservatives are added to the toothpastes to prevent the growth of microorganisms. The most commonly used preservatives are sodium benzoate, methylparaben, and ethylparaben.

*Sweetening agents.* These are added in the toothpaste to provide it a mild sweet taste. Earlier honey and sugar were 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.

Toothpastes made for children are usually made sweeter than that for adults. Xylitol is a sweetener that is also claimed to provide anti-caries activity.

*Coloring agents.* These provide an attractive color to the toothpaste. The Color Index is used to classify the color of the coloring agent. Titanium oxide and chlorophyll are the most commonly used coloring agents in the toothpastes.

*Table 12.3*

**Primary and therapeutic components agents of toothpaste**

| Primary components of a toothpaste | Therapeutic agents |
|---|---|
| Abrasives<br>● Alumina<br>● Aluminium trihydrate<br>● Calcium carbonate<br>● Sodium bicarbonate<br>● Sodium metaphosphate<br>● Calcium pyrophosphate<br>● Pumice<br>● Silica<br>● Bentonite<br>● Dicalcium phosphate<br>● Hydroxyapatite<br>● Kaolin<br>● Methacrylate<br>● Perlite (a natural volcanic glass)<br>● Polyethylene<br>● Zeolites | *Anti-caries agents*<br>● Fluoride<br>● Xylitol<br>● Calcium phosphate<br>● Sodium bicarbonate |
| ***Surfactants***<br>● Sodium lauryl sulfate (SLS)<br>● Sodium N lauryl sarcosinate<br>● Sodium stearyl fumarate<br>● Sodium stearyl lactate<br>● Sodium lauryl sulfoacetate<br>● Amine fluorides<br>● Dioctyl sodium sulfosuccinate | *Anti-plaque agents*<br>● Sodium Lauryl Sulfate (SLS)<br>● Triclosan<br>● Metal-ions<br>● Amyloglucosidase and Glucose oxidase<br>● Essential oils<br>● Chlorhexidine |
| ***Humectants***<br>● Water<br>● Glycerol<br>● Pentatol PPG (polypropylene glycol ethers)<br>● Sorbitol<br>● Xylitol<br>● PEG 8 (polyoxyethylene glycol esters) | *Anti-calculus agents*<br>● Pyrophosphate<br>● Zinc |
| ***Thickening / Binding agents***<br>● Carbopols<br>● Sodium alginate<br>● Sodium aluminum silicates<br>● Carboxymethylcellulose<br>● Silica thickeners Carrageenan | *Anti-dentine hypersensitive agents*<br>● Potassium salts |

| Primary components of a toothpaste | Therapeutic agents |
|---|---|
| ● Hydroxyethylcellulose<br>● Plant extracts (alginate, guar gum, gum arabic)<br>● Algae extracts (Carrageenan and Carbomer)<br>● Viscarine<br>● Xanthan gum | |
| ***Flavors***<br>● Aniseed<br>● Clove oil<br>● Eucalyptus<br>● Peppermint<br>● Spearmint<br>● Fennel<br>● Vanilla<br>● Menthol<br>● Wintergreen | *Anti-halitosis agents*<br>● Zinc |
| ***Preservatives***<br>● Alcohols<br>● Benzoic acid<br>● Phenolics (methyl, ethyl, propyl)<br>● Ethyl parabens<br>● Formaldehyde<br>● Methylparaben<br>● Polyaminopropyl biguanide | *Whitening agents*<br>● Abrasives<br>● Dimethicone<br>● Papain<br>● Sodium bicarbonate |
| ***Coloring agents***<br>● Chlorophyll<br>● Titanium dioxide | |
| ***Sweeteners***<br>● Acesulfame<br>● Aspartame<br>● Saccharine<br>● Sorbitol | |

**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 (Table 12.3). 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.

*Therapeutic agent*. Various therapeutic agents can be added to impart a specific property to the toothpaste and to produce a beneficial effect upon the oral tissue. The most commonly used agent is fluoride to prevent dental caries.

**Anticaries Agents.**

*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 (SnF2) — 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.

*Xylitol* is another sugar alcohol, which cannot be fermented by oral microorganisms, hence is used as a cariostatic agent.

**Anti-Plaque Agents.** The commonly added anti-plaque agent in toothpastes is *triclosan*. It is a synthetic non-ionic chlorinated phenolic agent with antiseptic qualities. It has an inhibitory effect on a broad spectrum of Gram-positive and Gram-negative micro-organisms. It acts on the cell membrane of the microorganisms and causes cell death due to leakage of cellular constituents. It is also effective against mycobacterium and strictly anaerobic bacteria, and against the spores and fungi of the Candida species. In spite 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.

Other metal ions used as anti-plaque agents are *zinc* ($Zn^{2+}$) and *stannous* ($Sn^{2+}$) ions. These ions inhibit the glycolytic sequence in oral anaerobic bacteria, thereby inhibiting their growth.

*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.

Certain enzymes such as *amyloglucosidase* and *glucose oxidase* are also used as anti-plaque agents. They inhibit the growth of microorganisms by activating the antibacterial lactoperoxidase-thiocyanate system in saliva.

*Essential oils* such as thymol, menthol, eucalyptol and methyl salicylate also have anti-plaque effects. They exert their antibacterial activity by altering the bacterial cell wall.

**Anticalculus Agents.** Anti-calculus agents added to the toothpastes exert their anti-calculus action by delaying dental plaque calcification. The commonly used anti-calculus agents are pyrophosphates (tetrasodium pyrophosphate, tetrapotassium pyrophosphate or disodium pyrophosphate).

These dentifrices inhibit the development of new calculus and has no effect on existing calculus.

*Pyrophosphate*. "Antitartar" toothpastes contain soluble pyrophosphates which prevent calcification of plaque. The percentage reduction in supragingi-val (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 tetra-sodium 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." Anti-dentinal hypersensitivity toothpastes are becoming increasingly popular these days. 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.

The most common anti-dentinal hypersensitivity agents used in toothpastes are *potassium ions*. It is claimed that potassium salts in dental preparations increase the concentration of potassium ions around the pulpal nerves, blocking the open dentinal tubules and thereby block action potential generation in intradental nerves. This can inhibit a nerve response from different stimuli.

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

**Whitening Agents.** Tooth whitening toothpastes are also becoming popular these days. It must be noted here that no therapeutic agent can change the natural color of the tooth. These agents, which are added to toothpastes for tooth whitening primary prevent staining of the teeth and help in the elimination of extrinsic and to some extent intrinsic stains on the teeth.

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.

*Zinc ions* are added to the toothpastes for its **anti-halitosis properties**. Halitosis is due to the formation of volatile sulfur-containing compounds (VSC) by the action of bacteria present in the oral cavity on sulfur-containing amino acids as substrates. Zinc ions exert their anti-halitosis effect by interacting with sulfur in the amino acids or the metabolism of sulfur-containing amino acids.

Toothpastes manufactured nowadays, serve multiple actions.

## 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 breath and provide a pleasant taste.

Mouthrinses are generally classified by FDA as either cosmetic or therapeutic or a combination of the two.

*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* 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. 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). 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.

*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: ethyl alcohol is commonly used both to stabilize certain active ingredients and to improve the shelf life of the product. To enhance antibacterial activity and taste. Also, to help keep flavoring agents in solution.

– Sweetening agents: saccharin.

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

– Preservatives.

– Water as the vehicle.

– 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.

**General Indications for Mouth Rinses.**

*Oral malodor.* The most common indication for the use of mouth rinses is oral malodor. Many chemical plaque control agents such as CHX, CPC, essential oils, triclosan etc. reduce the volatile sulfur-producing bacteria in the oral cavity, thus reducing oral malodor. These agents must be differentiated from mouth fresheners which do not kill the microorganisms causing malodor.

*Anti-plaque agents*. Although, mechanical plaque control is the primary method for controlling plaque accumulation, chemical plaque control agents may be used as an adjunct to mechanical plaque control. It must be remembered that chemical plaque control agents should not be advised to each and every patient.

Those patients, who are not able to maintain an adequate oral hygiene due to any reason or patients with periodontal diseases, may be advised to use these agents. The most commonly used anti-plaque agent is CHX.

*Anti-gingivitis agents.* Gingivitis is the result of plaque accumulation and resultant inflammation due to host-bacterial interaction. Adequate mechanical plaque control is the primary measure to reduce gingivitis. However, some mouth rinses may be advised to the patient to control gingival inflammation, including essential oils (Listerine), CHX etc.

*Calculus control.* In high calculus formers, agents which reduce the calculus formation are useful. These contain ingredients like zinc chloride, which retards calculus formation.

*Anti-carie agents*. Patients with high caries risk may get benefited from the use of mouth rinses congaing fluorides. Fluoride preparations such as sodium fluoride, acidulated phosphate, stannous fluoride or combination of fluoride sources may be advised in these patients.

# Chapter 13
# MECHANISM OF CARIES-PREVENTIVE ACTION OF FLUORIDES AND FACTORS DETERMINING ITS EFFECTIVENESS. FLUORIDE COMPOUNDS

The term topical (topical mineralizing, post-eruptive, local mineralizing) 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. This is complex of therapeutic measures recommended for:

– post-eruptive enamel maturation;

– prevention of initial clinical caries lesions;

– remineralization of initial caries lesions.

**Rationale for the Use of Topical Fluorides.** At the time of tooth eruption, the enamel is not yet completely calcified and undergoes a posteruptive 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.

**Role of Fluoride in Enamel Demineralization and Remineralization.** Fluoride interferes posteruptively with the carious process in various ways, such as the inhibition of demineralization, enhancement of remineralization, reduction of acid production in the plaque, and reduction of plaque adhesiveness.

Demineralization occurs when there is an imbalance between processes of mineral gain and loss. Fluoride may interact with these processes in several ways. Two kinds of fluoride and the mineral component of teeth interaction occur: incorporation into the crystal lattice and binding to crystal surface. 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. Fluoride ions when substituted into the hydroxyapatite crystals fit more perfectly than do hydroxyl ions. The interaction of fluoride with the mineral component of teeth produces a fluorohydroxyapatite (FHAP or FAP) mineral:

$$Ca_8[PO_4]_6[OH]_2 + 2F^- \rightarrow Ca_8(PO_4]_6OHF + F^- + OH^- \rightarrow Ca_8[PO_4]_6F_2 + 2[OH]^-$$

Fluoride absorption with subsequent ion exchange converts part or all of the surface to FA, which will show reduced solubility and lower rate of dissolution in acidic buffers (the critical pH of FAP (pH = 4.5) is lower than that of HA (pH = 5.5)) and good ability for precipitation of minerals. Fluorapatite (FA) is called the "firmly bound fluoride" and type of fluoride-apatite interaction — specific binding.

The incorporation of fluoride into the hydroxyapatite (HAP) lattice may occur while the tooth is forming or by ion exchange after it has erupted.

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. Most of the fluoride buried within the crystallites will have been acquired during the period of crystal growth, a process sometimes known as accretion.

The additional mechanisms for reducing of the enamel solubility are:

– under the influence of fluoride, larger crystals are formed with fewer imperfection thus stabilizing the lattice and presenting a smaller surface area/unit volume for dissolution [*Jenkins 1967*];

– enamel which mineralizes under the influence of fluoride has a lower carbonate content thus giving a reduced solubility [*Moreno et al., 1977*]. It has 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. 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.

Eventually, enamel formed has more perfect and larger crystals, less soluble in acid, and less likely to develop caries. Also the greater bonding potential of fluoride makes the apatite crystals more compact and more stable, thereby more resistant to the acid dissolution.

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.

The most important cariostatic role of fluoride is its action in the aqueous phase on the tooth surface and between the enamel crystals during demineralization and remineralization. The influence of fluoride on enamel depends on its availability around enamel crystallites: in fluid between crystal lattices, on the surface of the apatite, in liquid environment of the tooth and dental plaque fluid.

Fluoride should be available in the external environment or from readily soluble materials at the crystal surface. Fluoride is present in the saliva, plaque, and inter crystalline fluid, incorporated in the mineral crystals, and more or less bound to its surface and on the soft tissues in the oral cavity. This second type of fluorides uptake is known as nonspecific binding. In nonspecific binding, fluoride ions are absorbed to the crystal surface without reacting chemically with it. Nonspecifically absorbed fluoride ions might inhibit dissolution by restricting diffusion of ions from the enamel to the solution, and this has been proposed as the exclusive mechanism.

Fluoride accumulates in the plaque fluid and as $CaF_2$ on the enamel surface. Formation of $CaF_2$ is possible at fluoride concentrations greater than 100 ppm, and the amount increases with increasing fluoride activity, prolonged exposure, and lower pH in the solution:

$$\underset{\text{Hydroxyapatite}}{Ca_{10}[PO_4]_6[OH]_2} + 20F^- \leftrightarrow 10\ \underset{\text{Calcium fluoride}}{CaF_2} + 6[HPO_{4-}]_3 + 2[OH]^-$$

$CaF_2$ “globules” are covered with phosphate- and proteins rich protective layer and retained on enamel surface for a long time. By slow dissolution and hence prolonged retention, the solid $CaF_2$ is assumed to act as reservoir for fluoride to be released in to the liquid environment of the teeth. During the acid challenge, $CaF_2$ is dissolved. When the pH drops, fluoride and calcium are released into the plaque fluid. The dissolution of the $CaF_2$ seems to be the key to its caries-preventive effect, because only the free fluoride ion has an effect on enamel solubility.

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 fluorhydroxyapatite.

In an acidic environment, if fluoride is present, hydrogen fluoride (HF) is formed. The tooth surface acts as a so-called micropore filter, and $F^-$ and $H^+$ ions (HF) diffuse into the subsurface lesion, increasing the amounts of fluoride in the active lesion compared to the surrounding intact enamel. Within the lesion, the $F^-$ ions retard demineralization of the enamel crystals during acid challenge and enhance remineralization by crysyall growth and accumulation of fluorapatite on the crystal surfaces when pH rises.

HF is an undissociated, week acid that can penetrate the bacterial cell membrane. The entry of HF into the alkaline cytoplasmic comparnents results in dissociation of HF to $H^+$ and $F^-$ ions. This has two separate, major effects on the physiology of the cell. The first is that the released $F^-$ ions interacts with cellular components, including various fluoride-sensitive enzymes. The second effect is an acidification of the cytoplasmic compartment caused by the released protons.

The most important cariostatic role of fluoride is to enhance the precipitation of new minerals on exiting, partly dissolved enamel crystals. Subsequent to an acid challenge, as the pH rises externally and within the tooth

structure, if fluoride is present at low concentrations in the intercrystalline fluid, it will enhance either the precipitation of new mineral, incorporating calcium, phosphate, and fluoride, or the growth of crystals on existing, partly dissolved, enamel crystals. Either process will be dramatically enhanced by the fluoride present in the intercrystalline fluid. The crystalline surface so formed will then be more resistant to subsequent acid attack, as discussed earlier.

*Mechanism of action*. At low pH (5.5–4.5) fluoride ions presented in the environment, prevent the dissolution of FA:

– Fluoride ions bind calcium and phosphate ions and enhance the precipitation of new minerals on the surface of the enamel as FA and $CaF_2$. Formed FA and $CaF_2$ block the pathways of acid diffusion into the enamel, restrict diffusion of minerals from the enamel to the solution and serve as a source of ions for diffusion into the deep areas of the crystal lattice.

– The fluoride ions enhance crystal growth and larger crystals are formed. As a result — the fluoride ions retard demineralization of enamel.

Hereby if fluoride is present in the aqueous solution surrounding the crystals, it is adsorbed strongly to the enamel mineral crystals and thus acts as a potent protection mechanism against acid dissolution of the crystal surface in the tooth's subsurface region. Fluoride decreases the demineralization and increases the remineralization of the enamel in range pH 4.5–5.5, and hence the demineralization period is shortened.

At pH > 5.5 — environment remains supersaturated for FA and becomes supersaturated for HA and FA, FHA and HA are precipitated.

Thus, during a cariogenic challenge, both the concentration of fluoride at the crystal surfaces and 1 the concentration of fluoride in the liquid phase are important.

Demineralization (loss of mineral resulting from partial dissolution of enamel crystals) during the acid attack stage of caries is markedly inhibited if fluoride is present in solution at the time of the acid challenge. Fluoride diffuses with the acid from plaque into the enamel pores and acts at the crystal surface to reduce mineral loss. Fluoride present on crystal surfaces, for example, as FA deposited during a previous cycle, will be highly resistant to subsequent acid attack.

Fluoride present in solution at the crystal surface during a rise in pH following demineralization can combine with dissolved calcium and phosphate ions to precipitate or grow FA-like crystalline material within the tooth. Fluoride enhances this mineral gain (remineralization) and provides a material that is more resistant to subsequent acid attack. In the case of high fluoride concentrations, $CaF_2$ can be precipitated: This material slowly redissolves, providing a source of $F^-$ ions to inhibit demineralization or promote remineralization.

**Role in Remineralization.** Mineral deposition in enamel defects such as the caries lesion may result in complete or partial replacement of the lost mineral and is therefore called remineralization. The role of fluoride in

the remineralization process was found to be quite complex, although in theory it is relatively simple: In the presence of fluoride, apatite may precipitate as FA or FHA. Because FA and FHA are less soluble than HA, the thermodynamic driving force for their precipitation is greater.

When the impact of fluoride on lesion remineralization is studied, a distinction should be made between the effects in the remineralizing fluid of high doses of fluoride of short duration and the effects of a continuous low concentration of fluoride.

During and after short-term fluoride treatment, large amounts of fluoride are adsorbed in the lesion. Caries lesions in enamel contain considerably amounts of fluoride than does the surrounding intact enamel; i.e., the $F^-$ ions have great affinity for demineralized regions where free $Ca^{2+}$ and $PO_4^{3-}$ ions are available in abundance for "marriage." Frequently the $F^-$ ion "marries" both the $Ca^{2+}$ and $P0_4^{3-}$ ions, forming FA, but the favorite "wife" is the $Ca^{2+}$ ion, resulting in $CaF_2$. The marriage of $F^-$, $Ca^{2+}$, and $PO_4^{3-}$ (FA) is more stable than the marriage between the $F^-$ ion and the $Ca^{2+}$ ion ($CaF_2$), because they divorce easily during cariogenic challenge ($4.5 < pH < 5.5$). After cariogenic challenge, $F^-$ and $Ca^{2+}$ marry again at pH 7.0. These marriages (precipitation of FA and $CaF_2$), which are very important for remineralization, will be accelerated in the outermost region of the lesion, drawing away many of the free mineral ions from the inner parts of the lesion (subsurface lesion or lesion body) and effectively slowing diffusion toward the interior of the lesion. A high fluoride concentration results in delayed and incomplete mineralization in the lesion body compared to the remineralization that occurs at a very low fluoride concentration. Excess deposition of FA and $CaF_2$ may block the lesion pores, resulting in even more pronounced inhibition of diffusion.

Conditions are different in the continuous presence of fluoride at low concentrations, e.g., from fluoridated water (1 ppm F). Fluoride is now available concurrendy with calcium and phosphate and can diffuse into the lesion and precipitate as FA or FHA. Investigations have shown that low concentrations of fluoride do indeed accelerate the initial mineral deposition in lesions or softened enamel. At a constant concentration of 1 ppm F in the remineralizing solution, a twofold to threefold increase in the rate of precipitation was found.

Thus, fluoride regimens for the prevention of dental caries should be designed and used on the basis of the aforementioned principles. High-concentration topical preparations, used at intervals of months or years by the dentist, work not only initially on both demineralization and remineralization but also by providing fluoride as $CaF_2$ or adsorbed in early lesions and subsequently available over prolonged periods.

The most effective caries-preventive fluoride regimen, both from a theoretical perspective and as proved clinically, is the frequent (daily) low-concentration application of fluoride toothpaste and/or mouthrinses. Very low levels of fluoride (1.0 mg/L and less) in the oral fluids affect the rate of mineral dissolution and enhance redeposition of calcium and phosphate, forming acid-

resistant FA and fluoridated HA. Frequent, long-term topical use of low-concentration fluoride will result in almost complete remineralization of subsurface enamel lesions. On the other hand, use of topical fluoride agents with high fluoride concentration, such as APF gels and varnishes (1 % to 2 % F), will result in a faster remineralization and blocking of the "micropores" of the surface lesion, while the lesion body is less remineralized. In addition, more phosphate/pro-tein-coated $CaF_2$ particles will be formed on the enamel surface and in the pellicle. To date, there are no long-term data documenting the separate cariostatic effect of these two principles. However, frequent use (more than once a day) of low-concentration fluoride agents, supplemented by application of high-concentration topical fluoride agents (> 1 % F) at needs-related intervals, should be more effective than either of the two methods used separately.

**Posteruptive Effects of Fluoride.** The caries-preventive effects of fluoride are almost 100 % posteruptive.

The total posteruptive cariostatic effect of fluoride is a combination of several single effects:

– Reduction of mineral solubility through formation of FA on the crystal surfaces.

– Inhibition of mineral dissolution, primarily through its actions in the aqueous phase on the tooth surface and among the crystals in the tooth.

– Promotion of remineralization by fluoride available on the tooth surface as well as in the intercrystalline fluid.

– Reduction of acid formation by the acidogenic bacteria (primarily Mutans streptococci) of the plaques.

– Reduction of plaque adhesiveness and formation by reduction of surface energy, wettability, and extracellular polyglucan and polyfructan formation.

– Influence on cellular metabolism and growth.

Finally, it must be stressed that "clean teeth never decay," because plaque control is directed toward the etiology of dental caries: the cariogenic plaque. If the pH in the plaque fluid on the tooth surface drops frequently or nonstop to less than pH 5, then fluoride alone, regardless of concentration or frequency of application, cannot prevent formation of caries lesions.

## FLUORIDE AGENTS FOR TOPICAL USE

**Fluoride Compounds.** There is a great variety of fluoride compounds used in fluoride agents available to the public and to professionals. The main categories are:

– *inorganic compounds*: readily soluble salts, providing free fluoride (Sodium fluoride, Stannous fluoride, Acidulated sodium monofluorphosphate) and slowly soluble salts (Neutral sodium monofluorphosphate, calcium fluoride);

– *organic fluorides*, such as Aminoflouride and Silane fluorides.

**Sodium fluoride** is by far the most widely used flioride compound in topical fluoride agents for self-care as well as for professional use. Sodium

fluofide will immediately provide free F ions at low concentration in the saliva and in plaque, pellicle, and intercrystalline fluids and thereby interact with the carious process during dissolution and remineralization. Higher concentrations of NaF give greater reservoirs of $CaF_2$.

**Stannous fluoride** is used in means for self-care, and for professional application. Stannous fluoride provides free fluoride ($F^-$) ions and stannous ($Sn^{2+}$) ions, which also have an antimicrobial effect. Additionally, $SnF_2$ can produce stannous phosphate fluoride precipitates that should retard the carious process but tend to stain demineralized enamel. Unfortunately, this is a particular problem in caries-susceptible patients with poor oral hygiene.

In aqueous solution, $SnF_2$ is rapidly hydrolyzed and oxidized, reducing or eliminating its effectiveness: a fresh solution is therefore required for each treatment. However, in special gels, toothpastes, and prophylaxis pastes, $SnF_2$ is stable if stored in plastic tubes or cans.

**Acidulated sodium monofluorphosphate** is also used in toothpaste and prophylaxis paste formulations with low pH (approximately 3.0). There is therefore a chemical rationale for the observed clinical effectiveness of these topical agents. Because of its low pH, APF has an acidic taste.

APF readily etches the enamel surface, providing calcium ions that can interact with the fluoride and precipitate large amounts of $CaF_2$. Further, the hydrogen ions present complex with the fluoride, producing hydrogen fluoride (HF), which readily diffuses deep into the enamel.

APF is stable when stored in plastic container, but may etch the glass. Repeated or prolonged exposure of porcelain or composite restorations to APF may result in loss of contour, surface roughening, and possibly cosmetic changes.

**Sodium monofluorophosphate (SMFP)** is used mainly in toothpastes and gels for self-care and for professional application.

It is slowly soluble compound. The release of fluoride from SMFP occurs in the oral fluid and dental plaque in low pH. This is a slow two-step process. At the first stage, the MFP-ion ($FPO_3^{2-}$) is formed, which can bind to the surface of the apatite crystal, replacing the acidic phosphate ion ($HPO_3^{2-}$). Then, Monofluorophosphate (MFP) can be hydrolyzed in plaque and under acidic conditions, providing phosphate and fluoride ions (second stage).

The rate of production of fluoride from $FPO_3^{2-}$ obviously will depend on the level of phosphatase activity. It has been suggested that sodium lauryl sulfate (SLS), the detergent most commonly used in toothpastes, may inhibit oral phosphatases and thereby reduce, the cariostatic effect of MFP.

Monofluorophosphate seems to have no effect on acid production. Because the fluoride is covalently bound in SMFP, $FPO_3^{2-}$ is more compatible with dentifrice abrasives that react with free fluoride. From 1965 to 1985, when abrasives such as chalk and pumice comprised the bulk of toothpaste and prophylaxis paste formulations, SMFP was the major fluoride compound in these products.

**Calcium fluoride ($CaF_2$)** is insoluble in water thay why is non-toxic at high concentrations. $CaF_2$ (0.5–2.0 microns) blocks the micropores in the enamel, dentine tubules, cracks and spaces around restorations, forming a depot. $CaF_2$ in acidic medium releases fluoride ions and calcium.

**Amine fluoride** is an organic fluoride compound — used in toothpaste, gels, and mouthrinses for self-care and gels and prophylaxis pastes for professional use.

This is fluoride compound that readily provides free fluoride. Its enhanced reactivity has been attributed to the greater affinity of hydrophilic counter-ions to the enamel, which will reduce the surface energy and thereby plaque adhesiveness.

In addition, AmF provides a complexed store of fluoride ions and may enhance diffusion through carious enamel, releasing fluoride at appropriate times and sites.

**Silane fluoride** likes Amine fluoride, provides a complexed store of fluoride ions and may enhance diffusion through carious enamel, releasing fluoride at the appropriate time and site.

In contrast to other fluoride compounds, such as NaF, SMFP, and $SnF_2$, which dissolve in water and release fluoride ions, silane fluoride is insoluble. It reacts on contact with saliva, releasing small amounts of hydrogen fluoride (HF). HF penetrates enamel more rapidly than do fluoride ions. The HF molecules that have penetrated the intercristalline fluid of the porous enamel lesion react with water and provide free fluoride ions which will further retard dissolution and enhance remineralization.

Sometimes two or more of these compounds are combined in the same topical fluoride agent.

The cariostatic effect of fluoride is dose-related: the more fluoride ions are in contact with the enamel, the higher cariostatic effect.

The most effective caries-preventive fluoride regimen is: the frequent use (more than once a day) of low-concentration fluoride agent, supplemented by application of high-concentration topical fluoride agents ($\geq$ 1 % F) at needs-related intervals, should be more effective than either of two methods used separately.

Topical fluoride agents are available for self-care or for professional application.

# Chapter 14
# MEDICATIONS CONTAINING FLUORIDES FOR HOME AND OFFICE USE

Topical fluoride agents are available for self-care or for professional application (Table 14.1).

*Table 14.1*

| Fluorides Applied by Dentist / Professionally Applied | Self Applied Fluoride Agents |
|---|---|
| A. Topical solutions and gels.<br>B. Fluoride containing varnishes.<br>C. Fluoride prophylaxis paste.<br>D. Restorative materials containing fluoride.<br>E. Fluoride containing devices (slow-release agents). | A. Fluoride dentifrices.<br>B. Fluoride rinses.<br>C. Fluoride gels<br>D. Fluorinated tooth- picks<br>E. Fluorinated dental floss<br>F. Fluoride containing lozenges and chewing gum |

## PROFESSIONALLY APPLIED FLUORIDES

The fluoride concentration in topical fluoride agents for professional application is usually much higher Self Applied, ranging from about 1 % to 6.0 %, which will promote precipitation of calcium fluoride reservoir.

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.

*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.

**A. Aqueous Fluoride solutions.** The most common fluoride solutions for painting are neutral 2 % NaF (1 % F), 8 % $SnF_2$ (2 % F) and APF (1.23 % F).

Amine fluoride solutions are also used. Introduced in the 1940s, this way the first method pf professional fluoride application.

*Neutral 2 % sodium fluoride solutions.* It is available in both powder and liquid form. Solution 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.* It is recommended that a series of 4-weekly applications of 2 % NaF be given at ages 7, 11 and 13, coinciding with the eruption of different groups of 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 % 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. Patient is advised to avoid rinsing, drinking and eating for next half hour.
7. Second, third and fourth applications are recommended at intervals of approximately 1 week.

*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.

*8 % Stannous fluoride solutions.* $SnF_2$ is relatively unstable: an aqueous solution undergoes rapid hydrolysis and oxidation, which reduces or eliminates its effectiveness. Thus, a fresh solution is required for each treatment. The important difference s from the technique for NaF is that a thorough professional mechanical tooth cleaning (PMTC) is to precede every $SnF_2$ application, the teeth is kept wet with the solution for 4 minutes, making a saliva ejector essential, and applications at 6-month intervals are recommended.

Staining of active caries lesions, probably by the stannous component, has frequently been reported after topical application of $SnF_2$, particularly in patients with poor oral hygiene. On the other hand, it is considered a highly efficient measure for caries control in adults who are susceptible to root caries.

*Acidulated phosphate fluoride solutions.* APF solution contains a fluoride ion concentration of 1.23 %. The application technique is the same that for $SnF_2$.

APF is stable when stored in plastic container and should not be stored in glass container because it may etch the glass. It has an acidic taste. Because of

low pH, repeated or prolonged exposure of porcelain or composite restorations to APF may result in loss of contour, surface roughening, and possibly cosmetic changes. That why is not recommended to use an acidic topical fluoride agent in patients with these types of restorations.

The of caries-reducing effect in local treatment with fluoride solutions is about 30 %.

**B. Fluoride Gels and Foams.** Gels for professional use are similar to those for self-care but have a higher concentration. The most commonly used compounds are APF (1.23 %), neutral 2 % NaF (0.9 % F), and 2 % $SnF_2$ (0.5 % F). Thixotropic gels are 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.

The gel may be applied in prefabricated or customized trays, but syringes are also used for professional application, most frequently for approximal surfaces in elderly adults with exposed root surfaces.

*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) To improve the effectiveness of the gels in extremely high-risk patients, the teeth should be professionally cleaned (PMTC) and air-dried before application of the agent.

d) Enough gel, or foam, should be used to completely cover the teeth. Limit the amount of gel placed in each commercially available disposable mouth tray to no more than 2 ml or 40 % of the tray capacity. Limit the amount of gel placed in each custom fitted mouth tray to 5–10 drops.

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.

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 un-complicated and can be performed by a dental assistant.

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.

For patients with porcelain or resin restorations, neutral sodium fluoride is recommended to prevent etching of restorations.

The professional gel applications provide a reduction of dental caries about 25–30 %.

***C. Fluoride Varnishes.*** A fluoride varnish is a professionally applied adherent material. 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. 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.

Fluoride varnish for use as a topical treatment has a number of practical *advantages*.

– It is well accepted and considered to be safe.

– 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.

– Further, the application of fluoride varnish is simple and requires minimal training.

The use of fluoride varnish is contraindicated in patients with ulcerative gingivitis and stomatitis. There is a very small risk of allergy to one component of vanish, so for children who have a history of allergic episodes requiring hospital admission, including asthma, varnish application is contraindicated.

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.

There are two types of fluoride varnish: natural based and synthetic based:

– varnishes based on natural resins is the so-called “soft” (examples: *Duraphat, Fluoride*).

– varnishes on a synthetic basis, such as acrylates, polyurethane, epoxies and other materials (examples: Floucal, Fluor Protector, Biflurid-12, Belak, etc.).

Advantages and disadvantages of the basics:

– Varnishes on a synthetic basis are colorless, tasty, more fluid (they penetrate better into the proximal areas, micro pores of enamel), have high adhesion to cleaned and dried enamels, and create a long-term coverage of the tooth enamel surface (up to 2 weeks). But it is quite hard composition, and application of that varnish reduces the permeability of enamel, which can adversely affect the metabolic processes between enamel and saliva.

– Natural varnishes are less convenient when applied to the teeth (sticky), colored (usually yellow-brown), saved on the teeth for a shorter time. But they can even stick to wet teeth and create more permeable and soft covering.

In childhood, it is preferable to use soft permeable varnishes on a natural basis.

In the composition of varnishes, sparingly soluble fluorine compounds (calcium fluoride, silane fluoride) can be used. The concentration of fluoride in varnishes ranges from 0.05 to 6 %.

*Method of varnish application:*

1. Oral prophylaxis is done. While a thorough prophylaxis is not essential prior to application, removal of gross plaque is advised.

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.

Fluoride varnish can be successfully applied in most young children and there is no risk of over ingestion 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. Application of fluoride varnish is well accepted by young children, easy and safe and does not take long. It is the most common form of fluoride used to decrease dental caries.

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 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. Slow-release Fluoride agents.** Fluorides releasing dental 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.

*Intraoral slow-release devices.* The intraoral devices currently available are of two types: the copolymer membrane and the fluoride glass device. The copolymer membrane is used in the United States; depending on the amount of fluoride in the inner core the rate of fluoride release can be between 0.02 and 1.00 mg of F per day. The duration of release ranges from 30 to 180 days.

The fluoride glass device was developed in England. It is 4 mm in diameter and is attached to the buccal aspect of maxillary permanent molar. It releases F over a period of at least 1 year.

*Fluoride-releasing dental 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. 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.

The assortment of restorative materials, sealants, liners, and cements that contain fluoride and act as slow-release fluoride agents is continuously increasing. The significantly greatest fluoride release is from pure glass-ionomer cements, followed by resin-modified glass-ionomer cements, glass-ionomer cement-modified resin composites (compomers) and fluoridated resin composites and amalgams.

In addition to the slow release of fluoride, particularly from glass-ionomer cement materials, a further advantage is the possibility of replenishing the diminished fluoride reservoir from such sources as daily use of fluoride toothpastes, fluoride lozenges, and fluoride chewing gums. Both experimental in situ and clinical studies have shown glass-ionomer cement restorative material to be very efficient in preventing recurrent caries in caries-susceptible patients. Low-viscosity glass-ionomer cement is also recommended as a slow-release fluoride sealant for erupting molars, superseding acid-etch-retained resin sealants.

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.

## SELF-APPLIED FLUORIDE AGENTS

The fluoride concentration in topical fluoride agents for self-care, may vary from 0.012 % to 0.150 % F (in toothpaste and mouthrinses), while up to 1.000 % F is used in gels.

**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.

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 1997, the European Community recommended an upper limit of 1500 ppm (0.15 %) in toothpastes sold without prescription (Table 14.2).

*Table 14.2*

**Recommended use of fluoride toothpaste in children (WHO, 2017; IAPD, 2018; EAPD, 2019)**

| Age group | F, ppm | Frequency | Amount, g | Size |
|---|---|---|---|---|
| First tooth up to 2 years | 1000 | twice | 0.125 | Grain of rice |
| 2–6 years | 1000 (+)* | twice | 0.25 | Pea |
| Over 6 years | 1450 | twice | 0.5–1.0 | Up to full length of brush |

* For children 2–6 years, 1000 + fuoride concentrations may be considered based on the individual caries risk.

Since children can swallow between 0.12 and 0.38 mg of toothpaste per brushing (25 % to 30 % of toothpaste), to minimize the risk of fluorosis in children younger than 6 years only a limited amount of toothpaste should be used under adult supervision.

**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 cariostatic effects of fluoride toothpaste are also related to accessibility and fluoride clearance in the oral fluids. Accessibility is improved by:

– frequent mechanical removal of dental plaque, particularly on the proximal surfaces of the posterior teeth;

– deliberate application of fluoride toothpaste to posterior interdental spaces before approximal cleaning;

– thorough swishing with remaining toothpaste slurry after cleaning, followed only by one brief rinse with water.

**Fluoride Mouthrinses.** There are many commercially available Fluoride mouthrinses. The fluoride concentration is usually 0.025 % with the range from 0.001 % to 0.5 %. Neutral NaF 0.05 % solution for daily use and neutral 0.2 % NaF solution for weekly rinsing are the most commonly used. Other less extensively tested fluoride mouth rinses include those containing APF, stannous fluoride, ammonium fluoride and amine fluoride.

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.

Mouth rinses designed to be rinsed and spit out, either prescribed by your dentist or an over-the-counter variety.

Pharmacy-only fluoride mouthrinses typically contain 900 ppm F. They are designed to be used under adult supervision, for weekly/fortnightly use for one minute.

Over-the-counter fluoride mouthrinses generally contain 0.05 percent NaF (200–220 ppm F) and recommended for daily use.

The ADA recommends the use of fluoride mouthrinses, but not for children under 6 years of age because they may swallow the rinse.

A 10 mL volume should be swished around the mouth vigorously once each day for one minute (ideally just before bedtime) and then expectorated. After

rinsing, mouthrinse should be spit out, not swallowed. Patients should not rinse afterwards for 30 minutes.

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 tooth-paste.

**Fluoridated Gels for Self-Care.** Fluoridated gels were developed because their viscosity would make them easier manipulate and would readily permit their application in trays so that the entire dentition could be treated at the same time.

A Gel used as a vehicle for active agents such as fluoride compounds and chemical plaque control agents is a thickened aqueous system containing cellulose compounds for viscosity but neither abrasives nor foaming agents. As such, gels are generally compatible with the active agents. Some are formulated to be thixotropic; that is, they tend to flow when under the presser but remain viscous when not. That why thixotropic gels may penetrate better intraproximally.

Fluoridated gels may be used daily for self. The fluoride concentration is usually lower in gels for self-care (0.1 % to 0.5 % F). For self-care, fluoride gels are applied in trays, but some (e.g. $SnF_2$ gels), are applied with toothbrush.

The application time for self-administered gels should be no less than 4 or 5 min.

Because they relatively high fluoride concentration and risk of ingestion, the self-administration of fluoride gels is not recommended for children younger than 10 years (should be used under adult supervision).

Self-applied fluoride gels are recommended for patients who are highly susceptible to caries attack: 1) those undergoing orthodontic treatment; 2) those with xerostomia related to radiation therapy of the head and neck.

**Fluoridated Toothpicks, Dental Tape and Dental Floss.** The approximal surfaces of the molars and premolars and the occlusal surfaces of the molars have the highest prevalence of DMFs. To improve caries prevention and control, plaque control and topical use of fluorides, through both self-care and professional therapy, must be targeted to these specific key-risk surfaces.

Oral hygiene aid, specially designed for approximal plaque removal, can also deliver fluoride to these surfaces.

Wooden toothpicks are impregnated with 4 % NaF. The NaF crystals dissolve readily in contact with a saliva. To accelerate the release of fluoride, the toothpicks should be moistened in the saliva for a few seconds just before use. For teenagers and young adults, fluoridated dental tape is more suitable.

**Fluoridated Artificial Saliva.** By far the most caries-susceptible patients are those with xerostomia (< 0.1 mL unstimulated saliva per minute). Various artificial saliva products have been formulated as gels or sprays to relieve the extremely distressing subjective problems of dry mouth. The sprays are well accepted and applied 20 to 30 times a day. Because of the extremely high caries risk in patients with xerostomia, all artificial saliva products (sprays, gels) should contain fluoride in a concentration of 0.1–0.5 %.

# Chapter 15
# CALCIUM- AND PHOSPHATE-CONTAINING MEDICATIONS IN THE LOCAL PREVENTION OF DENTAL CARIES. COMBINED USE OF LOCAL PRODUCTS CONTAINING FLUORIDES, CALCIUM AND PHOSPHATES

## RATIONALE FOR USING CALCIUM AND PHOSPHATE COMPOUNDS IN THE PREVENTION OF DENTAL CARIES

The use of preparations containing calcium and phosphates aims to maintain the state of supersaturation of these ions in the environment surrounding the tooth (most often in plaque).

The oral fluid is a solution supersaturated with the basic mineral elements of enamel. When the plaque pH is dropped below a critical value the environment surrounding the tooth becomes an unsaturated solution that has a high demineralizing potential.

Salivary buffer systems help to restore supersaturation with hydroxyapatite. The activity of buffer systems depends on many factors, among which are the initial concentration of calcium and phosphate ions. If the pH of the plaque decreases many times, the reserves are depleted and the saturation of the environment surrounding the tooth with mineral ions will decrease.

The introduction of preparations containing compounds of calcium and phosphorus, allows you to artificially supplement the mineral supply of saliva and thus to maintain the supersaturation in the environment surrounding the tooth with repeated decreasing of pH. Therefore, many dentists consider calcium and phosphate ions as a "natural" basis for caries prevention.

It is recognized that as a result of topical application of Ca-P preparations, favorable changes occur both in enamel and in the oral fluid:

– calcium content in hydroxyapatites increases (respectively, the Ca / P coefficient increases);

– acid solubility and enamel conductivity decrease;

– metabolic processes in the system "enamel – saliva" are activated;

– the rate of salivation increases.

## CALCIUM AND PHOSPHATE COMPOUNDS IN MEDICATIONS FOR LOCAL CARIES PREVENTION

The clinical use of calcium and phosphate ions for remineralization has not been successful in the past due to the low solubility of calcium phosphates.

Insoluble calcium phosphates are not easily applied, do not localize effectively at the tooth surface and require acid for solubility to produce ions capable of diffusing into enamel subsurface lesions.

On the other hand, soluble calcium and phosphate ions can only be used at very low concentrations due to the intrinsic insolubility of the calcium phosphates, in particular the calcium fluoride phosphates. Soluble calcium and

phosphate ions do not substantially incorporate into dental plaque or localize at the tooth surface to produce effective concentration gradients to drive diffusion into the subsurface enamel.

Therefore, last two decades the following calcium-phosphate compositions have been formulated.

**Mineralin** — composition that used in the oral products named R.O.C.S. (*"**R**emineralizating **O**ral **C**are **S**ystems"*). Composition *Mineralin* contains calcium glycerophosphate and magnesium chloride, which is required for the action of metal-dependent enzymes hydrolyzing calcium glycerophosphate.

**Calcium Carbonate (Chalk).** Advantages of nanoparticles of the calcium carbonate are:

– small particles size provides their prolonged retention in the oral cavity and creates a depot effect;

– nanoparticles have a higher rate of dissolution and releasing of calcium ions;

– in the chalk dissolution the resulting carbon dioxide disappears, but the $Ca^{2+}$ and $OH^-$ ions remain, providing up of the pH till 9.1.

In recent times three calcium phosphate-based remineralization systems have been developed and are now commercially available: a casein phosphopeptide stabilized amorphous calcium phosphate (Recaldent™ (CPP-ACP), an unstabilized amorphous calcium phosphate (ACP or Enamelon™) and a bioactive glass containing calcium sodium phosphosilicate (NovaMin™). In that phosphate-based remineralization systems the specific form of the calcium phosphate helps overcome the limited bioavailability of calcium and phosphate ions for the remineralization process.

**Unstabilized 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. Amorphous calcium phosphate is readily soluble in saliva, delivering ions to the enamel.

The Enamelon TM technology is based on unstabilized amorphous calcium phosphate ACP, where a calcium salt (e.g., calcium sulphate) and a phosphate salt (e.g., ammonium phosphate) are delivered separately (e.g., from a dual chamber device) intraorally. As the salts mix with saliva they dissolve releasing calcium and phosphate ions.

The mixing of calcium ions with phosphate ions to produce an ion activity product for ACP which exceeds its solubility product results in the immediate precipitation of ACP or in the presence of fluoride ions, amorphous calcium fluoride phosphate (ACFP).

In the intraoral environment these phases (ACP and ACFP) are very unstable and rapidly transform to a more thermodynamically stable, insoluble crystalline phase (e.g., hydroxyapatitean, fluorhydroxyapatite).

However, before the phases transform calcium and phosphate ions should be transiently bioavailable to inhibit demineralization of enamel and promote enamel subsurface lesion remineralization.

Commercial product: “Enamel Care with liquid calcium”, “Nite White ACP”, “Day White ACP.”

**Casein phosphopeptide-amorphous calcium phosphate complexes CPP-ACP** (RECALDENT™). CPP-ACP products have provided a new direction to preventive dentistry. CPP-ACP is a milk product which helps in remineralization and prevents dental caries.

CPP is a peptide which contains elements that can bind calcium. Casein phosphopeptide can stabilize calcium phosphate present in the solution as amorphous calcium phosphate. 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.

Casein phosphopeptide can deliver amorphous calcium phosphate and can also help the ACP to bind with the dental enamel. Casein phosphopeptide can also decrease the count of Str. mutans as it has got the ability to integrate in the pellicle.

*Mechanism of action*. Casein binds nanoparticles amorphous calcium phosphate to form complexes (micelles) — CPP-ACP thus providing a pool of calcium and phosphate which can maintain the supersaturation of saliva.

CPP-ACP adheres to the surface of the epithelium, dental plaque and enamel pellicle where forms a depot of bio-available calcium phosphate. 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. Since CPP-ACP stabilizes calcium and phosphate in the solution, it can also help in the buffering of plaque pH and so calcium and phosphate level in plaque is increased.

CPP-ACP by the action of acids and bacterial enzymes releases in the environment surrounding the tooth ions of the calcium and phosphate. The half-life period for CPP-ACP on the tooth surface is 1–2 hours. Other part of the ACP, fixed casein, supports the activity of these ions. Therefore, calcium and phosphate concentration within the subsurface lesions is kept high which results in remineralization.

*Indications of CPP-ACP*. 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.

Amorphous calcium phosphate (CPP-ACP), which is available as Tooth Mousse, helps to remineralize the early carious lesions (demineralized areas of the teeth).

It has the ability to counteract the action of acids in cases of erosion.

It has been proposed that CPP-ACP has an edge over fluoride tooth paste when it comes to neutralizing acids in the oral cavity.

CPP-ACP can also block the dentinal tubules and in turn can reduce the sensitivity.

CPP-ACP alone or its combination with fluoride can be utilized as a prophylactic agent before the bonding of orthodontic brackets.

Recently developed ACP-filled bioactive composites are believed to be effective anti-demineralizing/remineralizing agents for the preservation and repair of tooth structures.

**Bioactive Glass (NovaMin$^{TM}$ Technology).** The NovaMinTM technology is based on calcium sodium phosphosilicate bioactive glass which release calcium and phosphate ions intra-orally to help the self-repair process of teeth.

When NovaMin is exposed to saliva in the mouth, sodium is replaced by hydrogen ions from the tooth environment, which leads to an increase in pH to 8.0. Calcium and phosphate migrate from the glass and calcium phosphate precipitates, which is transformed into hydroxycarbonapatite crystals.

NovaMin has been clinically proven to show the following benefits: anti-microbial and anti-inflammatory benefits, noticeable reduction of tooth sensitivity, remineralization of tooth, prevents the development of dental caries, reduction of gingivitis, noticeable tooth whitening.

But this technology appears to be at a very early stage of development.

Based on the in vitro studies, enamel remineralization improved with bioactive glasses. In vivo signs of remineralization such as increase in enamel hardness, the formation of an enamel-protective layer and reduced intensity of light backscattering were less evident with alternatives including fluoride, and casein phosphopeptide-amorphous calcium phosphate (CPP-ACP). Furthermore, no published studies could be found showing an anticariogenic efficacy of NovaMin in animal models or other caries model systems or randomized, controlled caries clinical trials.

**Topical Calcium and Phosphate Drugs and Products for Dental Caries Prevention.** Currently, variety of oral priducts with calcium glycerophosphate, ACP, CPP-ACP are available on the dental market. Calcium and phosporus ions can be delivered via tooth mousse, chewing gum (chewing gum increases the salivary stimulation), mouth rinses and toothpastes.

These oral products are included in individual protocols of primary prevention at high risk of caries (in particular, for people undergoing orthodontic treatment, for patients with hyposalivation), and are also used for remineralization of enamel in the pathology of hard tissues of the teeth (caries of enamel, hyperesthesia, erosion, etc.) after teeth whitening and professional oral hygiene.

The oral products can be used both in office and home prevention, in individual trays and in the simple application technique. The most effective of them are:

– GC Tooth Mousse (GC) contains CPP-ACP (casein phosphopeptide-amorphous calcium phosphate);

– R.O.C.S. Medical Minerals Gel contains calcium, phosphorus, magnesium, xylitol.

**Indications for use:**

For prevention:

– at high risk of dental caries;

– for orthodontic patients (during and after removal of braces);

– hyposalivation, dry mouth;

– at high acidity in the mouth (gastroesophageal reflux, pregnancy, other causes);

– after radiation therapy of the head and neck.

For treatment:

– for remineralization of initial carious lesions;

– to relieve hypersensitivity;

– after whitening (bleaching) procedures, ultrasonic or mechanical scaling or root planning.

**Contraindications:** CPP-ACP in Tooth Mousse is derived from milk casein. Do not use this material on patients with a proven or suspected milk protein allergy and/or with a sensitivity or allergy to benzoate preservatives.

**How to use.** Calcium and phosphorus containing gel or mousse can be used at dental office or at home.

**In-office application.**

I. *Custom tray application:*

1. Before use rinse the custom tray thoroughly under running water.

2. Extrude a generous layer of the mousse or gel into the tray and apply to the upper and/or lower teeth.

3. Leave the tray in the mouth for a minimum of 3 minutes.

4. Remove the tray.

5. Instruct the patient to use the tongue to spread the remaining mousse/gel throughout the mouth. Instruct the patient to retain for as long as possible (1–2 minutes) avoiding expectoration and delaying swallowing.

6. Ask the patient to expectorate and if possible avoid rinsing. Advise the patient not to eat or drink for 30 minutes following application.

II. *Non tray application:*

1. If necessary, remove any excess saliva on the tooth surface with a cotton roll or pellet. However, it is NOT necessary to dry the teeth with compressed air.

2. Apply a sufficient amount of the mousse/gel to the tooth surfaces using an application swab, gloved finger or in difficult interproximal areas using an Interproximal Tooth Cleaning Brush.

3. Leave the mousse/gel undisturbed for a minimum of 3 minutes.

4. Then instruct the patient to use the tongue to spread the remaining mousse/gel throughout the mouth.

5–6. See above.

**At home application.**

III. *Day time or Night time application after tooth brushing as recommended by a dental professional.*

1. Apply a sufficient amount of the mousse/gel to upper and lower teeth. A pea size amount for each arch is the minimum required. The material should be applied to the tooth surfaces using a clean dry finger or cotton tip. For difficult areas (between the teeth) use an Interproximal Tooth Cleaning Brush or dental floss coated with the mousse/gel.

2. Leave the mousse/gel on the teeth for a minimum of 3 minutes.

3. Then use your tongue to spread the remaining mousse/gel throughout the mouth. Hold in the mouth for as long as possible (a further 1–2 minutes) avoiding expectoration (spitting out) and delaying swallowing. The longer mousse/gel and saliva are maintained in the mouth, the more effective the result.

4. Expectorate thoroughly and if possible avoid rinsing. Do not to eat or drink for 30 minutes following application.

5. This procedure can be repeated 1–4 times a day.

The use of oral products in courses are recommended. Preventive courses depend on the intensity of dental caries:

– low risk of dental caries — 1 week 2 times a year;

– moderate risk — 2 weeks 2 times a year at intervals of 6 months;

– high risk of dental caries — a course of 2 weeks, 3 times a year at intervals of 3 months.

Remineralization therapy takes from 10–15 till 45 days according the doctor recommendations and on the need-related base.

**Combined use of calcium, phosphorus and fluoride.** The modern concept of enamel remineralization supports the combined use of calcium, phosphate and fluoride for the following reasons.

The addition of calcium and phosphate to the tooth environment is a necessary condition, but often (for example, due to the low pH) is not sufficient to achieve the hypersaturation with hydroxyapatite, the introduction of fluoride into this medium helps to achieve a supersaturation with Fluorapatite and thus to promote apatite precipitation.

Introduction only fluoride alone to the tooth environment may not be a sufficient to achieve a supersaturation with Fluorapatite due to the lack of calcium and phosphate ions coming during the enamel demineralization and brought in by saliva. Although fluoride enables enamel remineralization, the presence of calcium and phosphate ions in the supragingival plaque is also necessary to promote the process. Therefore, calcium and phosphate ions are often added in the oral environment from the outside (for example, in hyposalivation).

It is found that CPP-ACP with fluoride demonstrate a synergistic remineralization potential.

**Topical calcium, phosphate and fluoride oral products for dental caries prevention.** Oral products containing various compounds of calcium and phosphates (CPP-ACP, hydroxyapatite), combined with fluoride in a concentration 900–1450 ppm are currently available.

*GC MI Paste Plus* is a water based creme containing 10 % CPP-ACP with incorporated 0.2 % NaF (900 ppm) (CPP-ACPF: Casein Phosphopeptide-

Amorphous Calcium Phosphate Fluoride). When CPP-ACPF is applied in the oral environment, it will bind to biofilms, plaque, bacteria, hydroxyapatite and soft tissue localising bio-available calcium, phosphate and fluoride.

Saliva will enhance the effectiveness of CPP-ACPF and the flavor will help stimulate saliva flow. The longer CPP-ACPF and saliva are maintained in the mouth, the more effective the result. A topical *MI Paste Plus* containing bio-available calcium, phosphate and fluoride provides extra protection for teeth, helps neutralize acid challenges from acidogenic bacteria in plaque and helps neutralize acid challenges from other internal and external acid sources.

**Contraindications.** Do not use this material on patients with a proven or suspected milk protein allergy and/or with a sensitivity or allergy to benzoate preservatives.

**Directions for use indications:**

– Following in-office bleaching procedures.

– After ultrasonic, hand scaling or root planing.

– Following professional tooth cleaning (P.T.C.).

– Hypersensitivity prevention and control.

– As an alternative means of applying fluoride topically in children aged 6 and above.

– During orthodontic treatment.

– For high risk caries patients.

– To provide a topical coating for patients suffering from erosion, xerostomia or Sjögrens syndrome.

– For special needs adult patients.

**In-office application.**

I. *Custom tray application.*

1. Before use rinse the custom tray thoroughly under running water.

2. Extrude a generous layer of GC MI Paste Plus into the tray and apply to the upper and / or lower teeth.

3. Leave the tray undisturbed in the mouth for a minimum of 3 minutes.

4. Remove the tray.

5. Instruct the patient to use the tongue to spread the remaining GC MI Paste Plus throughout the mouth. Instruct the patient to retain for as long as possible (another 1–2 minutes) avoiding expectoration and delaying swallowing.

6. Ask the patient to expectorate thoroughly and if possible avoid rinsing. Any GC MI Paste Plus remaining on the surface can be left to gradually dissipate. Advise the patient not to eat or drink for 30 minutes following application.

II. *Non tray application:*

1. If necessary, remove any excess saliva on the tooth surface with a cotton roll or pellet. However, it is NOT necessary to dry the teeth with compressed air.

2. Apply GC MI Paste Plus to the tooth surfaces using an application swab, gloved finger or in difficult interproximal areas using an Interproximal Tooth Cleaning Brush.

3. Leave GC MI Paste Plus undisturbed for a minimum of 3 minutes.

4. Then instruct the patient to use the tongue to spread the remaining GC MI Paste Plus throughout the mouth. Request the patient to hold in the mouth for as long as possible (an additional 1–2 minutes) avoiding expectoration and delaying swallowing. The longer GC MI Paste Plus and saliva are maintained in the mouth, the more effective the result.

5. Ask the patient to expectorate thoroughly and if possible avoid rinsing. Any GC MI Paste Plus remaining on the surface can be left to gradually dissipate. Advise the patient not to eat or drink for 30 minutes following application.

**At-home application.**

III. *Day time application after tooth brushing as recommended by a dental professional or Night time application after tooth brushing as recommended by a dental professional*

*Note*: The overnight use is especially recommended for high-risk adult patients, but not recommended for children under 12.

1. Apply a sufficient amount of GC MI Paste Plus to upper and lower teeth. A pea size amount for each arch is the minimum required. The material should be applied to the tooth surfaces using a clean dry finger or cotton tip. For difficult areas (between the teeth) use an Interproximal Tooth Cleaning Brush or dental floss coated with GC MI Paste Plus.

2. Leave GC MI Paste Plus on the teeth undisturbed for a minimum of 3 minutes.

3. Then use your tongue to spread the remaining GC MI Paste Plus throughout the mouth. Hold in the mouth for as long as possible (a further 1–2 minutes) avoiding expectoration (spitting out) and delaying swallowing.

4. Expectorate thoroughly and if possible avoid rinsing. Any GC MI Paste Plus remaining on the surface can be left to gradually dissipate. Do not eat or drink for 30 minutes following application.

*MI Varnish with 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 bioavailable calcium and phosphate ions released in the oral cavity.

*Clinpro™ White Varnish* (3M ESPE) contains an innovative tricalcium phosphate (TCP) technology and 22,600 ppm fluoride. Clinpro White Varnish:

– 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;

Varnishes are professionally applied of at intervals, determined by indication and patient needs.

# Chapter 16
# THE USE OF SYSTEMIC FLUORIDE SUPPLEMENTS FOR DENTAL CARIES PREVENTION

Fluorides have unique external modifying effects on the initiation, progression, and arrest of caries. However, a prerequisite for fluoride to have an optimal effect is a combination of excellent mechanical and chemical plaque control directed toward the cause of dental caries: the cariogenic plaque.

The caries-inhibiting effect of fluoride (F) has been known for about 60 years. For 30 to 40 years following pioneering work by Dean and coworkers (1942), it was generally believed that the most significant caries-preventive effect of fluoride was preemptive.

Recommendations for use were based on the assumption that incorporation of fluoride in the enamel apatite lattice would confer on the enamel a resistance to acid dissolution; i.e., a high intake of fluoride during tooth formation and mineralization would result in enamel that was rich in fluoride, with enduring resistance to dental caries.

Preventive measures based on this assumption included fluoridation of public water supplies to the 1-mg/L level or, alternatively, supplying fluoride in salt or milk or in tablet form to children.

The above approach, i.e., systemic use of fluorides as an important caries preventive method, is no longer accepted because the preeruptive caries preventive effect is almost nonexistent.

Epidemiologic studies (Driscoll et al., 1982) have shown that, even where the water supply is optimally fluoridated, the topical effect of fluoride in the tooth environment is important. In addition, children with erupted permanent teeth who moved to a region with fluoridated water exhibited a reduction in caries incidence, ultimately demonstrating an incidence similar to that experienced by children born in the region. At the same time, there was an increasing recognition that caries is a disease resulting from an imbalance between processes of mineral loss and gain rather than an irreversible process of demineralization.

An increasing number of inconsistencies gradually emerged between the concept of enamel resistance and actual clinical and experimental observations. It became clear that high fluoride content in the dental hard tissues was of less importance than a moderate increase in fluoride concentration in oral fluids. Modern concepts of the mechanism of action of fluoride emphasize the importance of a daily supply to establish and maintain a significant concentration of fluoride in saliva and plaque fluid to control enamel dissolution.

There is general agreement today among scientists in the field of fluoride research that the caries-preventive and caries-controlling effects of fluorides are almost exclusively posteruptive, i.e. topical. The vehicle may be drinking water, slow-release tablets, or specific topical agents such as toothpastes, gels, or varnishes. Much current fluoride research is concerned with improving

the efficacy of topical treatments, based on an understanding of the mechanisms underlying the cariostatic action of fluoride (for review, see ten Cate and Featherstone, 1996).

Laboratory studies have, shown that fluoride not only reduces the equilibrium solubility of enamel (more or less apatite) but also exerts a wide range of effects on calcium phosphate chemistry, including the kinetics of dissolution and precipitation (ten Cate, 1994; ten Cate et al., 1995).

Fluoride also affects bacterial metabolism, particularly acid production and acidurance. It has also been shown that the formation of fluoride reservoirs, in the form of calcium fluoride ($CaF_2$) on the tooth surfaces and in the tooth environment, is of great importance (Ogaard et al., 1992).

**Natural Occurrence of Fluoride.** In biology, fluoride is usually classified as a trace element and belongs to the halogen group (fluorine, chlorine, iodine, and bromine). In biologic material 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.

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 [$CaF_2$]; 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 60.0 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, depending on the chlorinity. Concentrations may be altered locally by undersea volcanic activity. Thermal streams associated with volcanic activity may exhibit extremely high fluoride concentrations, ranging from 10 to 6,000 ppm in acidic spring waters. Most fluoride in water exists as free fluoride ions, but complexed fluoride increases with increasing salinity, reaching 50 % to 60 % in seawater.

**Intake 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. 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. In some subtropical or tropical countries even young children (1-to-6-year-old) drink a lot of tea every day, which may explain the relatively high prevalence of fluorosis in such regions.

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 amount of fluoride to the diet.

For children 1 to 6 years of age, as for older individuals, fluoride intake is dervied predominantly from beverages rather than from food. Fluoride intake is likely to be quite low when the predominant beverage is cow milk but will be considerably greater when consumption of fruit juices, fruit-flavored drinks, and carbonated beverages is high.

Fluoride intake from diet (including drinking water and beverages with fluoride levels less than 6 mg/L) and recommended use of fluoridated containing dental products such as toothpaste, 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. To prevent the development of visible and esthetically disturbing fluorosis, fluoride intake in infants and preschool children should be limited and controlled.

Studies have shown that 20 % to 60 % of toothpaste is swallowed and almost completely absorbed in children younger than 6 years of age. If a 3-year-old child uses 1 g of 0.15 % fluoride toothpaste per day and swallows 50 %, the total fluoride absorption should be about 0.75 mg per day, which, in combination with other sources of fluoride, such as drinking water, will increase the risk of visible fluorosis in the permanent incisors. Therefore, for children under the age of 6 years, only small amounts of fluoride toothpaste (0.5 g) should be used, and brushing should be followed by thorough rinsing with water.

**Toxicology of Fluoride.** Topical fluoride agents are safe and harmless if used strictly as directed. However, systemic intake of fluoride must be limited because fluoride is a toxic substance.

It has been concluded that if a child ingests a fluoride dose in excess of 15 mg/kg, then death is likely to occur. A fluoride dose as low as 5 mg/kg may be fatal for some children. Therefore, the probable toxic dose (PTD), defined as the threshold dose that could cause serious or life-threatening systemic signs and symptoms necessitating immediate emergency treatment and hospitalization, is 5 mg/kg of fluoride.

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 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 following recommendations should be followed for mouthrinses, toothpaste, gels and tablets:

– They should not be used by young children without supervision and presence of an adults.

– They should be kept out of the reach of young children.

**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.

**Treatment of Acute Fluoride Toxicity.** 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.

## 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.

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, i.e., the incisal edges and the occlusal surfaces of the molars and premolars. These preeruptive 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.

**Negative Effects: Fluorosis.** By far the best-known preemptive 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 that mottled enamel was an effect of fluoride on enamel formation and maturation, the condition was termed enamel fluorosis.

Dean et al. (1941, 1942) demonstrated a positive correlation between the fluoride concentration in the drinking water and the prevalence and severity of fluorosis. Numerous studies have subsequently confirmed that the risk of developing fluorosis is strongly correlated to the regular intake of fluoride during tooth mineralization and particularly during the maturation phase of the enamel.

## SYSTEMICALLY ADMINISTERED FLUORIDES

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. 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.

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.

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.

People who drink water that contains fluoride at levels around 1.0 mg/L have substantially less caries than do those who drink water with negligible concentrations of fluoride. (For review, see Burt and Fejerskov, 1996.) 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.

There is no doubt that water fluoridation is a highly effective means of reducing caries experience in a community with relatively high caries prevalence and insufficient preventive programs, and it is equally clear that it is safe and presents no great technical problems in existing municipal water supplies. While it is only one of several means of supplying fluoride to a community, no other method is as economical or as comprehensive when infrastructure conditions are favorable (i.e., an extensive water treatment system, people who drink the tap water, trained technicians and engineers, and availability of fluoride compounds).

**Fluoridated 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 state of knowledge on the subject, up to the mid-1970s, was summarized by Marthaler et al. (1978). The conclusions were that fluoride ingested via salt prevents dental caries in man, the cariostatic effect being similar to water fluoridation: The fluoride content of salt is adjusted so that urinary fluoride excretion levels are similar to those in areas with optimal water fluoride content.

The addition of fluoride to salt for human consumption was officially authorized in 1980-82. The cost of salt fluoridation is very low, within 0.02 and 0.05 € per year and capita. Recommended level of the fluoride in domestic salt is about 250 mg/kg.

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 % (Mexico, Colombia, Peru and Cuba). 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 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.

**Milk Fluoridation.** 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.

Disadvantages of milk fluoridation (summarized by Stamm, 1972):

– Because children from the lower socioeconomic groups tend to consume the least fresh milk, they would benefit the least.

– Any benefit ceases as an individual matures and drinks less milk.

– Because fluoride in milk is absorbed slowly, there is a very limited topical effect. (This must be questioned according to the recent study by Petersson et al., 2002.)

– Figures show that the procedure can be relatively costly, despite proponents' claims to the contrary.

In part, some of this criticism still may be valid. But school-based fluoridated milk programs in developing countries with high caries prevalence could be an alternative to water fluoridation, since most caries lesions develop during and soon after eruption. A definitive evaluation of fluoridated milk will be based on the results of ongoing large-scale experiments in Bulgaria, Chile, New Zealand, Italy, and Alaska.

**Fluoride Tablets and Lozenges.** Tablets and drops containing sodium fluoride, appeared in 1940–50-ies on the proposal F. A. Arnold and B. G. Bibby 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). Fluoride lozenges containing 0.25, 0.50, 0.75 and 1.00 mg of fluoride are also commercially available.

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.

However, the use of fluoride tablets should not be regarded as a public dental health measure, except for some at-risk age groups, such as 5.5 to 7.0 and 11.5 to 13.0 years, the ages at which the molars are erupting. To reduce the risk of fluorosis, fluoride tablets should not be used before the age of 5 years and absolutely not before the age of 3 years. Another problem with administration of fluoride tablets in public dental health programs for children is the limited compliance.

Because the aim of using fluoride tablets is to achieve a supplementary posteruptive cariostatic effect only slow-release lozenges, which prolong the fluoride clearance time in the oral fluids, should be recommended. An optimal effect should be achieved if the lozenges are used as a “dessert,” i.e., directly after meals. The 0.25-mg fluoride lozenges are recommended for children more than 5 years old, and 0.25- to 1.00-mg fluoride lozenges are recommended for selected young adults and adults according individual needs, particularly for elderly patients with reduced salivary secretion rates.

**Fluoride Chewing Gum.** Recently fluoride chewing gum has become commercially available (Fluorette and Fludent). It is sugarless; xylitol and sorbitol are used as sweeteners. Each peace contains 0.25 mg of fluoride.

For very caries-susceptible patients, fluoride chewing gum should be the preferred “systemic” agents, to be used for 15 to 20 minutes directly after every meal. It is recommended primarily for caries-susceptible adults with reduced salivary secretion rates and for caries-susceptible children and young adults, especially during the eruption of the molars.

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.

# Chapter 17
# DENTAL CARIES IN PITS AND FISSURES RISK ASSESSMENT AND PREVENTION POSSIBILITIES

**Epidemiology of Caries of Enamel Pits and Fissures.** Fissures and pits are more prone to the caries development than smooth surfaces due to the morphological complexity of these surfaces.

According to the recent data, the occlusal surfaces of the permanent molars are carious more frequently than are the proximal surfaces and the relative proportion of pit and fissure lesions has increased to 80 percent of the total new caries experience. So, 3-year-olds children have 76 % of caries lesions in the pits and fissures and 85 % of all cavities are localized on occlusal surfaces of molars in 17-year-old adolescence.

Active prevention of caries determines the second important change in the epidemiology of caries of pits and fissures of teeth — polarization of distribution the carious disease in the population: 80 % of the lesions are concentrated in 20 % of the population.

**Pit and Fissure Caries.** On the basis of epidemiological, clinical and microbiological studies molars have been recognized as teeth with a high risk of dental caries and occlusal surfaces as surfaces with a high risk of caries.

The initiation and development of occlusal caries is strongly correlated to the *morphology, eruption stage, and functional wear of the occlusal surface*.

Additionally, the following factors should be taken into considerations: *the mineralization level of the enamel pits and fissures in newly erupted immature teeth and the adhesion conditions for cariogenic microorganisms.*

**Morphology of the Occlusal Surfaces of the Molars.** Fissure caries is partly attributable to the extremely plaque-retentive morphology of the fissure systems. Hence, sites on the tooth surface which encourage plaque retention and stagnation are particularly prone to progression of lesions. These sites are:

– enamel in pits and fissures on occlusal surfaces of molars and premolars;

– buccal pits of molars;

– palatal pits of maxillary incisors.

Fissure pattern and its relation to structure within the depth of enamel is highly variable and usually is between 0.25 and 3.0 mm. Its width varies from 0.005 till 1.5 mm at top (orifice), and from 0.1 till 1.2 mm at the base (bottom). In cross section, most fissures have a wide opening, followed by a narrow cleft, approximately 1.0 mm deep (width: 0.1 mm) and reaching almost to the dentinoenamel junction.

However, there are some (less than 10 %) atypical fissures with a narrow opening and a bulbous widening at the base. Such fissures are regarded as sticky risk fissures.

Nagano classified occlusal fissure into five types on the basis of fissure morphology: V, U, Y, I, IK types (Fig. 17.1).

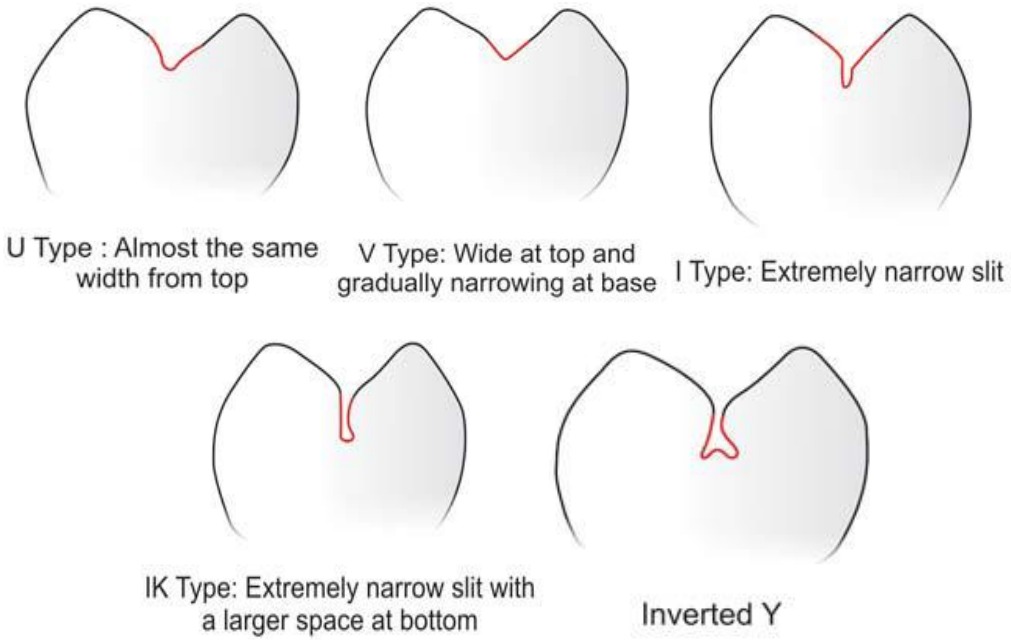


*Figure 17.1.* Types of fissures

**Role of Eruption Stage.** It has been estimated that about five times more plaque will reaccumulate on the occlusal surfaces of erupting molars compared to fully erupted molars with full occlusion and functional wear function 48 hours after professional mechanical toothcleaning (PMTC). That is the reason why occlusal caries is initiated in molars during the extremely long period of eruption (12 to 18 months) and why the occlusal surfaces of the premolars, which have only a 1- to 2-month eruption time, rarely are carious. Development of caries in molars occurs, on average, within 11 months of the start of eruption, i.e., during eruption (most is decayed within 3 to 9 months).

**Pre-eruptive Mineralization of Enamel Pits and Fissures.** Immediately after the eruption, enamel of pits and fissures has a relatively low level of mineralization.

The most intensively posteruptive mineralization of enamel on the occlusal surface of molars occurs during the first two years after tooth eruption, the final maturation takes about 4–5 years.

Enamel fissures matures slowly. The rate of maturation depends on the remineralizing properties of saliva and opportunities to contact with the surface of the enamel in the fissure.

The degree of penetration of saliva into the depths of the fissure (Z) is determined by capillary forces and depends on the width of the fissures (S), surface tension of liquid (y), contact angle (Θ), liquid viscosity (η) and time (t).

$$1.5\ Z = \frac{S\ \gamma\ \cos\theta}{6\ \eta}\ t$$

Therefore, enamel in open wide fissure has a good opportunity to get minerals from the saliva, but enamel in narrow deep fissure remains low level of mineralization.

**Adhesion of the Cariogenic Microorganisms in the Pits and Fissures.** If the formation of the microbial plaque on smooth enamel surfaces requires special adhesive properties of Streptococcus mutans, the plaque in the fissure can be created even by non-adherent microorganisms such as L. acidophilus, L. casei, A. viscosus, A. naeslundii, A. israeli, S. salivarius, S. sanguis, etc.

## DEVELOPMENT OF OCCLUSAL CARIES

It is a common clinical observation that caries on occlusal surfaces does not involve the entire fissure system with the same intensity, but rather is a localized occurrence. Each tooth type in the dentition has its own specific occlusal surface anatomy, and caries is usually detected in relation to the same specific anatomic configuration in identical tooth types. In maxillary molars, for example, the central and distal fossae are sites that typically accumulate plaque and hence are also the sites at which caries most often occurs. In general, occlusal caries is initiated at sites where bacterial accumulations are well protected against functional wear.

## PATHOGENESIS OF OCCLUSAL CARIES

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

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

– Lesion begins beneath plaque, with decalcification of enamel.

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

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

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

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

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

As enamel destruction proceeds, a true cavity forms, the outline reflecting the arrangement of rods in the areas. The cavity has the shape of a truncated cone. The particular anatomic configuration of the occlusal surface at the site of caries initiation explains why the opening of occlusal cavities is always smaller than the base. The dosed nature of the process obviously favors undisturbed growth of bacteria and hence accelerated destruction of the tissue. Occlusal enamel breakdown is the result of further demineralization from an initially established focus, rather than general demineralization involving the entire fissure system.

## DIAGNOSIS OF OCCLUSAL CARIES

The most common methods for diagnosis of occlusal caries are visual and tactile (i.e., probing) inspection, and radiographs.

However, because of the complicated three-dimensional shape of the occlusal surfaces, incorporating fossae, grooves, and fissures with a great range of individual variations, all these diagnostic methods have the potential to result in errors.

The caries lesion usually starts in the enamel on either side of the entrance to the fissures and is visible as a noncavitated white-spot enamel lesion. Gentle probing with a sharp explorer will damage the surface zone of such a lesion and initiate cavitation to the lesion body. A rule of thumb is to use sharp eyes and a blunt probe (or no probe at all) and to arrest the lesion by plaque control and fluoride.

New diagnostic tools, based on laser fluorescence or electrical conduction, used in combination with clinical visual inspection, tactile probing of cleaned occlusal surfaces, and bitewing radiographs, can improve the accuracy of diagnosis of occlusal enamel and dentin caries.

In sticky atypical fissures, none of these methods can accurately differentiate between non-cavitated and cavitated caries lesions in dentin until advanced, open cavities have developed, which can be verified visually and by probing.

It is of great importance to differentiate between noncavitated and cavitated dentin lesions, because the former can be arrested and treated by odontotomy and preventive restoration with fluoride-releasing resin-modified glass-ionomer sealants (so-called fissure blocking). Thus, for optimized accessibility and correct diagnosis of dentin lesions suspected to be cavitated, the fissures are opened to the bottom, with a pointed diamond bur.

## PREVENTION OF CARIES IN PITS AND FISSURES

Caries can be successfully prevented or at least significantly modified by plaque control, topical use of fluorides, and application of fissure sealants, in particular during eruption.

Historically, several methods have been used for prevention and preventive treatment of occlusal caries. In fact, there had been a long-standing interest in sealing as a method of preventing occlusal caries for more than 100 years.

One approach was proposed by Hyatt (1923) and Miller (1950). They attempted to fill the occlusal fissure with a sealant material that, by blocking up the fissure, would prevent bacteria and their substrate from coming into contact with that part of the tooth. Clearly, if successfully retained on the tooth, this material would have a good chance of preventing caries of the underlying enamel. The difficulty was to ensure the retention of the sealing material.

Hyatt (1923) recommended that the occlusal fissures of the erupting tooth be sealed with zinc phosphate cement as soon as possible. When the tooth was

sufficiently erupted, a minimal Class I cavity be prepared and the tooth restored with amalgam before it became carious. In other words, he advocated extension for prevention, according to Black's principles. Hyatt's argument was that it was almost inevitable that the permanent first molar would develop occlusal caries. There was considerable resistance to these proposals from the dental profession, which objected to cutting cavities in caries-free teeth. Hyatt's concept, sometimes called prophylactic odontotomy, never gained wide acceptance, probably because the procedure involved drilling the child's teeth.

**Alternative Methods for Prevention of Occlusal Caries.** Studies have shown that with intensified plaque control via a special daily toothbrushing technique combined with fluoride toothpaste, needs-related PMTC, and topical application of 2 % NaF solution during the 12- to 18-month eruption period, it is possible to achieve close 100 % caries prevention and arrest of enamel caries.

Fluorides have been found extremely effective in preventing caries on smooth surfaces of the teeth, but less effective on occlusal surfaces.

Use of CHX varnish has proven to provide a significant reduction in occlusal caries and in the amount of the cariogenic Mutans Streptococci in fissure plaque. Such varnishes can be used in the earliest stage of eruption until moisture control can be achieved and thus fissure sealants can be placed.

Miller (1950) tested the preventive action of black copper cement when used as a fissure sealant. However, the copper cement was not retained on the occlusal surface.

While investigating different methods of improving the marginal seal of acrylic resin restorative materials, Buonocore (1955) decided to test the effect of etching the tooth surface with an acid solution before application of the restorative material. This alteration in technique had a dramatic effect on the adhesion of the resin to the tooth, and acid-etching techniques were soon introduced to the field of fissure sealing.

# Chapter 18
# SEALING OF PITS AND FISSURES

Over the last few decades, several advancements have been made in caries prevention. Along with systemic and topical fluoride, the increased acceptance and use of pit and fissure sealants have without question had an impact on the prevention of caries. Sealants protect the occlusal surfaces, inhibiting bacterial growth and providing a smooth surface that increases the probability that the surface will stay clean.

Sealing is a one of the most popular methods of caries management of pits and fissures nowadays.

**Sealing** — creating an impermeable mechanical barrier between the microorganisms populating fissures and pits and their oral food sources using adhesive materials placed in fissures.

A pit and fissure sealant is a dental material that flows into the pit and fissures and bonds to the enamel surface mainly by mechanical retention.

**Purpose of the Sealing:**

1. To provide physical barrier to seal off the pit or fissure.
2. To prevent the bacteria and their nutrients from collecting within the pits or fissures to create the acid environment necessary for the initiation of dental caries.

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

1. A person who may be at moderate or high risk of developing dental caries for a variety of reasons.
2. A person with the incipient caries (limited to enamel of pits and fissures).
3. A person who has sufficiently erupted permanent teeth with susceptible pits and fissures.
4. A person who has existing pits and fissures that are anatomically susceptible.
5. A person with a deep or irregular fissure, fossa, or pit is present, especially if it catches the tip of the explorer (for example, occlusal pits and fissures, buccal pits of mandibular molar, lingual pits of maxillary incisors).
6. The fossa selected for sealant placement is well isolated from another fossa with a restoration present.
7. An intact occlusal surface is present where the contralateral tooth surface is carious or restored.

**Contraindications for the Sealing.** A sealant is contraindicated if:

1. Patient's behavior does not permit the use of adequate dry field (isolation) techniques throughout the procedure.
2. There is an open occlusal carious lesion.
3. Caries, particularly proximal lesions, exists on other surfaces of the same tooth (radiographs must be current).
4. A large occlusal restoration is already present.
5. If pits and fissures are well coalesced and self-cleansing.
6. Life-expectancy of primary tooth is limited.
7. When patients are allergic to methacrylate.

Depending on the clinical condition in the system of fissures of occlusal surfaces, following variants of sealing technology are recommended:

– Preventive sealing — isolation the healthy fissures on a healthy tooth surface using invasive or noninvasive sealing;

– Therapeutic sealing — isolation of fissures with an unclear diagnosis or initial caries using a noninvasive sealing;

– Preventive restoration — combination of preparation and restoration of small carious lesion with invasive sealing for surrounding healthy fissures.

The materials used for the sealing, called sealants, respectively. Sealant is a thin plastic coating placed in the pit and fissures of the teeth to act as a physical barrier to decay.

**Criteria for the Ideal Sealant:**

1. A viscosity allowing penetration into deep and narrow fissures even in maxillary teeth.
2. Adequate working time.
3. Rapid cure.
4. Good and prolonged adhesion/bonding to enamel.
5. Low sorption and solubility.
6. Resistance to wear.
7. Be compatible with the oral tissues (minimum irritation to tissues).
8. Cariostatic action.

There are mainly two groups of materials for the fissure-sealing technique — *Resin-based* and *Glass-ionomer cement*.

**Resin-Based Sealant Materials.** The first group is based on etching of the enamel surfaces with a phosphoric acid (about 35 %) and the use of a cross-linking, thermosetting dimethacrylate monomer (bis-GMA), which is diluted with methyl methacrylate or other co-monomers to increase the flow characteristics to reach the depth of narrow fissures. Etching is necessary to increase the roughness of the enamel surface because the retention of the resin-based sealant is mechanical. The resin forms so-called tags into the microroughness of the etched enamel surface to achieve mechanical retention.

A principal difference is the manner in which polymerization is initiated. The first marketed sealants, called first-generation sealants, were activated with an ultraviolet light source. Second-generation sealants are autopolymerizing (self-curing) and set on mixing with chemical catalyst-accelerator system. The third-generation sealants are phooinitiated with visible light (light-cured). Another recent innovation is the sale of fluoride-containing resin-based sealants and different glass-ionomer materials, which should be regarded as slow-release fluoride agents. Today, the third generation of fissure sealants, with or without fluoride, and chemical or light-cured glass-ionomer sealants are the materials of choice for fissure sealing.

During the last decade, the light-cured fissure sealants have been most frequently used. Commercially available sealants differ in whether they are free of inert fillers or semifilled and whether they are clear, tinted, or opaque.

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

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

Auto cured sealant appears to have equivalent documentation of performance compared to visible-light-cured sealant.

The use of an intermediate bonding layer, or the incorporation of the benefits of the advances of the past decade in dentine bonding agents into newly formulated pit and fissure sealants, is perhaps the most exciting new potential development for the future of pit and fissure sealant materials.

**Methods of Fissure Sealing.** There are two main technological methods of tooth preparation for sealant application:

*Noninvasive* — without physical invasion in hard dental tissues and with full preservation of tooth tissue.

*Invasive* — with physical invasion in hard dental tissues and minimal excision the enamel of pits and fissures.

**The Technique of Noninvasive Sealing with Using Resin-Based Fissure Sealants.**

*Step 1*: *Prepare the teeth*. Plaque and debris might interfere with the etching process or sealant penetration:

– Clean the pit and fissure surfaces.

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

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

– The surface is washed with water spray for 20–30 seconds and dried with compressed air.

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

*Step 2*: *Isolate the teeth*. Adequate isolation is the most critical aspect of the sealant application process. Salivary contamination of a tooth surface during or after acid etching will have a deleterious effect on the ultimate bond between enamel and resin.

– Use cotton rolls, dry angles, and/or rubber dam. Some of the disadvantages of Rubber dam include: discomfort during clamp placement, need for local anesthetic in some instances, difficulty in securely placing a clamp onto a partially erupted tooth, an increase in the cost and need for sterilization of the armamentarium.

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

*Step 3*: *Dry the surfaces*.

– Dry teeth with air for 20–30 seconds.

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

*Step 4*: Etch the surfaces. There are various etchant materials available, but the most frequently used etchant is 37 percent orthophosphoric acid. This is available as both a liquid solution and a gel. One should always apply the etchant onto all the susceptible pits and fissures of the tooth and extend it up the cuspal inclines well beyond (at least 2 millimeters) the anticipated margin of the sealant:

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

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

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

Step 5: *Rinsing and drying the teeth*.

– Rinse surfaces for 60 seconds.

– Check effectiveness of etchant by drying with air; the surface should become “chalky white.”

– If not, repeat etching procedure.

– Place new cotton rolls.

– Dry teeth with air for 20–30 seconds.

*Step 6*: *Application of sealant material*. During sealant application all the susceptible pits and fissures should be sealed for maximum caries protection. The sealant material can be applied to the tooth in a variety of methods. Many sealant kits have their own dispensers, some are pre-loaded, they directly apply the sealant to the tooth surface:

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

– Will polymerize in 60–90 seconds.

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

– Apply curing light to material.

– Will be polymerized in 20–30 seconds.

*Step 7*: *Evaluate the sealant*. The sealant should be visually and tactually inspected for complete coverage and the absence of any voids or bubbles. Small voids in the sealant can be repaired simply by adding new material to the void and polymerizing.

*Step 8*: *Occlusal evaluation*.

– Check occlusion with articulating paper or with green occlusal wax. If necessary, adjustment is performed with filled resins or a pear-shaped finishing bur.

*Step 9*: *Re-evaluation.*

– Recall the patient to evaluated the sealants on the six-month basis.

This technique is very sensitive to moisture. Therefore, it may be risky to use it during the eruption of the molars, until the distal fossae and distal margin of the occlusal surface are free of the gingiva. Unfortunately, almost all occlusal caries lesions are initiated during this period.

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

During the last few years, resin-based fissure sealants supplemented with fluorides have also become available. However, most fluoride is lost during the first few days, and the material, unlike glass-ionomer materials, cannot be recharged with fluoride.

**Glass-ionomer cement** is a water-based material that hardens following an acid-base reaction between fluoroaluminosilicate glass powder and an aqueous solution of polyacid. The reaction between glass-ionomer cements and the dental fissures is chemical. This chemical retention is somewhat weaker than the mechanical retention between resin and an etched enamel surface. However, conditioning of the enamel surfaces with polyacrylic acid can improve the retention of conventional glass-ionomer sealants by about 50 %.

A specific caries-preventive effect of glass-ionomer cements, not provided by resin-based sealants, is the release of fluoride; the cement can be recharged with fluorides from topical fluoride agents, such as gels and varnishes. Most of the released fluoride is NaF.

During the last few years, glass-ionomer sealants have been replaced with resin-modified glass-ionomer sealants, which achieve favorable physical properties similar to those of resin composites and resin-based sealants while they retain the basic features of the conventional glass-ionomer material, such as fluoride release and chemical adhesion. This type of material was created by incorporating water-soluble resin monomers into an aqueous solution of poly-acrylic acid.

Autopolymerized as well as light-cured resin-modified glass-ionomer sealants are available. However, the recently introduced light-cured resin-modified glass-ionomer sealant (Fuji III LC) is most frequently used. It is popular because of its fast setting reaction and because it is less sensitive to moisture than the etching-resin technique. This is of great importance because the light-cured resin-modified glass-ionomer sealants can be used as early as possible during the eruption of the molars as a combination of fissure sealant and a slow-release fluoride agent.

Light-cured resin-modified glass-ionomer sealants are used according to the following method:

1. Careful PMTC is performed as in the use of resin-based sealants.

2. The occlusal surface is chemically conditioned with 25 % acrylic acid for 30 seconds.

3. Debris in the fissures is removed with a probe.

4. The occlusal surface is washed for 30 seconds and dried.

5. Moisture control measures are carried out, and sealant is applied with a probe or a capsule.

6. The sealant is light cured for 30 seconds.

7. Occlusion is adjusted in a way similar to that described for resin-based sealants.

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

## INVASIVE PIT AND FISSURE TECHNIQUE

In deep, narrow, sticky fissures there may be suspected hidden caries in molars that are fully erupted. To achieve accessibility for correct diagnosis, such fissures are opened to the bottom with a pointed diamond bur.

If no cavitated lesion into the dentin is present, the opened fissure is sealed with either the etching-resin technique or light-cured resin-modified glass-ionomer sealant. Alternative materials could be flowable compomer or type II light-cured resin-modified glass-ionomer cement. This type of therapy is called fissure blocking or extended fissure sealing. Advantages of the invasive technique, where the fissures are widened with a small bur before the placement of sealants, are the following:

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

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

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

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

## PREVENTIVE RESTORATION

Preventive resin restorations (PRR) is a 20-year-old concept first reported by Simonsen and Stallard (1978). This ***Minimally invasive*** procedure involves removal of those areas of teeth which were affected by caries with following filling them using resin restorative material and finally covering all restorative material and any remaining fissured anatomy with sealant. This method is indicated where caries within a fissure has just reached the dentine.

The advantage of this approach is that the absolute minimum of tooth substance is removed. By avoiding the old philosophy “extension for prevention” the tooth preparation and replacing it with the idea of discrete removal of caries, there is a major reduction in intracoronal preparation and tooth structure loss.

In addition, the procedure avoids the unfortunate consequences of an error in diagnosis. If a healthy tooth is investigated, little harm is done, for it quickly becomes evident that no caries is present and the resulting cavity is very small. If the caries is more extensive than was originally supposed, this will become apparent during the procedure, and appropriate action can be taken.

The advantages of invasive sealants and PRR are:

1. There is minimal removal of the tooth structure, hence, greater the tooth strength.
2. There is no marginal leakage, with a reduced risk of recurrent caries.
3. Local anesthetic is not normally required.
4. The restoration can be completed in one visit and polishing is not required.
5. Caries in adjacent pits and fissures is prevented without fissure removal.
6. Pleasing aesthetics are obtained.
7. The restorations are cost-effective and can be easily repaired.

**Caries-Preventive Effects of Sealing.** Fissure sealants placed with the etching-resin technique are effective as long as they remain firmly adherent to the tooth. Early split-mouth studies of this technique resulted in about a 50 % caries reduction in populations with κ high incidence of caries. However, in populations with a low incidence of caries, less caries prevention may be expected. Thus, general use of fissure sealants in populations with a low prevalence of caries is not cost effective. In such populations, the use of fissure sealants should be restricted to caries-risk patients and molars with atypical sticky fissures as an integrated measure in needs-related caries-preventive programs.

In studies comparing the effect of glass-ionomer or light-cured resin-modified glass-ionomer sealants with resin-based sealants, the former have resulted in at least the same caries-preventive effect as the latter, in spite of their shorter period of retention. This may be attributed to the release of fluoride from the glass-ionomer sealants.

Because of their combined sealant and slow-release fluoride effect and lower sensitivity to moisture, light-cured resin-modified glass-ionomer materials should be regarded as a very efficient fissure protector rather than a long-lasting fissure sealant.

**Sealant Retention.** The long-term efficacy of sealants is well documented.

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

Sealants are lost most frequently fallout from the lingual surfaces of maxillary molars and the buccal surfaces of mandibular molars. This can be attributed to the shallower pits, which increase the difficulty of complete etching and retention.

Most clinicians find that retention rates are less for primary teeth (up to 50 percent are less). The theory behind this reduction in retention is the direction

of the enamel rods in primary teeth. The ends of enamel rods in permanent teeth form an angle perpendicular to the outer enamel surface, whereas the enamel rods in primary teeth often form an angle that does not allow for optimum retention.

Initial retention failure of sealants is attributed to technique errors, the most common of which is moisture contamination. Other technique errors that can affect retention are inadequate etching, incorporation of air bubbles into the sealant material (which weakens the material), and incomplete removal of debris from the pits and fissures prior to etching.

Sealant retention depends not only on proper application, but also on the eruption status of the tooth. When a tooth is not completely erupted, the retention rate is lower — possibly due to difficulties maintaining a dry tooth surface during application. Without doubt, the retention rate is lowered when an operculum is present over the distal marginal ridge of a molar.

Undoubtedly, sealants are susceptible to occlusal wear. This is a problem only if the seal at the margins of the sealant is not maintained.

Again, this emphasizes the importance of continued evaluation of the sealant.

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

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

Inform the patient or parent of the need for six-month recall appointments to monitor sealant retention. At the recall appointment, the sealed tooth should be categorized and treated according to one of the three following categories (Table 18.1).

*Table 18.1*

**Criteria for pits and fissures management at the recall appointment**

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

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

To achieve maximum benefit sealants should:

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

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

– Be used in connection with other preventive measures.

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

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

– Be monitored overtime and re-applied as needed.

## LITERATURE


1. *Шаковец, Н. В.* Герметизация ямок и фиссур = Pits and fissure sealing : учеб.-метод. пособие / Н. В. Шаковец, М. И. Кленовская. Минск : БГМУ, 2021. 55 с.

2. *Axellson, P.* Preventive materials, methods and programs / P. Axellson. 2004. Vol. 4. P. 263–368.

3. *Evidence-based* clinical recommendations for the use of pit-and-fissure sealants. A report of the American Dental Association Council on Scientific Affairs / J. Beauchamp [et al.] // JADA. 2008. Vol. 139, № 3. P. 257–267.

4. *A review* of novel dental caries preventive material: Casein phosphopeptide — amorphous calcium phosphate (CPP–ACP) complex / Farooq [et al.] // King Saud University Journal of Dental Sciences. 2013. Vol. 4. P. 47–51.

5. *A simplified* caries risk test in stimulated saliva from elderly patients / S. S. Garcia [et al.] // Gerodontology. 2008. Vol. 25 (1). P. 26–33.

6. *Irish* Oral Health Services Guideline Initiative. Pit and Fissure Sealants: Evidence-based guidance on the use of sealants for the prevention and management of pit and fissure caries. Irish Health Research Board, 2010. 53 p.

7. *Marya, C. M.* A Textbook in Public Health Dentistry / C. M. Marya. JP Medical Ltd, 2011. P. 103, 105, 273–292, 301–394.

8. *Samaranayake, L.* Essential Microbiology for Dentistry / L. Samaranayake. Churchill Livingstone, 2012. P. 277–286.

9. *Comparison* of five selective media for the growth and enumeration of Streptococcus Mutans / A. K. L. Wan [et al.] // Aust. Dent. J. 2002. Vol. 47 (1). P. 21–26.

10. *American* Academy of Pediatric Dentistry. Recordkeeping. The Reference Manual of Pediatric Dentistry. Chicago, Ill.: American Academy of Pediatric Dentistry, 2022. P. 521–8.

11. *Ergonomics* And Posture Guidelines for Oral Health Professionals. FDI, 2021.

12. *Upendran, A.* Dental Infection Control. 2022 Aug 8 / A. Upendran, R. Gupta, Z. Geiger // In StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing, 2022.

13. *Estimation* of salivary flow rate, pH, buffer capacity, calcium, total protein content and total antioxidant capacity in relation to dental caries severity, age and gender / P. Pandey [et al.] // Contemp. Clin. Dent. 2015. Suppl. 1. S. 65–71.

# CONTENT