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DENTAL PHYSIOLOGY

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Навчальний посібник адресований англомовним студентам стоматологічних факультетів вищих медичних навчальних закладів України. Структура посібника відповідає програмі викладання фізіології ротової порожнини у вищих медичних навчальних закладах. Посібник включає структурно-функціональну характеристику ротової порожнини та функціональні методи дослідження в стоматології, патофізіологічні аспекти захворювань ротової порожнини.

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The study manual is addressed to English-speaking students of Ukraine higher medical educational institutions dental faculties. The manual structure corresponds to oral cavity physiology teaching program in higher medical educational institutions. The manual includes oral cavity structural-functional characteristics as well as functional investigative methods in dentistry, oral cavity diseases pathophysiological aspects.

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CONTENT

SHORTENINGS LIST

ACE - angiotensin-converting enzyme
ACh - acetylcholine
ADH – antidiuretic hormone
AFP – atypical facial pain
APRP – acidic proline-rich salivary protein
ATP - adenosine triphosphate
cAMP – cyclic adenosine monophosphate
CE - cementoenamel
CNS – central nervous system
CT – computed tomography
CGRP - calcitonin gene-related peptide
DAG – diacylglycerol
DIC - disseminated intravascular coagulation
ECG - electrocardiogram
ECM - extracellular matrix
EEG - electroencephalogram
EGF – epidermal growth factor
EHP - exciting hyperpolarization
EMG – electromyography
EOD - electroodontodiagnostics
GABA - gamma-aminobutyric acid
GMP - guanosinemonophosphate
GPCR – G-protein-coupled receptor
GTP – guanosinetriphosphate
HLA – human leucocytic antigens
IDDM - insulin-dependent diabetes mellitus
IL – interleukin
IP3 – inositol 1,4,5- triphosphate
mcm – micrometer
MGF – mesoderm growth factor
MHC – major histocompatibility complex
MIR- masseter inhibitory reflex
mRNA – matrix rhibonucleic acid
MSG – minor salivary gland
MSG - monosodium glutamate
MSG - minor salivary glands
MU – motor unit
NE - norepinephrine
NGF – nerves growth factor
NICO – necrotizing intrabony cavitational osteonecrosis

NIA - National Institute of Aging
NKA - neurokinin A
NO – nitrogen oxide
NYP - neuropeptide Y
PET – positron-emission tomogram
PGE2 – prostaglandin E2
PHI - peptide histidine isoleucine
R- response
RCG - Route Central Generator
RPG – rheoparodontography
RNA – rhibonucleic acid
Slg A,M,G - secretory immunoglobulins A,M,G
TCA - tricycle antidepressant
TGF – transforming growth factor
TMD - temporomandibular disorders
TMJ – temporal-mandibular joint
TNF – tumor necrosis factor
USA – the United States of America
VIP - vasoactive intestinal peptide

Chapter 1. ORAL CAVITY ORGANS AND TISSUES STRUCTURAL-FUNCTIONAL CHARACTERISTICS

Oral cavity represents digestive tract initial part. It consists of vestibule and oral cavity as it is.

Oral vestibule is narrow fissure limited externally by lips and cheeks while internally – by teeth and gums, superiorily and inferiorily – by mucosa transducting to the gums from cheeks and lips.

Oral cavity properily is limited by oral floor and tongue down as well as hard and soft palate up. Lateral and anterior boarders of it are limited by teeth internal surface as well as alveolar process.

Oral cavity basement represents osseal-muscular skeleton consisting of maxilla with mandible with teeth, masticatory and mimic musculature. Oral mucosa except some areas which belong to the teeth crown is covered by mucosa.

BONE APPARATUS

It is represented by 15 bones 6 of which are paired (maxillas, zygomatics, palatine, lacrimal, nasal bones and inferior nasal conchas) while 3 are non-paired ones (vomer, mandible and sublingual bone). Bones participating in face basement formation and defining face are maxilla, mandible and sublingual bone. Two maxillas are surrounded by other face bones such as: palatal, nasal, lacrimal, inferior nasal conchas, zygomatics, vomer and mandible. All face bones except mandible and sublingual bone are interconnected between each other and are connected to the brain skull with sutures as well. Sublingual bone is connected with the skull basement by means of long ligaments.

Mandible

It forms joint with temporal bone. It is the only movable bone of facial skeleton. A great number of muscles are fixed on it due to which mandible is under permanent functional action. Also it explains its structure complicity. Mandible external and internal surfaces are rich in roughness, infoldings the origin of which is determined by action of the muscles attached to it, there is a canal within the lower jaw origined from mandubular foramen on ascending mandibular ramus medial surface (Juodzbalys; 2010).

Maxilla

It does not have masticatory muscles fixating points comparatively to mandible. Muscles locating on its surface belong to the mimic ones. That is why maxilla is not undergone to such a functional tension from muscles as mandible is. But it is under the mandible permanent influence conditions percepting pressure from it through the food piece or directly through the teeth.

From the first view, maxilla is seemed to be more fragile than mandible. This representation is possible due to presence of such air-containing cavities as maxillary (canine fossa, maxillary sinus, antrum of Highmore) and nasal cavities. Although despite

it it is capable to make strong resilience both to compress and to rupture. It is explained by contrforces (bone compact substance thickenings) presence in it. They are situated so that big tension appearing while food biting is redistributed to the jaw and then is transducted also to other bones connected to it.

TEMPORAL-MANDIBULAR JOINT

It is an articulation formed by temporal and mandibular bones. This joint structure has many common features with other human joints and it is characterized by some own anatomical and functional peculiarities at the same time.

Main elements: mandibular fossa, condylar process, articular disc, articular capsule, mandibular articular ligaments.

Mandibular fossa and articular tubercle are temporal bone part while mandibular head and condylar process belong to mandibular bone. Right and left articulations form one system physiologically, movements are performed at the same time in them.

Mandibular fossa is boardered by: articular tubercle in front, temporal bone tympanal part behind, zygomatic process outside.

Mandibular fossa fornix is formed by thin osseal plate boardering the joint from the skull cavity. The fossa posterior fornix boarders on tympanal cavity with middle and internal ear elements inside the cavity. Such a close anatomical bond of tympanal cavity and articular fossa encourages to inflammatory process transition to the mandibular fossa and the joint other parts. Mandibular fossa sizes are bigger than mandible head diameter that leads to incongruence (the head shape non-correspondence to the fossa shape). The joint articulating surface congruence is reached by the fossa size narrowing due to the joint capsule attachement not out of the fossa (like in other joints) but inside it – near temporal bone petrosal-tympanal fissure anterior limb as well by double-concaved disc.

Articular tubercle is formed by temporal bone zygomatic process posterior part thickening. Mandibular head slidens on the tubercle posterior surface at mandible vertical movements, head is set on the tubercle posterior surface near its apex.

Mandibular head has elliptoid shape, it is prolonged in the transversal direction and is narrowed in the sagittal one. The head shape and sizes have significant individual variability.

Articular disc is built from solid-fibered tissue. It has double-concaved shape determining the articulating surfaces congruence. A disc isolates mandible head from the fossa. That is why the joint cavity is divided into two floors – superior and inferior. The disc is located so that the head slidens on the tubercle posterior surface. It results in the following: maximal pressure during masticatory act is undergone to the tubercle but not to thin osseal plate forming mandibular fossa fornix posterior part. Articular disc being a soft flexible pad amortizes masticatory pressure force falling to the articulating solid congruenting surfaces.

Articular capsule represents elastic connective-tissular sheath regulating head movements in mandibular fossa. Joint capsule consists of two layers – fibrosal (external) and endothelial (internal).

The capsule anterior wall is attached to the articular joint frontal part, the posterior one – to petrosal-tympanal fissure while decreasing the joint fossa sizes. Joint capsule thickness is 0,4-1,7 mm. The thinnest parts are the capsule anterior and internal parts. The capsule biggest length is forward and externally due to which anterior mandible head fractures are observed significantly more often than the posterior ones.

Layers functions:

- endotheliocytes – produce synovial liquid determining sliding the articular surfaces and performing the joint biological protection from infection;
- fibrosal – gives hardeness, eleasticity and ability not to rupture even at the joint complete dislocations whereas it is often observed in other joints.

A space between the capsule posterior wall and the fossa posterior surface is filled with lax connective tissue encouraging to mandible movements back and performing the amortizing role at enforced functional loading to the joint.

The capsule posterior part probably is an antagonist of external pterygoid muscle tightening mandible disc and head forward.

Ligaments regulate movements in the joint and are divided into:

1) intracapsular: disc-temporal anetrior, disc-temporal posterior, disc-mandibular lateral, disc-mandibular medial;
2) extracapsular: lateral, sphenoid-mandibular, styloid-mandibular.

Joint ligaments especially the extracapsular ones prevent joint capsule stretching. They consist of fibrous non-elastic connective tissue. That is why their initial length is not restored after overstretching.

Ligament apparatus limits movements at inflammatory diseases, ligaments scar changings can cause practically complete loosing in the jaw mobility.

Blood supply

It is realized by internal carotid artery branches. Collaterals are weak to be developed between artery rami; the joint veins have wide anastomozes with ear veins.

Movements in temporal-mandibular joint

Under physiological conditions all movements of joint heads in the joint fossas are combined and have following components:

- vertical – corresponds to mouth opening and closage,
- sagittal – mandible movements forward and back,
- lateral or transversal – jaw replacement to the right and to the left.

Under norm mandibles movements are similar to the replacements by circle or ellips. Pathological changings and anomalies in dental rows and teeth lead to the movements amplitude lowering as well as to their number increasing.

Head mandibular movements peculiarity is a combination of forward and rotating movements in the joints. Any movement in the joint is origined from the forward one –

head sliding over the joint tubercle roll. Then the tubercle rotative movement round horizontal axe is added.

This characteristic functional peculiarity distinguishes temporal-mandibular joint from other joints in the human skeleton. It is determined by disc existence in the cavity. This disc divides the joint chamber into two chambers.

Forward movements appear in the superior chamber and the head replaces down over the articular tubercle roll. Rotative movements round horizontal axe take place at the same time in the horizontal chamber. Thus, the joint two parts isolated one from another with a disc are equal while the function performing because different-directed movements occur in the joint at the same time.

The second functional peculiarity of temporal-mandibular joint is that *movements are synchronous in both joints* because both joints (left and right) are interconnected by non-paired mandibular bone. This peculiarity must be taken into account at temporal-mandibular joint diseases diagnostics. So, for example, it should be done at one joint habitual dislocation when the second joint function gets disturbed.

Complicity in temporal-mandibular joint structure and function must be described in the aspect of taking the different food and variety of the movements essential for its grinding. Complete adenty results into the joint adaptation to new conditions because of mandibular movements amplitude changings.

Receptive function gets changed during implanting and joint disorders with special adaptive motor syndrome occurrence (Douglas; Avoglio; de Oliveira, 2010).

Alveolar processes

Alveolar processes are maxillary and mandibular processes carrying teeth.

Alveoles bony edges correspond to the teeth cervices contours and have waves-like shape though never reach tooth anatomical cervix – enamel boarder with root cement. Alveolar process osseal tissue does not differ practically from other bones of the human skeleton. The bone tissue consists of mineral salts (60-70%) and a little amount of water as well as of organic substances only 30-40% in weight the main component of which is collagen.

Alveolar process consists of external and internal cortical plates and is located between spongious tissue.

Spongious bone is formed by anastomozing trabecules the distribution of which corresponds usually to the direction of forces acting to the alveole during mastication. The trabecules redistribute the forces acting to the proper alveolar bone to the cortical plates. They are situated primarily horizontally in the alveoles lateral walls area while have more vertical course near their floor. Their amount is fluctuated in the alveolar process different locuses, gets decreased while aging and at the tooth absence. Osseal-cerebellar spaces filled by red bone marrow in the childhood while the yellow one – in the adulthood – are present between the trabecules.

Acid and alkaline phosphatase detected in the alveolar bone play an active role in osteogenesis and collagen formation.

Parodont neurotrophic and reactive functions are provided by numerous vertical canals in the spongious bone through which nerves, blood and lymphatic vessels pass.

Dentition

Humans and most other mammals develop two sets of teeth during a lifetime. The first teeth are called the deciduous teeth, or milk teeth, and their number is 20 in humans (figure 1, appendix).

General Eruption Pattern:

Both the deciduous and permanent dentitions have a general order, or pattern, of eruption.

Table 1

Deciduous Dentition: Normal Eruption Time (in months)

Eruption Age				
	Mandible	Order	Maxilla	Order
Central Incisor	6	1	7,5	1
Lateral Incisor	7	2	9	2
Canine	16	4	19	4
First Molar	12	3	14	3
Second Molar	20	5	24	5

As a general rule, mandibular deciduous teeth normally precede their maxillary counterparts in eruption (Table 1). It can also be said that the deciduous teeth normally erupt in order from the front of the mouth toward the back, even though the canines in each quadrant normally erupt after the first molars.

Teeth in primary dentition are smaller and fewer in number than permanent dentition to conform to the smaller jaw size.

Primary dentition: ~ 2 to 6 years of age

Mixed dentition: ~ 6 to 12 years

Permanent dentition: > 12 years

It takes three phases histologically different (Hulland, Lucas, Wake, Hesketh; 2000). As it can be seen, the permanent mandibular teeth normally precede their maxillary counterparts in eruption, as was also the pattern with the deciduous teeth (Table 2). If the first molar's eruption sequence is ignored, the permanent mandibular teeth exhibit a perfect anterior to posterior order. However, in the maxillary arch, not only is the first molar out of order but the canine normally follows both premolars.

Table 2

Permanent Dentition: Normal Eruption Time (in years)

Eruption Age				
	Mandible	Order	Maxilla	Order
Central Incisor	6-7	2	7-8	2

Lateral Incisor	7-8	3	8-9	3
Canine	9-10	4	11-12	6
First Premolar	10-11	5	10-11	4
Second Premolar	11-12	6	11-12	5
First Molar	6-7	1	6-7	1
Second Molar	11-13	7	12-13	7
Third Molar	17-21	8	17-21	8

Comparisons between Permanent and Deciduous Teeth (Goldberg; 2017)**:**

A. *External Considerations*:

1. The deciduous teeth are generally smaller than their permanent counterparts. This size disparity exists for crown and root portions of both anterior and posterior teeth.
2. The crown portion of the deciduous teeth is quite short inciso (occluso) gingivally, relative to its total crown-root length, when contrasted to the same dimensions of permanent teeth.
3. The crowns of deciduous teeth are wider mesiodistally, relative to their inciso (occluso) gingival height, when compared to the same dimensions of permanent teeth.
4. The crowns of deciduous teeth are more constricted faciolingually at the cervical line than are those of permanent teeth.
5. Crown relief has bigger variability sometimes.
6. Because of a greater occlusal convergence of the buccal and lingual surfaces, the occlusal tables of deciduous molars are relatively more constricted faciolingually than are the crowns of permanent molars.
7. There is a cervical ridge on both labial and lingual surfaces of deciduous anterior teeth, as well as on the buccal surface of deciduous posterior teeth. This ridge is normally much more prominent than any analogous structure found on permanent molars.
8. In comparison to the crown height occlusocervically, the roots of deciduous molars are relatively longer than those of permanent molars. They are, however, less substantial in their other dimensions, which in summary makes them longer and thinner.
9. The roots of deciduous molars reveal much more flare, or spreading, than do roots of the permanent molars. This flare creates additional space for the permanent premolar crown to develop. Their greater spread, coupled with the more slender shape and lack of a root trunk, make deciduous molar roots easier to fracture during extraction procedures. With other words, milk teeth have shorter and widely arranged roots (the permanent tooth germ is behind them).
10. The roots of deciduous molars branch almost directly from the base of the crown, so that there is no easily identifiable root trunk, as there is in the permanent molars. This feature also creates more space for the permanent premolar crown development.

11. The crowns of deciduous teeth are lighter in color. This is because they are more opaque and thus exhibit a whitish-white or even bluish-white cast, compared to the yellowish and grayish-white shades of permanent tooth crowns.
12. Bigger tooth cavity and its less thick walls.

B. *Internal Considerations*:
1. The enamel in the crowns of deciduous teeth is relatively thin, softer, fragile (more prone to fractures) and with bigger hydroxyapatite crystals when compared to permanent teeth (Low, Duraman, Mahmood; 2008).
2. Enamel has blue shade.
3. Enamel horizontal vallum (cingulum) is expressed in the crown cervical third.
4. Deciduous teeth enamel have more porous structure and it is hypomineralized (Sabel, Johansson, Kühnisch, Robertson, Steiniger, Noren et al.; 2008) because of which it is undergone to cariotic destruction (Imfeld; 1996).
5. Deciduous teeth higher enamel numerical density especially near dentine-enamel junction (Hueb de Menezes Olivera, Torres, Miranda Gomes-Silva, Chinelatti, Hueb de Menezes, Palma-Dibb et al.; 2010).
6. The dentine of primary teeth is also relatively thin, in comparison to permanent teeth but there were no reliable difference in dentinal tubules amount and diameter (Schilke, Lasson, Bauss, Geursten; 2000).
7. The differences are also in dentine and enamel composition in terms of type and amount of inorganic and/or organic phases, water concentration, microelements level and crystals size that can have significance in caries development in non-permanent teeth (Zenobio, Tavares, Zenobio, Silva; 2011).
8. Deciduous peritubular dentine is thcker than the permanent one, deciduous teeth possess smaller diameter and tubular density and thus less permeability in comparison with the permanent ones (Costa, Watanabe, Kronka, Silvia; 2002).
9. The pulp cavity is relatively larger in the deciduous teeth. The mesial pulp horns of deciduous molars are especially large.

It interesting to know about immune-modulating features of stem cells derivated from human exfoliated deciduous teeth (Alipour, Masoumi Karimi, Hashemibeni, Adib, Sereshki, Sadeghi; 2017), they are compared with the ones of bone marrow-derived mesenchymal stem cells (Alipour, Adib, Masoumi Karimi, Hashemibeni, Sereshki; 2013) and the ones from permanent teeth (Baghaban Eslaminejad, Vahabi, Shariati, Nazarian; 2010). Stem cells are taken from exfoliated deciduous teeth dental pulp (Mojarad, Amiri, Rafatjou, Janeshin, Farhadian; 2016).

MAXILLARY-FACIAL AREA MUSCLES PECULIARITIES

Maxillary-facial area muscles are divided into several independent groups: mimic, masticatory, of tongue, of soft palate, pharyngeal.

All mentioned muscles perform their role and participate in oral cavity various functions at the same time. For example, mimic muscles participate primarily in mimics, respiration and speech, in mastication – in less extent. On the contrary, masticatory muscles take part mainly in mastication, speech and less in respiration.

Mimic muscles

They are origined at the bone surface or from sublayed fascias and are ended in skin and are capable to cause face skin expressive movements (mimic) and reflect soul state. The mimic muscles majority are concentrated round oral foramen and eye fissure. Their muscular fibers perform sphincters role while the radial one – of the dilators (Table 3).

Table 3

Mimic muscles functions (Н.В. Головко, 2008)

The muscle name	***The muscle function***
Zygomatic muscle	Elevates corner of the mouth towards and up
Muscle of loughing	Toughens corner of the mouth laterally and leads to loughing fovea forming
Triangular muscle	Toughens corner of the mouth down at one-sided contraction and strengthens nasal-labial plica curvature while toughens all lip down at two-sided one
Superior lip square muscle	Toughens superior lip up, risens nasal-labial plica
Orbicular muscle	Closes or sharpens mouth; mouth orbicular muscles (located radially) antagonist
Canin muscle	Rises corner of the mouth at one-sided contraction while all inferior lip at at the two-sided one; triangular muscle antagonist
Inferior lip square muscle	Toughens inferior lip laterally and down
Inferior lip incisival muscle	Toughens corner of the mouth medially and down
Superior lip incisival muslce	Toughens corner of the mouth medially and up
Omental muscle	Rises and wrinkles chin skin as well as toughens inferior lip forward
Buccinator	Toughens corner of the mouth back, attaches cheeks to teeth and to jaws alveolar processes

Several muscles of facial expression act on the skin around the eyes and eyebrows. The occipital-frontal one raises the eyebrows. The eye orbicular one closes the eyelids and causes “crow’s feet” wrinkles in the skin at eyes lateral corners.

Several other muscles function is moving the lips and the skin surrounding the mouth. The eye orbicular muscle and buccinator, the kissing muscles, pucker of the mouth. The buccinator also flattens the cheeks as in whistling or blowing a trumpet and is therefore sometimes called the trumpeter's muscle. Smiling is accomplished primarily by the zygomatic muscles. Sneering is accomplished by the superior lip elevator and frowning or pouting largely by the depressor of mouth angle.

Blood supply

It is made by facial artery – external carotid artery branch.

Innervation

Innervation is realized due to facial nerve motor fibers.

Mimic muscles are close to masticatory muscles on their ***functions***.

They participate in: food capture, its supporing in oral cavity vestibule, oral cavity closure at mastication, speech creation, in new-borns – at sucking and at liquid food taking.

Masticatory muscles

Masticatory function is defined by the complex interaction of masticatory musculature, temporal-mandibular joint, teeth and nervous system during biting, jewing, swallowing and speech (Yamanel, Fukui; 2007). Masticatory muscles comprise temporal one, masseter, medial pterygoid and lateral pterygoid paired muscles (Table 4).

Table 4

Muscles of Mastication

Muscle	***Origin***	***Insertion***	***Action***
Temporal	Temporal region on the side of head	Mandible	Closes jaw
Masseter	Zygomatic arch	Mandible	Closes jaw
Pterygoid	Inferior side of the skull	Mandible	One closes jaw, one opens jaw

Masseter and medial pterygoid muscle serve first of all as an origin of powerful tension while the temporal and lateral pterygoid muscle are responsible for mandible stabilization.

Masticatory musculature functions in a complex with epihyoideal and subhyoideal muscles as well as with the ones of tongue, lips and cheeks. Neck muscles also influence indirectly on stabilization, participate into the head position changing during the mastication (Hannam, McMillan; 1994, P.O.Eriksson; 2000). Mouth closage id est mandible lifting appears due to two-sided symmetric activity of masseters, temporal and medial pterygoid muscles though muscles work non-symmetrically, with their bigger activity at the working side during the mastication (Bakke; 1993).

The gravitation action to mandible is equaled with the temporal muscles positive tone. They say that the last ones play important role in the mandible positioning in the space. Also masseters and medial pteryoid muscles get activated at the teeth closage. Mouth opening or mandible lowering is performed due to suprahyoid muscles (digastric muscle anterior belly, omy-hyoid and the mylohyoid one) with the lateral pterygoid muscles participance. Suprahyoid muscles are attached to the mandible and hyoid bone. When hyoid bone is fixed due to subhyoid muscles then suprahyoid muscles can participate in mandible lowering. Mandible symmetric protrusion (movement forward) is achieved by lateral pterygoid muscles two-sided action.

There are connections between masticatory function and skull, especially for mandible (von Cramon-Taubadel; 2011, Toro-Ibacache, Zapata Muňoz, O'Higgins; 2016).

Mandible retrusion (movement back) is performed by temporal muscles posterior part, suprahyoid muscles and masseter deep fibers. Laterotrusion (mandible shift on the right or on the left) takes place at contralateral pterygoid muscle contraction as well as at ipsylateral lateral pterygoid muscle contraction. Though laterotrusion is usually combined to the protrusion on the opposite side while antero-lateral movement forming.

As it is well-known, maxillary-facial region muscles are divided into 2 main groups: masticatory and mimic. They belong to skeletal muscles and possess the same features. Masticatory muscles contract mainly in auxotonic regimen i.e. with parallel tension and length changing. Masticatory muscles contracture can be developed due to masticatory muscles fatigue. Contracture means muscles retarded relaxation.

The four pairs of chewing muscles or mastication muscles are some of the strongest muscles of the body. The temporal and masseter muscles can be easily seen and felt on the side of the head during mastication. The pterygoid muscles, consisting of two pairs, are deep to the mandible. Masticatory musculature belongs to the force muscles. It means that they develop mainly force comparatively to other skeletal muscles which develop velocity. In course of masticatory musculature contraction force is developed. Such a force is necessary for mechanical action to the food piece, its crush, wearing down and grinding. Skeletal muscle with square in 1 cm^2 can develop muscular force in 10 kg. Transversal section sum for masticatory muscles ascending mandible on 1 side of face is equal to 19,5 cm^2, from the both sides – 39,0 cm^2. Thus, masticatory muscles absolute force is equal to 390 kg. At the same time, separate teeth parodont durability is weak. That is why, pain occurs in parodont during jaws enforced closure

and pressure further increasing reflectory stoppage is observed though muscular force has not exhausted yet.

Dental row masticatory center is dental-mandibular system region where food mechanical processing is performed maximally. In healthy people such a center represent small and large molars of another side from the one on which mastication takes place. During mastication left and right masticatory centers act in turns. At both masticatory centers function of loosing the crushing function is transferred to the first teeth which get adapted badly for this function performance under physiological conditions. That is why food crushing becomes bad and its processing with saliva becomes incomplete.

A representation about antagonists and synergists muscles myodynamic equilibrium has been given first by English scientist Rogers at the beginning of the XX-th century. To his point of view proper teething is influenced by balance of muscular forces acting from inside (tongue forces) and from outside (lips, cheeks muscles forces) while masseter and suprahyoid muscles force and action co-ordination act to jaw bones formation. Dental rows shape and size are determined primarily by growth direction during odontogenesis on one hand and by influence of muscular forces acting both under resting condition and under functioning on the other hand. Dental rows and occlusion proper forming are influenced by muscles anatomical peculiarities (shape, position, size) and by functional state (excitability, elasticity, tone et al.) as well.

Tongue muscles

Table 5

Tongue and Swallowing Muscles

Muscle	*Action*
Tongue muscles: • *intrinsic* • *extrinsic*	Change shape of tongue Move the tongue
Hyoid muscles: *a) Suprahyoid:* □ *geniohyoid,* □ *stylohyoid,* □ *hyoglossal* *b)Intrahyoid:* □ *thyrohyoid et al.*	Elevate or stabilize hyoid Depress or stabilize hyoid

Soft palate	Moves soft palate, tongue or pharynx
Pharyngeal muscles: *a) elevators;* *b) constrictors:* • *superior,* • *middle,* • *inferior*	 Elevate pharynx Constrict pharynx

Tongue is very important in mastication and speech. It moves food around in mouth, and with the buccinator muscle, holds the food in place while the teeth grind the food. Tongue pushes food up to the palate and back toward the pharynx to initiate swallowing. Tongue consists of intrinsic muscles mass, which are located entirely within tongue, and function to change its shape. The extrinsic muscles are located out off the tongue but are attached to and move tongue.

Tongue muscles anomaly (macroglossy) disturbs odontal-jaw system development. Tongue consists of muscles located in transversal, vertical and longitudinal directions. All muscles are interlaced between each other.

One can differentiate muscles origined from bones and the ones beginning in soft tissues – tongue proper muscles. The first muscles provide tongue replacement in all directions. They change the position and stretch oral cavity floor tissues while its shape changing. Tongue position changing is provided by omental-lingual, hypohyoid and stylohyoid muscles.

All tongue movements take place either at tongue muscles relaxation or contraction. Infrahyoid bone solid fixating is necessary often at this. Tongue proper muscles either make tongue flat, thicken it or give them groove-like shape at contraction.

When the suprahyoid muscles hold the hyoid bone in place from above, the infrahyoid muscles can elevate the larynx. To obtain this effect, place your hand on your larynx (Adam's apple) and swallow.

Pharyngeal muscles

The soft palate muscles close the posterior opening to the nasal cavity during swallowing, preventing food and liquid from entering the nasal cavity. Swallowing is accomplished by elevation of the pharynx and larynx. The pharyngeal elevators elevate the pharynx, and the pharyngeal constrictors constrict the pharynx from superior to inferior, forcing the food into the esophagus. Pharyngeal muscles also open the auditory

tube, which connects the middle ear with the pharynx. Opening the auditory tube equalizes the pressure between the middle ear and atmosphere. That is why it is sometimes helpful to chew gum or swallow when ascending or descending a mountain in a car or changing altitude in an airplane.

Mastication and its regulation

Mastication – is a complicated act (Клинеберг, Джагер; 2008, Rosentiel, Land, Fujimoto; 2006, Nallawsamy; 2007). Its essence is in consequent contractions of masticatory muscles, mandible, tongue and soft palate movements. Masticatory muscles are fixed to moveless skull part with their one end, with another end – to unique movable skull bone – mandible. They provide mandible status change as for maxilla while their contracting.

Mastication is origined from assessment of received food after which food piece irritates touch, temperature, gustatory, nociceptive receptors located in oral cavity. Besides, due to sense of smell impulses occuring in these receptors come through nervous pathways into medulla oblongata into the mastication center. Then they come to masticatory muscles through trygeminal nerve second and third branches, facial, glossal-pharyngeal and hypoglossal nerves. In parallel to the food getting smaller its washing with saliva occurs for better swallowing. Food getting smaller degree is under oral mucosa receptors control. Non-food elements are pushed at this by tongue (bones, stones, paper et al.). One should remember about necessity of careful food processing in oral cavity. It's an essential preventive measure for many diseases not only of alimentary tract. Sucking corresponds to mastication which is provided by mouth and tongue muscles reflectory contractions in babies.

Mastication act by its mechanism is partially arbitrary, partially – reflectory.

Human being can inhibit or enforce masticative movements freely as well as change their character. Food biting and mastication is performed at superior jaw teeth occlusion (contact) with inferior jaw teeth. Mandible performs rhythmic movements in 3 main directions: vertical, sagittal, transversal.

CNS activates separate motor units of those muscles which are required for the definite movement performance. All movements can be divided into conscious, reflectory and rhythmical. CNS different parts participate in masticatory movements formation. Facial motor cortex represents brain cortex higher area taking part in conscious movements generation. When the patient is asked to protrude his tongue and to open his mouth (for example, for the offprint taking) programs set choice and activation (similar to the computer programs) appear in basal ganglia. These programs send the signals in primary motor cortex in part in its facial zone. Facial primary cortex contains brain cortex specific zones the fibers of which pass through pyramidal pathway with shifting to alpha-motoneurons (for instance, through interneurons). Fibers of every

exit zone from facial primary cortex activates separate simple movement, for example, tongue movement forward, tongue replacement or the jaw shift on the right or on the left, mouth angle rising, mandible lowering. One and the same movement can be performed at facial primary cortex different zones participation. Cerebellum co-ordinates constantly the movements through the entrance signals coming into the motor zones. Every movement correction can be realized through shorter pathways (comprising the neurons less amount) the big number of which is origined from brain stem.

Rhythmic movements possess the features both of conscious and reflectory movements. Reflectory side of the rhythmic movement is in following: one must not think about the movement for its performance. For instance, we can masticate, breathe, swallow, walk without thinking about these processes but we are able to change these movements velocity and intensiveness at any instant consciously. Rhythmic movements performance and regulation are realized with the participation of especial spine and stem neurons sets. Every set is designated as a Route Central Generator (RCG). Mastication RCG is located in a medullary-pontic reticular formation. Swallowing is not a rhythmical process but it is also controlled with RCG located in a medulla oblongata.

Very active feed-back reactions are realized in oral cavity. Sensory feed-back is made at oral cavity mechanoreceptors. For example, parodont receptors transmit the signals about teeth contact level and direction; mucosa receptors give the information about food contact with the mucosa. Muscular spindles signalize about the muscle length and its length changing velocity at the mouth closage. Tendineal organ of Golgi tells about the enforcement developing with the muscle while the temporal-mandibular joint mechanoreceptors transmit the signals about the joint position.

Tongue, lips muscles and masticatory muscles contractive types and regimes at conversation

During mastication mandible displacement takes place due to masticatory muscles contractions occurring in tetanic regimen (mainly incomplete). Contraction type – auxotonic (accompanied by muscle length and tension changings). Lips participate in sounds formation; one can see isometric, isotonic and auxotonic (or auxometric) contractions. Regimen – tetanus.

Contractive types and regimens at mastication

Masticatory muscles contractive type is auxotonic, regimen is tetanic. Tongue: types – isotonic and auxotonic, regimen – tetanic.

Swallowing

Swallowing – is a complicated reflectory act due to which food is transported from oral cavity into stomach.

Swallowing Phases:

1) Oral arbitrary or voluntary – from food common mass in oral cavity small piece is separated which is pressed to hard palate wih tongue movements (Fox; 2004). Jaws are closed, soft palate is rised closing entrance into choanes. Palatal-pharyngeal muscles are contracted simultaneously with this. Septum is formed which closes passage between oral and nasal cavity in the result of these processes. Tongue moving ahead presses onto palate and pushes food piece into pharynx. Because of this food piece is pushed down into pharynx. Entrance into larynx is closed by epiglottis, vocal cord is closed to prevent food coming into trachea. As food piece comes into pharynx, soft palate anterior arch are contracted and together with tongue root prevent food returning into oral cavity. Shortly, a bolus of food is pushed by the tongue against the hard palate and posteriorly toward the oropharynx.
2) Pharyngeal-inarbitrary – can be described as a reflex that is initiated by stimulation of receptors in the oropharynx. It can be divided into several subphases:
   - the soft palate is elevated, closing off the nasopharynx;
   - the pharynx is elevated;
   - successive constriction of three pharyngeal constrictors from superior to inferior forces the bolus through the pharynx and into the esophagus;
   - the epiglottis is bent down over the opening of the larynx largely by the force of the bolus pressing against it;
   - as the inferior pharyngeal constrictor contracts, the upper esophageal sphincter relaxes,
   - allowing the bolus to enter the esophagus.

3) Oesophageal inarbitrary: this phase is responsible for moving food from the pharynx to the stomach. Muscular contractions of the esophagus occur in peristaltic waves. S wave of relaxation of the circular esophageal muscles precedes the bolus of food down the esophagus, and a wave of strong contraction of the circular muscles follows and propels the bolus through the esophagus. The peristaltic contractions associated with swallowing cause relaxation of the lower esophageal sphincter in the esophagus as the peristaltic waves approach the stomach.

Swallowing process as a reflectory act is performed due to irritation receptor endings of trygeminal, superior and inferior laryngeal, glossal-pharyngeal nerves located in soft palate and pharynx mucosa.

Swallowing center is located in medulla oblongata near respiratory center and is in reciproqual (antagonist) interrelations. Excitement respiratory center activity is inhibited in swallowing center, respiration is stopped in this moment that prevents food

particles passage into respiratory ways. Swallowing act afferent ways are superior and inferior pharyngeal, recurrent nerve and vagus fibres. They direct nervous impulses to muscles participating in swallowing.

The sensory impulses from pharyngeal opening are transmitted through the sensory portion of the trigeminal and glossopharyngeal nerve to medulla oblongata.

The swallowing process successive stages are controlled by neuronal areas in the medulla and lower pons that control swallowing and are collectively called the deglutition or swallowing center.

The motor impulses from the swallowing center to the pharynx and upper esophagus that cause swallowing are transmitted by the fifth, ninth, tenth and twelfth cranial nerves.

One must remember that the swallowing pharyngeal stage inhibits the medulla respiratory center for one to two seconds.

TEETH

Teeth are durable (Zaslansky, Friesem, Weiner; 2006), located in oral cavity and placed near 20% of its surface. The teeth cut, grind, and mix the food eaten. To perform these functions, the jaws have powerful muscles capable of providing an occlusive force between the front teeth of 50 to 100 pounds and for the jaw teeth 150 to 200 pounds. Also, the upper and lower teeth are provided with projections and facets that interdigitate, so that the upper set of teeth fits with the lower. This fitting is called occlusion, and it allows even small particles of food to be caught and ground between the tooth surfaces. Teeth perform senosory function by percepting and transducting the masticatory pressure to the periodontal receptors. Adaptation to different-solided food is regulated by functional activity of tongue muscles as well as the masticatory and mimic ones.

Reflectory bonds regulating salivary glands secretion and digestive tract activity are present between teeth, near-teeth tissues and oral cavity.

The tooth can also be divided into (figure 2, appendix): crown – the portion that protrudes out from the gum into the mouth, root – the portion within the jaw bony socket, neck – the collar between the crown and the root where the tooth is surrounded by the gum.

- *The anatomic crown* is the tooth portion covered by enamel.
- *The anatomic root* is the tooth lower two thirds.

- *The pulp chamber* houses the dental pulp, an organ of myelinated and unmyelinated nerves, arteries, veins, lymph channels, connective tissue cells, and various other cells.

Clinical crown: During eruption, the exposed crown extending from the cusp tip to the the gingival attachment area.

***Anatomic crown*:** Entire crown, extending from cusp tip to the cementoenamel (CE) junction.

Root. Root Formation. There should be an obvious cause of tooth eruption. But studies have not provided evidence for this. If a tooth that is continuously erupting (rodent incisor and guinea pig molar) is prevented the root still forms by causing bone resorption. Rootless tooth still erupts, some teeth erupt more than the total length of the roots and the teeth still erupt after root formation completing. Therefore root formation is accommodated during eruption and may not be the tooth eruption cause. One point of importance is that the tissue beneath the growing root resists the apical movement of the developing root. This resistance results in the occlusal movement of the tooth crown as the root lengthens.

A tooth represents hard cavitary formation. There are 2 main structures inside the tooth:

- ***tooth cavity*** – is filled with pulp;
- ***dentin*** – represents tooth solid basement – is similar to bone by its structure;
- ***enamel –*** covers dentin outside in the crown;
- ***cement –*** covers dentin in the root.

Teeth blood supply

Inflow

1) It is realized by maxillary arteries branches which in turn represents one of the external carotid ending branches.
2) Inferior alveolar artery leaves maxillary artery initial part and comes to the teeth with coming into mandible canal and sending dental rami to the teeth and near-teeth – to the parodont.
3) Maxillary teeth are supplied from maxillary artery ending part. Its branches:
    - posterior superior alveolar artery – penetrates into alveolar fossae at maxilla subtemporal surface (while shine vessels sending) and comes to the teeth while dental and near-dental branches sending to molars and parodont;

- subophthalmic artery – passing into maxilla one-named canal with branching into anterior superior alveolar arteries from which, in turn, dental and near-dental rami to the pre-molars, canines, incisives and parodont.

Blood outflow

1) First, blood comes into accompanying arteries one-named veins – anterior and posterior superior alveolar rami, inferior alveolar vein.
2) Then blood comes into large retromandibular vein (through maxillary veins) inflowing into internal jugular vein.

Arteries, veins and nerves are seen on the figure 3.

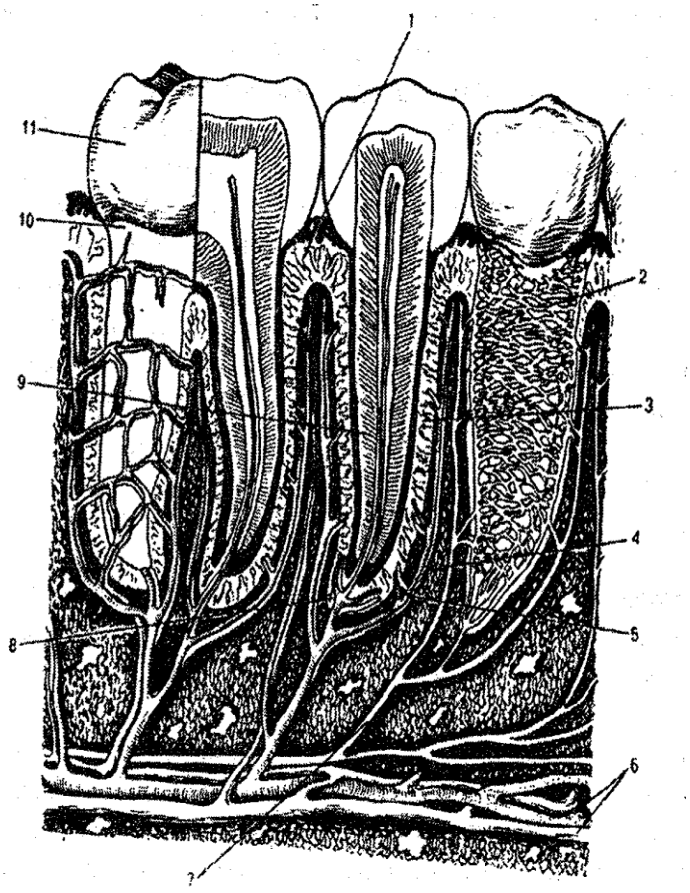

Figure 3. Dental-jaw segment structure (Гайворонский, Петрова; 2005). Designations: 1 – odontal-gingival fibers; 2 – alveolar wall; 3 – dental-alveolar fibers; 4 – alveolar-gingival branch; 5 – peridental vessels; 6 – jaw arteries and veins; 7 – nerve dental branch; 8 – alveolar floor; 9 – tooth root; 10 – tooth cervix; 11 – tooth crown.

Teeth innervation

1) It is performed by trigeminal nerve rami.

2) Autonomic (sympathetic) fibers from head autonomic nodes delt to trigeminal nerve are joined to these branches containing sensory fibers.

3) The last ones regulate trophycs of teeth tissues and parodont as well vascular lumen.

Superior teeth

1) Are innervated by maxillary nerve – the trigeminal nerve second branch (Tomaszewska, Zwinczewska, Gladysz, Walocha; 2015).

2) Subophthalmic nerve leaves it and passes the longest time in maxilla one-named canal.

3) Consequent branches outflowing from the last one: posterior superior alveolar branches coming to the molars with the one-named vessels; middle superior alveolar branch – to the premolars; anterior superior alveolar branches – to the canines and the incisives by one-named vessels course.

Inferior teeth

Are innervated by mandibular nerve from which inferior alveolar nerve comes to them following together with one-named vessels in mandible canal.

The nerves mentioned above form superior and inferior dental plexuses from which dental and gingival branches pass correspondingly to the teeth and parodont.

Different Teeth Functions

1. *Incisors* function as cutting or shearing instruments for food. The incisors as a group participate in all three of the major functions of the human dentition and have a greater role in esthetics and phonetics than any other group of teeth.

- Mastication - They function by biting, cutting, incising and shearing, thus breaking the food particles into smaller pieces suitable for grinding.
- Esthetics - Not only do the size, shape, color, and manner of placement of incisors directly contribute to a person’s appearance, but they provide the support necessary for the normal profile of the lips and face.
- Phonetics - They are necessary for certain sounds prononciation.

The central incisors functions in mastication are biting, cutting, incising and shearing. They also play an important role in the esthetics and phonetics functions of the human teeth. The lateral incisor supplements the central incisor in function. The mandibular central incisor functions are in biting, cutting, incising, and shearing, just as do their maxillary counterparts. The mandibular lateral incisor also complements the central in function.

2. *Canines*. The canines exhibit the greatest combined crown plus root length in each arch, and their root is very firmly anchored in alveolar bone. The thick facial plate of bone overlying the canine root is named the canine eminence. The canines are usually the most steadfast teeth in the mouth because of this bony support and the length of the root. Both the maxillary and the mandibular canine's role in mastication is mainly tearing, which is intermediate between the incising of the other anterior teeth, and the the posterior teeth grinding. They also contribute greatly to the cosmetic and facial support function, and play a part in phonetics as well.

3. Premolars. In function, the premolars mainly supplement the molars grinding during mastication, but there is still a tearing and piercing component, similar to that of the canine. The premolars contribute less to esthetics and phonetics than do the anterior teeth, but provide a greater share of these functions than do molars. In mastication, the first premolar functions basically as a grinding tooth, and contributes to the esthetics and phonetics roles as well. The two maxillary premolars are functionally alike. The mandibular first premolar actually is closer in form and masticatory function to a canine than to the other premolar in the mandibular arch.

4. Molars are located nearest the temporomandibular joint (TMJ), which serves as the fulcrum during function. The main masticatory function is grinding.

Pulp

As the principal source of pain within the mouth and as the major site of attention in endodontic treatment, the pulp warrants direct inspection (Pashley, Walton, Slavkin – electronic version). The pulp cavity of each tooth is filled with pulp. The dental pulp has most of its volume primarily composed of fibers and ground substance. These form the body and integrity of the pulp organ. Pulp represents any tooth soft part. It is connective tissue with abundant amount of nerves, blood and lymphatic vessels. Pulp does not contain elastic fibers comparatively to other types of connective tissue. The cells lining the surface of the pulp cavity are the odontoblasts, which, during the formative years of the tooth, lay down the dentin but at the same time encroach more and more on the pulp cavity, making it smaller. The pulp lives for the dentin and the dentin lives by the grace of the pulp. Few marriages in nature are marked by a greater interrelationship. Thus it is with the pulp and the four functions that it serves: namely, the formation and the nutrition of dentin and the innervation and defense of the tooth.

Pulp is characterized by highly-developed vascular net and rich innervation.

Blood supply

It is realized by arteries passing through the root canal apical foramen. Also some arteries come into pulp through the additional foramens especially in the roots apexes area. Thus, despite the fact that separate vessels have got little diameter, a common diameter of the vessels supplying pulp with blood is sufficient to its normal nutrition.

In root pulp arteries give some branches and only crown pulp is characterized by vast vascular net development. Vascular plexus from arterioles and capillaries anastomozing with each other gets formed under odontoblasts layer and inside the layer.

There are special vessels-reservoirs named as giant capillaries by course of which cone-shaped infoldings and sinuses get formed. Capillary net is the mostly branched in odontoblasts layer which are in a close contact with capillaries wall. Thus, odontoblasts high metabolic activity and plastic function are provided.

The presence of arteriovenous shunts in the pulp provides the opportunity for blood to shun, past capillary beds since these arteriole-venule connections are "upstream" from the capillaries. Alternatively, the arteriole-venule shunts could remain nearly closed (in a constricted state), and most of the blood would pass peripherally in the pulp to perfuse capillaries and the cells that they support. It has been suggested that the distribution of blood flow might change during pulp inflammation. Increased dilation of arteriole-venule shunts may produce "hyperemia," in which more blood vessels than normal are open and filled with blood cells; this may indicate more rapid blood flow or represent partial stasis. Further, this dilation of arteriole-venule shunts may "steal" blood from capillary beds causing waste products accumulation.

Capillary fenestration may indicate that these capillaries are more permeable to large molecules or that they allow more rapid fluid movement across the endothelium. However, studies on pulp capillaries suggest a lower than normal permeability to large molecules. On the other hand, a higher rate of fluid movement has not been ruled out.

Blood circulation in pulp is performed inside the tooth cavity having rigid walls. Blood volume pulse fluctuation must cause tissular pressure rising and physiological processes disorders in the tooth pulp as a result. But it does not appear due to arteries volume pulse oscillations transduction to the veins.

Pulp vascular wall antistagnative feature:

crown pulp veins summary lumen is bigger than in the apical foramen area.

Main result:

blood movement linear velocity is higher in the tooth root apical foramen than in the crown pulp.

Veins pulse oscillations are similar to the ones of brain veins. The tooth corresponding pulp venous vessels anastomize with the periodontal veins. Anastomoses rich net with periodontal veins provides pulpal circulation big functional possibilities. Anastomozes activity is expressed in blood periodic shunting from arterial vascular bed into the venous one at corresponding strong pressure changings in the pulpal chamber. Pain periodicity at pulpitis is connected with mentioned mechanism.

The anastomoses of pulpal, periodontal, and alveolar ***lymphatics*** may be important routes for the spread of pulpal inflammation into adjacent tissues during the removal of irritants and fluid from the pulp. These structural interrelationships have not received the attention they deserve. Finally, the extent and degree of anastomoses of

apical venules with those of the periodontal ligament and alveolar bone need investigation. Vessels may provide a route for local anesthetic movement during intraosseal or periodontal ligament injections rather than the fluid "dissecting" through perivascular tissue spaces. These same pathways have been implicated as routes of spread of inflammation from pulp to periodontal ligament and/or bone and vice versa.

The presence of pulpal lymphatics is disputed.

Innervation

It is performed by trigeminal nerve branches as well as sympathetic nervous fibers.

Part of myelin-free fibers belongs to sympathetic nervous system and contains norepinephrin. They are mainly vaso-motor, regulate arterioles tone and blood volume in pulp.

Myelin-free fibers significant part contains neuro-peptides accumulating in shine densed granules: cholecystokinine, leu- and metenkephaline, neuropeptides Y and K, vaso-active intestinal peptide (VIP), substance P, somatostatin and others.

It is considered that peptidergic fibers: participate in circulation regulating in pulp, provide noceoceptive sensitivity, regulate different neuromediators release, influence on inflammation development.

Several nerve bundles, each containing numerous unmyelinated and myelinated nerves, pass into each root via the apical foramen. The majority represents unmyelinated nerves, most of which are part of the sympathetic division of the autonomic nervous system; these have been shown to cause reductions in pulp blood flow when stimulated. The remaining nerves are myelinated sensory nerves of the trigeminal system. The myelinated nerve fibers branch extensively beneath the cell-rich zone to form the so-called plexus of Raschkow. The nerve endings terminate far short of the dentinoenamel junction; rather, endings are found only in tubules of the inner dentin and predentin, on or between odontoblasts. Some sensory axons exhibit terminal aborizations that innervate up to 100 dentinal tubules. Significantly, sensory nerves of the pulp respond to noxious stimuli with pain sensation only, regardless of the stimulus. This pain is produced whether the stimulus is applied to dentin or the pulp. Cavity preparation in the unanesthetized tooth is painful at any depth of dentin. How can this occur if there are no sensory nerves in the outer two-thirds of dentin? The answer probably lies in the hydrodynamic theory in which fluid movement within tubules stimulates distant sensory nerve endings.

Highly organized junctions have been demonstrated between some nerve fibers and odontoblasts. Although they do not appear to be typical synaptic junctions, their existence must be functional. It is unclear whether the activity is sensory or motor. An additional function of sympathetic nerves is the possible regulation of the rate of tooth eruption.

Sympathetic nerve activity influences local blood flow and tissue pressure by opening or closing arteriovenous shunts as well as arteriolar blood flow; this may

secondarily affect eruptive pressure. Activation of sympathetic fibers not only reduces pulpal blood flow but also decreases the excitability of intradental nerves. Thus, there is very intimate relationship between pulpal nerves and their excitability and local blood flow. Numbers and concentrations of nerves vary with the stage of tooth development and also with location. Very few nerves appear in the human pulp prior to tooth eruption. After eruption, the highest number of nerves is found in the pulp horns (about 40% of the tubules are "innervated"). The number of nerves per tubule drops off to about 4.8% in the more lateral parts of the coronal dentin to less than 1% in the cervical region, with only an occasional nerve in radicular dentin. Patterns of branching nerves seen with the light microscope would confirm numbers of nerves at different levels. There is little branching off the main nerve bundles until the coronal pulp. Regions of sensitivity also correlate in that coronal pulp and dentin are more painful to stimuli than are radicular pulp and dentin. The same stimuli applied to dentin were described as "sharp" when applied to coronal dentin but "dull" when applied to radicular dentin. Restorative procedures in rat teeth cause sprouting of pulpal and intradental nerves that may modify both dentin sensitivity and local inflammatory reactions.

Interestingly, removal of the pulp by extraction of the tooth or by pulpectomy and, presumably, pulpotomy results in the successive degeneration of the cell bodies located in the spinal nucleus of the trigeminal nerve, the main sensory ganglion, and the peripheral nerve leading to the tooth in the socket.

Functions

Formation of the dentin is the primary task of the pulp in both sequence and importance.

From the mesodermal aggregation known as the dental papilla arises the specialized cell layer of odontoblasts adjacent and internal to the inner layer of the ectodermal enamel organ.

Ectoderm interacts with mesoderm, and the odontoblasts initiate the process of dentin formation. Once under way, dentin production continues rapidly until the main form of the tooth crown and root is created. Then the process slows, eventually to a complete halt. Nutrition of the dentin is a function of the odontoblast cells and the underlying blood vessels.

Nutrients exchange across the capillaries into the pulp interstitial fluid, which, in turn, travels into the dentin through the network of tubules created by the odontoblasts to contain their processes. Innervation of the pulp and dentin is linked by the fluid and by its movement between the dentinal tubules and peripheral receptors, and thus to the sensory nerves of the pulp proper.

Defense of the tooth and the pulp itself has been speculated to occur by the creation of new dentin in the face of irritants.

The pulp may provide this defense by intent or by accident; the fact is that formation of layers of dentin may indeed decrease ingress of irritants or may prevent or

delay carious penetration. The pulp galvanizes odontoblasts into action or produces new odontoblasts to form needed hard tissue.

The defense of the pulp has several characteristics. First, dentin formation is localized. Dentin is produced at a rate faster than that seen at sites of nonstimulated primary or secondary dentin formation.

Microscopically, this dentin is often different from secondary dentin and has earned several designations: irritation dentin, reparative dentin, irregular secondary dentin, osteodentin, and tertiary dentin.

The type and amount of dentin created during the defensive response appear to depend on numerous factors. A second defensive reaction, inflammation within the pulp at the site of injury, should not be ignored.

In later life, the dentin stops growing and the pulp cavity remains essentially constant in size.

However, the odontoblasts are still viable and send projections into small dentinal tubules that penetrate all the way through the dentin; they are of importance for exchange of calcium, phosphate, and other minerals with the dentin.

Pulp provides any tooth normal activity as well as regenerative processes in it. The pulp contains a pool of reserve cells, descendants of undifferentiated cells in the primitive dental papilla.

These multipotential cells are likely a fibroblast type that retains the capability of dedifferentiating and then redifferentiating on demand into many of the mature cell types.

Beneath the odontoblasts, in the cell-rich zone there are concentrations of such cells. Also important are the reserve cells scattered throughout the pulp, usually in juxtaposition to blood vessels.

They retain the capacity, on stimulation, to divide and differentiate into other mature cell types. For example, mast cells and odontoclasts (tooth resorbers) arise in the presence of inflammation.

The unique cells differentiating to form the calcified tissue that develops under a pulp cap or pulpotomy when calcium hydroxide is placed in direct contact with the pulp are significant.

These unique cells are also frequently observed along the calcified tissue forming at the base of tubules involved with caries, restorations, attrition, or abrasion. This calcified tissue is not a true demonstrated the process of collagen synthesis and secretion by the fibroblast.

Not only fibroblasts are the principal producers of collagen, they also eliminate excess collagen or participate in collagen turnover in the pulp by resorption of collagen fibers. This has been demonstrated to occur intracellularly by the action of lysosomal enzymes, which literally digest the collagen components.

Trophyc function - crown and root dentin feeding as well as of cement is provided by odontoblasts processes (dentin partially and mainly root cement are supplied by blood through periodentium vascular wall).

Enamel trophycs, only in less extent, is also performed through odontoblasts processes.

Plastic function – is delt to dentinogenesis. It is expressed beginning from tooth formation start and lasts for all human life.

Protective – is provided by:

- endotheliocytes high absorbtive activity,
- pulp active inflammatory reaction to irritation, side substances penetrating and other phenomena accompanied by connective-tissular capsule forming restricting the injury zone as a result of which the damaged tooth is saved;
- defense cells.

Sensory – is realized by nervous fibers big amount presence in pulp.

Histiocytes and Macrophages. Undifferentiated mesenchymal cells (see the figure 4) around blood vessels (pericytes) can differentiate into fixed or wandering histiocytes under appropriate stimulation (Быков; 1997).

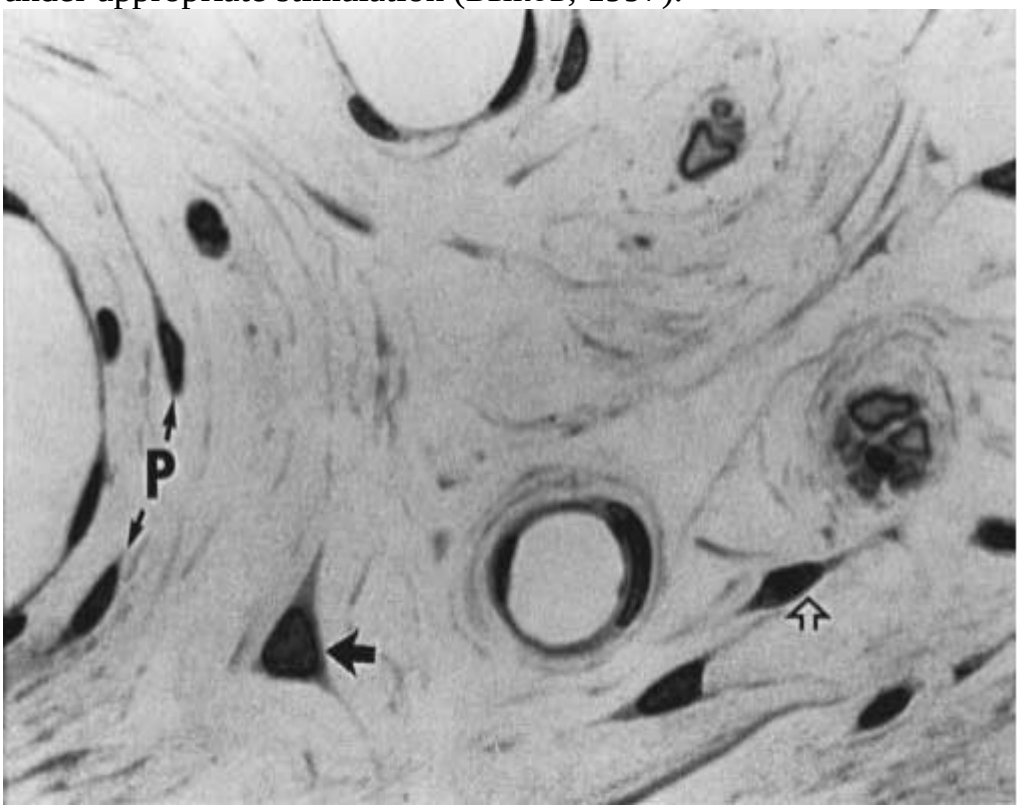

Figure 4. Pulpal fibroblasts showing spindle-shaped cytoplasm. Plump nuclei (dark arrow) usually indicate active collagen formation. Condensed nucleus is in a "quiet" cell, often termed a fibrocyte (open arrow). Pericytes (P) lie in close apposition to vessels and differentiate into other typical pulp cell types on demand (Быков; 1997).

Lymphocytes and Plasma Cells. These inflammatory cell types generally appear following invasion into the area of injury by neutrophils. These cells are not normally present in healthy pulp tissue but are associated with injury and resultant immune

responses— attempts to destroy, damage, or neutralize foreign substance(s). Their presence would therefore indicate the presence of a persistent irritant.

Mast Cells. Interestingly, mast cells are seldom in large numbers in normal, healthy pulps but are commonly found in inflamed pulps. The granules of these cells contain histamine, a potent inflammatory mediator, and heparin. These cells release these granules or degranulate into the surrounding tissue fluid during inflammation. Since these cells are generally found near blood vessels, degranulation of mast cells releases histamine close to vascular smooth muscle, causing vasodilation. This increases vessel permeability, allowing fluids and leukocytes to escape.

Odontoblasts. The principal cell of the dentin-forming layer, the odontoblast, is the first cell type encountered as the pulp is approached from the dentin.

Wandering histiocytes (macrophages) may also arise from monocytes that have migrated from vessels. These cells are highly phagocytic and can remove bacteria, foreign bodies (endodontic paste, zinc oxide, etc), dead cells, or other debris. Pulpal macrophages and dendritic cells thought to function like Langerhans' cells have been identified in normal rat pulp. These cells seem to be associated with pulpal immunosurveillance.

Polymorphonuclear Leukocytes. The most common form of leukocyte in pulpal inflammation is the neutrophil, although eosinophils and basophils are occasionally detected. It is important to know that although neutrophils are not normally present in intact healthy pulps, with injury and cell death they rapidly migrate into the areas from nearby capillaries and venules. They are the major cell type in microabscess formation and are very effective at destroying and phagocytizing bacteria or dead cells. Unfortunately, their participation often injures adjacent cells and may contribute to the development of wider zones of inflammation.

Shortly About Pulp:

- Innermost part of the tooth
- A soft tissue rich with blood vessels and nerves
- Responsible for nourishing the tooth
- The pulp in the crown of the tooth is known as the coronal pulp
- Pulp canals traverse the root of the tooth
- Typically sensitive to extreme thermal stimulation (hot or cold)

Effect of Posture on Pulpal Pain

Whenever an appendage is elevated above the heart, gravity acts on blood on the arterial side to reduce the effective pressure and, hence, appendage blood flow. This is why one's arm rapidly tires when working overhead. The reduced pressure effect occurs in structures in the head that, in normal upright posture, are well above the heart. When the patient lies down, however, the gravitation effect disappears, and there is a significant increase in pulp blood pressure and corresponding rise in tissue pressure over and above that caused by endogenous mediators of inflammation. In this position, an

irritated and inflamed pulp becomes more sensitive to many stimuli and may spontaneously begin to fire a message of pain. This is why patients with pulpitis frequently call their dentists after lying down at night. In the supine position, a higher perfusion pressure and, presumably, a higher tissue pressure develop in the patient, which cause more pulp pain. Patients often discover that they are more comfortable if they attempt to sleep sitting up, which again emphasizes the effects of gravity on pulp blood flow. Another factor contributing to elevated pulp pressure on reclining is the effect of posture on the activity of the sympathetic nervous system. When a person is upright, the baroreceptors (the so-called "carotid" sinus), located in the arch of the aorta and the bifurcation of the carotid arteries, maintain a relatively high degree of sympathetic stimulation to organs richly innervated by the sympathetic nervous system. Canine pulps showed large reductions in blood flow when the baroreceptor system was manipulated. If the human dental pulp is similar, it would result in slight pulpal vasoconstriction whenever a person is standing or sitting upright. Lying down would reverse the effect with an increase in blood flow and tissue pressure in the pulp. Lying down then increases pulp blood flow by removing both the effects of gravity and the effects of baroreceptor nerves which decrease pulpal vasoconstriction. Thus, the increase in pain from inflamed pulps at night or the transformation of the pain from a dull to a throbbing ache has rational physiologic bases.

Physiologically, as well as structurally, sharp contrasts set the periodontal ligament apparatus off from pulp tissue:

1) It is, for example, an organ of the finest tactile reception. The smallest contact on the tooth will stimulate its numerous pressor receptors. The pulp contains no such receptors. Proprioceptors of the periodontal ligament present the capability of spatial determination. It is for this reason that an inflamed periodontium can be localized easier by the patient than can an inflamed pulp.

2) Collateral blood supply, so lacking within the pulp, is abundant in this area. This rich blood supply is undoubtedly a major factor in the periapex ability to resolve inflammatory disease. In contrast, the pulp often succumbs to inflammation because it lacks collateral vessels.

3) The apical periodontium communicates with extensive medullary spaces of alveolar bone. The fluids of inflammation and resultant pressures apparently diffuse through this region more readily than is possible in the confined pulp space.

Enamel

The outer surface of the tooth is covered by a layer of enamel that is formed before eruption of the tooth by special epithelial cells called ameloblasts. Once the tooth has erupted, no more enamel is formed. Enamel is composed of very large and very dense crystals of hydroxyapathite with adsorbed carbonate, magnesium, sodium, potassium, and other ions imbedded in a fine meshwork of strong and almost insoluble

protein fibers that are similar in physical characteristics (but not chemically identical) to the keratin of hair. The crystalline structure of the salts makes the enamel extremely hard – much harder than the dentin. Also, the special protein fiber meshwork, although constituting only about 1 per cent of the enamel mass, makes enamel resistant to acids, enzymes, and other corrosive agents because this protein is one of the most insoluble and resistant proteins known.

Shortly:

- Structure: highly calcified and the hardest tissue in the body, crystalline in nature, enamel rods
- Insensitive—no nerves
- Acid-soluble—will demineralize at a pH of 5.5 and lower
- Cannot be renewed
- Darkens with age as enamel is lost
- Fluoride and saliva can help with remineralization and demineralized enamel lesions remineralization is important in clinics (Sharifi, Khoramian Tusi, Arab Pour, Majidi; 2016)
- Microhardness is decreased by iron administration and is increased by silicone oil, hydroxyapathite derivate chitosan administration (Tabari, Alaghemand, Rabiee, Khefri, Seyyed Ahadi, Nikpour; 2013).

Despite its great hardness and density, enamel has an appreciable porosity. Pore structure affects the mechanical properties of enamel. It also influences the interaction between light and enamel, understanding of which is necessary for development of transillumination techniques for caries detection and for optical matching of restorative materials and tooth tissue. However, interest in enamel porosity has above all been stimulated by the important role played by diffusion of acids and mineral ions in caries formation. Because enamel mineral exists as very small crystals, organized in an elaborate structure, the internal pores are small and variable in form, orientation and distribution. Microscopical information on pore structure tends to be prone to artefact but much information has been obtained by a range of less direct methods. Most studies have employed human or bovine permanent enamel.

The largest pores in enamel are associated with the prism junctions, but these constitute only a small fraction of the total porosity, most of which is associated with the prism bodies and tails. Here, most pores may exist as very narrow gaps between closely packed crystals but some, while still small, are elongated and tubule-like, and may well communicate with the prism-junction pores only through narrow intercrystalline pores. Organic matrix seems to be present within all pores and may alter pore sizes and modify diffusion. There are significant variations in porosity, apparently affecting both inter- and intraprismatic compartments, both within a single tooth (from outer to inner surface) and between tooth types. Pore structure affects the mechanical and optical properties of enamel. The formation of caries lesions is strongly influenced by the pathways for diffusion and by electrochemical effects arising from the charge on the pore walls.

It is appropriate to stress here the fundamental role of pore structure in this respect. The orientated laminar pores provided by the prism junctions provide planes of weakness, so that fractures in enamel propagate preferentially along the junctions. It has been found that enamel is more compressible at low loads and suggested that this was due to displacement of free water from pores. Permeability appears to be strongly influenced by matrix components.

Among the permanent teeth, canine enamel appears to be significantly less permeable than molar or incisor enamel. Enamel of deciduous teeth has lower mineral content than that of permanent teeth, except near the enamel dental junction. Diffusion coefficient is about 30 times less for permanent enamel than for deciduous enamel.

Observations on the membrane potential and streaming potential at different pH values suggest that the isoelectric point of enamel lies at about pH 4.

Enamel is permeable to water, ions, small organic solutes and dyes. Microscopical observations show that the prism junctions provide the main pathways, although, in inner enamel, some transport was observed within the prisms. Studies of fluid movement and electrical impedance show that permeability increases from the outer surface towards the enamel-dentin junction. Enamel forms an imperfect semi-permeable membrane in that, while water is transported through the tissue under the influence of an osmotic gradient, solute also moves in the opposite direction.

Inter- and intraprismatic diffusion represent two independent processes occurring in parallel, i.e. in separate compartments. However, it is highly unlikely that there is no exchange between the two pore systems. Irreversible binding is a possible difficulty in these penetration studies. Calcium, phosphate and fluoride, being components of the enamel crystals, may precipitate if the solubility product is exceeded. Reduction in slow diffusion for calcium with increasing calcium concentration is due to a surface diffusion mechanism. Diffusion of ionic solutes is affected, not only by pore size, but by interactions with the charge on the enamel solid (negative under quasi-physiological conditions). Because these interactions occur within narrow pores, enamel has the properties of an ion-exchange membrane. Ions having the same charge as the pore walls are partly excluded, while ions of opposite charge can enter the pores more readily and thus diffuse faster. Consequently, an electric potential difference (membrane potential) develops between two salt solutions with different concentrations separated by enamel. With gradients of salts such as KCl or NaCl (pH 6±7), the membrane potential has a negative sign, showing that the enamel has a negative charge. Phosphate, fluoride and various organic anions make the potential more negative, since these ions adsorb to the enamel and increase its negative charge. With a gradient of salts of divalent cations (e.g. Ca2+, Mg2+, Mn2+), the sign of the potential is reversed, because cation adsorption reduces or reverses the charge on the enamel.

Dentin

The main body of the tooth is composed of dentin, which has a strong, bony structure (figure 5), high toughness to fractures (Iwamoto; Ruse, 2003, Imbeni; Nalla; Bosi; Kinney; Ritchie, 2003). Dentin is made up principally of hydroxyapathite crystals similar to those in bone but harder comparatively to bone. These crystals are imbedded in a strong meshwork of collagen fibers. In other words, the principal constituents of dentin are very much the same as those of bone. Moreover, the dentin innerest layer (near odontoblasts bodies) is not unlamed and is represented only by collagen fibers and basal substance gluing them. The major difference is its histological organization, because dentin does not contain any osteoblasts, osteocytes, osteoclasts, or spaces for blood vessels or nerves. Instead, it is deposited and nourished by a layer of cells called odontoblasts, which line its inner surface along the wall of the pulp cavity. Dentinoblasts tips participating in dentin development are on pulp periphery. Only their processes are distributed in dentin while passing in the latest one in the special cavities – dentinal tubules. The calcium salts in dentin make it extremely resistant to compressional forces, and the collagen fibers make it tough and resistant to tensional forces that might result when the teeth are struck by solid objects.

Shortly:

- Softer than enamel, with load-independent hardness (Low, Duraman, Mahmood; 2008)
- Susceptible to tooth wear (physical or chemical)
- Does not have a nerve supply but can be sensitive
- Is produced throughout life
- With marked variations in structure in apical regions (Mjör, M.R.Smith, Ferrari, Mannocci; 2001)
- Three classifications: primary, secondary, tertiary
- Will demineralize at a pH of 6.5 and lower
- Possesses very high permeability (Mjör; 2009)
- Dental follicle represents ectomesenchymal tissue round developing tooth germ that gives stem cells and precursors for cementoblasts, periodontal ligament cells and osteoblasts (Morsczeck, Götz, Schierholz, Zeilhofer, Kühn, Möhl, Sippel, Hoffmann; 2005).

  Figure 5 (appendix).

Three classifications:

- Primary dentin forms the initial shape of the tooth.
- Secondary dentin is deposited after the formation of the primary dentin on all internal aspects of the pulp cavity.
- Tertiary dentin, or “reparative dentin” is formed by replacement odontoblasts in response to moderate-level irritants such as attrition, abrasion, erosion, trauma, moderate-rate dental caries, and some operative procedures.

Dentinal Tubules Role

- Dentinal tubules connect the dentin and the pulp (innermost part of the tooth, circumscribed by the dentin and lined with a layer of odontoblast cells).
- The tubules run parallel to each other in an S-shape course.
- Tubules contain fluid and nerve fibers.
- External stimuli cause movement of the dentinal fluid, a hydrodynamic movement, which can result in short, sharp pain episodes.
- Presence of tubules renders dentin permeable to fluoride.
- Number of tubules per unit area varies depending on the location because of the decreasing area of the dentin surfaces in the pulpal direction.

Dentin Sensitivity

Clinicians recognize that dentin is exquisitely sensitive to certain stimuli. It is unlikely that this sensitivity results from direct stimulation of nerves in dentin. As previously stated, nerves cannot be shown in peripheral dentin. Another speculation is that the odontoblastic process may serve as excitable "nerve endings" that would, in turn, excite nerve fibers shown to exist in deeper dentin, closer to the pulp. Neither odontoblastic processes nor excitable nerves within dentin are responsible for dentin's sensitivity. This led Brannstrom and colleagues to propose the "hydrodynamic theory" of dentin sensitivity, which sets forth that fluid movement through dentinal tubules, moving in either direction, stimulates sensory nerves in dentin or pulp. Further support for the hydrodynamic theory came from electron microscopic examination of animal and human dentin, demonstrating that odontoblastic processes seldom extend more than one-third the distance of the dentinal tubules. Work by LaFleche and colleagues suggested that the process may retract from the periphery during extraction or processing. Obviously, more investigation will be required before any definitive statement can be made regarding the distribution of the process. The tubules are filled with dentinal fluid that is similar in composition to interstitial fluid. The hydrodynamic theory satisfies numerous experimental observations. Although it cannot yet be regarded as fact, it has provided and will continue to provide a very useful perspective for the design of future experiments.

Cementum

Cementum characteristics:

- Thin layer of mineralized tissue covering the root surface.
- Softer than enamel and dentin.
- Anchors the tooth to the alveolar bone along with the periodontal ligament.
- Not sensitive.
- Bonelike, rigid connective tissue covering the root of a tooth from the cementoenamel junction to the apex and lining the apex of the root canal.

- It also serves as an attachment structure for the periodontal ligament, thus assisting in tooth support.
- It is avascular structure. One can differentiate cementum's types (Ho, Senkyrikova, Marshall, Yun, Wang, Karan et al.; 2009): cellular and/or acellular dependently on cementocytes (cement cells) presence or absence.

Afibrillar cementum covers cementum-enamel junction; this cementum type is formed by collagen, glycosaminoglycans and blood protein osteopontin.

There are three types of cementum-enamel junctions:

- an overlap – cementum overlaps enamel, such a cementum is known as coronal cementum and possesses lower elasticity than primary cementum;
- abutment – cementum butts with enamel;
- a gap depending on the sectioning plane.

PARODONT

Parodont represents a complex of tissues tightly connected between each other, surrounding and fixating the teeth: gums, epioosteum, dental alveole wall, peridentium, cement covering tooth root.

It is should be mentioned that life activity of every parodontal element is impossible out of this functional-morphological system. Disorder in form and function of any part of it is accompanied by corresponding reaction in parodont other tissues.

Blood supply

Mainly it is performed due to maxillary artery branches. Other arteries influencing in less extent are: facial, lingual, superficial temporal, internal carotid.

Maxilla dental organs are supplied from maxillary artery from which superior alveolar arteries come. The maxillary artery second branch coming into mandible canal is designated as inferior alveolar artery. It forms dental and interalveolar rami in mandibular canal. These branches determine mandibular tissues blood supply.

Parodont blood supply is performed by many collaterals which are formed by vascular anastomozes net with microcirculative systems of jaws alveolar process, tooth pulp and sublayed soft tissues. Vast vascular net as plexuses, loops and capillary glomeruli are located between alveole osseal wall and the tooth root. Amortizative (demppher) periodontal system forms due to it. This system is essential for masticatory pressure equaling by means of capillary anastomozes.

Maxillary veins are connected to the orbital veins through infraorbital vein and in turn to the skull venous sinuses through them. Besides, venous blood comes into jugular veins from parodont.

Innervation

It is performed by trigeminal nerve and vegetative nodes. Pterygo-palatinal node is parasympathetic one while superior cervical sympathetic node is the sympathetic one.

Maxillary teeth

They are innervated by superior alveolar nerves of the trigeminal nerve second branch.

Mandibular teeth

They take their innervation from the trigeminal nerve third branch and parodont major innervation is realized by inferior al.

***Functions*:**

1) supporting-maintaining function,
2) distributing pressure,
3) plastic,
4) trophyc,
5) teeth growth,
6) teething,
7) teeth changing,
8) barrier function,
9) sensory function.

Parodont fixates the teeth in the jaw. Force acts to the teeth both at mastication and out of masticatory loading as well as at other functional states. These forces try to replace the teeth out of their place. Parodont transfers the forces acting to the teeth to the jaw bones.

Plastic function is realized by cellular elements present in it. So, cementoblasts participate in secondary cement building while osteoblasts – in bone formation. Capillaries and nerves developed net determines its trophyc function – tooth cement and alveole walls feeding.

The duration of loading to the teeth creating by mastication and swallowing is in average near 30 min a day (not more than 2 hours). Mandible is usually moved down during sleeping so that the teeth are not in touching and the loading to the dental bed is absent. Masticatory force action depends on root size covered by gums and fixed in the alveole. Root is a clinical term here id est the tooth part really impeded into the alveole. The longer is “clinical root”, the stronger is a tooth support and it can be replaced only by significant force. On other hand, the bigger is a “clinical crown” (id est the tooth part placed above the gums) comparatively to the “clinical root” the less force can replace the tooth from the dental alveole. The forces acting at a functional loading, rebuild the bone.

New bone formation depends on: tension and value of the forces acting to the tooth, organism general state, general and local diseases in anamnesis, metabolism intensiveness et al.

Loading on parodont appearing while masticating depends on: food character, muscular force, jaws occlusion type.

Although only a part of parodont possible durability is used at mastication. Parodont reserve forces are possible to be increased by masticatory apparatus training (solid food mastication for instance).

Periodont (peridentium)

Peridentium (periodont) represents connective tissue placed between the alveole and the tooth root and maintains the tooth root in the osseal alveole. Its fibers as thick collagen fibers are looped in cement with one end and in alveolar process with another one.

Periodontal fibers are stretched in a very narrow fissure limited by the tooth root and alveolar process. This fissure is named as periodontal space or parodontal pocket (figure 6, appendix).

Composition:

1) intercellular space: fibers (figure 7), lax connective tissue;
2) cells: fibroblasts, osteoblasts, cementoblasts, osteoclasts, odontoclasts, little-differentiated cells, macrophagues, mast cells (labrocytes), leucocytes.

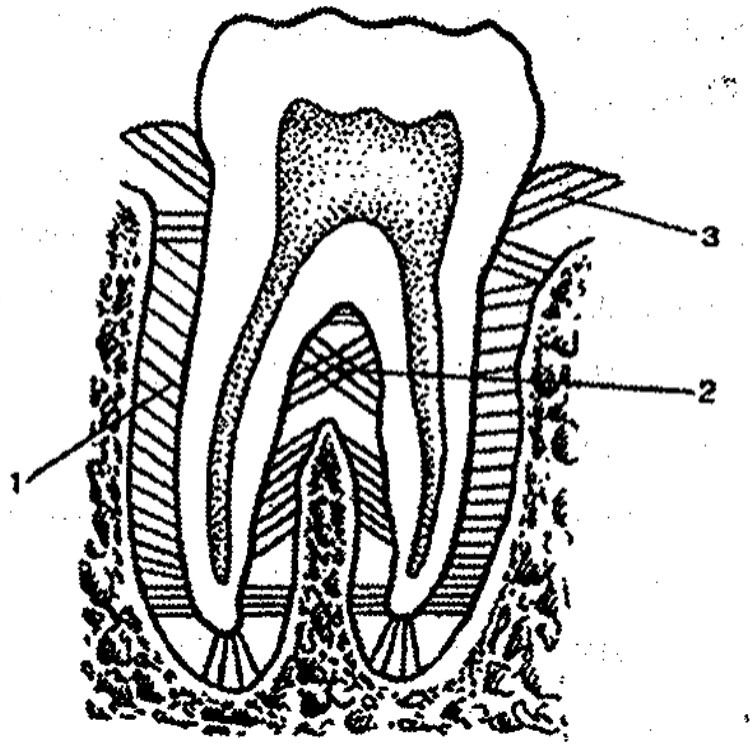

Figure 7. Peridentium structure. Designations: 1 – dental-alveolar fibers; 2 – interdental (interroot) fibers; 3 - dental-gingival fibers.

Blood supply

Periodont is characterized by vast circulation corresponding to high activity of its cellular and non-cellular elements renewal.

Main circulation sources are: superior alveolar artery, inferior alveolar artery.

Additional sources are: dental artery branches, supraperiostal arteries branches – in mucosa covering alveolar processes.

Plexuses: external – larger longitudinally located blood vessels, middle – from less blood vessels, capillary – near root cement.

Periodontal capillaries part has increased permeability. It is considered that it is connected to the necessity of water fast transport providing in periodont hydrophilic main substance and from it - for pressure adaptation to changeable masticatory loading (acting to the tooth) in periodontal space.

Veins collecting blood from periodont area are directed to osseal septi but do not repeat arteries course. There are multiplied anastomoses between arterial and venous vessels in periodont. Clinical importance has periodontal vessels connection with pulpar vessels as a possible way of infection spreading.

Innervation

Periodont is innervated both by afferent and efferent fibers. Afferent fibers come to periodont from two sources:

1) peripheral rami from dental nerve up to its inflowing into apical foramen;
2) branches of nerves penetrating osseal canals and directing towards root apex or crown.

Fibers from both sources are mixed while forming nervous plexus in the periodontal space.

Nervous endings are primarily mechanoreceptors and pain receptors (nociceptors). They look like curly oval incapsulated bodies, spindle-shaped and leaf-shaped structures or thin tree-branched free endings. The nervous endings biggest concentration is characteristic for root apex area except superior incisives.

Sympathetic fibers form endings as baskets round the vessels and probably participate in regional circulation regulating. Parasympathetic fibers are absent.

***Functions*:**

1. *Supporting (maintaining and amortizing)* – a tooth maintaining in the alveole, masticatory loading distribution due to fibers, major substance and liquid delt with it as well as the one in vessels.
2. *Participation in teething*. The surrounding fibers change from being parallel to the tooth surface to bundles that are attached to the tooth surface and extending towards the periodontium (bone). The periodontal ligament has contractile properties and changes drastically during eruption. During eruption, collagen fiber formation and turnover are rapid enabling fibers to attach and release and attach in rapid succession. Some fibers may attach and reattach later while the tooth moves occlusally as new bone forms around it and the fibers will organize and increase

in number and density as the tooth erupts. As the tooth moves occlusally it creates space underneath the tooth to accommodate root formation. Fibroblasts around the root apex form collagen that attach to the newly formed cementum. Bone trabeculae fill in the space left behind as the tooth erupts in the pattern of a ladder which gets denser as the tooth erupts. After tooth reaches functional occlusion periodontal fibers attach to the apical cementum and extend into the adjacent alveolar bone. The rate of tooth eruption depends on the phase of movement. Environmental factors affecting the final position of the tooth: muscular forces and thumb-sucking.

3. *Sensory* – due to multiplied sensory nervous endings; mechanoreceptors percepting loading encourages to masticatory pressure regulation, this function gets changed in non-dentate and implant patients (Salame, 2018, Sessle, 2006).
4. *Trophyc* – provides nutrition and alive activity of cement, partially (through additional canals) – of tooth pulp.
5. *Homeostatic* – regulating the: cells proliferative and functional activity, collagen renewal, cement resorbtion, cement reparation, alveolar bone rebuilding.
6. *Reparative* – taking part in reparative processes by cement formation both at tooth root fracture and at its superficial layers resorbtion; it has big potential of own renewal after damaging.
7. *Defense* – it is realized by leucocytes in part macrophages. Also peridentium produces antimicrobial peptides with antibacterial, antifungal and antiviral activity (Zasloff; 2002).

Gingiva

It is the part of the oral mucosa overlying the crowns of unerupted teeth and encircling the necks of erupted teeth, serving as support structure for subadjacent tissues (figure 8, appendix).

By other words, gums represent tissues covering tooth root near-cervical part and alveolar process attached to it.

Conditionally gums are divided into *two parts*:

1) mobile (free) – is attached to the tooth surface;
2) non-mobile (fixed) – is fixed with proper sheath fibers to the alveolar process epiosteum.

Gums are covered by multi-layered flattened keratinized epithelium. Gingival epithelium keratinization is a powerful defensive reaction to the mechanic, thermal and chemical stimuli. Oral mucosa proper plate high connective-tissular papillas penetrate gingival epithelium.

Capillary net is formed by vessels the wall of which is covered only by epitheliocytes several layers. The vessels come to the gingival papillas mucosa surface and form shoe-like capillary glomeruli. They provide densed attachment of gums margin to the tooth neck alongside with gingival edge vascular system.

ORAL MUCOSA

Knowledge about oral mucosa normal state maintaining represents necessary condition of its diseases exact diagnostics.

Under physiological conditions oral mucosa is primarily:

- smooth,
- bright (brightness is directly influenced by its moisturing with oral liquid),
- from pale-rosy to red by color (dependently on sublayed connective tissue vascularization degree).

Mucosa mobility is determined by well-developed submucosal layer presence.

The most mobile mucosa is (comparatively to gums and hard palate) located in: lips, cheeks, oral cavity floor, soft palate.

Oral mucosa consists of two layers: epithelium, plate.

As muscular plate is absent in oral mucosa then the plate properily is transducted to the submucosal background without strict boarder.

Oral epithelium is thick, multi-layered with the keratinization locuses in the areas which are undergone to big mechanic loadings: tongue dorsal surface, hard palate, gums.

Near 80% of all mouth surface is covered by keratinized epithelium while 30% - by non-keratinized one. Other 20% comprise the teeth. Oral epithelium has very high ability to regeneration and permeability even in vitro (Selvaratnam, Cruchley, Navsaria, Wertz, Hagi-Pavli, Leigh, Squier, Williams; 2001).

Keratinized epithelium envelops surface of masticatory mucosa (hard palate, gums) as well as some parts of covering mucosa (cheeks on teeth closage line) and specialized mucosa (on tongue dorsal surface in filiate papillas area).

Non-keratinized epithelium envelops surface of covering mucosa:

- oral cavity floor (Squier, Kremer; 2001),
- tongue ventral surface,
- cheek part,
- lips biggest part,
- some areas of specialized mucosa at tongue dorsal surface.

Oral mucosa proper plate is divided into two undistinctly boardered layers:

- papillar – enters the epithelium and is formed by lax connective tissue;
- net – deeper = represented by solid fibrous connective tissue.

Blood supply

It is performed through external carotid artery and its rami. Arteries pass parallelly to mucosa surface in submucosal basement. Rami making anastomozes to analogous vessels in proper plate net layer come from these arteries and supply big capillary plexuses in papillary layer. This plexus loops penetrate the papillas reaching approximately epithelium basal layer. The loops shape is defined mainly by connective-tissular papillas shape while their quantity – by the papilla volume. Fenestrated

endothelium covers capillaries part in some mucosal areas. Near 30% of gingival capillaries belong to this group while the capillaries majority in cheek mucosa has continuous endothelial covering.

Oral mucosa blood supply is more intensive than in skin in all mucosa areas: it is the most intensive in gums. There are many shunts between arterioles and venules in mucosa.

Veins accompanying the arteries inflow into the internal jugular vein.

Innervation

It is realized mainly by trigeminal nerve but also afferent fibers of facial, glossal-pharyngeal and vagal nerves participate in it. Such a rich innervation provides different substances reception and various reflectory reactions essential for mastication, salivation, swallowing and speech. That is why the innervation is primarily the sensory one.

Receptive apparatus

Receptive structures in oral mucosa are represented by:

a) free nervous endings;
b) specialized formations such as: Krauze's cones, Rouffini's bodies, Meissner's bodies, Merkel's discs et al.

Oral receptors 3 main groups:

1) chemoreceptors (gustatory),
2) proprioceptive,
3) somato-sensory: tactile, of coldness, of warmth, noceoceptive.

Receptors presence defines sensory function of oral cavity (Савченков, Пац; 2007). Their distribution varies at different areas. The biggest amount of gustatory receptors is located in tongue papillas, tactile – in lips and tongue papillas, temperature – in lips and tongue end, noceoceptors (pain receptors) – on soft palate, palatal arcs and transitional plica. Oral cavity as one of the most important reflexogenic zones has multiple bonds with organism different systems due to the closest contacts with brain performed through trigeminal, vagal and glossal-pharyngeal nerves.

Tactile reception

Oral mucosa tactile reception is an important part of somato-sensor analyzer. It is represented by touching and pressure receptors. These receptors are in strong functional interconnection with parodont mechanoreceptors and masticatory muscles proprioreceptors. Their interrelations define muscle participation in course of mastication act.

Tactile sensitivity study demonstrated receptors distribution unequality in maxillary-facial area different regions.

Tongue end and red lip limb have maximal sensitivity because these structures are the first structure for the analysis of substances coming into oral cavity.

Superior lip (mucosa and red limb) possesses more expressed sensitivity comparatively to the inferior one.

Hard palate mucosa has tactile sensitivity high level. It is of great importance in course of swallowing act (orienting mastication phase) and in course of food piece forming, swallowing.

Vestibular gum surface mucosa possesses minimal tactile sensitivity. One can see decreasing sensitivity gradient to the left and to the right from alveolar arch center in the gingival (gum) papillas region.

Sensitivity is bigger from the right side than from the left. Asymmetry is explained by innervation peculiarities: maximal neurons quantity is located on the right face side.

Minimal tactile sensitivity is characteristic for gums vestibular surface mucosa.

Conductive part

The information from oral mechanoreceptors majority comes through thick myelinic fibers (A-beta type) with 30-70 m/sec by velocity.

The neurons location:

- the first neuron - in the corresponding nerves sensory ganglii,
- the second one – in medulla oblongata,
- the third one – in thalamus.

Central (cortical) part

It is located in post-central gyrus (the fourth neuron) – in the I-st and in the II-nd sensory areas of brain cortex.

Tactile sensation study in regions covered by dentures that are denturing bed helps to develop individual peculiarities of adaptation to dentures in dental patients.

Pain (noceoceptive) reception

The *term "pain"* has different meanings. One can differentiate pain as usual sensory modality similar to hearing, taste, vision, that is a signal about reaching the physiological function boundaries out of which injury is located. The example of this pain definition is pain sensation appearance while trying to gnaw too solid nuts. Pain can be the result of pathologic processes, for example, pulpitis and periodontitis. Chronic durable pain can become the origin of new pathologic conditions for instance odontogenic trigeminal nerve neuralgia. Primarily pain is situated in injured tooth region but also it can irradiate to neihbouring jaw locuses, to eyeball, head frontal, temporal and occipital regions. Painful sensations also occur at mucosa inflammatory processes: stomatitis, glossitis, at galvanism phenomena et al.

Dental pain is considered to be the strongest because of the biggest amount of nociceptors to the square unit.

Nociceptors of face skin and oral mucosae are represented by free incapsulated nervous endings of different shape (hairs, spirals, plates). Expressed noceoceptive sensitivity is on mucosa at mandible vestibular surface in the lateral incisives area. The least noceoceptive sensitivity is on the gums oral surface. The pain sensitivity on the right is expressed bigger than on the left. Frontal teeth have the biggest pain sensitivity, the masticatory ones – the minimal one. There is a narrow locus on the cheek internal surface without pain sensitivity. One can see both free nervous endings and Meissner's bodies in the periodontal tissue.

Pain that is felt at a site other than the origin of the nociceptive stimulus is called heterotopic pain and can arise from sensations triggered within the CNS (central pain), pain that is felt at a different site along the same neurologic distribution (projected pain), and pain that is felt in a site of a different neural distribution (referred pain) (figure 9, appendix).

Atypical facial pain (AFP) is not as common as other diseases associated with facial pain, such as temporomandibular disorders (TMD).

The term was first used to describe patients with chronic facial pain who did not respond to neurosurgical procedures aimed at interrupting pain pathways in the peripheral and central nervous system (CNS). When surgical lesion making of somatic afferent nerve fibers and tracts was not effective, surgical procedures on the sympathetic nervous system pathways were performed and also failed. The model of pain as a sensation generated by a peripheral stimulus and relayed to the brain, and the lack of predictable effects of sectioning nerves suggested that a psychological abnormality was the likely cause. The absence of a local orofacial abnormality or ongoing injury supported this assumption. Variants of AFP, burning mouth syndrome and atypical odontalgia, have emerged as distinct conditions.

A significant percentage of patients with AFP ascribe the onset of pain to dental procedures that were of a routine nature: scaling, restorative, and endodontic procedures and dental extractions. Neuropathic pain may result from tissue injury that affects peripheral nerves, resulting in CNS changes, causing persistent pain. The absence of clear explanation for AFP and studies demonstrating the effectiveness of tricycle antidepressant (TCA) medication has been used to support a psychological explanation. Recently studies on brain activity indicate that pain processing in the CNS is different in patients with AFP than in control subjects. The hypothesis that AFP may be related to abnormal processing of information in the CNS is still speculation (Merrill; 1997, Woda, Pionchon; 1999).

Many studies of chronic pain have demonstrated that high scores of depression or somatization are stronger predictors of pain severity and pain persistence than physiologic or physical measures of pathology. For example, in patients with painful ***temporomandibular disorders (TMD)*** high scores on depression or somatization scales are greater predictors of persistent pain and dysfunction than any physical measure of pathology, including temporomandibular joint (TMJ) sounds or limitation in mandibular opening.

One uses local anaesthesia one type of which is conductive analgaesia in ***dentistry for analgaesia*** in the most often cases. It is based on nervous fibre physiological integrity law. Drug introduction disturbs nerve physiologic integrity that prevents excitement spreading in pharmacological blockade zone.

Temperature reception

Warmth receptors are histologically represented by Rouffini's bodies, coldness – by Krauze's cones. Coldness receptors are situated in epithelium or directly beneath it, warmth – primarily in proper oral mucosa inferior and superior layers. Mentioned peculiarities define oral mucosa higher sensitivity to the warmth than to the coldness.

Warmth sensitivity possesses increasing gradient from oral cavity anterior parts to the posterior ones, the coldness one – decreasing gradient in this direction and increasing gradient from posterior parts to the anterior ones.

Thus, oral cavity anterior part is responsible for heat release while the posterior one – for heat production (Будылина, Дегтярёва; 2000).

Coldness receptors dominance in oral cavity anterior parts and warmth receptors in the posterior ones is determined by their functions specificity and role in the organism thermoregulation processes.

Coldness sensory system being dominant in the organism thermoregulative processes answers to external environment changes faster and more adequate while the warmth afferentation give signals mainly about organism temperature homeostasis.

Cheeks mucosa has little sensitivity to coldness and, even less, - to warmth. Warmth perception is absent in the hard palate very center. Tongue posterior surface

central part does not act either to coldness, or to warmth stimuli. High sensitivity to temperature agents is a characteristic of tongue tip and lips red margin.

These areas exactly get irritated first of all at eating. Information about substances temperature can switch corresponding defensive reactions on from these areas if necessary.

If there is hot food in the oral cavity – much liquid saliva is released for the food cooling. Also processing with saliva can make the taken food warmer. Oral cavity thermoregulative function is delt with big heat release due to liquid evaporation from oral mucosa. One can say that this function is not significant in humans.

Oral mucosa temperature depends on a factors row:

- external environment temperature,
- external environment moisture,
- cellular metabolism intensiveness,
- tissues morphological-functional peculiarities,
- their vascular net state (in turn, it depends on capillaries amount and their filling degree as well as upon blood movement velocity in arterioles); mentioned circumstances explain different topography of oral cavity organs temperature indexes various topography;
- saliva evaporation from mucosa surface (at oral respiration for example).

Besides, saliva and oral cavity organs mucosa action, equaling the taken food temperature, is involved in a thermoregulation functional system.

It has been established that mucosa every locus possesses a definite temperature. Inferior lip skin average temperature is 33,1°C while the superior one is 33,9°C; the temperature lowers in a skin and lips red margin boarder zone. Oral mucosa temperature is higher in the distal parts and at a distance increasing from the middle line.

Any tooth temperature is also fluctuated in its various places with a definite regularity: it is lower at the cutting margin and masticatory surface (30,4-30,5°C) than in the near-cervical area (30,9°C). There is a tendency to the temperature gradual increase both on maxilla and on mandible as it has been established at jaws examination.

There are 2 methods for maxillary-facial area organs and tissues temperature investigation:

- contact electrical thermometry,
- thermal visiography (for the temperature assessment over the distance).

These methods have a definite value in a clinic because the thermometric indexes disorders can testify to the tissues trophycs changings as well as to the inflammatory processes in oral cavity.

Oral mucosa and maxillary-facial area skin temperature assessment importance:

1) The initial one – while prescribing the treatment with heat or coldness: at facial nerve injury the temperature can decrease to 8-10°C in the corresponding

innervation zones and usual heat procedures prescription can cause temperature dyscomfort or even pain under these conditions.

2) The teeth one – in a managing the tooth preparation rational methods in such a regimen when the heat trauma of enamel, dentin and pulp could be minimal.
3) Dentist should remember that the tooth heating appears at the decay cavity forming or while the tooth preparating under the crown due to resistance (friction) of the cutting (grinding) instrument. The tooth temperature increase higher than 45ºC can lead to enamel and dentin burning and result in pulp thermal trauma. Preventive procedures include: the instruments careful choosing while taking into account the burs and preparation discs size and shape, their rotation velocity as well as the material they are made of. Besides, a dentist must follow a proper work regimen. Essential conditions represent the preparation uncontinuosity and highly-speeded bur-machine applying. It is accompanied by the solid tissues co-grinding velocity rising, the cutting instrument pressure and vibration lowering and the tooth tissues burning is prevented at sufficient cooling. Cooling type, cooling system work in good working order, water flow proper direction to the contact place of cutting instrument with the tooth solid tissues are also of special importance.
4) Oral mucosa can undergo to the temperature stimuli significantly different from body temperature while eating. Cold meal (both solid and fluid) cause mucosa damage rather seldom because their taken amount is usually little and they are in oral cavity for short. Cooling influences on oral mucosa circulation by a following way: vascular spasm appears first, it is enforced at the cooling deepening and microcirculation is interrupted practically completely. Sharp significant cooling (with chlorethil for instance) does not destroy the tissues and their function restores after its action stoppage. Warms (heat) action to mucosa leads to hyperemy and further edema of the surrounding tissues. Hot dishes, stomatological instruments heated in course of work and other hot instruments having passed into mouth can cause mucosa limited necrosis. Then vesicle appears which is ruptured soon with an erosion formation.

Taste receptors

Papillas are specialized epithelial cells (Міщенко, Гончаренко; 1998) (figure 10, appendix).

There are <u>four types of papillae</u>:

- filiform (thread-shape),
- fungiform (mushroom-shape),
- foliate (leaf-shape), and
- circumvallate (ringed-circle).

All papillae except the filiform have taste buds on their surface. Some act directly by ion channels, others act indirectly.

- *Fungiform papillae* - as the name suggests, are slightly mushroom-shaped if to look at in section. These are present mostly at the apex (tip) of the tongue. They lay singularily among filiform ones. Connective-tissular background is very highly to be vascularized; blood lets light in their vessels through thin epithelium giving red coloring to the papillas. One can meet sometimes taste buds in the papilla apex epithelium (figure 11, appendix).

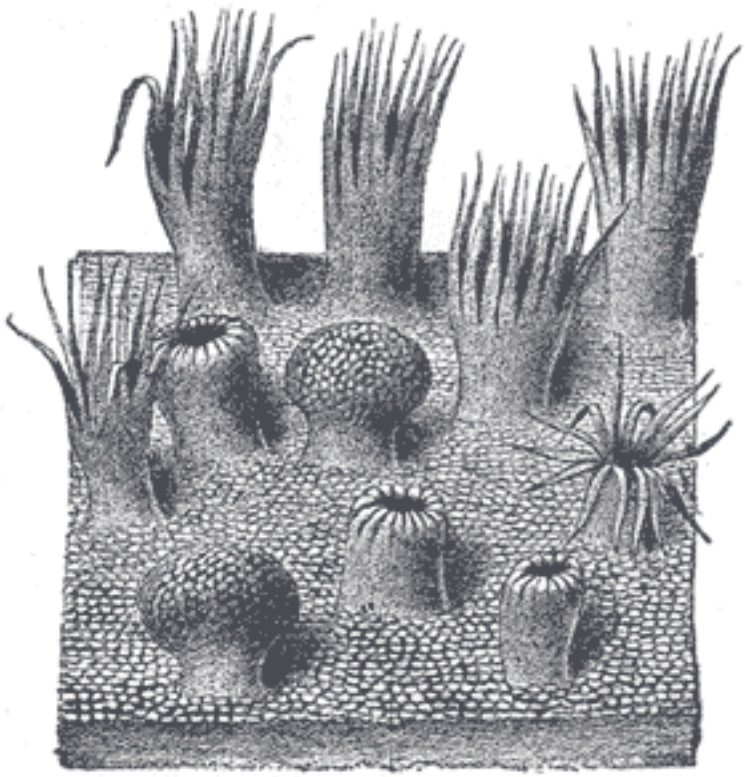

Figure 11. Tongue mucous membrane portion semidiagrammatic view. (Fox; 2004).

The epithelial prolongations stand straight on some filiform papillae, they are spread out in one and they are folded in in three papillae (Fox; 2004).

- *Filiform papillae* - these are thin, longer, cone-shaped papillae that don't contain taste buds but are the most numerous. They distribute at tongue back whole surface especially in the anterior parts. These papillae are mechanical and are not involved in gestation while forming the powerful abrasive surface due to which tongue pressures food piece to hard palate and participates in its grinding. Mucosa is covered by more flexible non-keratinized epithelium between papillas due to which its surface can change during food mechanical processing. Filiform papillas quantity decreases in the old due to iron and vitamins (B-group) insufficient coming with food.

- *Foliate papillae* - these are ridges and grooves towards the posterior part of the tongue. They are developed only in early childhood while they are rudimentary or absent in the adult. They are located 3-8 in amount at tongue lateral surfaces. These papillas are formed by mucosa parallel plicas, are divided by fissures into which serous salivary glands are opened. Epithelium contains taste buds at papillas lateral surfaces.
- *Circumvallate papillae* - there are only about 3-14 of these papillae in the most people and they are present at the tongue oral part back. They are arranged in a circular-shaped row just in front of the tongue terminal sulcus (y-like sulcus). These papillas are the biggest ones among all. Every papilla is surrounded by vallum, separated from it by deep sulcus on the floor of which serous salivary glands releasing ducts are opened. These glands secret encourages to the sulcuses rinsing; lipase is determined in it.

Smooth myocytes fascicles are present in the papillas connective tissue. They contract and thus encourage to the papillas lateral surfaces strengthening and provide the most complete and proper contact of food substances coming into the sulcus to the taste buds.

Taste is mainly a function of the ***taste buds*** in the mouth, but it is a common experience that one's sense of smell contributes strongly to taste perception (Guyton&Hall; 2000). In addition, the texture of food, as detected by tactual senses of the mouth, and the presence of such substances in the food as pepper, which stimulate pain endings, determine greatly the taste experience. The importance of taste lies in the fact that it allows a person to select food in accordance with desires and often in accord with the metabolic needs of the tissues for specific substances.

Taste Bud and Its Function

A taste bud has a diameter of about 1/30 millimeter and a length of about 1/16 millimeter. The taste bud is composed of about 50 modified epithelial cells, some of which are supporting cells called sustentacular cells and others of which are taste cells. The taste cells are continually being replaced by mitotic division of surrounding epithelial cells, so that some taste cells are young cells and others are mature cells that lie toward the center of the bud; these soon break up and dissolve. The life span of each taste cell is about 10 days in lower mammals but is unknown for humans.

The outer tips of the taste cells are arranged around a minute taste pore. From the tip of each taste cell, several microvilli, or taste hairs, protrude outward into the taste pore to approach the cavity of the mouth. These microvilli provide the receptor surface for taste. Each taste bud is flask-like in shape, its broad base resting on the corium, and its neck opening by an orifice, the gustatory pore, between the cells of the epithelium. The bud is formed by two kinds of cells: supporting cells and gustatory cells.

The supporting cells are mostly arranged like the staves of a cask, and form an outer envelope for the bud. Some, however, are found in the interior of the bud between the gustatory cells. The gustatory cells occupy the central portion of the bud; they are spindle-shaped, and each possesses a large spherical nucleus near the middle of the cell. The peripheral end of the cell terminates at the gustatory pore in a fine hair-like filament, the gustatory hair.

The central process passes toward the deep extremity of the bud, and there ends in single or bifurcated varicosities. The nerve fibrils after losing their medullary sheaths enter the taste bud, and end in fine extremities between the gustatory cells; other nerve fibrils ramify between the supporting cells and terminate in fine extremities; these, however, are believed to be nerves of ordinary sensation and not gustatory.

Taste Buds Location

The taste buds are found on three types of papillae of the tongue, as follows:

1) A large number of taste buds are on the walls of the troughs that surround the circumvallate papillae, which form a V line on the surface of the posterior tongue part.
2) Moderate numbers of taste buds are on the fungiform papillae over the flat anterior surface of the tongue.
3) Moderate numbers are on the foliate papillae located in the folds along the lateral surfaces of the tongue.
4) Additional taste buds are located: on the palate, a few - on the tonsillar pillars, on the epiglottis, and even in the proximal epiglottis.

The adult have 3000 to 10000 taste buds, and children have a few more. Beyond the age of 45 years many taste buds degenerate, causing the taste sensation to become progressively less critical.

Especially important in relation to taste is the tendency for taste buds subserving particular primary sensations of taste to be located in special areas:

- the sweet and salty – principally on the tip of the tongue,
- the sour taste – on the two lateral sides of the tongue,
- the bitter taste – on the posterior tongue surface and soft palate.

Taste sensory system

Gustatory sensitivity is oral mucosa sensory function specific peculiarity. Gustatory analyzer physiology knowledge is very important because change of its function may testify to serious disorders both in oral cavity and in other organism parts.

But gustatory analyzer role and its importance are difficult to be determined separately because natural adequate stimulus - food, coming into oral cavity – excites simultaneously other analyzer receptors. Thus, gustatory sensation is a complicated sum of excitements coming into cortex from gustatory, olfactory, tactile, temperature and nociceptive receptors. First of all, in oral mucosa tactile receptors are excited, later – temperature and then receptors answering to chemical food content. Impulses from them come into CNS through various fibres with different velocity. Result - dyspersion on excitement spreading through nervous centers. Different shades of gustatory sensations also depend on the complex of occuring excitations. Gustatory receptor cells are united in gustatory bulbs which are primarily located in tongue papillas: fungiformed, foliatae and vallate. Taste analyzer sensitivity assessment is performed by method of gustatory sensation threshold determining as well as by functional mobility method. Gustatory thresholds are defined separately for every stimulus from 4 main gustatory stimuli according to taste fields topography because separate tongue locuses possess different sensitivity to substances of various gustatory quality in the majority of people: tongue end is the most sensitive to the sweet, lateral surfaces – to the salty and the sour, root – to the bitter. It was established by means of functional mobility method that active lingual papillas amount is constantly changed according to alimentary tract functional state. Receptor mobilization maximal level is observed on an empty stomach, it is reduced after its irritation with food. This phenomenon is known as gastro-lingual reflex. Gustatory receptors play the effector role in this reflex. Some dental diseases for example glossalgia (pain in tongue), glossitis (tongue inflammation) and others may appear at alimentary tract disorders. There can be taste loss and gastro-lingual reflex disorder that can be used as diagnostic criterium. Gastro-lingual reflex study helps diseases ethiology assessment in these cases.

The sense of smell allows us to separate undesirable or even lethal foods from those that are nutritious. It also allows animals to recognize the proximity of other animals or even individuals among animals. Finally, it is strongly tied to primitive emotional and behavioral functions of our nervous system.

Primary Sensations of Taste

The identities of the specific chemicals that excite different taste receptors are still not all known. There are four general categories for practical analysis of taste called the *primary sensations of taste.* They are: sour, salty, sweet, bitter (figure 12, appendix).

We know that a person can perceive literally hundreds of different tastes. They are all supposed to be combinations of the elementary sensations in the same manner that all the colors we can see are combinations of the three primary colors.

Sour taste

The sour taste is caused by acids, that is, by the hydrogen ions concentration, and the intensity of the taste sensation is approximately proportional to the logarithm of the hydrogen ion concentration. That is, the more acidic the food, the stronger becomes the sour sensation. Like salty taste, it is produced by ion movement through membrane channels.

Sour taste signals the presence of acidic compounds (H+ ions in solution). There are three different receptor proteins at work in sour taste. The first is a simple ion channel which allows hydrogen ions to flow directly into the cell. The protein for this is EnAC, the same protein involved in the distinction of salty taste (this implies a relationship between salt and sour receptors and could explain why salty taste is reduced when a sour taste is present). There are also H+-gated channels present. The first is a K+-channel, which ordinarily allows K+ ions to escape from the cell. H+ ions block these, trapping the potassium ions inside the cell (this receptor is classified as MDEG1 of the EnAC/Deg Family). A third protein opens to Na+ ions when a hydrogen ion gets attached to it, allowing the sodium ions to flow down the concentration gradient into the cell. The influx of ions leads to the opening of a voltage-regulated Ca2+-gate. These receptors work together and lead to depolarization of the cell and neurotransmitter release.

Salty taste

The salty taste is elicited by ionized salts, mainly by sodium ion concentration. The quality of the taste varies somewhat from one salt to another because the salts elicit other taste sensations in addition to saltiness. The cations of the salts, especially sodium cations, are mainly responsible for the salty taste, but the anions also contribute to a lesser extent. Ions activate specific receptor cells for the salty taste. Different substances taste salty to the degree that they activate these particular receptor cells. The sodium ions pass into the sensitive receptor cells through channels in the apical membrane. This depolarizes the cells, causing them to release their transmitter. The anion associated with the sodium ion, however, modifies the perceived saltiness to a surprising degree: NaCl tastes much saltier than other sodium salts (such as sodium acetate). There is evidence to suggest that the anions can pass through the tight junctions between the receptor cells, and that the chlorine anion passes through this barrier more readily than the other anions. This is presumably related to the ability of chlorine ion to impart a saltier taste to the sodium ion than do the other anions.

Arguably the simplest receptor found in the mouth is the salt (NaCl) receptor. An ion channel in the taste cell wall allows Na+ ions to enter the cell. This on its own depolarizes the cell, and opens voltage-regulated Ca2+ gates, flooding the cell with ions and leading to neurotransmitter release. This sodium channel is known as EnAC and is

composed of three subunits. The sensitivity of the salty taste to amiloride in humans, however, is much less pronounced, leading to assumption that there may be additional receptor proteins except EnAC that may not have been discovered yet.

Sweet taste

The sweet taste is not caused by any single class of chemicals. A list of some of the types of chemicals that cause this taste includes: sugars, glycols, alcohols, aldehydes, ketones, amides, esters, amino acids, some small proteins, sulfonic acids, halogenated acids, inorganic salts of lead, inorganic salts of beryllium. Note specifically that most of the substances that cause a sweet taste are organic chemicals. It is especially interesting that slight changes in the chemical structure, such as addition of a simple radical, can often change the substance from sweet to bitter. The sweet taste is produced by interaction of taste molecules with specific membrane receptor proteins.

Bitter taste

The bitter taste, like the sweet taste, is not caused by any single type of chemical agent. Here again, the substances that give the bitter taste are almost entirely organic substances. Two particular classes of substances are especially likely to cause bitter taste sensations:

- long-chain organic substances that contain nitrogen and
- alkaloids (they, in turn, include many of the drugs used in medicines, such as quinine, caffeine, strychnine, and nicotine).

Some substances that at first taste sweet have a bitter aftertaste. This is true of saccharin, which makes this substance objectionable to some people.

The bitter taste, when it occurs in high intensity, usually causes the person or animal to reject the food. This is undoubtedly an important purposive function of the bitter taste sensation because many deadly toxins found in poisonous plants are alkaloids, and all of these cause intensively bitter taste, usually followed by rejection of the food. Like the sweet one, bitter taste is produced by interaction of taste molecules with specific membrane receptor proteins. Bitter taste is the most acute taste sensation and is generally associated with toxic molecules (though not all toxins taste bitter).

Threshold for Taste

The threshold for stimulation:

- of the sour taste by hydrochloric acid averages 0,0009 M;
- of the salty taste by sodium chloride 0.01 M;

- for the sweet taste by sucrose 0.01 M, it gets decreased at diabetes mellitus (Navabi; Farzad; Alaei, 2009);
- for the bitter taste by quinine 0.000008 M.

Note especially how much more sensitive is the bitter taste sense than all the others, which would be expected because this sensation provides an important protective function against many dangerous toxins in food.

Taste Blindness

Many people are taste blind for certain substances, especially for different types of thiourea compounds. A substance used frequently by psychologists for demonstrating taste blindness is phenylthiocarbamide, for which about 15 to 30 per cent of all people exhibit taste blindness, the exact percentage depends on the method of testing and the concentration of the substance. Interwoven around the bodies of the taste cells is a branching terminal network of taste nervous fibers that are stimulated by the taste receptor cells. Some of these fibers invaginate into folds of the taste cell membranes. Many vesicles form beneath the cell membrane near the fibers. It is believed that these vesicles contain a neurotransmitter substance that is released through the cell membrane to excite the nerve fiber endings to response to taste stimulations.

Taste Signals Transmission into the Central Nervous System

Taste impulses from the anterior two thirds of the tongue pass first into the lingual nerve, then through the chorda tympani into the facial nerve, and finally into the tractus solitarius in the brain stem. Taste sensations from the circumvallate papillae on the back of the tongue and from other posterior regions of the mouth are transmitted through the glossopharyngeal nerve also into the tractus solitarius but at slightly lower level. Finally, a few taste signals are transmitted into the tractus solitarius from the base of the tongue and other parts of the pharyngeal region by way of the vagus nerve. All taste fibers synapse in the nuclei of the tractus solitarius and send second-order neurons to a small area of the ventral posterior medial nucleus of the thalamus located slightly mesial to the thalamus terminations of the facial regions of the dorsal column-medial lemniscal system. From the thalamus, third-order neurons are transmitted to the lower tip of the postcentral gyrus in the parietal cortex, where it curls deep into the sylvian fissure. This lies slightly lateral, ventral, and rostral to the tongue area of somatic area 1.From this description of the taste pathways, it immediately becomes evident that they are closely parallel to the somatosensory pathways from the tongue (figure 13, appendix).

Taste Reflexes Integrated in the Brain Stem

From the tractus solitarius, a large number of taste signals are transmitted within the brain stem itself directly into the superior and inferior salivatory nuclei, and these in turn transmit signals to the submandibular, sublingual, and parotid glands to help to control the secretion of saliva during the ingestion of food.

Taste Adaptation

Everyone is familiar with the fact that taste sensations adapt rapidly, often almost completely within a minute or so of continuous stimulation. Yet, from electrophysiological studies, of taste nerve fibers, it is clear that adaptation of the taste buds themselves usually accounts for no more than about one half of this. Therefore, the final extreme degree of adaptation that occurs in the sensation of taste almost certainly occurs in the central nervous system itself, though the mechanism and site of this are not known. At any rate, it is a mechanism different from that of most other sensory systems, which adapt almost entirely at the receptors.

Gustatory receptors posses a unique feature – crossed adaptation: one taste increases sensitivity to another one. For instance, the sweet one – to the salty one (sometimes a human being likes using meat or salty cucumber after chocolate or cake).

SALIVARY GLANDS AND SALIVATION

Digestion in oral cavity is realized mainly due to salivary glands secretory function (Som, Brandwein; 2003). *Salivary glands secretory function* is provided by functioning of 3 pairs of large (parotid, sublingual and submandibular) and great amount of small glands disseminated in oral mucosa (Proctor; 2016, Hall; 2011).

Big salivary glands produce saliva which is a mixture of serous (watery) and mucous fluids, containing digestive enzymes. All of these salivary glands are alveolar glands, which have branching ducts with clusters of alveoli, which resemble grapes, at the ends of the ducts.

The largest of the salivary glands, the *parotid* (near the ear) glands, are serous glands located just anterior to each ear. Parotid ducts enter the oral cavity adjacent to the second upper molars. Parotid glands watery secretion lacks mucin (Levy, Koeppen, Stanton; 2018). Mumps is a type of parotiditis, an inflammation of the parotid gland, caused by a viral infection. The inflamed parotid glands become swollen, often making the cheeks quite large. The virus causing mumps can also infect other structures. Mumps in an adult male may also involve the testes and can result in sterility.

The *submandibular* (below the mandible) glands produce more serous than mucous secretions. Each gland can be felt as a soft lump along the inferior border of the mandible. The submandibular ducts open into the oral cavity on each side of the tongue frenulum. In certain people, if the mouth is opened and the tongue tip is elevated, saliva can squirt out of the mouth from the ducts of these glands.

The *sublingual* (below the tongue) glands, the smallest of the three paired salivary glands, produce primarily mucous secretions. They lie immediately below the mucous membrane in the floor of the oral cavity. Each sublingual gland has 10 to 12 small ducts opening onto the oral cavity floor.

Mixed salivary gland microscopic structure distinguishing features having been received under light microscope (Levy, Koeppen, Stanton; 2018)

Mixed salivary gland ultrastructural peculiarities are as follows as. Serous acinar cells situated in secretory end pieces (acini) have got apical zymogen granules containing salivary amylase and other salivary proteins. Mucous acinar cells secrete mucins into saliva. Intercalated ducts drain acinar fluid into somewhat larger ducts, so-called striated ducts, which empty into still larger excretory ducts. Single large duct brings the secretions of each major gland into mouth.

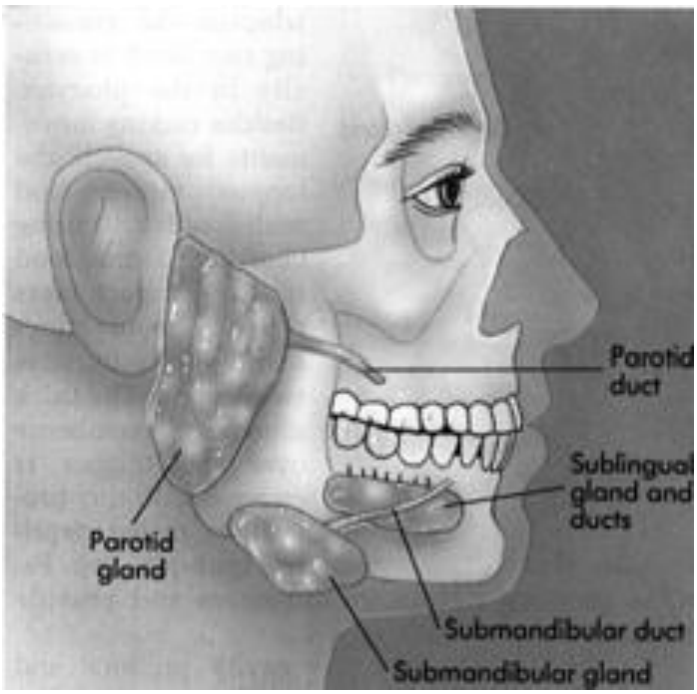

Figure 14. Big salivary glands with their ducts (Som, Brandwein; 2003).

Qualitative peculiarities of chemical content of saliva released by different salivary glands

Parotid glands (figure 14) – secrete liquid saliva. It contains more next substances (comparatively to other salivary glands): catalase – it catalizes H_2O_2 decomposition (hydrolysis): $H_2O_2 \rightarrow H_2 + O_2$; amylase; acid phosphatase; vitamins; antibiotics.

But it contains less coagulation and fibrinolysis factors.

Submandibular glands – contain organic substances big amount (amylase and especially mucin), radonic potassium, free sugars; substances participating in coagulation (thromboplastin, antiheparinic substance, fibrinase) and fibrinolysis (plasminogen activator and proactivator). There are proteins, identical to erythrocytes agglutinogens (they correspond to blood groups).

Sublingual glands – saliva with mucin dominance with powerful alkaline reaction. There are blood coagulation and fibrinolysis more active in it.

Table 6

Salivary glands each pair characteristics in humans

Gland	*Histologic Type*	*Secretion*[1]	*Percentage of Total Saliva in Humans*[2] *(1.5 L/d)*
Parotid	Serous	Watery	20
Submandibular	Mixed	Moderately viscous	70
Sublingual	Mucous	Viscous	5

Designations:
1 – Serous cells secrete ptyalin; mucous cells secrete mucin.
2 – The remaining 5% of salivary volume is contributed by lingual and other minor glands in the oral cavity.

Minor Salivary Glands

The minor salivary glands (MSG) lie within the submucosa of the oral cavity, palate, paranasal sinuses, pharynx, larynx, trachea, and bronchi. They are particularly concentrated in the buccal, labial, palatal, and lingual regions. The gingivae, anterior hard palate, and true vocal cords have relatively sparse concentrations of MSG. The concentration of MSG within the nasal cavity has been estimated at 7 to 10 glands /mm2, which is less than that of the palate but greater than that of the paranasal sinuses. Histologically, the MSG acini are either entirely mucinous (e.g., hard palate) or mixed seromucous glands (e.g., sinonasal, oral).

Salivation adaptive character to different alimentary and rejected substances

- at gustatory receptors irritation "alimentary saliva" is released rich in organic substances and enzymes;
- at action to thermoregulative apparatus – liquid "thermoregulative saliva" poor in organic substances is released;
- at rejected substances coming to oral cavity "washing or protective" liquid saliva is released;
- at emotional excitement salivation can be both enforced and inhibited.

Human being has continuous saliva secretion because at mucosae dryness mechanoreceptors irritation takes place and as a result – salivation.

Saliva volume changings

Hyposalivation reasons:

- depressions;
- fever states;
- liquid significant loosings;
- botulism;
- nitrogenemy;
- malignant anemy;
- facial nerve two-sided paralysis;
- progressive paralysis;
- diabetes mellitus.

Hypersalivation reasons:

- irritation with food;
- gingivitis;
- stomatitis;
- duodenal ulcer diseases;
- stomach ulcer disease;
- pancreatitis;
- medicines taking (pylocarpin, muscarin et al.);
- intoxication with Hg;
- intoxication with I;
- invasion with some parasite worms;
- parkinsonism;
- rabies;
- pregnancy;
- vomiting;
- strong irritation of rectum, urinary vesicle, gentitals;
- salivary glands reflectory dysfunction;
- cholinolytics;
- worrying;
- enforced sweating;
- watery exchange disturbances;
- after durable bleedings;
- after prolonged diarrhea.

Physical-chemical properties

Main properties are represented in the table 7. The parotids contribute about 45% (450 to 675 ml) of the total secretions, the submandibular glands about 45% (450 to 675 ml), the sublingual glands 5% (50 to 75 ml), and the minor salivary glands 5% (50 to 75 ml). The basal secretory rate is low (parotid gland = 0.04/ml/minute/gland and submandibular gland = 0.05 ml/minute/gland). During sleep, the flow rate is virtually nil.

Table 7

Saliva features

Quantity, ml/day, 1/3 – parotid saliva	1400-1500
Secretion velocity • at mastication • in the night • at food taking	0,03-2,4 ml/min 3,3-5,0 ml/min reduced increased up to 3-7 ml/min
Specific weight	1,002-1,020 or 1,001-1,017
Viscosity, puasa	1,10-1,32
pH • of parotid glands • of submandibular glands • at velocity secretion increase	6,75 (5,6-7,6) 5,81 6,39 it gets rised up to 7,8
Total protein, g/l	3,86 (1,56-6,30)
Amylase	One can receive 150 mg of crystallic amylase from 1500 ml of saliva or 0,2-0,3 g/l
Lyzosyme	1,7±0,2 or 0,15-0,25 g/l of mixed saliva

Salivary glands in new-borned secrete little saliva – 0,4 ml per minute in course of sucking, less – out off sucking. It is in average in 8 times less than in adulthood. Salivation volume is increased from 4 months and reaches up to 150 ml per day to 1 year (it is 1/10 of adult secretion). Amylase activity in new-borned saliva is low and it is increased in the second half-year, reaching adult level in course of 1-2 years after birth.

Figure 15. Saliva collecting (Som, Brandwein; 2003).
Unstimulated Whole Saliva
0.1 ml/min
Chewing Stimulated Whole Saliva
0.5 ml/min for women
0.7 ml/min for men

Saliva content

Saliva is a mixture of secretes (Humphries, Williamson; 2001). It forms *oral liquid* with the addition of epytheliocytes, food particles, mucus, lymphocytes, neutrophils and microorganisms (they are in oral cavity in large amounts). Daily saliva secretion is 0,5-2,0 liters. Its pH fluctuates from 5,25 to 8,0. Every gland contribution to oral liquid varies greatly (Nieuw Amerongen, Listenberg, Veerman; 2007).

Saliva contains up to 99,5% of water (98,5-99,0%). Dry substance part is 1,0-1,5% or in average 5-7 g daily. There are many organic and inorganic substances in solid residue. Organic substances comprise more than ½.

There are near 100 various enzymes in saliva. Saliva is considered to be oral cavity defender (Nieuw Amerongen, Veerman; 2002).

Salivary enzymes classification by their origin:

1) secreted by salivary glands,
2) forming in a process of bacteries enzymatic activity,
3) forming as a result of leucocytes decomposition in oral cavity.

The most important of enzymes are: alpha-amylase, lipase (Gelberg, 2014), maltase, proteases, peptidase, alkaline phosphatase, acid phophatase, hyaluronidase, glycolytic enzymes, adenylatecyclase, urease, ATPase, neuromidase, nucleases, superoxidedismutase, catalase, arginase.

Amylase comprises more than 10% from all salivary proteins and its content is 0,2-0,3 g/l. Alpha-amylase represents the biggest activity in alkaline environment while lowering the activity in acid environment. This enzyme name is alpha-1,4-glucan-glucanhydrolase of saliva. It represents metal-enzyme having a quaternary structure. It hydrolizes 1,4-glycoside bonds in starch and glycogen molecules as a result of which oligosugars, maltose and maltotrioses get formed. Alpha-amylase co-enzyme is Ca ion that stabilizes its secondary and tertiary structure. Calcium release leads to almost complete loosing the catalytic activity by this enzyme. Chloride-ion is a natural activator of this enzyme. Alpha-amylase also possesses antibacterial activity because it can split some bacteries membranes polysugars. Parotid glands produce near 70% of the enzyme. Starch digestion appears in oral cavity only partially because food is present only short time in it. Small intestine is a major place for starch digestion. Pancreatic amylase is more active than the salivary one. Salivary alpha-amylase is inactivated at pH 4,0. So, carbohydrates digestion start in oral cavity interrupts soon in stomach acid environment.

Maltase (alpha-glucosidase) decomposes disaccharide maltose up to glucose formation.

Lactoperoxidase is similar to milk peroxidase by its features.

Myeloperoxidase is a protecive enzyme of leucocytic (neutrophilic) genesis. Both enzymes inhibit free-radical oxidation while peroxides eliminating. Smoking inhibits peroxidase activity significantly.

Saliva is distinguished by high peroxidase activity.

Catalase has primarily bacterial origin. It splits hydrogen peroxide with oxygen and water formation.

Both catalase and peroxidase belong to iron-porphyrin antibacterial enzymes.

Alkaline phosphatase hydrolyzes phosphoric acid ethers and is important for enamel mineralization because helps in phosphorus adding to the organic substrates (Тарасенко, Непорада; 2008). Its optimal pH lies in alkaline range. This enzyme major source is sublingual salivary glands.

Acid phosphatase has the same action mechanism but works optimally in acid environment. Its work results in teeth tissue demineralization and parodont bony tissue resorbtion. It is encouraged by excess of organic acids forming during dental covering acidophilic microbs life activity that creates pH optimum.

Both phosphatase sources are: salivary glands, microorganisms and leucocytes.

Proteases, hyaluronidase, acid phosphatase, nucleases activity rising encourages to parodont tissues injury and lowers regenerative processes in them.

Microorganisms vegetating in oral cavity (especially in dental covering) can be the salivary proteases source.

Other substances present in saliva:

There are many organic substances in saliva. They are proteins – *albumins, globulins, amino acids.*

Proteins are dominant among the organic substances. Mostly they are represented by glycoproteids, the biggest part from which is *mucin* (from 2,0 till 5,0 g/l); mucin has such a huge viscosity only due to carbohydrate component presence (Rayment, Liu, Soares, Offner, Oppenheim, Troxler; 2001).

Protein concentration is higher is parotid gland saliva than in the submandibular one. Saliva contains the same proteinic fractions like blood serum – albumins, alpha-, beta- and gamma-globulins. But albumins amount is less significantly and beta-globulins amount is 4 times more. Salivary albumins comprise up to 10% from summary content of protein. Saliva specific proteins stabilize mineral substance and help in their passage into enamel. Saliva proteins take part in salivary pellicle formation at the enamel surface, performs its protection, bacterias agglutination as well as play important role in decay prevention.

Saliva contains immunoglobulin A and agglutinogens corresponding to blood groups found in submandibular salivary glands. Acid hydrolases – catepsines – can be released from oral mucosa damaged tissues as well as from lysosomes. Salivary proteinases excessive activity encourages parodontal tissues inflammation.

Low-molecular organic substances are represented by 20 free amino acids, various metabolites of proteinic (urea, creatinin, histamine et al.) and carbohydrate (glucose, syalic acids, lactate, pyruvate, citric acid) exchanges.

Vitamins B1, B2, B3, PP, B6, C and H are present in saliva.

There are following *hormones*: glucocorticoids, sexual steroids, thyroid hormones, epidermal growth factor (Gröschi; 2009), transforming growth factor-α, melatonin possessing anti-inflammatory and antioxidant features (Reiter; Rosales-Corral; Liu; Acuna-Castroviejo; Escames; Tan, 2015).

There is a direct dependence between hormones level in blood and saliva. Moreover, 10-15% of hormones content in blood is in saliva. Main endocrine factors and their effects are described in the table 8.

Table 8

The most important endocrine factors produced by human salivary glands

The factor name	The most important physiological effects

Kallikrein	• vessels dilation, • blood pressure lowering, • capillaries permeability rising up, • contraction of such inner organs smooth muscular tissue as uterus, intestine et al.
Renin	• vessels constriction with rising the arterial pressure, • aldosteron secretion increase
Nerves growth factor (NGF)	• providing the survival and differentiation of peripheral sympathetic and spinal sensory neurons in ontogenesis, • support of their differentiating, • neurons growth stimulating
Epidermal growth factor (EGF)	• epithelium proliferation and keratinization stimulating, • regeneration enforcement, • stomach secretion inhibiting
Mesoderm growth factor	• stimulates epitheliocytes proliferation, • stimulates fibroblasts division, • causes angiogenesis (microvessels hyperplasy)
Parotin	• calcium level lowering in blood, • bone tissue and dentin unlaming enforcement, • hematopoiesis activation, • leucocytosis, • macrophagal system stimulation
Endothelial growth factor	• vessels tone maintaining, • angiogenesis
Erythropoietins	• the only erythropoiesis specific stimulator (mainly it is synthesized in submandibular salivary glands)
Trasylol	• participates in oral cavity proteases activity regulating by their inhibiting that is important for decreasing the excessive protheolytic activity appearing during pathological processes development

Parotin represents parotid glands hormone of proteinic nature. Its molecular weight is more than 100 kDa. It was separated from the glands tissues for clinical application. Its hypocalciemic action is determined by enforcing calcium passage to the unlamed tissues – teeth and bones. Also it rises calcium and phosphorus metabolism

intensiveness. Teeth mineralization enforcement under parotin action determines its clinical application at parodontitis and fulcral apparatus diseases. Parotin increases teeth pulp odontoblasts functional activity. It is seen by proteins and nucleic acids accumulation in them. Macrophagal system stimulation belongs to important physiological effect of parotin. Parotin possesses insulin-like effect to carbohydrates and lipids metabolism.

Nerves growth factor (NGF) is one of the most important bioregulators of salivary glands. It represents a protein consisting of polypeptide chains with a molecular weight near 140 kDa. Submandibular glands serve as NGF main synthesis source. Control of this substance synthesis and secretion to saliva and blood is realized by thyroid, suprarenal and sexual glands. Its secretion stimulating is performed mainly by alpha-adrenoreceptors. There exist sexual differencies in NGF content: it is higher in males than in females and this increase coincides to the puberty start. Chemicals called neurotrophins promote neuron growth in a developing fetal brain, NGF was the first neurotrophin to be identified. NGF is known to be particularly important in the embryonic development of sensory neurons and sympathetic ganglia. NGF is required for the maintenance of sympathetic ganglia in the adult nervous system, and there is evidence that neurotrophins are required for nature sensory neurons to regenerate after injury. NGF is believed to influence on cholinergic transmittance in CNS structures greatly. Also wound-healing action is of interest. Given data indicate to NGF taking part in regulating the neurotrophyc processes necessary for nervous structures normal development and functioning. Fibroblasts can produce NGF.

Epidermal growth factor (EGF) represents a peptide interacting with specific membrane receptors located in epitheliocytes, endotheliocytes, fibroblasts, chondrocytes.

Submandibular salivary glands ducti cells are EGF main source. Besides, EGF synthesis is also performed by cells of other epithelium-origined glands (pancreas), macrophages and fibroblasts. EGF is present not only in saliva but also in other alimentary juices (stomach, pancreatic). There exists a tight connection between EGF level in blood plasma and salivary glands functional activity.

EGF biological action is expressed in epitheliocytes proliferation and maturation stimulating. It is thought that this action mechanism determines pre-termed teething at its injection to the new-borned animals. EGF significant content in saliva causes trophyc influence on oral mucosa, provides supporting the epithelium regeneration physiological level as well as helps in injuries healing acceleration. EGF makes protective action to stomach mucosa that is delt to stomach secretion inhibiting and preventing the stress ulcers formation. It is important to note that EGF stimulated collagen synthesis.

EGF is a peptide with a neurotrophyc action. It realizes normalizing action to the tissues with disturbed trophycs on the background of salivary glands induced hypertrophy. Immobilization stress enforces significantly EGF secretion from

submandibular salivary glands and its level in blood plasma. This peptide level is higher in male salivary glands than in the female ones.

Mesoderm growth factor (MGF) is secreted by submandibular and parotid salivary glands (more by the parotid ones).

NO (nitrogen oxide) is produced in salivary glands in nervous endings. L-arginin (NO donor) is able to increase submandibular gland mass and to decrease alpha-amylase activity. L-arginin also has stress-protective action to salivary glands.

Kallikreins or kininogenases represent proteases, serine proteinases. They possess narrow substrate specificity at interaction to proteins. Blood plasma kallikreins split bradykinin from this protein at action to kininogen but tissular kallikreins (and salivary in part) release kallidin. Salivary kallikrein characteristic feature represents its ability to release kinines in the alkaline environment. The enzyme is in its active form in saliva differently from plasma and pancreatic forms.

Tissular kininogenases possess wider action spectrum than the plasma ones. Their activity in permeability increase exceeds plasma kallikrein in hundreds times. Glandular (secretory) kallikrein causes activation of pre-kallikrein, kallikrein and plasminogen of blood plasma that is accompanied by fibrinolytic activity rising as well as trypsine activation. Also kallikrein is capable to release arachidonic acid and stimulate synthesis of prostaglandin E2 and thromboxane A2 in epitheliocytes culture due to phospholipase A2 activation. May be, kallikrein participates in oral cavity blood circulation local regulation (Морман, Хелер; 2000). It dilates blood vessels of glandular tissue and enforces blood stream necessary for actively synthesizing gland. This substance possesses chemotactic action, inhibits neutrophils emigration, activates migration and mitogenesis of T-lymphocytes, stimulates lymphokines production, enforces fibroblasts proliferation and collagen synthesis as well as encourages histamine liberation from mast cells. Kallikrein-kinine system components are messengers in some effects initiated by inflammatory agents particularly pain, exudation and proliferation. Tympanal chord stimulation induces kallikrein production. Kinine system activation appears under many damaging factors action such as trauma, hypoxy, allergic process, ionizing radiation, toxins.

There are gender differencies in kallikrein content in salivary glands: females have bigger amount of this enzyme in their glands than males. Males castration helps in its content increase in salivary glands.

Renin is an enzyme with molecular weight equal to 40 kDa (Inagami; 1998). Renin influences on salivary glands secretory function. This enzyme possesses a defensive function and is capable to stimulate reparative processes that have huge biological role in stress situations. Renin triggers formation of renal angiotensin-II (Kim, Iwao; 2000) which represents the strongest vasoconstrictor in the human organism (Вандер; 2000, Corvol, Jeunemaitre; 1997). Blood serum renin-angiotensin system

activation causes vasoconstrictory effect and determines blood pressure durable increase. Also renin increases aldosteron secretion.

Transforming growth factor (TGF) has two major form: properily TGF (TGF-α) and TGF-β. Except salivary glands, TGF-α is secreted by: activated macrophages, skin keratinocytes, hypophysis cells, some brain neurons, placenta, embryonal kidney (Hammerman, O'Shea, Miller; 1993), fetus nasal-pharyngeal zone, embryo auricular vesicles, mammals and human uterus decidual cells. TGF-α participates in the cells proliferation regulating (Fozekas; 1998) as well as in tumor cells growth. It is considered that TGF-α represents EGF "neoplastic form". TGF-β acts as a vessels growth modeling factor at new-formation together with fibroblasts growth factor FGF which stimulates angiogenesis with its direct influence on endotheliocytes as well as represents mitogen for neuro-ectodermal and mesenchymal cells (Пальцев, Иванов; 1995).

Proteinases inhibitors are: contrykal, trasylol, gordox, ingitryl.

They inhibit Hageman-dependent (intrinsic) prothrombinase formation way, prekallikrein, plasminogen and Hageman's factors activity. Also they can prevent cellular protheolysis. Saliva contains proteinases inhibitors not only of plasmic but also of local origin. Trypsin-like enzymes such as salivain, glandulain, kallikrein-like peptidase activity is low in saliva. It is defined by alpha-1-proteinase inhibitor and alpha-2-macroglobulin.

Histatines **–** low-molecular histidin-riched proteins possessing bacteriocydic and fungicydic activity against several microorganisms such as Streptococcus mutans and Candida albicans are secreted and released by large salivary glands (Rothstein, Helmerhost, Spacciapoli et al.; 2002).

Leptin and ***ghrelin*** **–** peptide hormones participating in immune regulation by influence on energy balance through net of cytokines, hormones and neuropeptides are also produced in salivary glands (Dixit, Taub; 2005).

Pellicle-forming proteins forming hydroxyapathites and thus playing protective role (Nieuw Amerongen; Veerman, 2002).

One can say that almost all Mendeleev's table is in saliva (even gold!).

Dominant *cathions* are: P, Na, Ca, Mg. K level is 4-5 times higher in saliva than in blood. Ca, Mg content in mixed saliva corresponds to their concentration in blood plasma. Potassium release with saliva can characterize sympathetic nervous system tone. Sodium and potassium content in saliva depends on secretion velocity. Sodium level is less in children saliva comparatively to the adult one. Calcium salivary concentration can be significantly higher than the blood one. Ionized and non-ionized calcium content correlation is 0,5 in average. Salivary magnesium level is lower than the blood one and depends on saliva flow velocity. Saliva is different with phosphates high content. Salivary phosphate is present in free ionic form and bond with protein. Inorganic

phosphate in saliva corresponds to almost 2/3 from all quantity of this mineral. Bicarbonate level is higher than in blood (Matsuo; 2000).

As its has been stated above, saliva is saturated with calcium, magnesium, phosphate and chlorine ions high concentrations of which cause the ions transfer to the enamel. It is crucial for enamel maturation after teething and makes it hard and resilient to decay development. This function is important for enamel normal chemical content maintaining. There can be enamel remineralization due to salivary ions at enamel damage as well as decay initial stages development.

Demineralization:

- mineral salts dissolve into the surrounding salivary fluid:
  - enamel at approximate pH of 5.5 or lower
  - dentin at approximate pH of 6.5 or lower
- erosion or caries can occur

Remineralization:

- pH comes back to neutral (7)
- saliva-rich calcium and phosphates
- minerals penetrate the damaged enamel surface and repair it:
  - enamel pH is above 5.5
  - dentin pH is above 6.5

Main *anions* are: chloride, phosphate, bicarbonate (Puy; 2006), sulphate.

Chlorides are dominant among anions. Chlorine ion function is to activate alpha-amylase. Salivary bicarbonate has double origin: it can be formed both due to glandular tissue metabolic function and due to active transport from blood plasma with the acinar cells.

The ones in less concentrations are: fluoride-ion, rhodanide-ion.

Fluoride-ion and rhodanide-ion are met in non-significant quantities in saliva (5,26-1,53 mcmol/l). Thiocyanates (rhodanides) (CNS) perform antibacterial function. Their concentration correlates positively to age. Smokers have increased level due to cynilic acid passage with tobacco smoke.

Such *microelements* as Cu, Fe (Buche, Guso, Bertoli, Souza, Guimarães, Brancher; 2016), Mn, Zn, F, Br, I, Co and the others are present also in little concentrations in saliva. Zn, Cu, Br, Co, Mg perform co-enzymatic function as well as take part in metabolism processes and teeth mineralization (fluorum, stroncium et al.). There are several Zn-containing enzymes such as alcohol-dehydrogenase, glutamate-dehydrogenase, lactatedehydrogenase.

Salivary glands are capable to accumulate iodine and play significant role in thyroid hormones iodification. Iodine level is much higher in saliva than in blood.

Salivary fluorides play role of hydroxyapathite stabilizers. Fluorum is included in a crystallic net in osseal tissue and teeth mineral fraction. Dental enamel represents a

tissue saturated in the organism with fluorum in the biggest extent. Fluorum lowered level in a drinking water (less than 0,9 mg/l) encourages teeth decay development. Inhabitants high morbidity with teeth caries is observed in the regions with fluorum insufficient content. Water fluorification, fluorides-containing remineralizing mixtures and fluorum-containing pastes application represents one of the most effective methods in teeth decay prevention.

Easy fluorosis is not accompanied by calcium level changing in children saliva while hard fluorosis is accompanied by its significant decreasing.

Thus, saliva mineral content represents one of the major factors defining teeth solid tissues mineralization processes as well as their resistance to cariesogenic influencings.

Saliva role in digestion*:* it gives the beginning to food chemical processing. Saliva lubricates food for swallowing greater ease and begins starch digestion (Levy, Koeppen, Stanton; 2018). It occurs due to amylase acting on polysacharides (starch) while their destruction to maltose (Pedersen, Bardow, Jensen, Nauntofte; 2002).

Salivary amylase (starch-splitting enzyme) is present in the saliva serous part. This enzyme breaks the covalent bonds between glucose molecules in starch and other polysaccharides to produce the disaccharides maltose and isomaltose. Maltose and isomaltose have a sweet taste; thus digestion of polysaccharides by salivary amylase enhances the sweet taste of food. Under other enzyme maltase influence maltose destruction to glucose can occur. But enzymes action is very limited because food is very little time in oral cavity.

One of the most important digestion rules: careful (durable) food mastication due to which saliva can influence on food (in oral cavity) more effectively.

Only about 50% of the total carbohydrates are digested in the mouth. Most starches are contained in plant cells, which are surrounded by cell walls composed primarily of the polysaccharide cellulose.

Humans lack the necessary enzymes to digest cellulose. Cooking and thorough chewing of food disrupt the cellulose covering and increase the efficiency of the digestive process.

Even though humans can not digest cellulose, it is important to normal digestive function. Cellulose provides bulk, or fiber, in the diet. The presence of this bulk facilitates movement of material through the digestive tract by providing mass against which the muscular wall of the digestive tract can push.

In the 1950s some nutritionists dreamed that all the nutrients we need could be eventually reduced into a single tablet and that we no longer would have to eat food. It is now known that indigestive bulk is very important to the normal function of the digestive tract. For example, bulk in the diet is important in the prevention of colon cancer (clinical clue).

The serous part of saliva dissolves molecules, which can only stimulate taste receptors when in solution. It also helps lubricate the food. The mucous secretions of the submandibular and sublingual glands contain a large amount of mucin, a proteoglycan that gives a lubricating quality to the secretions of the salivary glands.

Saliva formation mechanism

Saliva is formed both in acinuses and in salivary glands ducts. Secretory granules are in glandulocytes cytoplasm (Агаджанян, Смирнов; 2007). Granules size, amount and location are changed in course of secretion. They move to cellular apex from Golgi complex.

Saliva formation first stage is realized in acinuses – primary secrete forming containing amylase and mucin. Ions content in it insignificantly differs from their concentration in extracellular space.

Secrete content changes significantly in salivary ducts:

- sodium ions are actively reabsorbed and
- potassium ions are actively secreted.

As a result, sodium amount in saliva becomes less and potassium – bigger.

And after reabsorbtion and secretion secondary saliva gets formed.

Electrolytes secretion into saliva is under parasympathetic control via acinar cholinergic receptors.

Their stimulation activates cytoplasmic guanylate cyclase and the conversion of GTP to GMP, necessary for the transport of sodium.

Sodium enters along the basal cell membrane following an electrochemical gradient, which is the result of acetylcholine-induced enhanced permeability of the basal cell membrane to potassium and sodium.

The result of this process is the formation of an isotonic, high-sodium, low-potassium fluid.

The saliva becomes hypotonic as sodium is reabsorbed and potassium is excreted in the striated ducts, which are histologically, ultrastructurally, and functionally similar to the renal distal convoluted tubules.

This sodium reabsorption is influenced by aldosterone.

Ducto-acinar salivary apparatus is shown schematically in the figure 16.

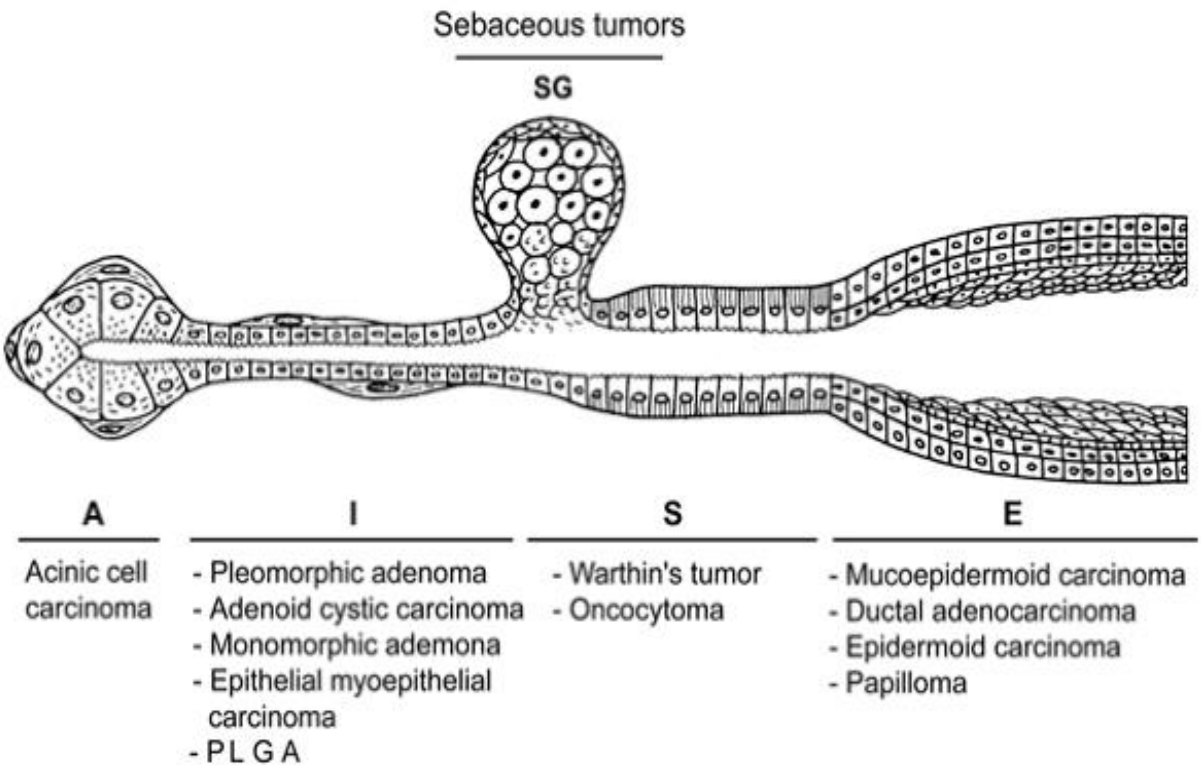

Figure 16. Schematic representation of the ducto-acinar salivary apparatus (Som, Brandwein; 2003).

Secretion is triggered by electrical processes in a gland. Secretory cells resting potential is -10-35 mV while reaching -80 mV in salivary glands ducti cells. Secretory cells electrical activity is characterized not by depolarization but by hyperpolarization of cellular membranes – so called exciting hyperpolarization (EHP) of cellular membranes. EHP of secretory cells is determined by iodate and chlorine pumps activity. These pumps are situated in basal membrane and pump iodum and chlorine ions from blood into cytoplasm that results in cytoplasm electrical negativity rising id est to the cell hyperpolarization. EHP or resting potential rising up to -40-50 mV is registered at tympanal chord parasympathetic fibers irritation and acetylcholine release in their endings in sublingual salivary gland. Salivation latent period fluctuates in the limits of 1-30 sec after food taking that depends on alimentary stimulus type as well as on digestive center excitability. Saliva secretion continues during mastication and reduces significantly after its stoppage.

All salivary glands belong to the merocrine type by secret releasing mechanism: secretory product as molecules penetrates apical membrane without damaging it. Nervous impulses passage to the secretory cells leads to following events.

Water, electrolytes and low-molecular organic substances (amino acids, monosugars, fatty acids) come from blood vessels through intersticium to the secretory cells by primary and secondary (liquid filtration) ways.

Organic substances synthesis passed with water through cell on endoplasmic net is performed in granules.

Acetylcholine releasing from parasympathetic nervous endings influences only on the secretory cell basal membrane activating primary active transport of chlorine and iodate anions into the cell (chlorine and iodic pumps through basal membrane). It leads not only to EHP but also to cytoplasm osmotic pressure increase in the acinar cell. It is also getting increased because sodium ions diffund additionally into the cell through basal membrane due to electrical negativity increase in it that provides water passage into the cell by osmosis law.

Water passage to the cell leads to hydrostatic pressure increase in it that allows liquid exit through the apical membranes pores the formation mechanism of which is in a study process.

Myoepitheliocytes contraction also encourages the secret release into the acinus lumen from the glandulocytes. Potassium and sodium ions also diffund from the secretory cell into acinus through apical membrane right after chlorine and iodum ions pumping with the one-named pumps (figure 17).

Primary secretion is performed in secretory ends pieces while cells lining ducts modify primary secretion (Levy, Koeppen, Stanton; 2018).

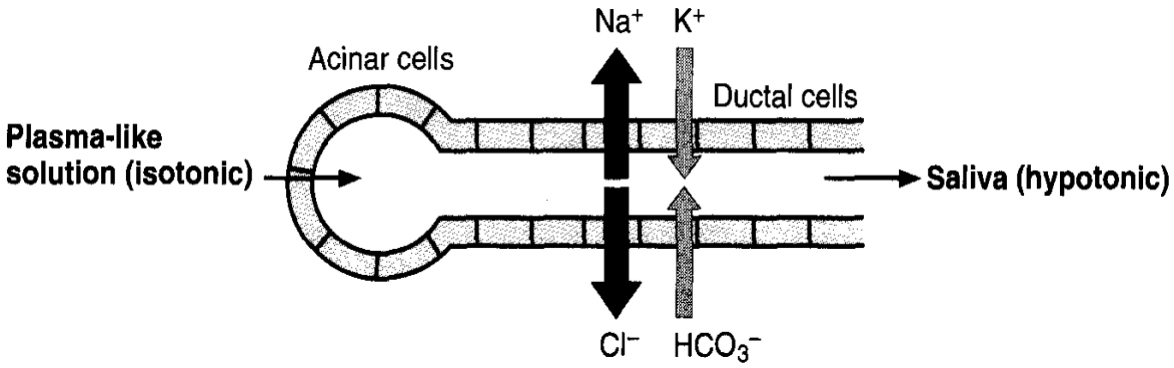

Figure 17. Saliva modification by ductal cells (Costanzo; 2002).

Acinus and ducts role in saliva production

1) The acinus:
   - produces and initial saliva with a composition similar to plasma;
   - this initial saliva is isotonic and has the same sodium, potassium, chlorine and bicarbonic ions concentrations as plasma.
2) The ducts modify the initial saliva by the following processes:

- the ducts reabsorb Na and Cl ions; therefore, the concentrations of these ions are lower than their plasma concentrations;
- the ducts secrete potassium and bicarbonate ions; their concentrations of these ions are higher than their plasma concentrations.

Salivation is physiologically controlled almost entirely by the autonomic nervous system. Either parasympathetic or sympathetic stimulation will produce secretions; parotid or submandibular secretions probably never occur without autonomic input. Parasympathetic stimulation predominates, and parasympathetic denervation results in glandular atrophy. Conversely, sympathetic denervation causes little, if any, effect. Vasoactive intestinal polypeptide (VIP) and acetylcholine are released from parasympathetic nerve terminals in salivary glands and contribute both to vasodilation during secretory activity (Levy, Koeppen, Stanton; 2018).

Thus, saliva is characterized by:

- high volume (relatively to the small size of the salivary glands);
- high potassium cathion and hydrocarbonate anion concentrations;
- low sodium cathion and chlorine anion concentrations;
- hypotonicity to plasma (Levy, Koeppen, Stanton; 2018);
- presence of alpha-amylase, lingual lipase, and kallikrein.

Saliva becomes hypotonic in the ducts because the ducts are relatively impermeable to water. Because more solute than water is reabsorbed by the ducts, the saliva becomes dilute relatively to plasma. Salivary composition differs dependently on salivary flow rate and some data are represented on the figure 18.

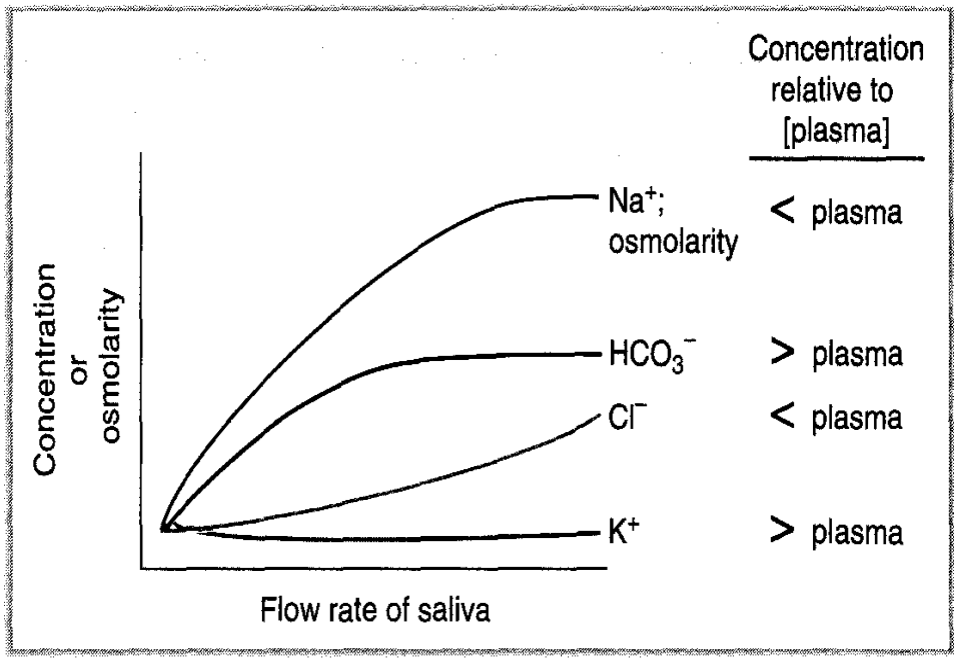

Figure 18. Composition of saliva as a function of salivary flow rate (Costanzo; 2002).

- At the lowest flow rates saliva has the lowest osmolarity and the lowest sodium cathions, chlorine and bicarbonate anions level, but has the highest potassium cathions concentration; it is the least like the initial acinar secretion.
- At the highest flow rates (up to 4 ml/min) the composition of saliva is closest to that of plasma, initial acinus concentration; it has the lowest potassium level, the highest sodium and chlorine concentrations.

The only ion that does not "fit" this contact-time explanation is bicarbonate; its secretion is selectively stimulated when saliva secretion is stimulated.

Protective function:

a) Saliva moistens and softens food, covers mucosae with a sheath and thus protects them from mechanic damagings with hard food and facilitates swallowing.

b) Mechano-, thermo- and chemoreceptors strong irritation and action to nociceptors lead to releasing of great number of saliva poor on enzymes and performing next tasks: ability to the fastest removal of harmful substances out off oral cavity, normalization of coming products temperature.

c) Saliva permanent flow plays cleaning role also while preventing the pathogenic microorganisms attachment to the epithelium and teeth surface; it also encourages food residues removal and these residues are as a nutritive environment for microorganisms.

c) *Buffer* saliva features are essential and allow neutralizing acids and alkalis of removable substances. Also it is important for neutralizing the acids producing by pathogenic microorganisms (that prevents enamel demineralization). There are 3 main buffer systems namely bicarbonate, phosphate and proteinic. Bicarbonate buffer powers 80% of saliva buffer capacity. Buffer features are connected with alkaline salts existence in a secretion (sodium and potassium chloride et al.). Salivary pH under norm is 5,7-7,4. At hypersalivation pH is increased up to 7,8. Saliva pH is important for all oral cavity tissues and formations state and functional activity assessment. In neutral environment saliva covers teeth equally with special membrane formation on them. Saliva is mineralizing liquid. But at its pH decreasing (for example, at caries) it becomes demineralizing liquid from the mineralizing one (mineral ions do not come to enamel and even come out of it). Under such conditions mucin forms solid covering on teeth which is difficult to be removed. At pH 6,5 and lower oral liquid is deficient on Ca content and these ions leave enamel. Far result – is dental plaque. At pH equal to 7,0-8,0 saliva is oversaturated with Ca^{2+}-ions. It creates optimal conditions for these ions coming to enamel. Durable taking of sweet drinking (coffee with sugar, strawberry yoghurt) encourages enamel demineralization especially in people with saliva hyposecretion. Saliva pH can restore to its initial level for several minutes in the people with labile buffer system. pH determining methods used in dentistry are: pH-meters and litmus papers. Also saliva buffer functions play an important role for neutralizing the

stomach acid content that can pass to oral cavity while regurgitating. Saliva acid-alkaline equilibrium influences actively on teeth enamel re- and demineralization, dental covering formation, oral cavity defense mechanism expression, parodont tissues and mucosa state. Acid-alkaline homeostasis disorder encourages proteins protheolytic degrading in part of dental pellicle components proteins as well as enamel demineralization enforcement. These data prove the necessity of oral cavity good hygiene as well as of rational feeding for saliva pH disturbances unfavorable results prevention.

d) Saliva is saturated with ions of calcium, phosphate and chlorine high concentrations of which encourage these ions transfer into enamel that is crucial in enamelogenesis after teething and makes it hard and resilient to decay development. *Mineralizing function* is important for enamel normal chemical content maintaining.

e) Epidermal growth factor (EGF) presence provides epithelium regeneration physiological level maintaining and encourages damaged mucosa healing acceleration both of oral cavity and of caudally located areas of alimentary tract. It should be mentioned that this substance content is significantly less in saliva than in other animals.

f) Saliva cools excessively hot food avoiding oral mucosa thermal damaging.

g) There is a *secretory immunoglobulin "A" (SlgA)* in an oral liquid (Strober, Kelsall, Fuss et al., 1997) and saliva (Fathi; Asadi Farsani; Adeli, 2018). Its content in saliva is much higher than in serum. It is a glycoprotein by structure. It is synthesized locally by plasma cells formed from B - lymphocytes, mainly, in a submucosal layer. Parotid glands are main source. It interferes with antigens introduction, has antibacterial and virus neutralizing activity. SlgA is formed at the interaction of plasmocytes synthesizing lgA, and secretory component the synthesis of which is performed by salivary glands ducti epitheliocytes. Secretory lgA has higher molecular weight than serum lgA (390000 and 150000 Da correspondingly). It protects mucosae coverings and prevents penetrating the microorganisms into the tissues. SlgA antiadhesive features determine its antibacterial and antiallergenic features. SlgA prevents adhesion of allergens, microorganism and their toxins at the surface of mucosae epithelium that blocks their penetrating into organism internal environment. The persons with defect of the given immunoglobulin have often inflammatory diseases of oral cavity because of oral cavity local immunity weakening. SlgA ability to protect mucosae from side antigens is determined by its high resilience to proteinases, incapability to bind complement components that prevents its damaging action to mucosae. *Serum lgG and lgM* passage into oral cavity at oral mucosa inflammatory processes is of great importance because their coming encourages to local immunity enforcement (Strober, Kelsall, Fuss et al.; 1997).

h) In saliva there are the *components of a complement (C3, C4)*, playing the important role in the phagocytosis reactions and also stimulating the cell and humoral immunity reactions. They get in saliva from circulation through odontal-gingival sulcus.

i) Such a stability is defined by saliva content, bacteriocydic and bacteryostatic substances containing in it:

- *Enzyme lysozyme (muromidase)* plays important role in oral cavity homeostasis supporting. It splits beta-1,4-glycoside bonds between residues of N-acetylmuramic acid and 2-acetamino-2-desoxy-D-glucose of glycosaminoglicans and proteoglicans. This enzyme bacteriolytic action is delt with muramic acid destruction in some bacteries wall changing its permeability that causes their content diffusion in surrounding environment. It represents an alkaline protein consisting of 129 amino acid residues. Its molecular weight is 15000 Da in average. The enzyme concentration is fluctuated from 1,15 till 1,25 g/l in saliva. This enzyme decomposes bacterial wall plasmatic membrane and protects oral mucosa from pathogenic bacteries thus. Also it stimulates leucocytes phagocytic activity and participates in tissues regeneration. This enzyme source is parotid and submandibular salivary glands. Its content in mixed saliva is higher than in all other human liquids. Saliva lysozyme activity determining allows assessing the salivary glands functional state as well as saliva protective features at pathological processes in oral cavity.

- Salivary *lactoperoxidase* makes bacteriocydic action (participates in gram-negative bacteries lysis).

- *Myeloperoxidase* is an enzyme encouraging lipid peroxidative oxidation that results in the bacteries death.

• *Lactoferrin* is capable to bind two iron atoms and bicarbonate for bacteriostatic activity (Fine; Furgan; Beydouim, 2002, Nieuw Amerongen; Bolscher; Veerman, 2004).

• *Mucin* encourages bacteries big amount fixation to desquamating epitheliocytes. It belongs to protective proteins, stabilizes saliva minerals while supporting its micellar content and forms protective sheath on the teeth enamel – pellicle. It is believed that ionic bonds between calcium and proteins prevent calcium salts sedimentation. Ca and hydrophosphate ions can not form oversaturated solutions at mucin presence. Mucin gives the saliva viscous mucosal character because of carbohydrate component. Syalomucins negative charge provides their adsorbtion of teeth enamel hydroxyapathites. Salivary glands release the secret containing different concentration of mucin that depends on mucoid cells quantity. Submandibular and sublingual glands secret containing mucin is viscous and saliva secreted by parotid gland is not viscous because it does not contain mucin. Sublingual saliva is the most viscous.

• There are *beta-lysins* in oral liquid which penetrate here from blood and cause bacterial cellular membrane lysis. They exhibit maximal activity to anaerobes and spores-forming microorganisms.

• Saliva contains *interferons* having the ability to suppress viral replication, possessing the antitumorogenic features.

• Salivary *protheolytic enzymes* of wide activity spectrum can injury some bacteries membranes.

•*Lithium ions, zyanides* presence and other components also lead to microorganisms death.

Participation in hemostatic reactions also can be described as protective function.

In short, saliva contains substances influencing on all links of hemostatic process both with stimulative and inhibitory action. It can influence on vascular-platelet hemostasis, coagulative hemostasis and fibrinolysis.

It is known nowadays that saliva accelerates platelets aggregation. Aggregates become larger. Latent period gets shorter, platelets aggregation velocity gets bigger under saliva influence. It activates also blood clot retraction probably due to substances enforcing this process. Though, may be, this effect depends upon salivary calcium. For example, it can play important role at denturing, influence on interrelation between oral cavity tissues with the denture material at the inflammatory processes in mucosa, parodont tissues and salivary glands. Finally, aggregation under saliva influence can be caused thromboxane B_2 presence in it, which is considered to be the most powerful proaggregant.

Saliva possesses expressed procoagulative features. They get increased while aging. All salivary glands saliva contains a substance resembling of a tissue thromboplastin properties. It contains in a great number especially in the mixed saliva containing blood cell and desquamated epithelium. However, parotid saliva, as well as centrifuged and released from cells oral liquid, also contains a tissue thromboplastin. Besides there is in saliva an incomplete thromboplastin representing the complex of negatively charged phospholipids (cell membranes breaks). One can find almost all blood coagulation factors containing in blood plasma in saliva in a small concentration and also the fibrinolytic system components are found out there.

Thromboplastic activity is maximal in oral liquid, submandibular and sublingual saliva and minimal in the parotid one. Saliva coagulational activity is the mostly expressed in the elderly. It’s not an exception that keratinization process increasing while aging leads to the desquamating epitheliocytes increasing and thus encourages its thromboplastic features rising up. Although thromboplastic features increasing is also characteristic for other tissues and blood of the old. At the same time, oral liquid centrifugating lowers its thromboplastic potential insignificantly but their presence testifies to this hemostatic component glandular origin. Other procoagulants found in

oral liquid are antiheparinic factor, fibrinase (XIII), factors identical to the plasma factors IV, V, VIII, IX, X. Probably, part of them is filtrated from blood plasma.

Saliva possesses antithromboplastic and antithrombinic anticoagulants. They are less active than the procoagulants and do not play significant role in the pathological processes.

In parotid and mixed saliva composition plasminogen and plasmin are absent, but there are plasminogen activator and pro-activator. On its properties the plasminogen activator resembles tissue activator. It is quite possible, that it gets in saliva due to diffusion from blood. Besides the desquamated cells and leucocytes, being destructed, allocate trypsin-like and other proteases capable to lyse a fibrin. The fibrinolytic agents result in a vessels recanalization that is accompanied by circulation restoration in an injured oral cavity. At the same time, the fibrinolytic agents presence in saliva can render negative action as well. Maximal fibrinolytic activity is a characteristic for mixed saliva, then submandibular saliva while minimal – for the parotid one.

j) One can belong *antioxidative enzymes* (catalase, peroxidase and others) functioning belongs also to saliva protective function; their levels are in opposite correlation with periodontitis and gingivitis development (Golpasand, Zakavi, Etemadi, Ataii; 2018), they have bigger levels in smokers for damages compensation (Moballegholeslam; Mahjoub, Taghibakhsh, Bijani; 2018).

k) *Tumor necrotic factor alpha (TNF-α)* (Chaudhuri, Krishnankutty Nair, Ashok; 2018).

Salivation regulation

It is performed by complicated-reflectory and humoral ways. Special place in regulation has *complicated-reflectory* mechanism. It consists of conditioned-reflectory and unconditioned-reflectory one.

Conditioned-reflectory salivation regulation way is connected with food appearance, its smell (in humans and animals), communication about it and other conditioned stimuli (pictures, writings, symbols) delt with alimentary motivation.

Unconditioned-reflectory way appears as an answer to oral cavity mechano-, chemo-, thermo- and gustatory receptors irritation. Nervous impulses flow comes from these receptors through the V-th, VII-th, IX-th and X-th pairs of cranio-cerebral nerves to medulla oblongata in salivation center. Efferent fibres of given reflectory acts go from this center to salivary glands. They can carry information to salivary glands through sympathetic and parasympathetic fibres that innervate salivary glands. Sublingual and submandibular salivary glands are innervated by preganglionar parasympathetic nervous fibres coming in composition of tympanal chord (facial nerve branch) to corresponding ganglions located in glands body. Post-ganglionar nervous fibres innervate glands secretory cells and vessels.

Parotid glands are innervated by pre-ganglionar parasympathetic fibres of inferior salivatory nucleus of medulla oblongata coming in the composition of the IX-th pair in auricular node (figure 18). Post-ganglionar nervous fibres are directed to secretory cells and vessels. Sympathetic innervation is represented by pre-ganglionar nervous fibres from lateral corns of spine the IInd-IVth thoracic segments and ends in superior cervical node, then post-ganglionar fibres to salivary glands come. Receptors are muscarinic and beta-adrenergic.

Sympathetic nerve excitement leads to small saliva amount release containing mucin that does it viscous and dense. At parasympathetic nerve – on the contrary, saliva becomes fluid and its amount is big. Sympathetic trunk stimulation on human neck causes sublingual gland secretion but this influence does not interfere to parotid gland. Some biochemical mechanisms of salivary secretion are represented on the figure.

Salivary glands autonomic innervation is rather rich (Proctor, Carpenter; 2007).

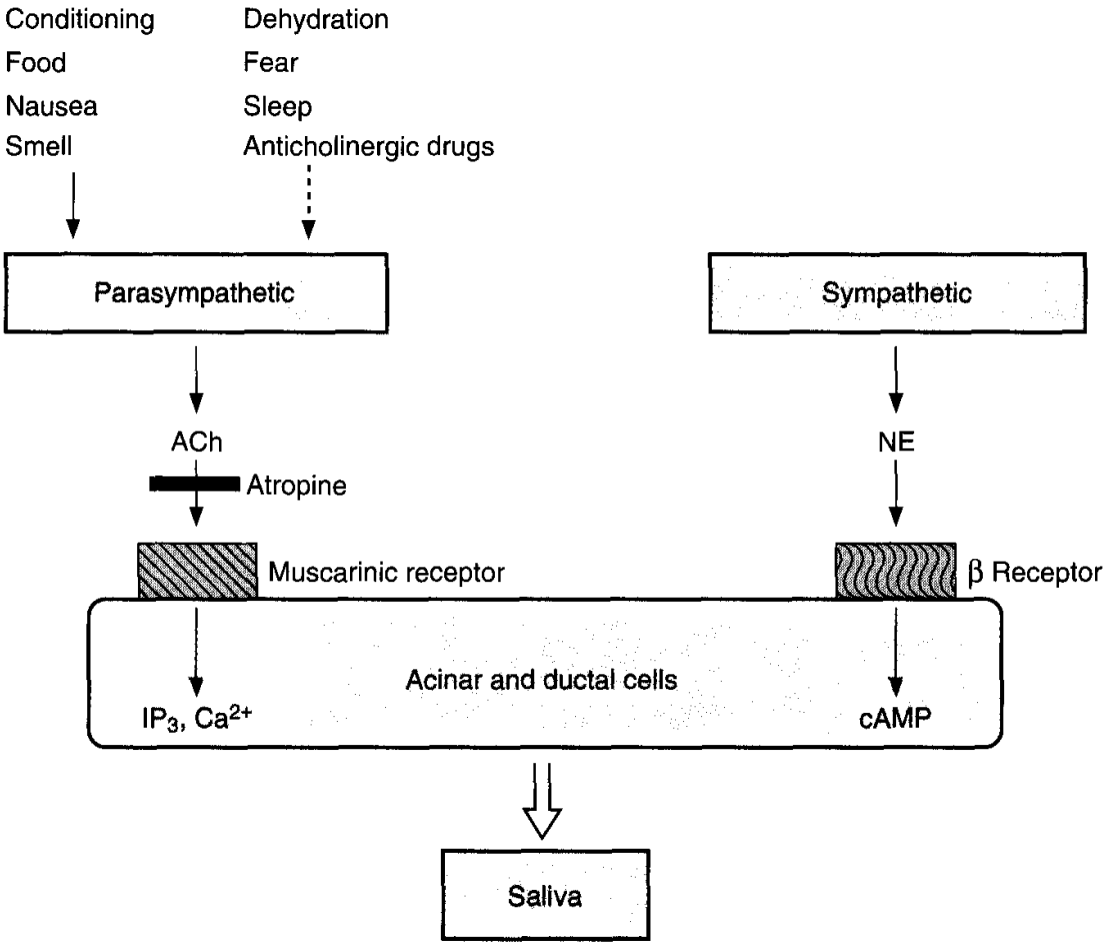

Figure 19. Salivary secretion regulating. ACh = acetylcholine; cAMP = cyclic adenosine monophosphate; IP3 = inositol 1,4,5-triphosphate; NE = norepinephrine (Costanzo; 2002).

Hypothalamic anterior and posterior nuclear groups participate in salivation regulation. Salivation reflectory regulation is not unique though it is main one.

Salivatory centers of medulla oblongata consist of two symmetrically-located neuronic pulls in reticular formation spreading from every side of facial nerve nucleus till n.ambiguus anterior part. This neuronic formation rostral part – superior salivatory nucleus – is connected to sublingual and submandibular glands while the caudal one – inferior salivatory nucleus – with parotid.

There is a tight functional connection between heart-vascular, respiratory and salivatory systems. For example, such a complicated reaction as vomiting act involves: salivation, swallowing, spastic respiratory movements, cardiac-vascular reactions, abdominal musculature, diaphragm.

Cortical regulation is also possible and can be proven by conditioned reflex possible development.

Humoral mechanism is delt with: hypophyseal, pancreatic, thyroid, sexual hormones action. But it is worthy to mention that these glands have modulating action more than the triggering one. Aldosterone acts on the ducatl cells to increase the reabsorption of Na and the secretion of potassium ions (analogous to its actions on the renal distal tubule). Excessive salivation occurs due to salivatory center irritation by carbonic acid. For example, this reaction is present at asphyxia. Saliva releasing may be stimulated by vegetothrophic pharmacologic substances – pilocarpine, proserine, atropine. Saliva production can decrease too. It may be connected with noceoceptive and emotional reactions, with fever states, at systematic sleeping pills usage, diabetes mellitus, anaemia, uraemia, salivary glands diseases.

LIPS

Superior and inferior lips limiting oral fissure represent plicas on the basement of which there is muscular layer – mouth orbicular muscle (Bentsianov, Blitzer; 2004). They are covered by skin anteriorily, mucosa – posteriorily. There is an intermediate lips red margin between skin and mucosa on free margin.

Every part has a structure of skin, is covered by multi-layered flattened keratinized epithelium, has hairs, sweat and lard glands. Muscular fibers providing this part mobility penetrate derma.

Intermediate part (red margin) is characterized by epithelium significant thickening, thin transparent corneal layer presence, disappearing hairs and sweat glands with saved lard glands (especially in corners of the mouth and on superior lip). Capillary blood lets light through the epithelial layer defining red color of this lip part. As an intermediate part has only singular lard glands and absent salivary glands its surface can get dry and cracked. That is why it requires seldom moisturing while lips licking.

Newborns lips intermediate part is covered by epithelial outfoldings (pillis) which are considered to be thought as adaptation for sucking.

Mucosal part represents typic mucosa covered by non-keratinized epithelium. Lips mucosal part has many shine salivary glands. They are complex alveolar-tubular proteinic mucosal glands with dominating mucosal cells the amount of which is reduced from the middle line to the periphery.

Blood supply

It is provided by facial artery representing external carotid artery branch.

Innervation

Sensory nerve is trigeminal, motor nerve is facial (Hotta, 2016).

Functions:

1) sensory – due to big amount of tactile, temperature and pain receptors;
2) speech formation;
3) food capture;
4) food keeping in oral vestibule;
5) oral cavity closage at mastication.

TONGUE

The **tongue** is a large, muscular organ that occupies most of the oral cavity (figure 20, appendix). The major attachment of the tongue is in the posterior part of the oral cavity. The anterior part of the tongue is relatively free, and its most anterior attachment is to the floor of the mouth by a thin fold of tissue called the frenulum.

Blood supply

It is performed by lingual artery – external carotid artery branch.

Innervation

It is provided by sensory and motor nerves. Mucosa receives sensory (general and taste) innervation from trigeminal, facial, glossal-pharyngeal and vagal nerves. Muscles are innervated by sublingual nerve.

Functions

Tongue has a definite role in mastication. It takes an active part in a food mixture, definition of its place for getting smaller on teeth. The tongue moves food in the mouth and, in cooperation with the lips and cheeks, holds the food in place during mastication.

It also plays a major role in the process of swallowing. The tongue is a major sensory organ for taste, as well as one of the major organs of speech.

ORAL CAVITY MAJOR FUNCTIONS

Oral cavity multiple functions are in great dependence on salivary functions (Edgar, Dawes, O'Mullane; 2012, Lightenberg, Veerman; 2014). As a whole, oral cavity protective mechanisms are divided into two groups (Mistchenko, Tkachenko; 2005):

1) non-specific defense factors – acting against all microorganisms and side substances;
2) specific (immune) – influencing only on definite microorganisms and proteins.

One can differentiate three mechanisms of non-specific defensive factors action:

1) mechanic:
   - non-damaged mucosa barrier function;
   - microorganisms washing with saliva;
   - splitting;
   - mucosa clearance during food taking;
   - desquamated epithelium adhesion on the cells;
2) chemical – different biologically-active substances (described below);
3) physiological – phagocytosis.

Antitoxic, bacteriolytic and bacteriocydic substances presence in saliva does not provide complete defense against the pathogenic action of coming microflora. Barriers have a crucial role in these processes.

Barriers represent special physiological mechanisms encouraging preventing the organs and tissues from a contact with the damaging agents, side substances, toxins, venoms, viruses et al.

As a whole, all barriers (conditionally) are divided into external and internal ones.

The external ones include: skin, lungs, alimentary tract, liver, kidney.

The internal barriers represent so-called histo-hematic ones: hemato-encephalic, hemato-ophthalmic, hemato-lymphatic, hemato-pleural et al.

Barrier function can be described as a protective subfunction or as a separate one. It is realized mainly by oral epithelium. It is achieved by a row of factors:

- epithelium significant width,

- multiplied intercellular bonds presence,
- few-permeable, chemically and mechanically stable corneal layer (when it is present),
- its superficial layer constant loosing,
- antimicrobial substances fats release and synthesis,
- epithelium permanent washing by saliva possessing powerful antimicrobial compounds and growth factors,
- calprotectinpeptide that is produced by epitheliocytes and neutrophils and possesses powerful antimicrobial activity; its expression is characteristic in the biggest extent for mucosa covered by non-keratinized epithelium.

The barrier quality depends greatly on the layers quantity and epitheliocytes in oral cavity. Barrier function is provided by constant changing and loosing the cells of external layer. These cells are damaged and have high regenerative ability that defines rather short terms of its renewal. The strongest barrier is observed on tongue covered by keratinizing multi-layered epithelium. Gingival barrier is much weaker because gums are covered by one-layered epithelium that makes it vulnerable in the biggest extent, permeable for the injuring agents. Though the gingival weakness is compensated by big amount of the cells having the ability to phagocytosis and located in the gum submucosal layer. Such cells amount is much less in the tongue submucosal layer. Phagocytes get involved into the defensive reactions at damaging or deficiency in the epithelial barrier functioning.

Oral cavity protective function systemic mechanisms are functional integrity of behavioral, conditioned- and unconditioned-reflectory, barrier and immune-chemical reactions. Information about threat to tissues integrity occurs at super intensive influencings onto mechano-, thermo- and chemoreceptors of tongue, lips, cheeks mucosa, palate, periodont and others. Besides, at oral cavity tissues injury special chemoreceptors (chemonociceptors) percept substances forming in course of cells destruction and direct the information to CNS. On the base of this information compensatory mechanisms get formed the ending aim of which is to provide tissues integrity, to protect organism from injury.

One of protective mechanisms is *behavior* directed to injured factors avoiding (head turn, jaws closure, running from irritator, avoiding the dangerous places et al.). Defensive behavior may be passive and active. Pricking up, covering, harboring, avoiding something (remind children's behavior in dental clinics) belong to behavior passive forms; aggression, resistance belong to the active ones. But the most important urgent oral cavity protective mechanism is salivation – saliva releasing as answer reaction to removable substances coming. Protective function is determined by mucosa impermeability to various microbes and viruses except to the ones of tularemy and foot-and-finger. Besides, microbes and their life activity products are released in a process of

a permanent desquamation of epithelium. Oral cavity normal microflora represented by bacteries species big number avoids multiplicating the microbes invaded from the external environment.

Alongside with alimentary or removable substances toxins and microbe flora (particularly the pathogenic one) come into oral cavity. There are more than 400 types of bacteries in a human mouth, some of them may be the reason of infectioning of gums and osseal tissue below them.

The resident microbial flora in the oral cavity typically contains 1010 bacterias. Over 500 bacterial species are today recognized as normal inhabitants of the oral cavity. However, only 150 microbial species have been isolated and cultured from root canals. The endodontium is a sterile cavity and the ingress of oral microbes to establish infection is quite difficult when compared to other dental tissues, as the microorganisms have to penetrate the enamel and dentine and overcome the host responses. Furthermore, they have to survive in the limited space, nutrients and distinct residents alongside other root canal microorganisms by genetic exchange, mutation and highly modified functions. Therefore, though all the bacteria in the oral cavity can invade the root canal, only a few microbes have been identified in infected root canals.

There exist rather favorable conditions in oral cavity for microflora development – food residues existence, weakly alkaline saliva environment (pH), humidity, optimal temperature. Microorganisms are up to 70 per cent of dental covering. It was estimated that approximately 250 microbe cells are in 1 mg of dental covering dry mass, 1 ml of saliva contains more than 10^8 of microbes. Facultative and anaerobic diphteroids represent 80% among resident (permanent) microflora. Microbes and viruses distribution in oral cavity is unequal - their main part is located in dental-gingival pockets, mucosa plicas and interdental spaces. The facultative and anaerobic diphteroids are dominant on the tongue back. Neisserias are present constantly in oral cavity and reach 3-5% from all bacterias amount. Lactobacilles, streptococci and flagellas represent usually near 1% of general flora. Spirohets are characteristic for gingival liquid where their quantity is 1-5%. The biggest part is represented by polyforms, mycoplasms and candidas.

The synergistic mechanisms between the various endodontic pathogens involve an interplay of various factors, like providing nutrition, inhibition of phagocytosis (i.e. preventing the opsonisation and inflammation, destruction of phagocyte), secretion of growth factors and enzymes, decrease in the local oxygen concentration and oxidation–reduction potential and local pH in the root canal. These mechanisms facilitate the survival and pathogenesis of obligate and facultative anaerobes.

Bacterial antigens can form immune complexes antigen-antibodies activating complement system releasing various biologically-active substances. Phagocytosis got activated as well as neutrophilic chemotaxis. Gram-negative bacteries lipopolysaccharides enforce immune answer as dextran produced by veilonellas.

Bacterial antagonism existing in oral cavity represents important protective factor. But autoinfection appearance can be at oral mucosa barrier function significant weakening.

Mucosa barrier function weakening takes place as a result of: various dystrophic processes, infection diseases, other diseases, burnings, mechanic traumas, chemical irritation, medicines action.

Pathogenic microflora is of essential importance at gums injuries. Special attention should be payed to parodontosis development. Gums inflammation, in course of which they become sensitive to different irritators action and bleed – it is the first stage of this disease, affecting millions of people. But not only gums are affected at this disease. But while sick gums are exfoliated out off teeth deeper and deeper pockets get formed where infection penetrates destructing osseal tissue. Teeth are sitting in their nests undensely and that's why finally human being loses them. But simultaneously parodontosis may accelerate other diseases development in organism or make their course complicated. How defense from pathogenic microflora is performed in oral cavity? In course of oral cavity microflora it was established that it possesses relative stability preventing pathogenic microorganisms spreading.

Dendritic cells, immature ones or Langerhans' cells in part, are present in oral mucosa and perform antigen-presenting function. Their maturation is enforced by bacteries. Mucosa epitheliocytes can perform presentation of MHC II molecules (Kvale, Brandtzaeg; 1995).

Non-specific and specific protective mechanisms realizing at especial lymphoid tissue participance get involved in the reaction in the case if saliva components and tissular barrier did not deal with a microflora pathogenic action.

Mucosa takes part in local immunity providing. This function is weaker than in caudally located parts of alimentary tract. But in oral cavity itself food antigens as well as microbial antigens act to the organism tissues first of all. Oral cavity mucosa has cellular elements participating both in afferent and efferent link of immune reactions (cells of Langerhance, macrophages, lymphocytes, plasmocytes).

Tonsils structure is similar: lymphocytes mass with their spheric accumulations named follicles is located between connective tissular fibers (Seeley, Stephens, Tate; 1999).

Lingual tonsil that is in lympho-epithelial pharyngeal ring compound represents immune system specialized structure located in oral cavity. Palatal and lingual tonsils represent the most expressed accumulations of lymphoid tissue. They realize detoxication of infection-toxic substances. The authors majority consider that there exist two lingual tonsils. Their second function is in hemopoiesis: they save as a home for lymphocytes coming partially into the lymphatic vessels, partially into oral cavity and pharynx. Probably, lymphopoiesis takes place in the follicles and in diffusally disseminated connective tissue as well. Lymphocytes get destroyed after coming into

oral cavity that is accompanied by releasing the lysosomal enzymes accelerating the chemical processes encouraging pathogenic microflora detoxication.

Tonsils are called as “guarder points” avoiding the infection distribution into deeper parts of the organism.

Palatine tonsils have clinically important features: free or faucal surface is directed to pharynx cavity and is covered by mucosa with multi-layered or flattened epithelium.

Tonsil has 16-18 deep fissures called as lacunes or crypts. Tonsil external surface is bond with pharynx lateral wall by tonsillar capsule. Many connective fibers connected one with another by meatus of trabecules or septi, forming densely-looped net, pass from this capsule into tonsil parenchyma. Alveoles are filled with lymphocytes (sometimes they are grouped in follicles), mast cells, plasmocytes and the others. Lacunes penetrate tonsil thickness. They have branches of the first, the second, the third or even the fourth orders. Lacunes walls are covered by flattened epithelium desquamating in many places giving hemorrhagies. This epithelium represents basement of so-called corks.

One can mention following clinically important factors. Prophound, narrow and branched (like trees) lacunes releasing is easily to be disturbed because of this and because of lacunes estuary scars constructions. Moreover, these estuaries are partially covered by mucosa flattened plica (Gis’ plica) in palatine tonsil anterior-inferior part. These morphological-topographical predispositions create favorable conditions for inflammation occurrence in palatine tonsils superior pole. There is one predisposed factor for inflammation development: palatine tonsil lobule can lie in a soft palate higher than the palatinal tonsil. Lymphadenoid tissue as granules (follicles) or shine pointed formation is also present in pharynx posterior wall.

Tonsils function – protective – “home” for B-lymphocytes. They represent peripheral lymphoid organ.

Valdeyer-Pirogov’s ring (pharyngeal) comprises: 2 palatine tonsils, 2 tubular, 1 pharyngeal (naso-pharyngeal) – it possesses strong receptive apparatus, 1 lingual, mentioned less accumulations of lymphoid tissue.

Although tonsils represent themselves mature organs up to birth, pharyngeal lymphoid ring structural elements development is not finished to this time. It is maximal from 3 till 7 years, pharyngeal tonsil gets gradually less beginning from 12 years, it represents only little residues of lymphoid tissue up to 16 years while it is atrophised in the adult.

The antibodies have the special value in an oral cavity protective functions (Кузник; 2001).

Phagocytosis also plays an important role in an oral cavity. 1/80 of all blood leucocytes are allocated in an oral cavity for one day from a gingival blood while this

index is increased in 2-10 times at inflammations. There is a leucocytic formula of saliva. 95-97 % of it neutrophils comprise, 1-2 % - lymphocytes and 2-3 % - monocytes. Immature dendritic cells capture and phagocityze bacterial antigens (Lanzavecchia; 1996) and HLA II in rather little amounts under the organism intact conditions or do not carry them at all (Kleijmeer, Oorschot, Geuze; 1994). Dendritic cells maturation, migration (and even elimination) from mucosa are triggered by TNF-α, IL-1β and lipopolysaccharides (Austyn; 1996). Immature dendritic cells level can change at smoking (in different directions by various authors) (Barrett, Williams, Scott; 1991) or remain unchanged dependently on smoking intensivity as well as can vary under some pathological conditions. Alcohol taking enforces smoking influence.

The oral mucosal epithelium serves as a barrier on the way of any antigens penetration, including cancerogens. The appreciable amount of neutrophils, and also monocytes is located under an epithelium, through which they migrate from the vessels of an own plate in a gingival sulcus break. Neutrophils migration velocity makes 30 000 in 1 minute. One can find out T-lymphocytes and B- lymphocytes in oral mucosal epithelium. Langerhans' cells, amounting about 2 % of cell population play an important role in maintenance of oral mucosal epithelium barrier function. They, mainly, are in a status of constant movement that facilitates a meeting with an antigen. There are dendrite antigen-presenting cells, epitheliocytes and others in oral mucosal epithelium.

Defensins – represent bacteriocydic and fungicydic peptides localized in oral mucosa in part capable enforce local and systemic inflammatory answer due to their signal function (Yang, Chertov, Oppenheim; 2001, Dale, Krisanaprakornkit; 2001).

Cathelicidin (LL-37 or FALL39) – antibacterial protein with molecular weight 18 kDa is present in oral non-keratinized flattened epithelium as well as in saliva (Murakami, Dorschner, Gallo; 2002) and gum tissues and possesses bacteriocydic action.

The oral cavity plays an important role *in* ***hemostasis regulation*** as well. Oral cavity organs release substances influencing on vascular-platelet hemostasis, blood coagulation and fibrinolysis.

The first stage of any trauma and inflammation is accompanied by hypercoagulation (to catch the microbs in fibrin net), the second one – hypocoagulation (to solve them in this net).

Finally, it should be mentioned in conclusion that alimentary organs chronic diseases sometimes are accompanied by appearance of antibodies in circulating blood that react to food proteins antigens and glycoproteids of milk, eggs, fish, citruses, chocholate and other foods. These antibodies to foods participate in alimentary allergy pathogenesis (toxic-allergic stomatitis). But we would like to pay your attention to the fact that antibodies against alimentary antigens are in blood of healthy people too. That's why feeding according to blood group can be mentioned as important way of oral cavity

diseases prevention as well as other factors of a healthy life style (physical activity, harmful habits et al.).

Oral mucosa has a definite set of power for physical loadings due to turgor, ability to stretching. This function is defined mainly by tonofilaments presence in epitheliocytes cytoplasm, fibrous elements number (elastic fibers in part) as well as tissues satiation degree with water and fatty cellulose development.

Now let's touch *oral cavity participation in hemostatic processes* which can be also described as defensive function (Кузник, Пинелис, Хавинсон; 1999).

Saliva-producing tissues can influence on saliva aggregative features. It can be predisposed that antiaggregative activity of salivary glands tissues is connected with prostacyclin-like substances presence in them (prostacyclin is known as the most powerful antiaggregant). Saliva influence on thrombocytes aggregation can also depend on antiaggregative features of the tissues of salivary glands and parodont possessing mainly the proaggregative functions. Microcirculative hemostasis disorders will encourage developing the parodontitis, salivary glands inflammatory processes as well as influence on the interrelations between mucosa tissues and dentures materials at prosthetic treatment.

Physiological bases of measurements at prolonged bleeding after tooth extraction

They include:

1) careful local haemostasis by means of making tampons with thrombin, haemostatic spongea and epsilone-aminocapronic acid;
2) in parallel to this – replacement therapy taking into account blood coagulation factors deficiency.

Physiological basement of patients preparation to tooth extraction at blood diseases

Such patients can have complications as bleedings after operations. That's why doctor before dental manipulation performance must ask the patient whether he had prolonged bleedings at wounds or operations. If it is necessary the patient must be consulted in hematologist. Dental operations should be performed at in-patient department in separate patients with blood diseases.

Dentist should follow systemic approaches 1st stage at hemophilia in the patient:

- anamnesis taking;
- X-ray examination;
- special protective plates making from plastic mass (such plates are placed right before the operation or in the operation day).

The IInd stage – operational one. Local anaesthesia should be done with thin needle application. All manipulations on tooth extraction are sparing with maximal mucosa ruptures. They perform hemostasis with haemostatic sponge application,

thrombin, aminocapronic acid and protective plate. They perform replacement therapy by hemopreparations injecting (plasma +necessary coagulation factors).

The IIIrd stage – post-operational. Replacement therapy continuation is observed.

Complications occurring after tooth extraction in patients with blood coagulation disorders

There can be long-termed massive bleedings from oral mucosa at its traumas and especially after tooth extraction in such patients. Tooth is bigger in patients with haemophilia. It is delt with teeth mineralization disorder on the background of total decalcination due to frequent bleedings. Frequent bleedings lead to clots appearance in a cavity and thus to bacterias and inflammation development.

These hemostatic physiological factors role is in following: saliva washing oral mucosa encourages local hemostasis. It is well-known that wounded surface on oral mucosa occurs every day during eating and possibility of blood vessels is quite big. But bleedings in oral cavity are stopped quickly due to active salivary procoagulants and, first of all, thromboplastin.

At the same time, oral mucosa high regenerative ability during small traumas under physiological conditions is provided mainly due to fibrinolytic agents in saliva. These agents help to mucosa clearance from fibrin plicas and desquamated epitheliocytes.

The question about tight interconnection and interrelation between hemostatic processes and other protective blood and tissular systems (antioxidant, immune, complement, non-specific resistance) is having been discussed during last 20 years in a scientific literature. The same interrelations between mentioned reactions are also present in oral cavity both in saliva and in tissular level. One can find free radicals, antioxidants, immunocompetent cells, non-specific defense factors in given substrates. Their activity and level are changed in saliva and tissues at some pathological reactions.

For example they have stimulating influence on microcirculative hemostasis state, blood coagulation and fibrinolysis at stomatites, parodontitis, jaws fractures and other pathological processes in oral cavity saliva (and corresponding tissues). Saliva thrombocytoactive and coagulational features enforcement at different-origined inflammation processes in oral cavity encourages local hemostasis as the result of which fibrin gets formed. Fibrin helps wounded surface to repair. But this reaction must not have excessive character because increased fibrin formation can be unfavorable phenomenon which disturbs tissue inflamed locus feeding and encourages microflora growth in this locus (fibrin represents very favorable nutritive environment).

At the same time, fibrinolytic features increasing at this is of essential importance because it helps in reaching the tissues clearance from different metabolism products and fibrin coating. Besides, saliva active fibrinolytic agents can encourage oral cavity tissues tolerance to hypoxy. Although excessive fibrinolytic features increasing in saliva can play also negative role leading to premature fibrin removal (id est in alveole of extracted tooth) and thus to slow reparation down. Such a reaction is essential for wound

clearance from non-alive tissues and products of their decomposition in the first inflammation days when hyperfibrinolysis takes place in saliva. Then when wound is clean and connective tissue granulation has begun excessive fibrinolysis can be unfavorable. Under such conditions we must inhibit fibrinolysis.

According to literature data the biggest amount of pathological processes in oral cavity are accompanied by hypercoagulation. Such a reaction is considered to be the signal about injury.

One can determine anticoagulant hemostatic link weakening at excessive influencings (inflammations, tissues injuries at woundings, traumas and others) in oral cavity. As a result, the reaction observed transmits to disseminated intravascular coagulation (DIC).

Uncontrolled bleeding from tooth extraction, periodontal surgery or other oral and maxillofacial surgery procedures can occur in patients with either platelet disorders or coagulopathies **(**Basi, Schmiechen; 1999, Bodner, Weinstein, Baumgarten; 1998, Garfunkel, Galili, Findler, et al.; 1999, Troulis, Head, Leclerc.; 1998**)**. Oral manifestations include petechia, ecchymosis, and spontaneous oronasal bleedings that occur in the absence of any significant trauma. Purpura found in conjunction with gingival enlargement and malaise may underlie leukemia. If facial spider telangiectasias are evident and the skin or conjunctivae are jaundiced, cirrhosis of the liver must be assumed. Purpura can occur in patients with HIV infection, indicating to thrombocytopenic purpura. Ecchymosis of the mucosa or skin in a patient with a history of vascular occlusive disease may be indicative of Coumadin therapy. The medical history should be thoroughly reviewed to explore whether there are any past or present episodes indicative of a hemorrhagic diathesis.

Two dilemmas can occur in dental patients:

1) unknown performing a surgical procedure on a hemorrhagic subject, and
2) performing necessary extractions or surgeries in a patient with known coagulation disorder.

DIC (disseminated intravascular coagulation syndrome) – is the result of quantitative movements in hemostasis system which leads to the new state. Main region for all disorders development is microcirculatory vascular bed. Under one conditions this state has local, under the others – more generalized character. DIC-syndrome at salivary glands diseases, stomatites, mucosa injuries at jaws traumas, surgical influencings in this area and other processes development are more often of local character. During tissues injury, for example of salivary glands, their decomposition products come to the blood stream. It becomes key factor of DIC-syndrome development. Such a reaction in oral cavity has such course like at Artus' or Sanarelli-Shwarzmann's reaction. They are the most typical DIC-syndromes variants in dentistry.

The first phase of any trauma or inflammation is accompanied by hypercoagulation while the second one – with hypocoagulation. DIC-syndrome is rather rapid in oral cavity and is accompanied by autoimmune reactions. The postponed

stabilized fibrin (for example, in a removed tooth alveola) is a matrix for development of connective tissue, that promotes the reparative processes and fast healing up of wounds in oral cavity. Fast fibrinous clots formation interferes with an infection hit into the oral cavity wound depth. Quite often after odontectomy operation alveolar bleeding arises because of fast dissolution of a fibrinous clot. It is promoted by stress experienced by many patients at the reference to dentist. The similar picture can arise also at operative procedures in oral cavity, at mandible fractures, gingival fissure liquidation and others. The fibrinolysis inhibitors local application in course of them promotes not only fast bleeding stoppage, but also leads to earlier operational wounds healing.

Thus, oral cavity protective mechanisms providing the integrity of tissues of alimentary channel initial part and organism in a whole are very complicated system.

Regulative function

It is determined by such biologically-active substances presence in it as parotin, trasylol, kallikrein, nerves growth factor, epidermal growth factor. Other substances action has been discussed above while oral cavity endocrine function describing.

Excretory function

Exchange products (urinary acid, creatinin), medicines, hard metals, halogens are excreted with saliva.

Watery-salty homeostasis regulation

It is connected with releasing the liquid containing ions of sodium, potassium, calcium, chlorine and others.

Oral mucosa absorbtive function

It is rather expressed. This function provides permeability of some oral cavity mucosa area. So, thin mucosa at oral floor area is permeable for iodum, potassium, sodium, separate amino acids. Drugs (nitrong, glycerin, nitroglycerinum) can be absorbed very rapidly round to portal vein. This circumstance can be used for urgent care. These medicines are applied sublingually (for instance for stenocardia fit).

Mucosa epitheliocytes possess characteristic perspiration id est water passage through their thickness. This phenomenon is provided by intercellular space presence. It is a crucial factor of mucosa permeability at the area of gingival sulcus where gingival liquid is accumulated. This liquid is similar to blood serum by its content and contains electrolytes, enzymes, cells. Under normal conditions it performs a barrier function for the beneath-located parodontal tissues.

Oral mucosa excretory function

Metabolic products release appears both with saliva and with all mouth surface mucosa. Protein exchange products (ammonia at liver insufficiency, urea – at liver and kidney, acetone – at diabetes mellitus) can be released through oral cavity. It can help in diagnosis putting. Especially this excretory function is enforced when the one of other excretory organs (kidney, liver) is damaged. Hard metals like Hg, Pb can also be released. Hg intoxications sign is grey red margin and oral mucosa, Pb (lead) intoxication is accompanied by black gums, lips red margin and gums oral surface.

One can say that salivary glands switch the excretory function for compensation at main excretory organ (kidney) excretory function insufficiency. Permanent unpleasant smell from mouth in the patient is a result of urea big quantities release with saliva (urea is transformed into ammonium under salivary substances action).

Oral cavity role in speech breathing and speech creation

Human respiratory system besides its main function – lung gas exchange providing – participates directly in speech sounds creation. Acoustic effects main creative ways are air stream stoppage by rhythmic voice cords. Tonal and noisy sounds occur while air passaging with too large velocity through narrowings formed in this or that place alongside respiratory tracts. Thus, speech appears due to respiratory system actions, providing necessary pressure and air flows in speech-forming tract as well as due to this tract elements movement managing air streams. Oral cavity organs for example lips, tongue and teeth participate in acoustic effect creating because expiration in course of communication occurs through mouth.

Respiratory apparatus activity in course of speech is called ***speech respiration.*** Normal speech with correct and distinct sounds prononciation is tightly connected with dental rows integrity and dentition (Valcheva, Arnautska, Dimova, Ivanova, Atanasova; 2018) though it can be harmful habit (Arathi; 2012, Gill, Naini; 2011). Teeth loss especially the anterior ones leads to lisping, pronounced sounds clearance decreasing and even to losing of possibility to prononciation of separate from them. There may be salivation and saliva releasing through the gaps forming despite absent teeth. Speech defects can be also determined by disorders of salivary glands functions (dryness in mouth), masticatory musculature (muscles contracture and motor nerves paralysis), temporal-mandibular joint (mandible contracture) as well as congenital or acquired defects of maxillary-facial region organs, organs anomalies and uncorrect denturing.

One of main reasons of speech function disorder represent dental rows defects especially of dental-maxillary system frontal region. Sound generation distortion, energy consumptions changes under speech activity are observed. That’s why dentist in course of denturing must choose denture construction at which speech activity becomes optimal as for clarity of generated sounds and minimal energy on its loss.

Human being has no specific speech organs. He uses respiration, mastication and swallowing organs for speech-forming. But he has specialized vocal apparatus (larynx and vocal cords) for speech vocal selectiveness. Organs participating in speech-forming are divided into 2 groups: respiration organs (lungs with bronchi and trachea) and organs directly participating in voice-forming. One can differentiate *active* (movable) having the ability to change their volume and shape of speech tract and create obstacles from expired air in them; and *passive* (motionless) without such ability.

Active speech-forming organs:

larynx, pharynx, soft palate, tongue, lips.

Passive organs:

teeth, hard palate, nasal cavity, additional sinuses.

All these structures from the point of view of speech-forming peripheral mechanism one can imagine as 3 interconnected parts: generatory, resonatory, energetic.

There are 2 resonators: tonal – larynx, noisy – due to fissures creating in oral cavity.

Other resonators classification: 2 modulating – mouth and pharynx; 1 non-modulating – nasopharynx with additional sinuses.

2 energy sources:

- skeletal intercostal muscles, diaphragmal, abdominal;
- tracheobronchial tree smooth muscles.

Vascular reactions in sound-forming have vessel reactions in respiratory ways and vocal tract mucosa. Resonator function in sound-forming process depends on these parts blood filling state.

Respiratory ways and vocal tract mucosa glands secretion also influences on speech-producing. Its increasing influences on vocal tract resonatory features. Excessive secretion in naso-pharynx inhibits nasal sounds reproduction causing nasolaly. Hypersalivation influences on all sounds in which oral cavity, teeth, tongue and lips participate. This is the sphere of speech-forming odontogenic aspect. Every dentist should pay the attention to this aspect. Vocal tract is important executive part of speech-forming system. Here phonemic and whispered constituents of speech are formed. This part activity mostly is under competention of dentist. Dental rows integrity injury (especially incisives) leads to dental sounds forming changing and inhibiting (whistle, lisping). Pathological structures on tongue back leads to sounds reproducing inhibiting and disorders in labial (of lips) region. Changed occlusion influences greatly onto phonation result. It is especially expressed at opened, crossed occlusions, prognathy and progeny.

Phonation disorders receive corresponding names at different changings in oral cavity. Disorder delt with cleft palate (hard palate fissure) is called palatolaly**.** At anomalies in tongue structure and function occuring articulational disorders receive the name glossolalies**.** Uncorrect teeth structure and their localization in alveolar arches especially of anterior group (incisives and canines) are often reason of dyslalies. All mentioned dentist must take into account while medical manipulations performance in oral cavity performance.

Surgeon-dentist must forecast the possibility of speech-forming function disturbances in course of operations in oral cavity organs. Articulation mechanism knowledge is of essential importance for orthopedic dentist. Removable dentures production, especially at wide adenties or complete teeth absence leads to articulation correlations changing in oral cavity. Naturally, it influences on vocal apparatus resonator function. Occlusion overstating at denturing, artificial teeth uncorrect installation and even well-done denture always lead to speech-forming retardation at the first stages of adaptation. Patients with removable dentures often complain about these or those dyslalies signs: sound-production inhibiting, additional whispering, whistle and lisping. All this is necessary to take into account at dentures constructing and creation, especially for people which use speech actively in their working activity (artists, singers, lecturers, dictors, teachers). Famous statement "to train somebody's voice" to singer, artist, dictor or teacher means to tune respiration and articulation by definite behavioural measures usage.

Oral liquid, its difference from saliva and physiological role

Oral liquid - is a mixture of: saliva, gingival liquid, inspired and expired air liquid part, nasopharyngeal mucus, upper respiration ways mucus.

Comparatively to saliva, oral liquid can change its content and its physical-chemical features in very wide limits.

Role: mucosa and teeth prevention from dryness, protection from food physical and chemical action, thermoregulation.

BLOOD CIRCULATION AND ITS REGULATION PECULIARITIES IN MAXILLARY-FACIAL REGION

Blood circulation in dental pulp occurs inside its cavity having the walls. Pulse fluctuations of blood volume in closed cavity can cause tissular pressure increasing and as a result physiological processes in pulp disorders. But it doesn't occur due to arterial volume pulsal fluctuation transmission to veins. Pulp vascular net possesses effective antistagnational features. Sum crown's pulp veins space is more than in apical foramen region that's why circulation linear velocity in root apical foramen region is higher than in pulp. Veins pulse fluctuations are the similar to brain veins fluctuations. Pulpal abducting venous vessels anastomosize with periodontal veins and such rich anastomozes net with periodontal veins increases blood circulation system opportunities in pulp.

Blood circulation regulation of this region is performed by nervous, humoral and myogenic mechanism. *Nervous mechanism:* tonic impulsation comes to these vessels from vascular-motor center through nervous fibres coming from superior cervical sympathetical node. Vasoconstricting reactions into maxillary-facial region and dental

pulp are determined by noradrenaline releasing in sympathetical nervous fibre that acts through vascular walls alfa-adrenoreceptors. If it acts to beta-adrenoreceptors – vessels are dilated.

Maxillary-facial region and oral cavity organs vascular space may be also changed under influence of *humoral factor:* hormones (adrenaline and others), cellular metabolism products and electrolites.

Finally, there is a proper vascular tone regulation *myogenic mechanism* in this blood circulation region. Muscular type vessels (arterioles and precapillary sphincters) hypertony leads to functionning capillaries amount decreasing that in turn prevents intravascular blood pressure increasing and enforced liquid filtration in tissues, i.e. it serves as tissues physiological protection from oedema development. This myogenic blood circulation regulative mechanism plays important role in dental pulp acvtivity providing. Such a mechanism is essential for pulp located in closed space and limited by dental cavity walls for microcirculation regulation under norma and pathology for instance at pulp inflammation (pulpitis). Weakness of this vascular myogenic tone regulative mechanism is one of factors for oedema development in pulp, parodont and other oral cavity tissues. Myogenic vascular tone is significantly decreased at functional loads to tissues that lead to regional blood supply increasing and "working hyperaemia" development. At parodontosis, when parodontal tissues blood supply is disturbed, functional loadings decreasing microvessels myogenic tone (for instance, mastication) may be used for medical and preventive aims to parodont trophycs improvement. It has essential importance because vascular tone functional changes play dominant role in course of parodontosis development.

Humoral action to circulation in oral cavity may give many drugs absorbing there.

Any dentist should remember that oral cavity is a powerful reflexogenic zone afferent impulsation from which can change heart activity and vascular tone and that's why doctor shoud apply sparing manipulations there. Any dental manipulation is a complicated emotional-painful factor. Practically all people are afraid of such manipulations. Such noceoceptive factor may influence on heart-vascular system state. And such influence may be sometimes even more significant than medical procedure itself. It is especially actual for patients with heart-vascular diseases for instance suffering from hypertonic disease. These patients have expressed haemodynamic changings at dental influencing, in course of its waiting. Sometimes it is accompanied by crisis, giddiness, faint as a result of brain circulation disorders. Special place have noceoceptive irritations causing significant changings in blood circulation system. Such disorders may vary dependently on painful syndrome intensivity and organism reactiveness. One patients have tachycardia (as a rule, at hypersympathicotony), others – bradycardia (as a rule, at hyperparasympathicotony) as answer reaction to manipulation. Even such process as tooth preparing (in course of orthopedic treatment) in healthy

people may cause changes connected to organism individual features. All of these must be taken into account by dentist in his daily job.

MAXILLARY-FACIAL AREA ORGANS INNERVATION

Trigeminal nerve, its second and third rami (maxillary and mandibular nerves correspondingly) represent common sensory nerve for oral cavity (lips and teeth in part) as well as tongue anterior 2/3. Branches forming dental plexuses leave them and give rami to the tooth pulp, peridentium and gum. Cheek and gum mucosa is innervated by buccal nerve in superior teeth area. Palatal mucosa is innervated by palatal and nasal-palatal nerves moving away from pterygoid-palatine node. Mucosa of oral cavity floor and gum is innervated by lingual nerve in mandibular area. Glossal-pharyngeal nerve together with superior laryngeal nerve branches (vagal nerve) innervates tongue back mucosa. Mainly myelinized nervous fascicles penetrate root pulp through tooth root apical foramen. Their multiple branching is observed in pulp root part. Nervous fibers net is the vastest under odontoblasts layer where subodontoblastic nervous plexus is formed. Myelin-free nervous fibers leave this plexus, pass through odontoblasts layer and penetrate dentin as bushes. Peridentium innervation is performed by double way. First, myelin nervous fibers penetrate into peridentium and are impeded both into fibrous fibers fascicles and into connective tissue layers in the root apical third area. These fibers part comes alongside the periodontal fissure. Second, innervation in the peridentium middle and near-cervical thirds is realized by nervous fibers penetrating from alveole osseal walls. Peridentium on its whole course (from circular ligament till the tooth root apex) has dissemination of many free sensory nervous endings various by their structure. Big amount of such endings is determined in peridentium near-cervical part, significantly less – in the tooth circular ligament.

Chapter 2. FUNCTIONAL METHODS USED IN DENTISTRY

\Linguodiagnostics

Linguodiagnostics is a pathological process assessment in corresponding organ according to tongue appearence, covering on it, relief. Taking into account tongue appearence (its consistence, movement, colour), tongue coating assessment (on colour, thickness, shape, character: humid, dry), surface relief (smooth, tubercular, coated by

small papillary infoldings) one can receive the information characterizing inner organs functional state.

Under norm tongue is: flexible, soft, red, covered by thin white tunic (coating or bloom). Pale, white tongue is often met at: anaemias of different aethiology, chronic enterocolitis. Bright red tongue is characteristics of: inflammatory processes, B_{12}-deficient anaemia (figure 21, appendix), chronic gastritis, pellagra (vitamin PP deficiency).

Grey and black tongue is a differential diagnostic criteria of:

- mercury intoxication (grey, asphalt-like);
- lead intoxication (black).

Black ("hairy") tongue – a disease characterizing by different-expressed tongue filiate papillas keratinization and hyperplasy (figure 22, appendix). It is rather seldom state appearing mainly in men, primarily in the middle and old age.

Tongue with tubercular surface relief (filia-formed papillas are in hypertrophia state, folia-formed ones are relief), so-called hypertrophic gastritis is met at: hyperacidic gastritis, normacidic gastritis.

Smooth tongue with elements of desquamation, epithelium atrophy is particularly connected with hypoacidic gastritis (figure 23, appendix).

Swelling (oedematic) tongue or with cracks, plicas can testify to water-salty exchange dysorders, that one can often see at: chronic pancreatitis, enteropathies.

Very often tongue covered by white coating is observed at: gastritis acuting, cholecistitis acuting, colitis acuting.

Yellow covering of different intensivity is a characteristic of: liver infections, viral pathology, candidosis (figure 24, appendix).

Grey or black covering is observed at: hypovitaminoses, chronic gastroenteropathies.

Hyper- and hypokeratinization focuses alternation takes place at desquamative glossitis (figure 25, appendix).

One should use the rule at linguodiagnostics:

- tongue examination at daily light;
- tongue investigation in relaxation state;
- preliminary taking into account the character of received food (tea, milk, coffee, medicines usage).

The investigated person to whom the linguodiagnostics is performed washes his mouth by water and shows his tongue. The investigator assesses tongue appearance and colour, its covering and relief.

Methods Characterizing Mastication Effectiveness

To calculate parodontium endurance and every tooth role in mastication there have been offered special tables, which got the name of static systems of calculating the mastication effectiveness (Flis, Omelchuk, Raschenko et al.; 2008). The degree of every tooth participation in the act of mastication is defined by a constant, expressed as percentage, in these tables. During tables charting the role of every tooth is measured by the value of:

- the masticatory and cutting surfaces,
- roots number,
- the distance at which they are located from the jaw angle.

There have been offered a couple of tables charted by one and the same principle.

In Ukraine, the static system of calculating mastication effectiveness, worked out be *N.I.Ahapov*, has gained ground (Table 9). N.I.Ahapov recognized the mastication effectiveness of the whole dental apparatus as 100%, and as a unit of mastication capacity and parodontium endurance he took the small incisor, comparing all other teeth with it. Thus, every tooth in the table has a constant mastication coefficient.

N.I.Ahapov amended the table, while recommending to take into account teeth-antagonists at calculating the mastication effectiveness, and at their absence – counting as 0%.

Table 9

Mastication Coefficients of Teeth (according to N.I.Ahapov)

Teeth	Teeth	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**Total**
Mastication coefficient, %	Upper jaw	2	1	3	4	4	6	5	-	25%
Mastication coefficient, %	Lower jaw	1	2	3	4	4	6	5	-	25%

In N.I.Ahapov system the value of every tooth is constant and does not depend on the condition of its parodontium. For example, the role of a canine tooth in mastication is always determined by one and the same coefficient, no matter if it is stable or pathologically mobile. This is a serious drawback of the system.

There have been attempts to compile new statistic systems, in which the endurance of parodontium to mastication pressure would also depend on the degree of parodontium affection. Thus, I.M.Oksman based the offered system of calculating the mastication effectiveness of the dental system on the anatomico-physiological principle. Estimation is given to every tooth, even the 3rd molar. At that, the area of the masticatory or cutting surface, the number of tubercles, roots, parodontium peculiarities and the

presence of the last tooth in the dentition are taken into account. The lower and upper lateral incisors as weaker in functional aspect are taken as a unit. The upper central incisors and canine teeth are taken as two units, premolars – three, the 1st molars – six, 2nd – five, 3rd upper molars – as three, lower – four units. As a result calculation a corresponding table has been charted (Table 10).

Table 10

Mastication Coefficients of Teeth (according to I.M.Oksman)

Teeth	Teeth	1	2	3	4	5	6	7	8	Total
Mastication coefficient, %	Upper jaw	2	1	2	3	3	6	5	3	25%
Mastication coefficient, %	Lower jaw	1	1	2	3	3	6	5	4	25%

Except for the anatomico-topographic peculiarities of every tooth, I.M.Oksman recommends taking into account its functional value in connection with parodontium affection. Therefore at the mobility of the 1st degree teeth are to be estimated as normal, at the mobility of the 3rd degree they are to be considered absent. Also, single-rooted teeth with evident signs of apical chronic or acute periodontitis are to be estimated. Carious teeth, which should be filled, are referred to valid, and those with destroyed crown – absent.

Calculating the mastication effectiveness of the dental apparatus by I.M.Oksman is more expedient than by N.I.Ahapov, as it takes into account the value of every tooth not only in compliance with its anatomico-topographic data, but also with its functional peculiarities.

V.Y.Kurliandsky offered a static system of calculating the condition of the supportive apparatus of teeth, named by him a ***parodontogram***. It is obtained by means of putting the data about every tooth into a special scheme. As well as in other static schemes, in a parodontogram every tooth with unaffected parodotium is given a conditioned coefficient (Table 11). The difference between it and I.M.Oksman's and N.I.Ahapov's tables lies in the fact that conditional coefficients are derived not from anatomico-topographic data, but on the basis of the gnathodynamometric data of Haber.

Table 11

Coefficients of Parodontium Endurance to Load

Upper jaw	1	1	2	2	3	3	54	45	76	67	8	8
Lower jaw			21	12	3	3	54	45	76	67	8	8

Coefficient	1,25	1,25	1,0	1,0	1,5	1,5	1,75	1,75	3,0	3,0	2,0	2,0

The parodontogram not only reflects a comprehensive picture of parodontium affection (at alveolar process atrophy in part) but also allows outlining a plan of prosthetics and prophylaxis of further destruction of the dentognathic apparatus. Parodontium endurance co-efficients are derived on the basis of Haber's data, obtained more than 50 years ago. As is well known, this method takes into account only the endurance of parodontium to vertical load, which is absolutely insufficient for the characteristics of parodontium shock-absorbing capability. Haber's data, in addition, raise doubts, as the supporting apparatus of teeth is allotted with a very big summary endurance (1408 kg).

Parodontium endurance coefficients, as many biological characteristic, have considerable variability. They can not be characterized by mean values, obtained as a result of insignificant number of measurements. Proving (V.A.Naumov) has shown that the precervical third of tooth has the biggest area, the apical – the smallest. Molars are an exception in this rule, as in them the middle third has the biggest surface, followed by the apical and only then by the precervical. Thus, parodontium capability of taking up mastication pressure is not identical at different levels.

Static methods appeared not to be acceptable for detecting the degree of mastication effectiveness derangement, and not only because they insufficiently precisely characterize the role of every tooth in mastication and taking up mastication pressure, but also because they do not take into account:

- the occlusion type,
- mastication intensity,
- mastication pressure force,
- saliva influence and
- the role of the tongue in the mechanism of bolus formation.

As a conclusion, stathic methods - are based on coefficient determining for each tooth. This coefficient determines the destiny of its participation in mastication processes. If to take dental row masticatory effectiveness of a healthy person as 100% and for the unit of masticatory ability – small incisive tooth, then every tooth will have its coefficient. Dental row half on every jaw performs 25% of work under mastication conditions. At masticatory effectiveness determining one should exclude not only absent teeth but their antagonists too. Masticatory coefficient is expressed in per cents on formula:

5 6 4 4 3 1 2 1 2 1 3 4 4 6 5 -a

7 6 5 4 3 2 1 1 1 2 3 4 5 6 7 -b

5 6 4 4 3 1 2 1 2 1 3 4 6 5 -a

where a – masticatory coefficients, b – order teeth number

At mastication decreasing on 40% - it is a limit after which there are digestion process disorders. It serves as absolute evidence for denturing.

Thereby, to take into account the influence of all the mentioned factors there were offered functional (mastication) tests, which allow getting a more correct idea of mastication function.

Helman's probe

S.E.Helman worked out and simplified the mastication test technique. Instead of hazelnut he took almond weighing 5 g and offered a patient to chew it during 50 seconds. To the product, which might be used in the mastication test, certain demands were made. Particles, formed after mastication, should not dissolve in saliva, contract in volume after drying on water bath, and stick together. These requirements were substantially met by almond, which was offered for the purpose.

Mastication act physiology investigation under norm and at teeth loss gives the possibility to follow mastication function variation under different irritative agents action and at dental rows different defects. Jaws structural features and dental archs form directly depend on their function.

Mastication effectiveness determining method is used in clinical orthopedic practice for: diagnosis making, denture construction choosing, treatment quality analysis as well for scientific investigations.

Such method was first proposed by S.I.Helman's, then it was modified by *I.S.Rubinov's* which demonstrated reflectory acts significant role in food processing in oral cavity. This probe determines masticatory apparatus effectiveness. One proposes to masticate 0,8 g of forest nuts and to determine swallowing reflex time. Positive dynamics is characterized by swallowing appearance time shortening.

One should use for work: nut, Petry cup, glass, watering can, gauze, sieve with foramens diameters in 2, 4 mm, scales, second watches, sand bath.

The Helman's probe performance. To scale one nut. The investigated person takes it in his mouth and with the signal "to start" begins to masticate. In 30 sec with the signal "to stop" the mastication is stopped. Masticated mass is spitted, mouth is washed by water, which is spitted in the same cup. Cup content is strained through gauze, dry into sand bath, then sow through sieve. Non-sowed residues are scaled.

Formula: X= (Px100)/H, where

H- initial nut weight;

P - residue weight;

X –mastication disorder per cent

Mastication effectiveness (ME) is determined by means of subtraction of masticatory function disorder per cent from 100.

ME=100-X

Gnathodynamometry

For *parodont resiliency* determining to pressure and *masticatory muscles force determining* one can use gnatodynamometry method that is performed by special apparatuses– gnatodynamometers. They have plates for teeth through which pressure is transmitted to spring while mouth closing. This pressure is registered on the scale. It was established that frontal teeth resiliency is approximately equal to 60 kg, masticatory ones – 180 kg. Parodont resiliency depends on masticatory muscles and parodont individual development, their functional state according to gender, age et al.

The most widely-spread ciphras for parodont resiliency is follows as:

For men

Dental formula	1 2 3 4 5 6 7 8
Maxilla	12 7 17 21 22 37 34 21 in total 342 kg
Mandible	7 7 17 21 22 37 34 21 in total 332 kg

For women

Maxilla	8 5 12 15 16 27 24 16 in total 244 kg
Mandible	5 5 12 15 16 27 24 15 in total 238 kg

Both in men and in women parodont resiliency of symmetrically located teeth is equal with exception for superior premolars (left – 27, right – 25 kg).

Masticatory movements repeating in a definite order as a result of which there occur food bite, reducing to fragments, wearing out and food piece forming are in a composition of so-called *masticatory cycle.* Under resting state mandibule usually is lowered and dental rows are diverged so that between first superior and inferior incisivi was a space in 1-6 mm. Masticatory muscles are relaxed and stretched at this.

Muscular stretching is accompanied by constantly acting proprioreceptors irritation that reflectorily causes different muscular groups tonic contraction. As a result of this mandible can save for long definite orientation as for the maxilla. Such localization of jaws one as for another is an initial and it can seen as protective reflex. Food bite and mastication is performed at mandibular and maxillary teeth occlusion. Mandible in course of mastication does rhythmical movements in 3 main directions oriented vertically (up till occlusion and down over the distance of 40-50 mm from superior dental row), sagitally (forward on 5-15 mm and at usual mastication 2-3 mm

ahead), transversally - on the right and on the left. All mandible movements in all directions are accompanied by simultaneous sliding and rotation of articulational heads.

Masticacyography

Masticacyography is mandible masticatory movements assessment method.

Method *principle* is in air fluctuation registrating in a closed system in course of mandible movements.

They use for masticacyogram record: gum cuff, three-way adaptor, squeezing, Marey's capsule, kymograph, nut.

Gum cuff is placed under mandible while its fixating on a head. They open squeezing through gum tube, flow the cuff, connecting it with Marey's capsule and write masticacyogram on kymograph while food mastication. The registration is begun at the moment of food introduction in mouth and finish in a swallowing moment.

Masticacyogram consists of masticatory waves curves or oscillations and is represented on the figure 26.

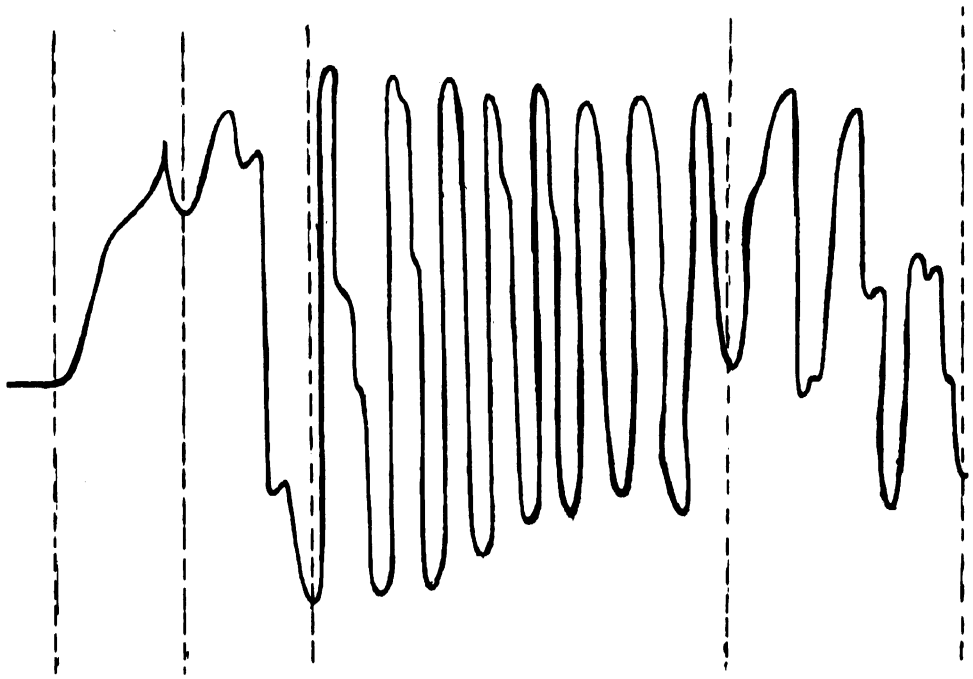

Figure 26. Masticacyogram.

Feeding act phases:

- I-st phase – of rest.
- 2-nd phase – of feeding.
- 3-rd phase – oriented mastication.
- 4-th phase – main mastication phase.
- 5-th phase – feeding piece forming and swallowing.

Masticacyogram significance

One can determine due to it: masticatory cycle time before swallowing, separate phases duration, masticatory movements number, mouth opening amptitude.

For example, mastication time under norm is 14 seconds. One can see this ciphra on masticacyogram. But reason that caused mastication cycle and its separate phases disorder and their change by means of this method is impossible to be determined.

Electromyography

EMG APPLICATION IN DENTISTRY DIFFERENT BRANCHES

EMG can be applied for secreted saliva amount assessment (Nederkoorn; Smulders; Jansen, 1999).

Local electromyography is used in surgical dentistry at masticatory (chewing) muscles dystrophies and hypertrophies; in stomatoneurology – at traumatic and infectious injuries of maxillary-facial region nerves; in children dentistry – for determining the soft palate muscles bioelectrical activity in children under norm and congenital developmental anomalies.

Stimulatory electromyography is used in stomatoneurology and surgical dentistry at face nerve injuries for its conduction and impulses spreading velocity through the nerve determining as well as for the assessment of expression muscles paresis degree.

Interferential electromyography has the biggest spreading in various dentistry branches. For example, it is used in therapeutical dentistry for the registration of masticatory muscles contraction force regulation at parodontites because there are functional-dynamic disturbances of masticatory apparatus at this disease. It is usually performed in parallel with gnatodynamometry for the assessment of mandibular (lower jaw) force during chewing. In surgical dentistry interferential EMG is used at:

- jaws fractures;
- maxillary-facial region inflammatory diseases (phlegmones, abscesses, osteomyelitis);
- during myoplastic operations at expression muscles pareses.

In orthopedic dentistry this method is applied for study of bioelectrical activity of chewing muscles under the condition of complete teeth absence in course of adaptation to the new removable dentures. Interferential EMG is used in children dentistry for

zygomatic and masticatory muscles coordinating correlations reorganization control at bite anomalies treatment.

EMG application in therapeutical dentistry

EMG investigations are performed at parodontitis for registrating the changings of masticatory musculature contractive force regulation because masticatory apparatus functional-dynamic disorders are observed at this widely spread dental disease. It is performed with gnatho-dynamometric probes (mandible force determining) which allow to know about correlation between muscles excitement intensivity and their force effects. There are disorders of correct alternations of bioelectrical activity and bioelectrical resting periods during mastication in patients with inflammatory-dystrophic parodontitis and with periodontitis.

EMG application in surgical dentistry

All mentioned below 3 EMG types are applied during operations. Global or interferential EMG is used at jaws fractures, mandibular-facial region inflammatory processes (phlegmones, abscesses, periostitis, osteomyelitis), during myoplastic operations at tongue and mimic musculature strong paralyses. The local one – at masticatory muscles dystrophies and hypertrophies; in stomato-neurology during mandibular-facial region nerves traumatic and infectious injuries. The stimulational one – in stomato-neurology and surgical dentistry at facial nerve determining for its conductance and excitement velocity distribution determining as well as for mimic musculature paresis degree assessment.

EMG application in prosthetic dentistry

Interferential EMG is applied for masticatory muscles bioelectrical activity assessment at teeth complete absence during adaptation to complete temporary dentures. Denturing with complete temporary dentures leads to masticatory muscles bioelectrical activity increasing during mastication with dentures and after their removal. During adaptation to complete temporary dentures all mastication time is reduced due to masticatory movements quantity decreasing and one masticatory movement time shortage. Masticatory muscles adaptation to new conditions on EMG indexes takes place during first 6 months of dentures usage. Pathological wiping off with EMG bite hightening admitted boarders are under control at bite height increasing after orthopedic treatment of teeth. Central occlusion height increasing in admitted limits (8-10 mm) leads to temporal muscles resting bioelectrical activity. Such an activity appearance in proper masticatory muscles is a symptom of excessive (more than 10 mm) bite increasing. EMG investigation allows objective assessing the effectiveness of occlusion straightening out. It is also allow to control symmetrical muscles activity co-ordination.

EMG application in children dentistry and orthodonthia

Interferential EMG is applied for control of temporal and masticatory muscles functions co-ordinational correlations re-organization during bite anomalies treatment. The local one – for soft palate muscles bioelectrical activity study in children under norm and at congenital developmental anomalies. After soft palate fissures operative removal EMG is applied for operative determining the possibility of speech restoration prognosis as well as for control during muscles special trainings with myogymnastic exercises special complex.

Electromyography method principle

It is a functional method that allows registering graphically the electrical muscular activity at the muscles excitement. With other words, it is the registration method of skeletal muscles electrical potential oscillations occurrence under rest, at tonic tension and arbitrary movements. At muscular contractions visual observation under muscle or nerve irritation one tells about electrical excitability investigation.

EMG allows studying the structure and function of neuro-motor apparatus consisting of functional elements named as motor units (MU). MU is an integrity of motoneuron and muscular fibers group innervated by it. On EMG potentials oscillations in neuro-muscular endings (motor plates) occurring under impulses action from medulla oblongata and spine motors are fixed. EMG record is performed at paper and tape movement velocity equal to 4-5 cm/sec and 20 cm/sec for oscillations quantity estimation.

The curve receiving at this method usage is named electromyogram. It is the result of interference of multiply action potential that appears asynchronically in different muscular fibers and is registered by means of intracellular electrodes.

There are 3 main electromyogram kinds:

- interferential – muscular biopotentials are taken off from large surface while applying the electrodes on skin;
- local – separate motor units activity is registered by means of needle electrodes;
- stimulatory – the registration of electrical muscle answer to the stimulation of nerve innervating it.

At EMG with needle electrodes it is necessary to make EMG record under rest and at the investigated muscles weak tension. If it is necersary one can investigate muscles reaction to nerve electrical irritation or doctor can apply dosated muscular loading, reaction to fatigue and different pharmacological substances (for example, proserin, at myasthenia).

Muscular potentials bringing out is performed by means of electrodes:

- needle – they are involved in the muscle and bioelectric potentials of separate muscular fibers registration;
- surface – they register summary muscles activity from many muscular fibers.

At EMG analysis one should take into account:

- altitudes level;
- potentials oscillations frequency;
- common oscillograms structure (osclillations monotony or division into volleys, volleys form, duration and frequency).

Parameters of motor units are different because non-equal amount of muscular fibers are included in motor unit. That is why for taking information about state of motor unit of a given muscle it is essential to register not less than 20 action potentials. Action potential duration is 5-13 msec. Under norma in a resting state (at local bringing out with needle electrodes) the bioelectrical potentials oscillations don't increase (at summary EMG one can see low-altituded weak oscillations up to 10-15 mcV). Reflectory tonus increasing is accompanied by insignificant rising up of electrical activity (up to 50-100 mcV). At arbitrary tension frequent high oscillations (1000-2000 mcV) are occured. In healthy people summary EMG dependently on muscular force has altitude up to 400 mcV and frequency up to 400 fluctuations per 1 second.

EMG have different picture at movement disorders which are connected with anomalies of central and peripheral nervous system and muscular apparatus as itself. The muscular bioelectrical activity changes are delt with pathological process topics, severity and course stages. EMG helps at diagnostics of central, segmentary, neurithic and myopathic motor disturbances, it helps to determine typical disorders at early disease stage under conditions of low-expressed symptoms. It also gives the opportunity to observe process dynamics and treatment effectiveness. Sometimes in neurological practice they use ***electroneuromyography*** – complex investigation method including in it:

- registration and analysis of muscles and nerves stimulated potentials parameters (stimulated potentials parameters latent period, form, altitude and duration);
- functioning motor units determining;
- impulses transmission velocity through peripheral nerves motor and sensory fibers et al.

At interpherential EMG one should determine such parameters as:

- altitude;
- duration and
- temporary course of bioelectrical activity during functional probes;
- symmetrical muscles activity correlation;
- activity distribution in muscles of one and different groups.

Acquaintance with EMG qualitative analysis

Qualitative EMG analysis – EMG character describing:

- saturated;
- non-saturated;
- EMG rounding curve character – activity slow or sharp increasing and decreasing;
- activity phases number.

Acquaintance with EMG quantitative analysis

This analysis includes:

- activity and rest phases duration;
- temporary intervals between activity beginning in different muscles;
- total electrical muscular activity level (the most important parameter) – is determined by EMG oscillations altitudes measurement and by means of special devices. Moda (the most common oscillations numeral, number that are repeated the mostly often in the variation row) is usually taken as the level of summary EMG oscillations altitude. It's necessary to measure all main oscillations during definite time period (for example, for 0,5 sec) and to determine the altitude meaning the most often meets from peak to peak. The second way of summary oscillations altitude assessment is to measure 10 most expressed oscillations at a definite time period with farther estimation of their middle meaning. Then the altitude of this section must be compared with proper meaning of calibrating signal and EMG altitude received must be expressed in mcV. The received EMG summary altitude is a conditional quantity but very important because it's a proportional to (it's correlated to) isometric muscular contraction intensivity at any assessment way.
- Oscillations frequency – under norm is great (100 oscillations per second) and isn't connected with muscular contraction force. Thus, EMG looks like saturated one. In such cases EMG is not analysed.

Examples of EMG are given on the figures 27-29.

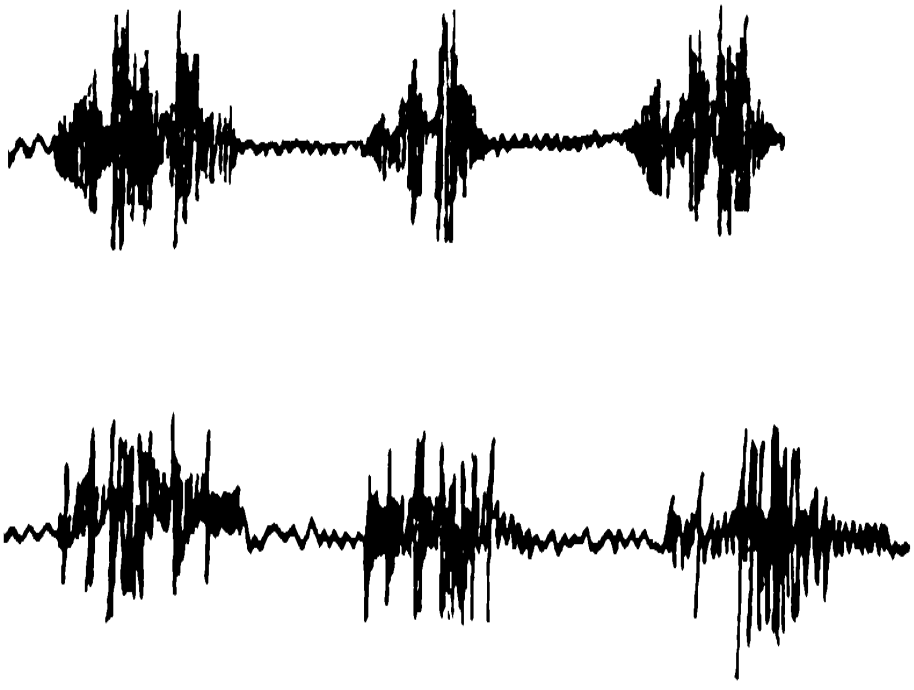

Figure 27. Masticatory muscles EMG (usual one-sided mastication).

Figure 28. Scheme of EMG summary amplitude estimation. Horizontal lines run on peaks of most frequent amplitudes; vertical line is summary amplitude.

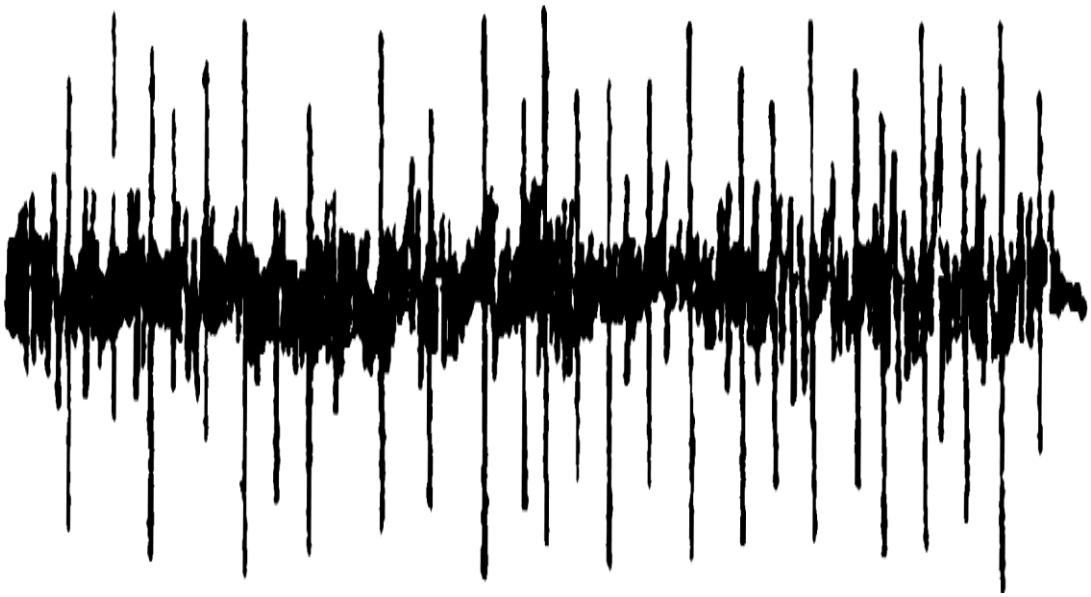

Figure 29. Non-saturated EMG (it is possible to estimate frequency and altitude on it).

Chronaxymetry

Current is coming also through nervous fibers located in it at electrical current application on muscle. That is why by chronaxy level determining one can tell about motor nerve fibers injury. Irritation threshold (rheobase) and chronaxy of nervous fibers are lower than in muscles. That is why at normal muscle chronaxy determining we in fact measure chronaxy of nervous fiber innervating it. And that is why the excitement occurs first in the nervous fibers and than are transmitted to the muscle at muscular irritation.

If nerve is injured than nervous fiber is degenerated and under such conditions electrical stimulus applying to the muscle shows muscular fibers chronaxy (such a chronaxy is longer in time).

Chronaxy and rheobase indexes are in oppositely-proportional dependence on the tissue excitability level. They can vary significantly at neuritis and neuralgias of trigeminal and facial nerves as well as at myosites of mimic and masticatory musculature.

Besides, excitement conductance velocity through the peripheral nerves are lowered significantly at neuritis and polyneuritis of different ethiology that allows the nerves damage gravity and level determining.

Chronaxymetry is widely used in dentistry nowadays.

Methods with electrical current usage

Electroodontodiagnostics (EOD)

Electrical current acts to pulp through enamel and dentin. It is easily and exactly to be dosated. Current does not damage tooth pulp. That is why it can be applied many times. Tooth electroexcitability assessment is in fact the investigation of corresponding sensitive nerves and tooth pulp.

Electroodontodiagnostics represents a current applying for the tooth pulp excitability determining with a diagnostic aim. The tooth reaction to the electrical irritation gives an opportunity to determine its electrical excitability changes specific picture at various pathological processes.

It has been detected that healthy teeth have equal excitability and react to current force 2-6 mcA independently on their grouped dependence. If a tooth threshold is less than 2 mcA it testifies to hyperexcitability (at parodontosis). At pulpits on the contrary nervous fiber threshold is bigger than the threshold of muscle and is more than 6 mcA. Excitability reducing up to 100-200 mcA is pulp death sign. Parodont receptors act under such conditions.

Electroodontodiagnostics is a leading investigative method at the pathological states majority. It is so because it allows:

- testify about pulp damage degree;
- follow the pathological process dynamics;
- control the treatment prognosis and
- forecast the disease result.

Oral mucosa is highly-sensitive to electrical current because it possesses good electrical conductivity. It is defined by:

- its abundant blood supply;
- corneal layer absence;
- tissues big hydrophyly.

One can use potentials leads from body surface in clinical practice. The records received are called correspondingly to the potentials origin: electrocardiogram (ECG), electroencephalogram (EEG), electromyogram (MG) and so on.

It is known from the first Galwani experiment that different-named metals are so-called galvanic current origin which can irritate alive tissues. It must be taken into account by dentist while denturing and teeth filling with different-origined metals (gold, unrusted steel, amalgames) acting as electrodes; saliva is an electrolyte. Metals ions release to the saliva creates the condition for different-sized microcurrents appearance in oral cavity.

The appearing current force depends on:

- saliva pH,
- metallic surface state,
- metallic dentures quality,
- their distance between each other.

Tooth solid tissues electrical features determining is performed in dental practice for acute and chronic pulpitis diagnostics. This methodics is rather complicated. It requires measurements taking into account. They are of individual peculiarities of teeth morphological shape and geometric sizes. Also there must be obligatory following the most possible parameters stimulus.

Nowadays one uses also possibility of oral mucosa biopotentials measurement for its functional state assessment. There was detecting the summary biopotentials age dynamics as well as their level change at parodontosis, oral mucosa diseases which is of important diagnostic value.

Dentist can touch with potentials occurrence between similar metals (for instance, amalgame) of different content or between crowns made of the same metal if there is metal filling under them.

Appearing microcurrents can be the reason of such a phenomenon named as *galvanism*. Galvanic currents appearing in oral cavity define increased irritability of taste reception and gustatory sensations some perversion at different metals presence. Sometimes pathological process is developed in years after denturing. It depends on the patient individual reactivity.

Galvanism clinical symptoms are rather different. They can be divided into two big groups: subjective complaints which occur directly right after metallic fillings and crowns fixation in oral cavity. “Metallic taste” and some others belong to them. They are usually stopped in several days. Complaints which are appeared in prolonged time (sometimes in several years) belong to other group: metallic taste, pain.

Galvanism the most frequent symptoms are as follows as:

- oral mucosa permanent burning of different location (80%);
- metallic and sour taste appearing usually in 3-5 months after denturing (70%);
- salivation disturbances (58%);
- headache (47%);

- insomnia (19%);
- pain in abdomen area (8%);
- vomiting (3%);
- sparks into eyes (1%).

As a rule, several symptoms appear at once. Patient sometimes can not determine them distinctly but has a discomfort.

Oral mucosa chronic inflammation can be developed:

- reddish color,
- tongue papillas swelling,
- erosions and
- ulcers.

Microelements and metals ions big quantity will pass into oral cavity and saliva from the metals (especially from the gold) as a result of electrical-chemical processes. Local inflammatory processes will develop as a result of their toxic action to oral mucosa receptor apparatus. Taste sensitivity to the sweet, sour and salty is lowered and distorted. It can lead to food mechanic and chemical processing disorder as well as to speech disturbance. Besides, there can be chronic gastral-intestinal diseases acuting at such a saliva passage into alimentary tract and saliva microelements action to stomach and intestine mucosa.

Current force appearing between different-origined metals correlates to the subjective complaints degree. Galvanism is strongly expressed at force 80 mcA while it is weak at 25-80 mcA and the complaints are practically absent at 5 mcA. Galvanism phenomena disappear after different-natured metals changing to the one-natured.

Electrical current is applied in dentistry also for treatment.

Galwanization

Galvanization represents continuous constant current usage of low tension (30-80 V) and little force (up to 50 mcA).

Constant current action to oral cavity mucosa leads to:

- vessels dilation,
- blood stream acceleration,
- vascular wall increase,
- hyperemy,
- temperature rising.

These reactions encourage local metabolism activation as well as the one of epithelium and connective tissue. The receptors irritation in the action zone leads to their excitability changing. Afferent impulsation causes local, segmentary and generalized reflectory reactions into CNS that leads to the inner organs functions changing such as arterial pressure, heart contractions rate et al.

Medical electrophoresis represents a method of medicines injection into the teeth tissues.

It is the most widely-spread galvanization form.

Three phenomena are on the basement of electrophoresis method:

- electrolytic dissociation,
- electrolysis,
- electrical osmosis.

This curative method effectiveness is rised significantly due to the combined action of constant current and medicines ions (particles) injected by means of it.

Electrophoresis possesses following advantages comparatively to other physio-therapeutic methods:

- the injected medicine saves its specific action in the organism and usually does not cause general toxic action;
- electrophoresis allows injecting one or several substances at once to any place by its location and size;
- medicines ions depot is created under the active electrode in the skin thickness (in the depth up to 3-5 mm) and these ions are present in the organism longer;
- medicines ions are excreted slowly from the organism;
- electrophoresis does not disturb tissues physiological life activity in a given area;
- electrophoresis gives an opportunity of local stimulation at the pathological focus superficial location;
- the injected medicine quantity can be dosated by changing the electrode size, solution concentration, current force as well as the action duration;
- the medicine can be excreted from the organism to the hydrophilic pad while changing the constant current polarity.

There can be root canals electrophoresis. It is applied at tooth caries (decay). Some drops of the applied medicine, then cotton-wool globule washed with the same solution is introduced into the cavity after the decay cavity mechanic and medical processing as well as the pulp chamber washing-up. A thin isolated cable end is placed between cotton-wool and the cavity wall. The tooth cavity is closed by wax (for its hermetization and the electrodes fixating). The second electrode with a pad 6x8 cm is fixed to the forearm. Current force is up to 2 mA, the session time is 10-20 min. It is necessary to perform daily.

Gums electrophoresis. A special gingival electrode consisting of basin-like can and lead plate with a thin cabel molded to its middle in chlorvynil isolation is covered with a gauze from the concave surface. The gauze is folded up into 6-8 layers and is washed by the medicine. This gauze is placed directly to the gum mucosa. The second electrode with a pad is placed to the forearm. Current force is 2-4 mA, the session duration is 20 min. It is necessary to perform daily.

Temporal-mandibular joints area electrophoresis. The electrodes with two pads are placed to two temporal-mandibular joints. Current force is up to 5 mcA, the session duration is 15-20 min. It is necessary to perform daily.

Finally, constant electrical current is used for noceoceptive sensations prevention at various stomatological manipulations. Constant current anesthesizing action is delt to

electrotone phenomena development in the tissues causing their excitability changing at current passage. Excitability is increased under catod (catelectrotone) while its decreasing under anode (anelectrotone). There is a phenomenon of Verigo's cathodic depression – excitability is decreased under cathode too at current durable passage.

Methods of blood supply assessment

Rheodentogram

This method reflects circulation state in pulp under physiological and pathological conditions.

Rheoparodontogram – demonstrates parodont vessels structural changings and functional state in course of parodontitis, gingivites, treatment ways and methods assessment. Its configuration is changed in course of these diseases,

Oral mucosa rheogram – under norm very weak low-frequented oscillations are registered. In course of chronic recidivating aphthosis rheogram altitude isn't changed but rheogram configuration character is changed.

Rheoparodontogram

For rheoparodontogram registration dental devices are put on corresponding areas of alveolar process.

In healthy people one can see non-similarity of rheoparodontogram shape at different parodontal areas.

Incisors characteristics:

- steep ascendance,
- sharp apex,
- steep descendance,
- expressed incisura.

Molars characteristics:

- gently sloping ascendant,
- apex like plataux or blunt angle,
- non-distinct incisura expressiveness,
- descendant gently sloping.

Canines and premolars features:

- rheogram has intermediate character between mentioned above.

Rheographic index:

- for incisors – 5,3-6,3,
- canines – 6,3-7,0,
- premolars – 5,6-6,4,
- molars – 6,3-8,0.

Parodontogram ascendance average duration:

- for incisors – 0,13-0,15,
- canines - 0,15-0,19,
- premolars – 0,14-0,18,
- molars – 0,17-0,20 sec.

Descendant duration:

- incisors – 0,72 sec,
- 0,80 sec – molars.

Rheoparodontogram can be changed in course of *pathological conditions in parodont*:

- curve apex is like blunt angle;
- incisura and dicrote are expressed non-distictly and are replaced to its apex;
- descendant duration is increased;
- rheographic index is reduced;
- ascendance duration is increased;
- two last features testify to local blood circulation disorders.

Rheodentogram

For rheodentogram registration electrodes are fixed on mucosa from vestibular side (active) and from palatal side alongside root of the examined tooth (passive electrode). Rheodentogram reflects objectively blood stream state in pulpal area under physiological and pathological conditions.

In course of pulpitis rheodentogram altitude is increased (figure 32). Intact tooth RDG is shown on the figures 30 and 31. Atherosclerosis is shown on the figure 33.

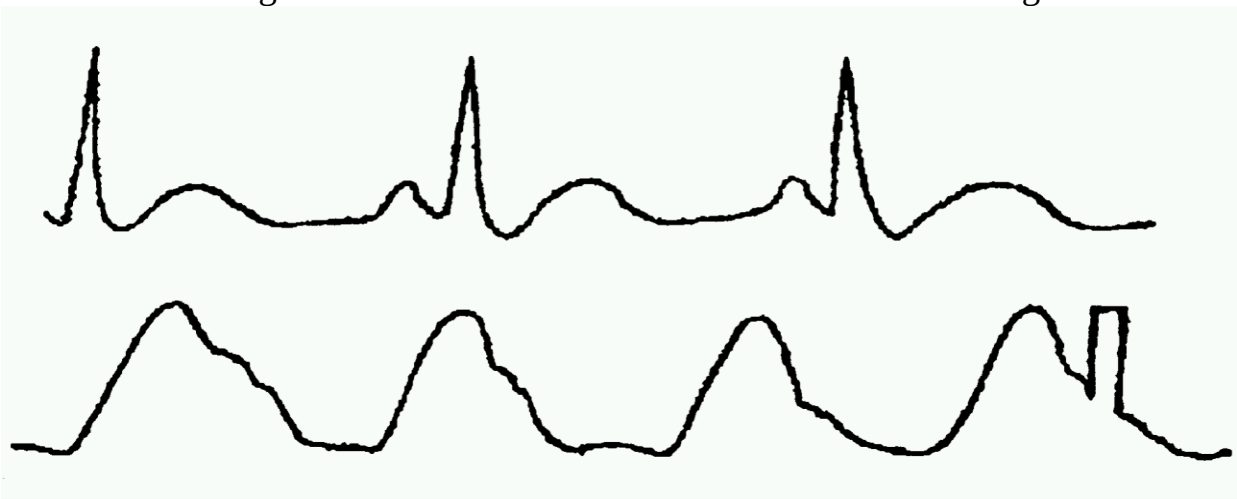

Figure 30. Intact tooth rheodentogram.The first wave – ECG.

The second wave – RDG.

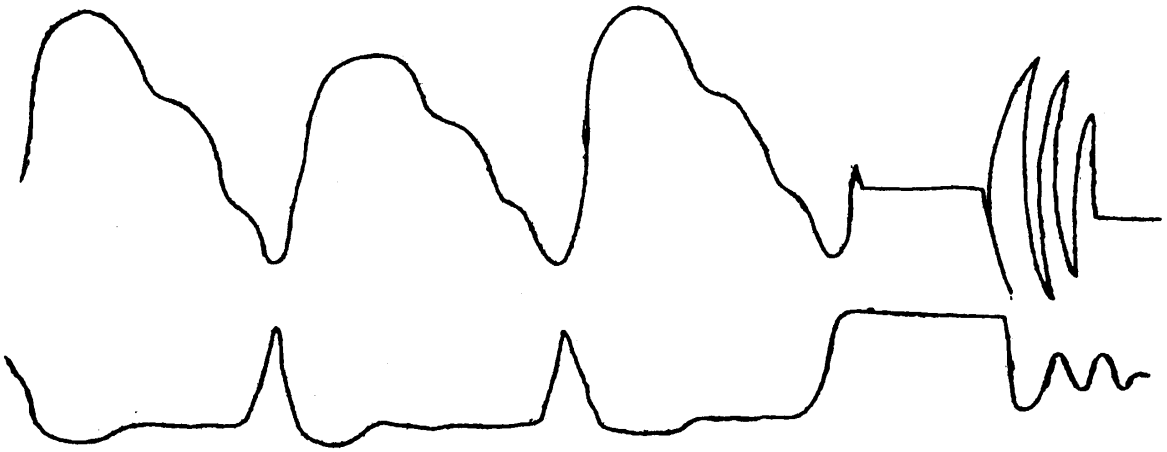

Figure 31. Rheoparodontogram of person with intact parodont.

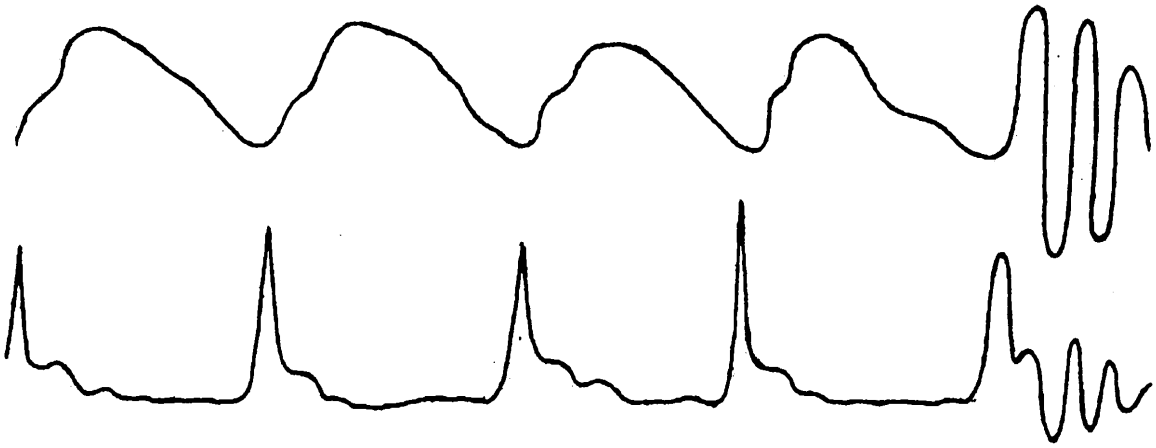

Figure 32. Rheoparodontogram of sick person with parodontitis of hard degree.

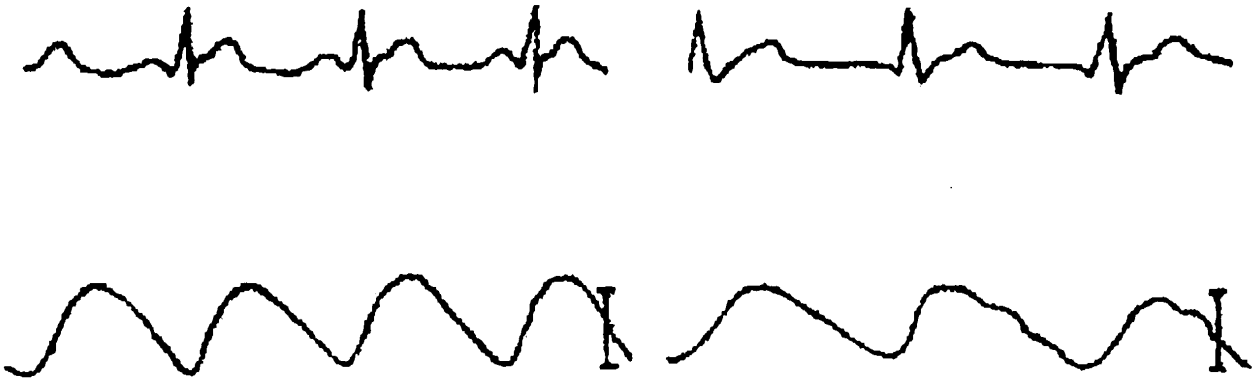

Figure 33. Rheoparodontogram at isolated (a) and generalized (b) atherosclerosis of parodontal vessels.

The first wave is ECG. The second wave is RPG. Calibrating signal is 0,1 Om.

Neurophysiological assessment of trigeminal nerve reflexes

The trigeminal nerve is one of the largest cranial nerves, sending fibers to innervate all muscles controlling jaw movements and the skin of virtually the whole face and skull. The trigeminal nerve and nuclei (the trigeminal complex) have interesting anatomical and physiological peculiarities. No Renshaw cells have been identified in the trigeminal motor nucleus and, therefore, trigeminal motoneurons have no recurrent inhibition. The trigeminal sensory nucleus is a complex structure, divided into many subnuclei extending from the mesencephalon to the spinal cord. One of the most striking anatomical peculiarities of the trigeminal complex is that the first order sensory neurons for proprioceptive information are located inside the central nervous system, in the mesencephalic nucleus, when comparable sensory neurons from other muscles in the body are located in the dorsal root ganglia, half covered only by the blood–brain barrier. Vibration, which causes a decrease of the H reflex in limb muscles, induces potentiation in trigeminal motoneurons. The size and connections of the bulbospinal sensory nucleus testify of the importance of the trigeminal neurons as a relay station for cutaneous somatosensory inputs in their way to higher order structures and to the integrative motor centers of the reticular formation. As it is the case with other cranial nerve nuclei, trigeminal motoneurons receive bilateral inputs from cortical neurons. They also receive ipsilateral projections from muscle spindle afferents, and bilateral projections from cutaneous afferents. Sensory inputs to the neurons of the mesencephalic nucleus come from muscle receptors of trigeminal muscles and, possibly, also from those of the extraocular muscles. Those to sensory neurons of the bulbospinal trigeminal nucleus come from cutaneous receptors, with a relatively well-defined somatotopic distribution. These neurons also receive modulatory inputs from the basal ganglia through the superior colliculus and the nucleus raphe magnus, but the exact relationship between the nuclei of the reticular formation and the neurons of the trigeminal sensory nucleus is still unclear.

The trigeminal complex and their central projections are the site of a variety of lesions that may or may not be accompanied by clinical manifestations. Clinical assessment of motor and sensory dysfunction in the trigeminal complex may be difficult. Motor dysfunction is only identifiable in severe lesions, there are no objective means for the assessment of sensation in the face, and the mandibular reflex is not usually accompanied by a visible movement of the chin. On the contrary, cutaneous reflexes are most helpful for clinical assessment. Typically, brainstem reflexes mediated by the trigeminal nerve are consensual, which means that unilateral stimulation leads to responses in both sides. Because of that, a defect in the afferent branch of the reflex gives rise to abnormalities in both sides while a defect in the efferent branch leads to abnormalities in the affected side to stimulation of either side. Radiological neuroimaging techniques may also not provide good resolution for topographical diagnosis within the brainstem.

For the reasons above, neurophysiological tests remain as most valuable methods to assess the functions and dysfunctions of the trigeminal nerve and nuclei. The

following is a review of the clinical applicability of brainstem reflexes mediated by the trigeminal nerve, such as the blink reflex, the corneal reflex, the masseteric inhibitory reflex, the mandibular reflex, and other neurophysiological responses that may help in understanding the pathophysiology of symptoms and signs of lesions involving the trigeminal complex. The clinical utility of trigeminal nerve reflexes is not supported by data originating on evidence-based medicine. However, this is a common situation in neurophysiology, in which the tests can be considered as a continuation of the clinical exam.

2. ***Methods***

This review is not intended to describe specific technical and methodological aspects of trigeminal reflexes. Therefore, only a short summary of them is included here. The reader interested in these aspects can find information in previously published reviews.

2.1. *Trigeminal nerve conduction studies*

Despite the fact that branches of the trigeminal nerve are accessible in many sites, the possibilities for the study of motor and sensory nerve conduction are only limited, and most electrodiagnostic studies rely on the assessment of reflex muscle responses. A few methods have been described to assess sensory nerve conduction in the mandibular branch of the trigeminal nerve, with consistent results in healthy subjects and still scarce demonstration of abnormalities in patients with neuropathic lesions. Recording requires needle electrodes inserted below the zygomatic arc at a depth sufficient to reach the foramen ovale (4–4.5 cm). Electrical stimulation can be applied to the mental nerve at the mental foramen or to other terminal branches of the mandibular division.

2.2. *Trigeminal-trigeminal reflexes*

The mandibular reflex, or jaw jerk, is the only monosynaptic reflex available to electrophysiological testing in the cranial and facial muscles. It is ordinarily elicited by a mechanical tap over the mandible with a reflex tendon hammer that electronically triggers the oscilloscopic sweep. The reflex responses are recorded simultaneously from the right and left masseter muscles using two pairs of surface electrodes, the active one placed over the muscle belly at the angle of the mandible, and the reference one placed over the mastoid process or the ear lobe. Since reflex latencies vary with successive trials, comparison of simultaneously recorded right-sided and left-sided responses is more meaningful than absolute values, which are of the order of 6–8 ms in normal subjects.

Stimulation of the stretch receptors induces not only the jaw jerk but also inhibitory effects that can be easily observed as a silent period when the stretch is applied during masseter contraction. The silent period to chin taps begins at about 10–12 ms and lasts for 20–40 ms, although latency and duration are both dependent partly on the strength of the tap and the level of sustained activation of the masseter muscles. The masseter inhibitory reflex (MIR) can be obtained by applying electrical stimulation to the mentalis nerve, which is a more standardized technique to examine reflex inhibitory circuits on trigeminal motoneurons. The electrical stimulation of the mental nerve

induces a twophase inhibition, MIR1 and MIR2. The latency of the MIR1 is of about 10–14 ms and the latency of the MIR2 is of about 40–50 ms. If the MIR is to be used for electrodiagnostic purposes, the subject must exert a stable background voluntary contraction, and the amount of inhibition should be quantified, which often requires rectification and averaging of EMG activity in a sufficient number of trials.

2.3. *Trigeminal-facial reflexes*

The best known and most commonly used of all brainstem reflexes is the blink reflex elicited by an electrical stimulus applied to the supraorbital nerve. This causes an involuntary closure of the eyelids, and the reflex response can be recorded with EMG electrodes over the orbicularis oculi muscles in the lower eyelid. Surface electrodes are used to apply single stimuli of an intensity 2–3 times the perception threshold to the supraorbital nerve over the supraorbital notch. Careful placement of the electrodes may help with reducing the stimulus artifact that could interfere with the appropriate assessment of response latencies. The response consists of two separate components: an early ipsilateral R1 response that appears at a latency of 10–13 ms, and a later bilateral R2 response that appears at a latency of 38–45 ms. Because of the differences in the circuitry of the R1 and R2 responses, an interesting possibility emerges from the study of the onset latency of the responses: The latency of the R1 depends more on the trigeminal and facial conduction times than on the intra-axial synaptic connectivity, with only 1 or two interneurons in the circuit.

The reverse occurs with regard to the R2, which latency is more dependent on interneuronal synapsis than on peripheral nerve conduction time. Therefore, a delay is observed predominantly in the R1 response in lesions involving the peripheral nerve and in the R2 response in lesions involving the trigeminal complex at the brainstem.

However, a delay in the R1 response can also be found in clinical manifest or silent pontine lesions in multiple sclerosis. The blink reflex can also be obtained by stimulation of infraorbital and mental nerves. The responses are less reliable and the R1 is not consistently obtained. However, they can be of help for the assessment of the site of the lesion in the trigeminal spinal nuclei in brainstem vascular lesions. Mechanical or electrical stimulation of the lips can induce the perioral reflex. Responses to mechanical stimuli, recorded with EMG surface electrodes placed on the orbicularis oris, reveal that the reflex is composed of a bilateral early response, at 11–18 ms, followed by a late response, at 24–45 ms. In adults, perioral responses are usually accompanied by responses in the orbicularis oculi at a similar latency. Although the reflex is of interest, interindividual variability and poor consistency has probably prevented a wider use of the perioral reflex. In infants, perioral reflexes can be used for the study of the physiological mechanisms underlying the sucking reflex.

2.4. *Trigeminal evoked potentials*

Evoked potentials to electrical stimulation of branches of the trigeminal nerve are difficult to obtain because of the artifact usually induced by the electrical stimulus. This was a technical impediment in the exam of possible trigeminal nerve conduction abnormalities in the posterior fossa. The scientists obtained reproducible responses with

needle stimulation of the infraorbital nerve. However, this invasive technique did not reach a widespread use. Nowadays, some of the methodological problems limiting the recording of trigeminal nerve evoked potentials have been overcome with the introduction of laser stimulation. A laser CO2 stimulus induces the activation of epidermal skin receptors of pain with no skin contact and little or no artifact. Laser-induced trigeminal nerve evoked potentials have been studied in normal subjects and in patients with a variety of disorders. The limitations of the technique lie in the fact that it permits to assess conduction in small fibers but not in large fibers and that the information comes from brain centers activated after some elaboration of sensory information.

2.5. *Other tests involving the activation of trigeminal neurones*

2.5.1. The blink reflex excitability recovery curve

While nerve conduction and synaptic connectivity between the trigeminal nerve and the facial nuclei can be assessed with the blink reflex, a single stimulus does not tell us much about the excitability of the whole circuit, which is regulated by inputs from various sources, including the basal ganglia. To test the excitability of the brainstem interneurons in the blink reflex pathway, Kimura devised the method of paired stimulation. Two stimuli of the same intensity are delivered. The first stimulus (conditioning) leaves a trace of excitability changes in some brainstem interneurons and induces a reflex response, whose size reflects the number and density of inputs reaching the facial motoneurons. The second stimulus (test), applied after a certain time period, goes through the same interneurons. However, their availability to transmit the inputs would have changed because of the change in excitability induced by the first stimulus. The excitability recovery curves are different for the early and late blink reflex responses probably because of the different number of synapses involved in the two of them. Therefore, an abnormality in the excitability recovery of the R2 component with no changes in that of the R1 component would indicate a disorder of the excitability of brainstem interneurons mediating the R2 component.

2.5.2. Prepulse inhibition of the blink reflex

The availability of interneurons of the blink reflex pathway to inputs from the supraorbital nerve is also transiently modified by the arrival at the brainstem of sensory inputs from different sources. The phenomenon of prepulse inhibition, which was described initially in relation to the startle reaction, consists in the inhibition of the response to a stimulus when this is preceded by a weak stimulus of the same or different modality at specific leading intervals. In the case of the blink reflex to supraorbital nerve electrical stimuli, the R1 response is enhanced or not modified, while the R2 is significantly suppressed when the electrical stimulus is preceded by low intensity acoustic stimuli or peripheral nerve electrical stimuli with an interval of 100 ms. An abnormal blink reflex inhibition by a prepulse may indicate an abnormal processing of sensory inputs at brainstem level.

Chapter 3. JAWS, ORAL MUCOSA AND SALIVARY GLANDS AGE PECULIARITIES

In a childhood

Osseal apparatus

Maxilla and mandible bones development begins in antenatal period.

Mandible

It consists of two halves united with connective tissue in antenatal period. Mandible uniting begins in neonatal period. It lasts till the end of the first year of life. Mandible has body and alveolar process up to this time. But alveolar process with dental follicles has weak development. Jaw ramus is wide and short, articular joint is located approximately at the level of alveolar process. Then rami get developed in parallel to jaw body and articular heads are formed. Mandible rami growth in length is accompanied by angle changing between them and jaw body: very blunt angle becomes more pointed in the adult. The changings are fluctuated in ranges 140-105°.

Maxilla

It is developed weak in new-born. It is short, wide and mainly consists of alveolar process with teeth follicles located in it.

Jaw bones of children are rich in organic substances and contain mineral substances less than the ones of the adult. It is the explanation of bigger elasticity and lower fragility of children bones. Children jaws bones have rich circulation, wide nutritive (Havers') canals of osteon, thin and tender structure of osseal joints. Red bone marrow in children is less resistant to different irritations than yellow bone marrow. Jaw bones epiosteum is thickened in a childhood.

Alveolar process

Alveolar process of maxilla and mandible gets developed synchronically to the teeth development and teething. Age sizes of jaw bones these parts are determined by teeth quantity and formation degree. Dental rows state gives clinical information about jaw bones growth. Jaws alveolar processes do not develop and do not grow at congenital adenty.

One can tell about 2 periods in jaw bones development.

1) Jaws accelerated growth in 4,5-6 years when jaw is prepared to frontal constant teeth teething. Spaces are formed between milky teeth because constant frontal teeth are bigger comparatively to milky ones.
2) It coincides to constant masticatory teeth development and teething and is characterized by jaw body growth in corresponding parts. This period is origined from 6 years and lasts till 12-13 years when the second molars are teethed. In 16-

18 years at third molars teething jaw growth is enforced in corresponding locus. Growth in vertical direction occurs simultaneously to the one in horizontal direction: body is increased, mandible ascendant rami grow, articular heads and articular fossas, nasal meatuses and maxillary sinuses get formed.

Hard palate

It is almost flattened in new-borned. It acquires shape like cupule while aging. Palatinal processes are united with connective tissue in new-borned. Bone tissue invades gradually from palatinal processes side like spines. Palatinal suture is penetrated by osseal spines coming toward each other by the moment of teeth changing. Connective tissue layer is decreased while ageing and suture becomes winding. 3-6 transversal palatinal plicas come on the right and on the left from suture in palate anterior part. These plicas are more often curved and even can stopped or be divided into branches. These plicas are well-developed in new-borned and are rather essential for sucking. They are less expressed in the people of average age and even can disappear.

Maxillary sinuses

Maxillary sinuses in new-borned are like small fossa or pressures into nose external wall. They appear only on the 5^{th} month of antenatal period. Maxillary sinuses get increased the most intensively during the first 5 years of child life. Their development is retarded from 5 till 15 years of life. Only after all permanent teeth teething they acquire the shape which is characteristics of adult person. Left sinus can be bigger than the right one. Sinuses size is bigger in boys than in girls.

Oral mucosa

In early postnatal period (up to 1 year)

All parts of it have similar mucosa structure. It is defined by low differentiation of epithelium and connective tissue. Epithelial layer is thin and consists of 2 cells layers – basal and spines-shaped, epithelial papillas are not developed. Epithelium is rich in glycogen and RNA in all oral cavity parts, big amount of acid glycosaminoglycans are defined in epithelium and connective tissue.

Basal membrane is thinned in all parts in undergone to different injured stimuli very easily.

Proper plate is represented by lax connective tissue, the fibrous structures of which are low-differentiated.

Mentioned peculiarities of mucosa define its non-durability and easy possibility to be wounded in this period. At the same time, tissues qualitative content provides its high ability to regeneration. Maternal antibodies transmission through placenta as well as hormones, enzymes and other biologically-active substances determine rather high child

organism resistance to viral and bacterial stomatites occurrence during the first year of life.

In a period of first childhood (up to 1-3 years)

distinct regionary differencies become clear in mucosa. They are determined by its morphological-functional peculiarities. There are low content of glycogen and epithelium formation stabilizing in epithelium of tongue, lips and cheeks. Proper plate collagen and elastic fibers are located without definite orientation. They have shine structure delt with low degree of maturation. Acid glycosaminoglycans content is reduced but cellular elements content is increased with their primary localization in a region of vessels.

Cellular elements existence in a combination with big amount of blood vessels encourages to high vascular wall permeability in these areas.

Many labrocytes (mast cells) located perivascularly appear in mucosa connective tissue. Plasmocytes and histiocytes amount remains insignificant.

Gums and hard palate mucosa epithelial covering is more solid. It is connected with bigger epihteliocytes hardening as well as keratinization and parakeratosis zones existence.

Glycogen disappears. May be, it is used during keratinization.

Basal membrane and fibrous structures of proper plate in gums and hard palate mucosa are harden comparatively to the ones in babies. It is connected with separate fibers and fascicles oriented location.

In 3-12 years or in a period of pre-school and junior school age

Oral mucosa qualitative and quantitative changings occur determined by the character of metabolic processes in a child organism. Epithelium volume, glycogen and RNA content in it are increased comparatively to early child age. Basal membrane becomes solid and rough. Reticular and elastic structures amount grows in mucosa proper plate, collagen maturates.

Connective-tissued base cellular content gets changed. Lymphoid-histiocytic elements (forming perivascular infiltrates) amount is increased.

Lymphoid-histiocytic (round-cellular) accumulations appearance is a characteristic of immunological movements. It deals with proteinic exchange changing. Antibodies-producing cells (plasmocytes) grow first. Then proper antibodies (immunoglobulins) are produced. Similar mucosa structures qualitative reconstruction in this age is probably connected with organism sensibilization and protective mechanisms formation.

In elder school age

After 12-14 years, in elder school age, histological and histochemical changings as well as functional peculiarities are connected with hormonal regulation factors influence due to active sexual development and sexual maturation.

Salivary glands

Salivary glands function from the very moment of birth. Fist, salivary secretion is non-significant. It determines some dryeness of oral mucosa in children in the first months of life. But beginning from the 5-6th months salivation gets enforced significantly. Sometimes children do not manage to swallow saliva and it flows form their mouth non-arbitrary (physiological salivation).

Parotid gland projection in new-borned and children of early age differs from the one of the adult. Duct is located below, has non-direct course and is opened on 0,8-1,0 cm from masseter muscle anterior limb. Parotid gland is more round, comes forward less and passes up to mandible angle. Facial nerve lies superficially.

Secretory processes in salivary glands are especially active during puberty due to organism hormonal reconstruction.

While aging

Age Changes in Dental Tissues

1) Dispelling myths about aging:

- teeth loss,
- taste sense weakening,
- salivary activity decrease.

2) Most changes remain within the limits of homeostatic function.

3) Normal activities may not be seriously affected.

Changes and Consequences of Aging:

They are concerning teeth, gums / jaws, **s**alivary glands, tongue, taste**, s**wallowing**, s**peech.

Teeth changings:

teeth loss, enamel, dentin, pulp, cementum, periodontal ligament.

Tissues in our mouth change as we grow older. Tooth enamel can be eroded and dissolved by the action of acids from foods or produced by microbes in the mouth. Once the protective enamel is gone, the interior of the teeth can be attacked. The resulting infection will lead to tooth decay and eventual loss. The remaining teeth may become darkened and more brittle. Soft tissues lose their ability to stretch and to heal. The amount of saliva produced by glands in your mouth is frequently reduced resulting in dry mouth conditions that affect our ability to taste and also speeds up the processes of tooth decay. With age, teeth become less white and more brittle. Oral hygiene habits and use of tobacco, coffee and tea also have profound effects on tooth color. Teeth also can darken or turn yellow due to the thickening of the underlying dentin. Brittle teeth tend to be susceptible to cracks, fractures and shearing.

Over a lifetime, the enamel layer is subjected to wear due to chewing, grinding and ingestion of acidic foods. In severe cases, the enamel may be completely worn away and the underlying dentin attacked by acidic oral fluids, leaving teeth with only a fragile, brittle, enamel shell. These teeth are easily chipped or broken. Inside the tooth pulp, the number of blood vessels and cells decrease and fibroses increase with age. Thus, the capacity to respond to trauma may also decrease. Teeth undergoing inherent aging changes are symptomless, whereas teeth subjected to trauma are often painful.

Teeth Loss:

- Not a normal part of aging.
- A consequence of oral disease:
  - caries,
  - periodontal disease,
  - often association with systemic diseases.

Tooth loss is not part of the normal aging process, but is a consequence of oral disease. Aging does not cause oral diseases, but oral diseases, including tooth loss, are more common with age. This is partially due to older persons having been alive longer and consequently, having had a longer time for the effects of poor oral hygiene to accumulate. In partl, this is due to changes that are normally associated with aging such as:

1) Changes in the soft tissues that surround the teeth.
2) A depression of the immune system.
3) An increase in the number of systemic diseases.

4) Decreased ability in older persons to perform adequate oral hygiene and self care as a result of age-related diseases, such as stroke, arthritis, Parkinson's disease, dementia, or Alzheimer's disease.

5) Dry mouth due to greater use of prescription and over-the-counter medications. Edentulousness is on decline in the USA.

Factors Affecting Teeth Loss

The following factors which affect tooth loss: education, income, smoking history, race and ethnicity.

The education level was shown to have a significant effect on tooth retention among the elderly. Significantly more individuals with college educations were shown to retain their functional dentition after the age of 64 than those with lesser educational levels.

Income level was also a significant factor. Individuals with higher incomes had higher rates of tooth retention. Income levels reflect maximum pre-retirement incomes. Income levels are often highly correlated with education, so this and the previous chart probably illustrate confounded trends. It is also important to realize that education and income also correlate with the level of dental care sought and provided over the individual's lifetime.

It is interesting to note that there was no significant difference between regular or daily and occasional smokers or not daily. Those who had given up smoking or former smokers and those who had never smoked demonstrated significant gains in tooth retention.

Race and ethnicity were also shown to affect tooth retention.

Enamel

- Healthy teeth go through cycles of demineralization and remineralization throughout life.
- Aging results in physical loss of tissue through wear and acid erosion, not replaced.
- Other changes of properties include permeability, mineralization and light transmission.

Enamel is the hardest substance in the body – a translucent layer of highly ordered hydroxyapatite, a mineral made up of calcium phosphate. It is 90% mineralized. Throughout life, tooth enamel goes through cycles of demineralization and remineralization. But, if it is destroyed, it is not replaced.

As the people age, enamel undergoes several changes.

These changes include:

- thickness,
- permeability,
- mineralization and
- light transmission which affect the color of the enamel (normal enamel is translucent, the underlying dentin is white to yellowish, light passing through the enamel is reflected by the dentin, giving your teeth their color).

Other changings:

- enamel is getting thinner with age due to a lifetime of wear and tear as well as acidicerosion from acid foods or acidic bacterial metabolites,
- even excessive horizontal tooth brushing can result in abrasion of the enamel,
- as noted, enamel is normally translucent, but may take on color from smoking, food like blueberries, beverages especially red wine, tea and coffee or medications like tetracycline that are adsorbed on the surface,
- despite these changes, enamel in older people continues to absorb fluoride or remineralization, making continued use of fluoride toothpastes or fluoride treatments an important part of dental hygiene-care.

Dentin

Age Changes:

- Increase of volume at the expense of pulp
- Increase of secondary and tertiary dentin, more dead tracts and sclerotic dentin found
- Increase in secondary dentin makes the teeth appear darker

Dentin is softer than enamel, but still harder than bone. It consists mainly of apatite crystals of calcium and phosphate, which is only about 70% mineralization and contains proportionally more collagen fibers and cells. Dentin is produced by the odontoblasts, cells that line the pulp cavity of the tooth. Dentin is an alive tissue which has an ability for constant growth, repair and reacts to physiologic or functional and pathologic or disease stimuli. Primary dentin is formed during tooth formation. Once a tooth becomes functional, subsequent formation of dentin results in secondary dentin, somewhat less mineralized and with more irregular tubules than the primary dentin.

Tertiary or reparative dentin forms in response to injury: dental abrasion, attrition, cavity preparation, erosion or dental caries.

Tertiary dentin appears to have less structural organization and strength than the original dentin which it replaces. Although the exact form and the regularity of tertiary dentin appears to be dependent on the intensity of the external stimulus. Dentin tubules hypercalcification can also occur in response to injury and is referred to as sclerotic dentin. Sclerotic dentin amount increases with age, proceeding from the root apex towards the crown. There are also obvious optical changes in the tissue, which becomes translucent, while normal dentin is normally opaque.

Undergoing numerous other alterations associated with caries, aging or induced through treatment or medications:

- demineralization,
- deproteinization,
- hypermineralization.

Age changes in dentin include:

1. Increase of thickness and volume at the expense of pulp.
2. Decline in number and density of odontoblasts and other cell types with decreases in the root being greater than those in the crown region.
3. An increase of secondary and tertiary dentine, more dead tracts and sclerotic dentin found.

Aging teeth develop secondary dentin over time that also makes the teeth appear darker.

Pulp:

- decrease in size with age,
- fewer blood vessels and nerves,
- decreased capacity to respond to trauma,
- dminished immune competence.

Pulp is a loose connective tissue containing cells, ground substance, collagenous fibers, nerves and blood vessels that is encased in solid, bonelike wall of dentin and cementum. Pulp contains a variety of cells, chiefly odontoblasts that produce the fibers and ground substance of the tissue, histiocytes that produce macrophages and mesenchymal cells that differentiate to produce the other cell types.

Pulp also includes a variety of immunocompetent cells, especially Class II major histocapability complex (MHC) expressing cells and macrophages. Mast cell lymphocytes, plasma cells and eosinophils typically occur only near sites of inflammation.

While aging, pulp cavity gets reduced by deposition of secondary dentin and could conceivably be obliterated. The pulp gradually becomes smaller with fewer blood vessels and less nerve tissue supplying the teeth. As a result, teeth have less fluid content and become brittle and may be easily broken or chipped. The number of blood vessels and cells in the pulp decrease and fibroses get developed. Often the residual connective tissue sheaths of degenerated nerves and blood vessels increase with age. Thus, capacity to respond to trauma may also decrease. Odontoblasts degeneration begins near the root apex and advances towards the crown. The density and composition of those pulpal cells expressing macrophage-associated antigens becomes more variable with aging. Collagen fibers form cross-links, darken and produce toxic substances via the Maillard reaction such as lipid peroxidases that interfere with structural and functional properties of other molecules.

Cementum:

- mineralized connective tissue,
- covers teeth roots,
- deposition throughout life,
- deposition increases in response to stress,
- resulting in increased thickness in the elderly in some extent.

Unlike bone, cementum is not vascular and exhibits little turnover. Cementum grows slowly throughout life by surface apposition. Deposition appears to occur in response to functional stress. In older animals, as in elderly humans, cementum may make up a greater proportion of the tooth. The cementum layer widens first in apical region. In functional teeth, this may help compensate for loss of enamel around the gum line, although impacted teeth in the elderly also show increased width of the cementum layer.

Cementum Age Changings

Hypercementosis:

a) It is an abnormal thickening of cementum, may be:
- diffused or
- circumscribed.

b) It may:
   - affect all teeth of the dentition,
   - be confined to a single tooth, or
   - even affect only one tooth parts.

c) If the overgrowth improves the functional qualities of the cementum, it is termed a cementum hypertrophy.

d) If the overgrowth occurs in nonfunctional teeth, it is termed hyperplasia (e.g. ostitis deformans paget).

Although root resorption is not a part of the normal functional activity in the permanent dentition, most teeth show minute areas of resorption which may extend through the cementum and into the root dentin.

The resorptive activity may have been initiated by trauma from occlusion, orthodontic forces or for unknown reasons. Generally, the defect is repaired by rapid deposition of cellular cementum once the initiating factor is removed.

Periodontal Ligament

- Experimental evidence - width decreases with age but is very responsive to function
- Increase or decrease of load due to loss of teeth influences width

Little is known about age-related changes to the periodontal ligament in adults outside of the obvious effects of tooth loss and periodontal disease. While these may require long periods to develop, they are not associated with normal aging. Other aging-related factors, such as tissue fragility and slow healing, may also play important roles. It has been suggested that its width decreases with age, but as the ligament is very responsive to function any increase or decrease of load due to loss of teeth will influence on its width.

Age changes in periodontal connective tissue

- Gingival connective tissue becomes more dense and coarsely texture
- Decreases in the number of fibroblast, cellularity is related to growth and regeneration rather than to age
- Decrease in fiber content and a relative increase in the size of interstitial compartments containing blood vessels, but contradictory
- Collagen synthesis: with aging fibers become more stable showing increased thermal stability, insolubility and mechanical strength
- Changes in width of the periodontal ligament

Summary of Aging Changes in Teeth

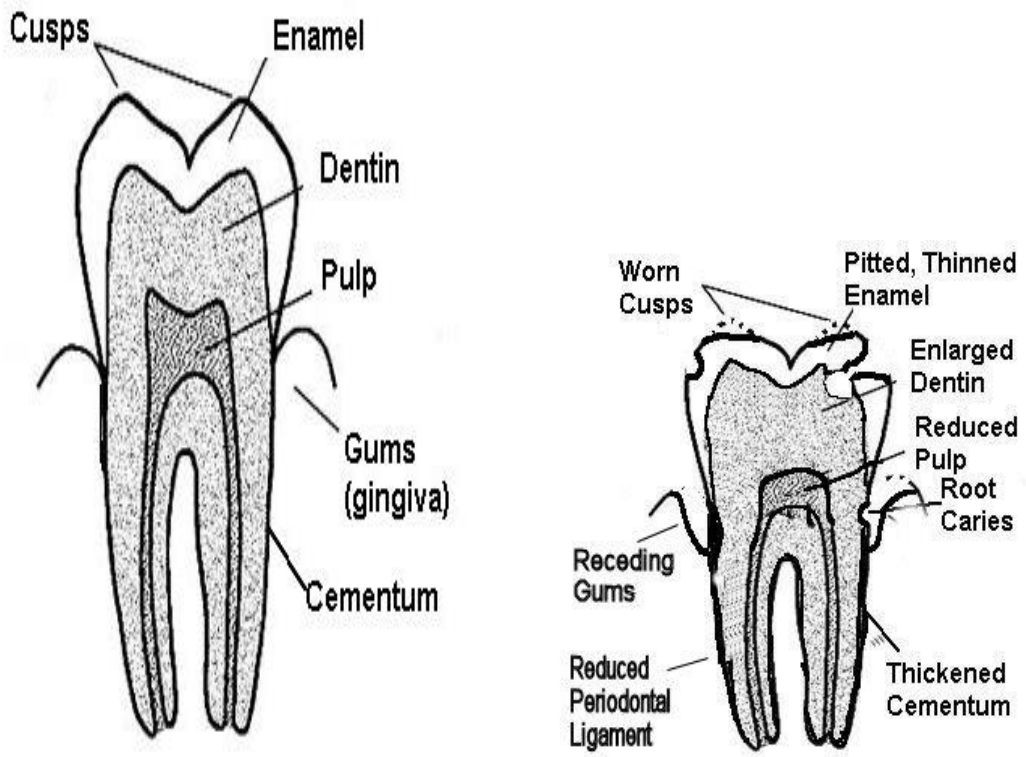

Figure 34. Summary of Aging Changes in Teeth

All of these changes can occur in teeth and many are reducible or preventable with proper dental care (figure 34). Cusps become worn from abrasion. Enamel can become thinned and pitted by erosion and wear. Caries can occur in reduced enamel layer. Infections may spread into the interior of the tooth, leaving enamel unsupported. Inside the tooth, the dentin layer enlarges and the pulp is reduced. More tertiary and sclerotic dentin can occur. Reduction in pulp results in reduced nutritive, reparative and immunological functions. The gumline recedes forming pockets and exposing roots. Root caries occur in exposed the cementum. Periodontal ligament retreats apically.

Dentogingival Junction

- Longer-in-the-tooth with age
- Apical migration of attachment or periodontal ligament
- Rate increases with gingival disease

We get longer-in-the-tooth with age, the result of receding gums and the apical migration of the periodontal ligamentary attachment. The rates of gum recession and ligamentary recession increase with gingival disease.

Alveolar Bone and Other Connective Tissues

- Loss of teeth means loss of bone.
- Loss of alveolar bone leads to loss of vertical dimension.
- Osteoporosis – seen particularly in females after menopause.
- Effects are exaggerated by malabsorption syndromes.

Oral Mucosa

- Epithelium gets thinner, more fragile, less keratinised
- Loss of collagen and elastin from fibers also weaken mucosa
- Increase in pathological change - loss of tongue papillae and taste buds
- Fordyce's spots increase and minor salivary glands diminish
- Lesions more common and slower to heal
- Inflammations, irritation and infections

Age-related changes in the oral mucosa are similar to those in skin.

a) Epithelium becomes:
   - thinner and
   - more fragile due to keratin loss.

b) Loss of collagen fibers also weakens mucosa.

c) Increase in pathological change results in loss of
   - tongue papillae and
   - taste buds.

d) Fordyce's spots or ectopic sebaceous glands increase and minor salivary glands diminishing.

e) Soft tissue lesions are more common in older adults and tooth loss may occur.

f) Chronic inflammation such as candidiasis or fungus growth and denture irritation also occur more often.

g) Wound healing is decreased due to reduced vascularity or blood flow to the area and immune response while aging.

h) In edentulous persons, changes in the mucosa are exacerbated by the presence of removable dental prostheses.

Lamina Propria

Histologic view of lamina propria is presented on the figure 35.

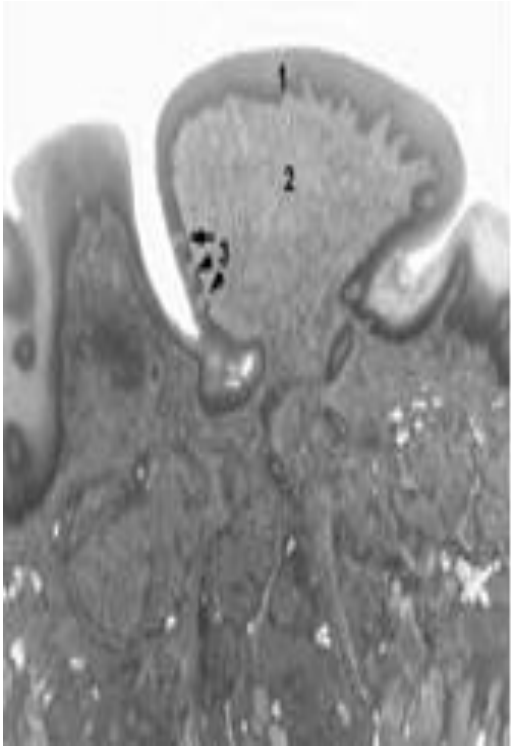

Figure 35. Oral mucosa lamina propria (own plate)

Own plate variations

a) Collagen density.
b) Size and organization of collagen fibers bundles.
c) Quantity of elastic fibers.

Changings

a) Gingival tissue handles like mucosa and is more readily traumatized by the dentist or by a prosthesist

b) The tissue is getting:

- more friable and
- more difficult to handle

c) Collagen lack means:

- more difficulty in differentiating the muco-gingival margin

So, Aged Oral Mucosa (changings can be seen on the figure 36):

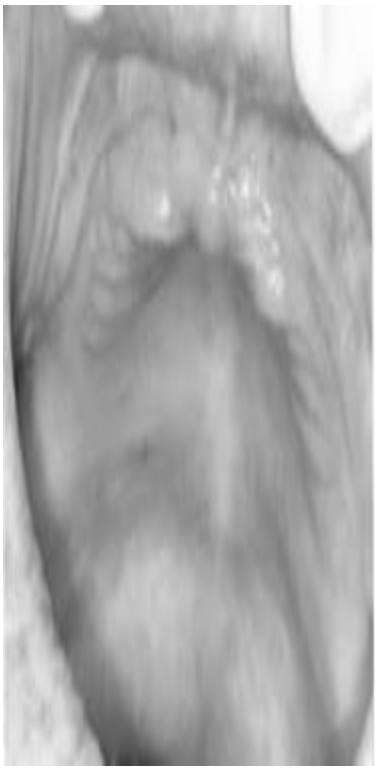

Figure 36. Aged oral mucosa:
increasingly thin, smooth and dry, satin-like, edematous appearance with loss of elasticity and stippling, more susceptible to injury.

Tongue

Functional changings

- Clinical changes with loss of filiform papillae
- Disturbance of the sensory elements resulting in deterioration in the sense of taste
- Burning sensation
- Dietary deficiencies can lead to atrophic changes of the mucosa
- Mucosal changes may result from systemic influences
- Sublingual varicosities
- Increased susceptibility to candidal infection
- Decrease rate of wound healing.

  These functional changes are resulted from following morphological changings:
- Diminished keratinization
- Alteration in the morphology of the epithelium-connective tissue interface
- Thinning of the epithelial cells layers
- Reduction in the thickness of the lingual epithelium > atrophy

Salivary Glands

- Major - Minor Salivary Glands*:*
  - Function Loss,

- Fatty Degeneration and
- Fibrosis.

In both major and minor salary glands, there are anatomical changes that occur with aging. Acinar cells number that produce saliva atrophy or decrease with age. The remaining tissues of the glands show an increase in fatty tissue and fibroses. However, healthy, unmedicated older adults do not have reduced saliva flow. This is because the salivary glands have a high reserve capacity. As the volume of secretory cells or acini declines with age, the relative volume of ducts in salivary glands increases.

- Saliva: changes in quantity and in composition.

Saliva: Changes in chemical composition, physical properties resulting in effects on taste and effects on environment such as more plaque and cavities. Until the 1990s, it was generally believed that salivary production decreased with age. But, earlier studies did not appreciate that other factors, such as medications and smoking history, will result in decreased salivary flow independent of age. More recent studies have produced conflicting results. While some appear to show declines, others appear to show no declines but have increases in salivary flow with age. Many studies, recent as well as older ones, are flawed by small sample sizes. Sometimes these are with fewer than 10 subjects or restricted range of ages. One study included no individuals over 49; another had none under 73; yet, both purporsed to investigate effects of aging on salivary activity. For example, these scientists (Janket, Wightman, Baird et al.; 2005) consider that salivary flow rates appear to decline with age when external factors such as medications, smoking or disease are not accounted.

Salivary Hypofunction Results:

- dry and friable oral mucosa,
- decreased antimicrobial activity,
- diminished lubrication,
- difficulty with mastication,
- deglutition,
- gustation,
- impaired retention of removable dentures,
- caries development,
- oral fungal infections,
- pain.

The composition of saliva also changes. For example, one of the following charts shows a decline in sodium levels. Potassium, total protein or acidic proline-rich salivary protein (APRP) fraction remains changes-free. Declines in some the concentrations of kallikrein, sIgA, low-molecular mucin and high-molecular mucin have also been reported. This could reflect decreases in the capacity of the oral cavity to fight off infections.

Increased lipid peroxide or free radicals levels in both oral tissues and saliva may contribute to plaque development and tooth erosion. Plaque is a biofilm – a structured organization of microbial cells, both motile and sessile in a matrix of water, organic and inorganic materials. Saliva both sustains and flights plaque. It is a medium for transport of bacteria within and between mouths, but it also helps prevent cell reattachment or agglutigens, kills or antimicrobial and flushes bacteria and dietary material from the tooth surface (Terpenning, Bretz, Lopatin et al.; 1993).

Not All Salivary Glands Respond the Same During Aging

Not all salivary glands respond the same during aging. Secretory activity of parotid glands may even appear to increase. However, the difference in parotid activity shown here is not statistically significant. Differences in submandibular and labial or minor gland activities are significant and valuable.

Taste

- Greatest decline in ability to detect salty, bitter and fine tastes
- Only slight reductions in the ability to detect sweet and little or no change for sour
- Ability to taste also affected by smoking, dentures and certain medications
- Loss of taste buds as an individual ages has been a controversial issue

A reduction of taste is not usually life-threatening, but it can alter one's eating habits. It is generally agreed that older adults show reduced taste discrimination ability, but the cause for this decline in function is not well understood. Ability to taste is also affected by nutrition, smoking, dentures, diseases, oral hygiene and certain medications. The sense of smell and fine taste starts to diminish gradually when persons are in their 50s, because of neural degeneration. Smoking may accelerate the process. Since the ability to taste is closely related to smell, taste perception may be altered in older adults. Loss of fine taste does not appear to be related to loss of lingual taste or deficits in cognition. Like olfactory function, taste perception is reduced with normal aging. Compared with younger persons, the elderly tend to perceive tastes as being less intense. The fact that many seniors take medicine regularly may compound the problem. Elderly people who take an average of three medications require two to 15 times more of a taste to experience it as vividly as do younger people.

Not only do different aged persons have different thresholds for detecting tastes. The relative pleasantness or unpleasantness of those tastes may change.

Not only are sweet, sour and salty taste thresholds higher in the elderly, but the elderly perceive these tastes, especially salty, as less unpleasant. This can lead to the use of too much salt or to exposure to spoiled or unsafe foods.

Although perception thresholds are far below the levels found in normal food, these differences may still be great enough to contribute to nutritional withdrawal resulting in "an anorexia of the elderly" to provide greater positive reinforcement at meal times.

Men are more prone to losses than women. Differences between young males and females are not significant in any of the tests. Since the changes in sensitivity to each tastant varies, it is possible that each sensory capability has a different pattern and perhaps mechanism of aging.

Reasons for decline in sense of taste are unclear or contradictory data:

- Possible decline in number of taste buds
- Possible decline in density of taste buds
- Possible decline in sensitivity of taste buds
- Possible decline in neural processing or retrieval
- All of the above also possible!

A smaller number of taste buds could explain why the elderly are only capable of recognizing the flavors of certain foods. The density rather than absolute number of taste buds, however, could provide an even better explanation of the observed. Researchers have speculated about whether or not a person's taste bud density changes as he or she ages, causing gradual changes in the ability to recognize certain flavors of foods.

During the course of a lifetime, taste buds are constantly renewed on approximately a 10-day cycle. It has been suggested that with aging, less vigorous replacement of taste cells may reduce loss of perception in elderly.

Receptor cells found in taste buds are known to degenerate when they lose their connections to the brain, so aging changes in the Central Nervous System (CNS) could not only affect the ability to interpret taste sensations, but the capacity to receive these in the first place.

Whatever the cause, the elderly have less specified taste acuity than the young, for which the noise hypothesis provides an explanation, either at a neural level, at a psychological level, or at both levels. However, an explanation in terms of physiological aging cannot be excluded. Although it seems that renewal and redundancy in the taste system preserve gustatory function in old age, it is not clear that the functioning of aged taste buds is not impaired.

Swallowing

- Reduced chewing effectiveness
- Decreased tongue strength due to less muscle and an increase in fatty and connective tissue in the tongue
- Atrophy of the alveolar bone with lost dentition
- Increased swallowing time with age
- Swallowing disorders may be prevalent

A number of age-related changes have been noted in the oral, pharyngeal and esophageal phases of swallowing.

These changes include:

- increase in fatty and connective tissue in the tongue;
- atrophy of the alveolar bone with lost dentition;

- reduced chewing capabilities;
- decreased esophageal muscle tone.

Even fully dentate elder persons are less efficient in mastication than younger individuals. Other studies show that while there may be some reduced chewing effectiveness, decreased tongue strength and increased swallowing time with age; there does not appear to be any real change in the ability to swallow with age.

Swallowing slows with age however temporal changes in the esophageal phase are more significant than those during the pharyngeal phase of swallowing. Older people are less able to prepare food adequately, they swallow larger-sized food particles which offer reduced masticatory efficiency and is exacerbated by compromised dentition. Swallowing requires well co-ordinated neuromuscular activity, adequate saliva and an intact mucosal barrier. In young adults, the oral phase of swallowing requires from 1,5 to 2,0 seconds. This increases 50 – 100% in older adults.

Swallowing disorders may or may not be inherent to aging. However, they are relatively prevalent in the elderly population. This is because dysphagia is associated with many disorders that are much more common in the older population. Conditions developing as a consequence of stroke, motor neuron diseases, Parkinson's, and arthritis can all result in dysphagia.

Speech and Enunciation

- Speech is a complex process
- Aging changes – slowness, deepening of voice
- Effects of edentulousness & dentures
    - 50% over 85 lack natural dentition
- Dysphasia and aphasia are results of disease, not aging

Speech is a complex process involving visual and auditory input, central processing and motor output, not only to the vocal muscles but also to the facial muscles. The facial muscles produce expression and are thus, an integral part of vocal communication. Speech patterns change with age. The voice becomes deeper and may develop an increasing thremor, speech becomes slower and may develop an abnormal prosody. Enunciation of consonants becomes imprecise.

Close to 50% of Americans age 85 and over have no natural teeth. There are unique problems associated with the edentulous state. Functionally, the teeth aid in mastication and enunciation, however, ill fitting dentures can interfere with speech patterns, and the removal of dentures can aid in speech.

Changes in pronunciation and enunciation are quite common in the aged. Dysphasia and aphasia are less common. Dysphasia is difficulty in speaking or in understanding language. Aphasia is loss or impairment of the capacity to use words as symbol of ideas. Both are caused by brain lesions, often a consequence of stroke, and not by changes in the mouth.

Chapter 4. SOME APPLIED ASPECTS

CHANGINGS IN ORAL CAVITY AT ENDOCRINE GLANDS PATHOLOGY

This function is of great clinical importance. Oral cavity expressions can be observed at other organism system (Eversole; 2000) in part at endocrinal pathology.

Odontogenesis is rather durable process. That is why the probability of its tissues injuries at endocrine pathology is quite high.

Adenohypophyseal, parathyroid and thyroid hormones participate in intrasecretory regulation of teeth growth and development. Neurohypophyseal somatotropin is major stimulator of dental tissues histogenesis (Reisine, Bell; 1995). Teeth calcination is tightly connected with parathyroid hormone (Strewler; 2000), teething – the thyroid one (Ng'ang'a, Chindia; 1990).

Changes in oral cavity occur at endocrine glands hypofunction, hyperfunction or dysfunction. Stomatological expressions of some endocrine disorders are considered to have big diagnostic value. It is so because they are often manifesting. It means that they appear before general clinical symptoms. It should be mentioned that sometimes expressions on oral mucosa have many similar features with its changes at other, non-endocrinological diseases.

The mostly often oral mucosa injuries (catarrhal stomatitis, epithelium desquamation et al.) are found at sexual glands dysfunctions as a result of estrogens secretion disturbances.

Juvenile gingivitis. It is observed in fellows and girls in puberty, at menstrual cycle disorders. Oral mucosa changes are the result of gonadotropins action. Dentury anomalies, non-satisfactory oral cavity hygiene are among local predisposition factors. Gum mucosa is edematic, hyperemic and bleeds easily. Gingival papillas, gum marginal limb hypertrophy is observed mainly in mandibular frontal teeth.

Gingivitis of the pregnant is also worthy to be described. It is developed because of pregnancy or its course is hardened during pregnancy. It is expressed in interdental papillas hypertrophy and sometimes also – of gum marginal limb. "Non-real" pockets are formed, gums get bleeded. Gravity of its course can be changed. Sometimes hypertrophied papillas reach teeth crown cutting limb or their masticatory surface. It causes pain and bleedings while eating. Gingivitis symptoms being present before pregnancy are hardened in the biggest cases. The gum is more often transformed into its normal state after labours but sometimes (at bad oral cavity hygiene, dental stone, bad dentures, denturing anomalies) and other local traumatizing factors gum hypertrophy is remained non-changed.

Knowledge about dental peculiarities of oral cavity changes at endocrinal disorders will encourage the early disease stage exposure as well as proper assessment

the local expressions of general pathology and also adequate correction methods choosing.

Adenohypophyseal hypofunction. Retarded teething and child lacked development are observed at hypophyseal dwarfness. Teeth are formed properily because disorders are mainly expressed in the period when their germs formation was completed. Constant teeth pulpal chambers are wide, secondary dentin formation is retarded. Jaws bones are little. Skin hypoelasticity helps radial winkles appearance round mouth.

Adiposal-genital dystrophy is characterized by odontogenesis retardation in parallel to organism development retardation.

In a case of *adenohypophyseal hyperfunction* the biggest changing are observed at hypophyseal giantism and juvenile acromegaly.

Hypophyseal giantism is characterized by expressed odontogenesis acceleration. Crowns can be of usual size, but their roots are prolonged.

Juvenile acromegaly is described by distinct bright mandible development especially of its ascendant branch. Teeth can be of normal size, but their roots look shortened. Sometimes macroglossy is happened.

Thyroid dysfunctions

Obtaining an understanding of thyroid dysfunction is of significant importance to the dentist for two reasons. First, the dentist may be the first to suspect a serious thyroid disorder and aid in early diagnosis. The second reason is to avoid possible dental complications resulting from treating patients with poorly controlled hyperthyroid conditions. Although a rare occurrence, a thyrotoxic crisis—a true medical emergency—can occur associated with hyperthyroidism and dental procedures. The patient with hypothyroidism presents risks less critical in nature, but a few precautions are noteworthy.

Hypothyroidism

Neonatal cretinism is characterized by:

- dwarfism,
- a broad flat nose,
- thick lips,
- large protruding tongue,
- poor muscle tone,
- pale skin,
- retarded bone age,
- teeth delayed eruption,
- malocclusions.

The long-term effects of severe hypothyroidism on craniofacial growth and dental development have also included impaction of the mandibular second molars, owing to failure of normal resorption of the internal aspect of the ramus.

Endemic cretinism (hypofunction in early childhood) is characterized by such changes in oral cavity as macroglossy, milky and constant retarded development, enamel hypoplasy, predisposition to caries, lips size increasing, dryness, cracks. Hypothyroidism in older children and adults is characterized by a dull expression; puffy eyelids, face, and hands; rough skin and brittle, coarse hair; enlarged tongue; slurred speech; and increased sensitivity to cold. Juvenile hypothyroidism has been reported to include evidence of delays in shedding of deciduous teeth, root development, and eruption of permanent teeth as well as retarded skeletal growth (Ng'ang'a, Chindia; 1990). Two years of treatment with L-thyroxine results in dental and skeletal changes.

Hypothyroidism may occur from Hashimoto thyroiditis. This disorder represents chronic inflammatory disease of the thyroid in which autoimmune factors play a prominent role. It occurs in females during middle age and is the most common cause of sporadic goiter in children. Autoantibodies are indicative of disease but cytotoxic T cells probably destroy parenchyma. The most prominent feature is a diffuse goiter, which may be symmetrical or asymmetrical.

Hyperthyroidism

Thyroid hyperfunction is characterized by preliminary teething, tendency to caries, hypogevsia (gustatory sensations decreasing), multiplied sulcuses existence on tongue (Perusse, Goulet, Turcotte; 1992). A small number of patients may exhibit a raised, reddish asymptomatic mass in the dorsal posterior tongue area, near the foramen cecum. This is called lingual thyroid and represents a mass of normal thyroid tissue left along the path of the thyroglossal duct. This appears in females more commonly than in males. Before removal or biopsy of this mass, it should be confirmed that the thyroid gland is present and functional. In many cases, the lingual thyroid is the only functional thyroid tissue present. Hyperpigmentation of the oral mucosa has not been reported.

Somatotropin hyperpoduction

Paget's disease of bone usually occurs in people over 40 years of age (Delmas, Meunier; 1997). It is a condition in which osteoclastic activity and compensatory disorganized bone overgrowth occur side by side (figure 37, appendix).

The patient complaints of maxilla progressive enlargement or much less frequently mandible. Teeth become spaced, if dentures are worn these cease to fit properly as a result of dental arch increase in size.

Pain occurs occasionally in connection with jaw lesion, sometimes it is rather severe. Jaws lesions incidence is often uncertain.

Mandibular overgrowth and malocclusion are oral manifestations, and sometimes are the first sign of this idiopathic disease. Long bones may bow, and there is often associated pain.

Diagnosis can be confirmed by biopsy, radiographs, and chemistry (normal calcium, high alkaline phosphatase). There might be some clinical confusion with acromegaly, since both conditions may present with the Class III malocclusion. But acromegaly is a reflection of anterior pituitary adenoma with an increase in growth hormone that stimulates condylar cartilagenous reactivation and mandibular growth. Other endocrinopathies, such as diabetes mellitus, occur simultaneously.

Estrogen deficiency

Osteoporosis is an extremely common condition of aging, particularly in postmenopausal women (Eastell; 1998). Osteoporosis is not a primary disorder of calcium metabolism. It may result from inadequate stimulation of osteoblasts (postmenopause, disuse) or excess catabolic agents (adrenal cortex overactivity). Pain and fractures are common findings. It has been shown that osteoporosis affects jaw bones; however, the clinical implications are not clear.

Parathyroid dysfunctions

Parathyroid main function is a regulation of calcium metabolism and inorganic phosphorus level (Silverman, Eversole, Truelove; 2001). That is why these glands play important role in tooth solid tissues mineralization regulating.

Hypoparathyroidism

The dental manifestations occur only at the congenital form, which affects the teeth at the developmental stage, causing mottling (hypoplastia) and discoloration (figure 38, appendix).

There are no dental findings in acquired hypoparathyroidism. It is the mostly often expressed in enamel hypoplasy. Permanent incisives and the first permanent big molars are the most probable “targets” for injury. Hypocalcemia takes place during their mineralization.

Hyperparathyroidism

Hyperparathyreoidism (Reklingauzen’s disease or fibrous osteodystrophy) is described by generalized skeleton injury due to osseal tissue reconstruction and its replacement with fibrous one. Progressive resorbtion of bone tissue takes place in pocket process together with teeth making wobbly and loosing.

Diabetes mellitus

Oral cavity changes are often manifesting. Oral cavity changings are often the first diagnostically valuable signs of the disease thus the dentist can put the diagnosis.

Xerostomy is the patients' complaint from the very beginning of the disease. The manifestation is different in various patients. Hyperdypsia and hyperphagia (enforced thirst and appetite correspondingly) are also among symptoms. At examination: oral mucosa is weakly wet or dry, brilliant, with insignificant hyperemia. Dryness is considered to be the result of dehydration.

Dentist should keep in his mind that xerostomy is also observed at other diseases and states: Mikulich disease, syaloadenitis, syalodohitis, Shegren's syndrome, nasal respiration disorders, nervous system disturbances and others.

Catarrhal stomatitis and glossitis – is the result of mucosa infectioning, easily possibility to be wounded because mucosa barrier function got significantly decreased, dysbacteriosis takes place. Hyposalivation encourages this. Patients complain about pain while eating especially of hot and solid food. Mucosa is dry, hyperemized, erosions and hemorhagies and other mechanical injuries are visual sometimes.

Fungal stomatitis, mycotic angular stomatitis. Fungal injury is rather stable. Mycotic angular stomatitis is the mostly often observed. Cracks covered by white-grey crusts are appeared in mouth angles. Fungal injuries are considered to be the result of dysbacteriosis on the background of organism resistance decreasing. Metabolic acidosis (due to pyruvate and lactate accumulation) helps to this (Adrogue, Madius; 1998; Гринстейн, Гринстейн; 2000).

Mucosa paresthesia at diabetes occurs together with its dryness. This symptom is also observed at alimentary tract and nervous system diseases. At diabetes mucosa burning is accompanied by skin itch in genitals area and other body areas.

Neuritis, trigeminal nerve neuralgia belongs to neurological disorders observed at diabetes mellitus.

Patients have gustatory sensitivity disorders: decreasing to sweat, salty and to sour (in less extent). Taste sensitivity disorders are functional and they come to norm after the treatment performed.

Trophyc changings can be present in oral mucosa: trophyc ulcers with prolonged course and retarded regeneration. One can see legs, arms and other body parts gangrene.

Diabetes is associated with several oral manifestations, primarily those related to infections, inflammations and poor wound healing. Hyperglycemia, ketoacidosis, and vascular-wall disease contribute to the increased susceptibility of uncontrolled diabetics to infection and the decreased ability to manage infections. Xerostomia and effects associated with reduced salivary flow rates are also problematic.

These conditions have significant implications for dental care. Several studies have reported an increased incidence and severity of gingival inflammation, periodontal abscess, and chronic periodontal disease in diabetic patients (figure 39, appendix) (Clark, Lee; 1995).

Microvascular disease affects blood flow and leukocyte migration adversely and predisposes to premature periodontal disease, abscess, and delayed wound healing (Grossi, Genco; 1998) in the periodontium. The research supports strongly the cessation of cigarette smoking to aid maintenance of periodontal health, especially in the patients with diabetes (Soskolne; 1998). Data also support the concept that in diabetes-associated periodontitis, the altered host inflammatory response plays a critical role. An unexpected high level of gingival crevicular fluid mediators was found among subjects with insulin-dependent diabetes mellitus (IDDM), even in the patients with gingivitis and mild periodontitis. Diabetics had significantly higher gingival cervical fluid levels of both prostaglandin E2 (PGE2) and interleukin (IL)-l beta when compared to nondiabetic controls with similar periodontal status. These findings suggest that IDDM is a significant risk factor for more severe periodontal disease, because as compared to nondiabetics, diabetic subjects react with an abnormally high degree of inflammation to an equivalent bacterial burden. Other findings suggest that both hyper- and hypoglycemia might directly impair the biologic functions of periodontal connective tissues through cell–matrix interactions. Well-controlled diabetic patients, as measured by blood glycosylated hemoglobin levels, have less severe periodontal disease than poorly controlled diabetics. Recent findings indicate that effective control of periodontal infection in patients with diabetes (both insulin-dependent and insulin-independent) reduces the level of advanced glycosylation end-products in the serum. If this is confirmed via additional studies, periodontal infection control must be considered as an integral part of medical management of diabetic control. Oral candidiasis occurs more frequently in diabetics than in nonaffected populations, because of altered response to infections and xerostomia and an altered oral flora (figure 40, appendix).

The patient should be referred to an infectious disease specialist for appropriate management. Burning tongue may be associated with fungal infections, such as candidiasis, or peripheral neuropathies associated with diabetes. A cytologic smear can confirm the diagnosis of oral candidiasis and proper treatment can be initiated. The diagnosis of peripheral neuropathy should be concluded after other probable causes have been ruled out by consultation with the patient's physician and an oral medicine specialist. Xerostomia may result from hyperglycemia and subsequent polyuria that depletes the extracellular fluids. The overall effect is reduction in secretion of saliva. Adequate salivary flow is recognized to be an essential component for normal mastication, taste, and swallowing functions. Saliva plays a critical role in the lubrication and protection of the oral mucosa, harmful acids neutralizing that can lead to

dental caries and destroying microorganisms. Diminished flow can increase susceptibility to oral ulcers, bacterial, viral or fungal infections, and dental caries.

Diabetes insipidus

Diabetes insipidus is a condition caused by a neurohypophyseal lesion, either inflammatory or neoplastic in nature, in which water renal keeping is impaired owing to deficient antidiuretic hormone (ADH) release. Vasopressin, also called antidiuretic hormone, affects the control of water retention and its release in coordination with the activity of the thirst center that regulates fluid intake. Via actions on receptors in the distal tubules of the kidney, ADH keeps water inside the organism and concentrates the urine. This action assists in maintaining the constancy of the osmolarity and volume of body fluids.

Oral manifestations

Infiltrative lesions of the neurohypophysis as a result of Langerhans cell histiocytosis have implications for dental treatment. Histiocytes may infiltrate the posterior pituitary, resulting in decreased output of ADH, with resultant polyuria. Retro-orbital infiltrates of histiocytes may lead to exophthalmosis, and osseous infiltrates are typically found in the skull and jaws. In addition, osseous infiltrates can be identified via conventional dental radiography (Seow, Thomsett; 1994). In addition to jaw lesions, one might observe loosing of the teeth or teeth “floating in space” (figure 41, appendix).

Both hyperfunction and hypofunction of the adrenal glands can have profound effects on dental management of affected individuals. The following sections address primary and secondary adrenal insufficiency and hyperfunctioning of the adrenal cortex.

Itsenko-Cushing’s syndrome

Itsenko-Kushing’s syndrome represents suprarenal cortex hyperfunction. Changes similar to the ones at diabetes mellitus are observed in oral cavity: trophyc ulcers on tongue, cheeks mucosa and mouth other parts (Wehling; 1997). Candidosis is often present (figure 42, appendix).

Addisson’s disease

Pigmentation both of skin (at places which are undergone to light action) and mucosae (on lips, on tongue and gums limb, cheeks mucosa) as blue or grey-black small spots and strips takes place (Davenport, Kellerman, Reiss, Harrison; 1991). The oral manifestations include diffuse patchy brown macular pigmentation of the oral mucosa

(Werbel, Ober; 1993). Oral mucosal changes may be the first manifestations of the disease, with skin hyperpigmentation following. Patients demonstrating diffuse oral pigmentation should be interviewed regarding onset. Reason: melanin significant accumulation on skin and mucosa.

Thus, ***changes in oral cavity are the mostly often observed at***: diabetes mellitus, dysfunctions of sexual glands, hypophysis, thyroid, parathyroid and suprarenal gland cortex.

That is why timed and correct assessing the local symptoms of these diseases by dentist will promote their early diagnostics and correspondingly earlier, more adequate and as a result – more effective therapy.

NUTRITION

Nutrition is the most important biogenic factor of the environment. Qualitative or quantitative inferiority of nutrition may be the reason of disturbance of food status of man and as a result of many alimentary diseases.

Food status is a physical, mental, psychic and physiological condition of a man, which is caused by previous and present nutrition and characterized by a complex of somatometric, metabolic, functional and clinical indices of health and capacity of work.

There are 4 kinds of food status: usual food status, optimum, deficient, surplus food status.

The usual food status corresponds to normal nutrition, which meets all the requirements of rational nutrition. During this food status all functions of the organism run normally, normal self-renewal and adaptation of the organism to the environment take place. The normal food status is characterized by normal health level and a high level of capacity for work. The usual food status provides preservation of the human body structure and functions, human adaptation and health in usual life conditions.

The optimum food status is formed under special food rations, which provide high level of the human organism bioresistance under the extreme and stressful conditions. This status provides work carrying out by man in unusual conditions without disturbance of his health.

The deficient food status is characterized by presence of changes in health condition which appears after complete or incomplete (partial) starvation. In this situation a deficient quantity of food substances enter the organism of a human being. The deficiency can be both qualitative and quantitative.

The surplus food status develops during consumption of a high quantity of food or its components exceeding a physiological need. An excess of food as well as its deficiency, may be both qualitative and quantitative. Such a situation causes pathology. The deficient and surplus food statuses result from nutrition disturbances.

A nutrition disturbance is a pathological condition caused by an absolute or relative deficiency or excess of one or several nutrients in the food ration. The

insufficient or surplus food status can cause different disturbances of the organism structure and functions, decrease of capacity for work; somatic pathology appears.

Nutrition disturbances diseases are divided into 2 large groups:

1) diseases of insufficient nutrition,
2) diseases of excessive nutrition.

Diseases of insufficient nutrition include quantitative insufficiency of nutrition (malnutrition) and qualitative insufficiency of nutrition. The qualitative insufficiency of nutrition can be proteinic insufficiency, deficiency of fat, deficiency of mineral substances (deficiency of macroelements and microelements), vitamin deficiency, (avitaminosis and hypovitaminosis) and imbalanced nutrition.

Diseases of the nutrition excess represent quantitative excess of nutrition (obesity) and qualitative excess of nutrition. Hypervitaminoses can be nutrition qualitative excess example.

FEEDING RATION AND STOMATOLOGICAL PROBLEMS DEVELOPMENT

Feeding ration quantitative and qualitative content can be as pathogenetic factor in some dental diseases occurrence especially teeth caries. Excessive feeding does not influence directly on oral cavity organs state but metabolism diseases accompanying teeth and mucosa damaging appear at this.

Raw and damp, solid food taking, its careful mastication encourage to the teeth surface clearance and prevent dental covering formation. People taking porridge-like food have dental covering that can lead to caries or parodontosis.

Nutrients correlation disorder in a feeding ration can be the reason of developing the diseases with expression in oral cavity. So, fermentation processes develop at carbohydrates excessive taking that favors multiplication of the microbes creating oral cavity acid environment. It is accompanied by covering formation on the teeth, enamel dissolving that helps in the teeth cariotic damage. That is why carbohydrates dominance in a feeding ration needs vitamin B increased concentration as well as thorough care for the teeth. Taking food with proteins excessive content creates alkaline environment in oral cavity that can result in the gums diseases (gingivitis). Proteins insufficiency leads to the hypovitaminosis of B-group vitamins.

Oral cavity and teeth state is rather sensitive indicator of vitamins deficiency in a feeding ration. It is explained by their abundant blood supply and capillaries vast net. Capillaries endotheliocytes react subtly to the vitamins content in blood. Vitamins play important role in oral mucosa protection and its regeneration. Bacteries present in oral cavity cause inflammation rather easy at avitaminoses because of mucosa resistance weakening. The pathological symptoms always appear first at the foci where mucosa is undergone to the mechanic action at mastication.

Vitamin A deficiency causes oral mucosa epithelium keratinization and small submucosal salivary glands atrophy that result in hyposalivation. Mucosa gets dry,

cracks appear on it. These cracks become infected easily that leads to inflammatory processes development.

Vitamins B-group is expressed usually by oral mucosa inflammation, atrophic areas presence on tongue, tongue edema, fissures appearance in mouth angles (angular cheilitis).

Vitamin C significant deficiency causes zynga (scorbut) in the adult. The disease is characterized by spontaneous bleedings from gums. Gums get swollen, hyperemized, cyanotic-red. As a rule, secondary infection is added that enforces hemorrhagies and bleedings. The teeth are covered by infected and thus stinky covering. Grey covering is present on the gums margin. Painful ulcers get formed. Gums and interdental papillas necroses occur if the inflammation lasts long.

Vitamins D deficiency during odontogenesis disturbs enamelogenesis.

Undamaged tooth contains: F, Zn, Fe, Ag, Mn, Si, Sn, Ba, Cr, Sr, Ti, Ni, Al, B, Pt, Va and other elements. Odontal-jaw system changes can be determined by food insufficient mineralization (Ca, P), microelements insufficiency or excess (especially I and F). While passing the organism through alimentary tract they influence actively on different physiological processes in part on bones and teeth mineralization, their resilience or predisposition to decay both during odontogenesis and in the formed tooth.

Special attention must be paid to feeding the patients with disturbed normal conditions for food taking. Following conditions can be described in this group: oral cavity integrity disorder as a result of traumas and congenital defects as well as the disturbances caused by mouth retarded opening for instance due to temporal-mandibular joint diseases and jaws fixating at fractures treatment. Mastication act is usually disturbed in such patients that leads to food non-proper mechanic and chemical processing in oral cavity. Food must be heated up to 40-60°C to them, have fluid consistence allowing its injecting through zond. It is essential to reach such a food balance on different nutrients, vitamins, microelements content as well as on calorage. It is possible to have nutrients parenteral injecting in the separate cases of jaws-facial area hard injuries. It is prescribed for rather short time – up to 10 days. Highly-effective proteinic medicines, water-soluble vitamins and mineral salts can be injected by parenteral way.

ORAL CAVITY PATHOLOGICAL PROCESSES CONSEQUENCES

The clinicians must always remember about possibility of many diseases oral genesis.

Pathological foci in the teeth and tonsils can lead to: heart-vascular activity changes in part arterial pressure rising, myocardium infarction (sometimes), endocarditis, myocarditis, pericarditis; skin trophyc changes; memory weakening;

reason-free hyperhydrosis; reason-free subfebrile temperature; hemorrhagies in brain (sometimes).

Chronic focal odontogenic infection can result in local and disseminated damages of nervous system such as: meningitis, encephalomyelitis, multiple sclerosis, radiculitis et al.

Pathological changes in oral cavity can cause chronic diencephalites. Under such conditions oral cavity sanation encourage to speedy self-being improvement or complete recovery.

Different inflammatory diseases of teeth and palate tonsils serve as a source of very stable headache appearance. Its location depends often on inflammatory process area. It has been established that pathological foci in maxillary incisives are accompanied by pain in frontal-temporal area while in molars – in parietal-occipital area. Headache can acquire a diffused character at maxilla teeth inflammation. Headache appearing at the teeth damage first of all is determined by trigeminal nerve the second and the third rami sensory endings irritation as well as their multiple bonds with the vegetative nodes in the head area. Such headaches leave the patients after extraction of the sick teeth or purulent cysts on their roots. Acute and chronic tonsillitis is characterized by splitting pain in the back of the head accompanied by shoulders plexalgy, tension and pain in occipital-cervical musculature as well as round or oval focus of pain hyperesthesia on the occipital skin area. Pathological processes developing in oral cavity can encourage some diseases of inner organs, cause or maintain various complications. So, pathological mobility or teeth loosing lead to food non-proper processing in oral cavity that first of all is reflected to stomach and intestine motor and secretory activity. But digestion disorders in oral cavity caused by mastication dysfunction at teeth loosing did not always give life to one or another pathology in other parts of alimentary tract.

Mastication insufficient function can be compensated by other digestive system organs function. At the same time, one must take into account that every organ has its own compensation limits especially at pathological process presence in a stomach or intestine.

In turn, such processes in alimentary tract are reflected always in one or another degree to oral mucosa state. It is non-occasionally that human tongue is examined by the doctor during the patient's observation from the ancient times. This interaction is realized by morphological, physiological and humoral bonds of alimentary tract different organs and its initial part – oral cavity. Oral mucosa is an exceptional stimulation zone. None of human body areas has such a powerful exit of brain stem afferent ways. Oral cavity possesses the widest extero-receptive zones of vegetative-somatic reflexes. It is so-called avanpost of brain stem sensory centers with a tonizing and visceral-signal function. That is why oral cavity irritation especially of pharynx posterior wall can cause the shifts in the patients' state.

There are known cases in a clinical practice when pharynx posterior wall irritation led to consciousness restoration at sopor (underconscious state). On other case, one knows the sad examples of action to oral cavity leading to the patient's state weakening or even death. There existed the cases of sudden death at oral mucosa burning with alkalis and acids, clinical death cases from anesthesia of pharynx and tongue back with dicain et al. Cry and dyspeptic disorders during teething can be connected to oral mucosa irritation in some children as well. All mentioned reactions mechanism is explained by trigeminal-vagal and vagal-vagal reflectory bonds. Tongue ancient-chineese method is based on these reflex bonds. This method is following: 18 tongue slow movements in one and 18 movements in another side. Mentioned attempt favors liver and gallbladder diseases course. We must remember that pathological foci in maxillary-facial area as a whole and in oral cavity in part can cause or harden diseases and syndromes course out of them because of all organs systems tight interconnections (figure 43, appendix).

Every person must take care of his/her oral cavity, a dentist can put non-stomatological diagnosis first and direct the patient to further examination and treatment to corresponding specialist. On other hand, a qualified doctor of non-stomatological profile can help sometimes in dental problem proper and termed diagnosis and thus therapy.

GLOSSARY

A

Absorption – the taking in or reception of gases, liquids, light, heat, or solutes, such as the movement of digested molecules across the intestinal wall and into the bloodstream, the movement of substance through the skin, and the movement of fluid into the lymphatics from the intestinal fluid.

Acidosis – condition characterized by a lower than normal blood pH (pH of 7,35 or lower).

Ageusia is a loss of taste sensation.

Alkalosis – condition characterized by a higher than normal blood pH (pH of 7,45 or above).

Amylase – one of a group of starch-splitting enzymes that cleave starch, glycogen, and related polysaccharides. Salivary amylase is found in saliva while the pancreatic amylase is found in panctreatic juice.

Antibodies – proteins found in the plasma and saliva that are responsible for antibody-mediated (humoral) immunity; immunoglobulin proteins (G, M, D, E, A, D) secreted by B-lymphocytes that have been transformed into plasmic cells (plasmocytes); their synthesis is induced by specific antigens and they combine with these specific antigens but not with unrelated antigens.

Antibody-mediated immunity – immunity resulting from B cells and the production of antibodies.

Anticoagulant – chemical that prevents coagulation or blood clotting; an example is antithrombin.

Antigen – a molecule able to induce the production of antibodies and to react in a specific manner with antibodies.

B

Best response - the stimulus that elicits the highest spiking rate from a neuron and is used in defining classes of taste neurons, such as sucrose-best.

Buffer – chemical that prevents changes in pH when either an acid or base is added to a solution containing the buffer; it realizes this by either combining with hydrogen proton or by releasing hydrogen proton into solution.

C

Canine – a tooth having a crown of thick conical shape and a long, slightly flattened conical root; there are two canine teeth in each jaw, one on either side adjacent to the distal surface of the lateral incisors, in both the deciduous and the permanent dentition.

Cathelicidin (LL-37 or FALL39) – antibacterial protein with molecular weight 18 kDa is present in oral non-keratinized flattened epithelium as well as in saliva and gum tissues and possesses bacteriocydic action.

Chemotopy - spatial separation of taste receptors within the oral cavity or taste neurons within an area of the central nervous system, according to chemosensory sensitivities.

Common chemical sensation - elicited by activation of somatosensory pain pathways with a chemical stimulus.

Cover dentin – the tooth mass ivory-coloured. Calcified tissue that is not as hard as enamel but harder than cementum. About 20% are organic matrix, mostly a fibrous protein collagen, with some elastin and a small amount of mucopolysaccharide; the inorganic fraction (70%) is mainly hydroxyapathite, with some carbonate, magnesium, and fluoride. The dentin is transversed by closely packed tubules running from the pulp cavity outward; there are processes from the odontoblasts within the tubules.

D

Defensins – represent bacteriocydic and fungicydic peptides localized in oral mucosa in part capable to enforce local and systemic inflammatory answer due to their signal function.

Deglutition – the swallowing act.

Dental articulation - The contact relations between maxillary and mandibular teeth during jaw movement.

Dental bulb – the papilla, derived from mesoderm that forms the part of the primordium of the tooth that is situated within the cup-shaped enamel organ.

Dental canal – the chamber of the dental pulp lying within the root portion of a tooth.

Dentate - having natural teeth.

Dentin – one of the hard tissues of the teeth. It covers the pulp cavity and is itself covered on its exposed surface by enamel and on its root surface by cementum.

Dentition - the natural teeth in the dental arches.

Denture - A removable or non-removable dental prosthesis replacing natural teeth and their associated tissues.

Digestion – the breakdown of carbohydrate, lipids, proteins, and other large molecules to their component parts.

Dry mouth – a condition that occurs as a result of reduced salivary flow from a variety of causes, including Sjögren's syndrome, connective tissue diseases, diabetes, excision or absence of a major salivary gland, or radiotherapy to the head that destroys the salivary glands. It causes swallowing and speech difficulties, inflamed gums and teeth, and increased incidence of dental caries, and loss of denture stability in people who have lost their teeth. Patients with their own teeth should be given sugar-free nonacidic saliva substitutes, strict dietary advice, and chlorhexidine mouthwashes; they require special monitoring by their dentist. Medical name is **xerostomia**.

Dysgeusia - a distorted, frequently unpleasant, taste perception.

F

Flavor - the combination of taste, smell, and other sensory aspects of food.

Free articulation - articulation that is unobstructed by cuspal interference.

Functional impression - an impression modified by masticatory loads and adjacent muscular activity.

G

Gerodontics - the treatment of dental problems of aging persons.

Gingiva /gum – dense fibrous tissue, covered by mucous membrane that covers the alveolar processes of the upper and lower jaws and surrounds necks of the teeth.

Gnathodynamometer - an instrument for measuring the force acted at closing the jaws.
Gnathology - study of the jaws functions and related disorders.
Group function - multiple contacts between the maxillary and mandibular teeth on the working side during lateral movements.

H

High lip-line - the highest level which the margin of the upper lip achieves in function.
Hypodontia, oligodontia or partial anodontia - absence of some teeth.

I

Immunity – the ability to resist damage from foreign substances such as microorganisms and harmful chemicals for example toxins released by microorganisms.
Immunoglobulins – proteins that have antibody functions and provide humoral immunity; they belong to classes A, D, G, M, E.
Innate immunity – immune system response that is the same on each exposure to an antigen; there is no ability to remember a previous exposure to a specific antigen.
Interferons – small proteins inhibiting the multiplication of viruses inside host cells and also possessing radioprotective, antitumorogenic and immunomodulative properties.
Interleukins – biologically-active substances that participate in interleucocytic integration (in part, during non-specific resistance and immune reactions).

J

Jaw relation - A relation of the mandible to the maxilla.

L

Lymphocyte – a type of mononuclear leucocyte; the cell responsible for humoral and cell-mediated immunity.

M

Macrophage – any large mononuclear phagocytic cell that contributes to both specific and non-specific immunity.
Mastication – chewing act.
Masticatory system - the oral structures engaged in mastication.
Milk occlusion – the relation of the upper and lower deciduous teeth when they are in contact in children.
Milk tooth – one of the first set of teeth comprising 20 in all, that empts between the mean ages of 6 and 28 months of life. Synonyme: **deciduous dentition**.
Modality - sensation associated with one sensory system, such as vision, hearing, or taste. Multimodal responses are responses to stimuli that activate different sensory systems.
Molar – a tooth having a somewhat quadrangular crown with four or five cusps on the grinding surface; the root is bifid in the lower jaw, but there are three conical roots in the upper jaw; there are six molars in each jaw; three on either side behind the premolars in

the permanent dentition; in the deciduous dentition there are but four molars in each jaw, two on either side behind the canines.

N

Neck of a tooth – a continuous anatomical irregular curved line marking the junction of the crown and the root of a tooth.

Non-working side or contralateral side - the side opposite to the working side.

O

Occlude - to bring the mandibular and maxillary teeth into contact.

Occluding surfaces - those surfaces of the teeth, or tooth substitutes, which make contact with those in the opposing jaw.

Occlusal surfaces - the surfaces of molar or premolar teeth which would normally occlude with an opposing tooth.

Occlusion - any contact between teeth of opposing dental arches.

Oral vestibule – that part of the mouth bounded anteriorly and laterally by the lips and the cheeks, posteriorly and medially by the teeth and/or gums, and above and below by the reflections of the mucosa from the lips and cheeks to the gums.

P

Pantograph (oral) - a set of tracing devices attached to the mandible and maxilla which records mandibular movements is three planes.

Papilla (interdental) - the interdental gingival tissue.

Parotid gland – the biggest salivary gland among big salivary glands.

pH scale – a measure of the hydrogen ion concentration of a solution; the scale extends from 0 to 14,0; a pH of 7,0 being neutral. A pH of less than 7,0 – acidic, and a pH of greater than 7,0 – basic.

Phagocytosis – process of substances ingestion and digestion by cells such as other cells, bacteria, cell debris, and foreign particles.

Pocket – a diseased gingival attachment; a space between the inflamed gum and the surface of a tooth, limited apically by epithelial attachment.

Polyphagia – excessive eating.

Prototypal taste stimuli - stimuli that represent taste qualities in relatively pure form, such as sucrose for sweet or quinine for bitter.

Pulp – the soft tissue inside a tooth, consisting of connective tissue, blood vessels, nerves, and lymphatic vessels.

R

Root of tongue – the posterior attached portion of the tongue.

Root of tooth – that part of a tooth below the neck, covered by cementum rather than enamel, and attached by the periodontal ligament to the alveolar bone.

Rooting reflex – in infants, rubbing or scratching about the mouth causes a puckering of the lips.

S

Saliva – a fluid containing enzymes and mucus, produced by the salivary glands and released into the oral cavity.

Salivary gland – gland opening into the mouth and producing saliva.
Selective grinding or spot grinding - the planned adjustment of the occlusal forms of teeth by grinding.
Sublingual gland – one of a pair of big salivary glands located below the tongue.
Submandibular gland - one of a pair of big salivary glands located below the mandible.

T

T-cell – a type of lymphocyte that provides cell-mediated immunity (in contrast to B-lymphocytes that provide humoral immunity through the secretion of antibodies); there are three subpopulations of T-cells: cytotoxic (killers), helpers and suppressors.

V

Vallate papilla – one of eight or ten projections from the dorsum of the tongue forming a row anterior to and parallel with the terminal sulcus; each papilla is surrounded by a circular trench (fossa) having a slightly raised outer wall (vallum); on the sides of the vallate papilla and the opposed margin of the vallum one can see numerous taste buds.
Vermilion border or **lips red margin –** the red margin of the upper and lower lip, which starts at the exterior edge of the intraoral labial mucosa ("moist line") and extends outward, terminating at the extraoral labial cutaneous junction; a thinly keratinized type of stratified squamous epithelium deeply penetrated by well-vascularized dermal papillae.

W

Watery saliva – saliva containing water or resembling it by its consistence. This state can be observed at vagotony.
Wisdom tooth – eithth permanent tooth in maxilla and mandible on each side; the most posterior tooth in human dentition.

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