Translation from
Romanian by
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Paediatric dentistry

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CONTENTS

1 *DEVELOPMENT PERIODS OF CHILDREN*

CHILDHOOD PERIODS

The childhood is a stage of life situated between birth (0 years) and 18 years, that is characterized by quantitative accumulations and qualitative leaps, which provide appearance of new qualities and functions, that offer to the individual a better adaptation and correlation to the environment.

Childhood periods and stages by *Roland Planche*

I. **The first childhood** (0 - 3 years – the small childhood) contains many stages:

- pre-birth period
- the moment of birth
- a new born baby (0 - 30 days)
- nursling (suckling) (30 days – 12 months)
- small child (1- 3 years)

II. **The second childhood** (3 - 6/7 years) – the middle childhood, preschool child

III. **The third childhood** (6/7 – 14/16 years) – the big, large childhood, contains two stages:

- first stage 6/7 – 10/11 years for girls; 11/12 years for boys
- prepuberty 11 – 14 years for girls; 12 – 15 years for boys

IV. **Puberty** (14 – 16 years)
V. **Adolescence** (teenage 16 – 20 years)

The physiological and physio-pathological particularities of the childhood

I. The first childhood (the small childhood):

▪ **pre-birth period**: in this period the genetic fund (genetic patrimony) and the intrauterine peristasis factors have an important part.

Troubles: cellular undifferentiation; the absence of facial buds union; troubles of bronchial membranes resorption

Prevention: genetically advice; the avoidance in the first part of pregnancy, of radiation, anaesthesia, alcohol, tobacco, drugs; the treatment of mother's acute and chronically infections.

▪ **the moment of birth:** is a traumatically moment. The compression of cephalic extremity causes hypoxia and goes to a lower mineralization of primary teeth enamel (circular Stein caries).

▪ **0-6 month**: absence of the child's immunity;

▪ **30 days – 1 year**: the neuro-vegetative phenomena prevail. Maternal milk feeding is very important and it is possible due to the inborn suckling reflex.

Advantages: nourishing principal contribution; immunity factors contribution; the harmonious psycho-somatic development; the continuation of mother-child affective symbiosis; superior maxillary development; the first physiological mandible mesial movement; in opposition, artificial feeding is unfavourable (use of an inadequate baby's dummy produced traumatical stomatitis Bender); deficiencies in oral hygiene of the child and Candida albicans are favourable for infectious stomatitis; 3-4 months: permanent teeth mineralization; around 6 months: being the deciduous teeth eruption (gingivitis by eruption, eruptive stomatitis Riga); 0-12 months: pathogenical interrelation between artificial feeding, repeated dyspepsia.

The stages of: new-born (0-30 days) and suckling (1-12 months) are the most important aspects of growth and development, instalment general and cranio-facial.

▪ **1-3 years**: the closing the fontanella; ossification of skull (1-2 years); slow rhythm of growth; perfecting of the locomotor's function; development of a second system of signal – speaking; contacting with children's community and the first contagious diseases of childhood; metabolic troubles (2,3 – 3 years) and graves hypo-calcifications of teeth; sequential continuation and finalising eruption of primary teeth; the first occlusion elevation (physiological highest of occlusion) by the clinical eruption of the first temporary molar (at 18 months); installing of the vicious habits; the function of maxillo-facial's apparatus is more complex; circular caries of primary teeth (sweetening in excess the artificial food with suckling bottle and by deficiencies in: oral hygiene and child nutrition).

This stage (1-3 years) is favourable to make correctly the oral hygiene, nutritional hygiene and general fluoridation (in function of fluoride concentration in air, water, food) properly.

II. The second childhood (middle childhood): the general rhythm of growth – slowly (slow down); psycho-somatic development accentuated (increased); contact with pre-school's community – contagious diseases of childhood, favourable conditions for: - teeth hypoplasia; teeth hypo-calcification; caries (about accent's dehydration and loss of mineral salts); the development of childhood leads to a certain autonomy marked a strong psycho-somatic development.

From a stomatological point of view: the appearance of vicious postural attitude; the appearance of the first caries (deficiency of oral, functional and nutritional hygiene) the first stomatological consultation.

That is a favourable period for: teaching, strengthening and improving correctly oral and nourishing hygiene habits;

prevention and intercepting treatment of caries and dental-maxillary anomalies at individual and community level; continuing the fluoridation: general and local.

III. The third childhood (the big childhood): initial development is slow; temporary teeth risalisa and exfoliation; sequential eruption of permanent teeth.

The pre-puberty stage (10 - 11/12 years): for boys prevail stature growth; for girls prevail weight growth; very active metabolism; hormonal period, when general affections (tuberculosis, osteosclerosis, osteoporosis) may appear; pre-pubertal rickets (through deficiency D, C vitamin, malnutrition); chronic acidosis; phosphate diabetes; temporary teeth risalisa and exfoliation; sequential eruption of permanent teeth; coexistence of the temporary and permanent teeth.

IV. The puberty stage: passing from childhood to adolescence: the appearance of secondary sexual character difference debut between sex (the children's morphology is typical for this stage); maturation of the sexual function; profound psycho-somatic transformation, which are reflected in behaviour; psychic crisis.

From a stomatological point of view: sequential eruption of permanent teeth; finalising of the permanent teeth eruption (M2 included); temporary teeth risalisa and exfoliation; coexistence of the temporary and permanent teeth; change's period of teeth – instability in occlusal relation; the second (M1) and third (M2) physiological occlusion elevation (highest of occlusion).

Troubles: wrong dental care habits during the mixed dentition (persistence of vicious habits, dysfunction, and deficiency of oral and nutritional hygiene) result in: caries (symmetric caries with growth prevalence); gingivitis of eruption; periodontal irritation events; pre-pubertal periodontitis; transitory or permanent dental alveolo-maxillary anomalies.

This period is favourable for: prevailing correct and consistent habits and preoccupations concerning: the oral, nourishing and functional hygiene.

V. The adolescence: passing from puberty to young adult (16-20 years): the psychic characters are entirely developed; the residual growth is possible.

The young adult: the destiny develops in terms of emphasising the independence and the individual autonomy; the individualisation of a psycho-somatic equilibrium, more or less harmoniously, between desires and reality.

DYNAMICS OF DEVELOPMENT OF THE DENTITION IN SYSTEMIC CONCEPT

The development of dentition (odontogenesis) is a complex biological process of teeth formation and development between 6^{th} week intrauterine (i.u.) and 20-25 years.

Odontogenetic evolution depends on: genotypes; peristasis factors; time and space action period goes to phenotypes.

This process spreads in the context of dental-maxillary apparatus development (ontogenesis) which includes 3 types of biological processes: the growth under the influence of the genetic factors; the development under the influence of: peristasis, neuro-endocrine, nutritive and partial-functional factors; the modeling under the influence of the functional factors.

Periods of ontogenesis of dento-maxillary apparatus

A. Organogenesis (0-3 months i.u.)

B. Morphogenesis (4 months i.u. – 14/16 years) with two phases:
- foetus-genesis (4 months i.u. – till birth)
- after birth – till 14/16 years

C. Maturation (16 – 20/25 years) and modeling (16 – 60/70 years)

In the third week i.u. life the primitive oral cavity is formed. There are five brachial arches in its near neighborhood.

In the second month i.u. from the first arch (mandibular) and cells multiplication from cephalic extremity, five facial buds occur:

- naso-frontal bud (with 1 internal naso-bud and 2 external naso-buds)
- maxillary buds – 2
- mandibulary buds – 2

The odontogenesis

In 6-7th week i.u. life, in the superior and inferior maxillary we can see the epithelium growing large and forming basal lamina. At this level, the phases of odontogenesis are by MC Donald, Pinkham:

A. 4 proliferation phases:
  1. the initiation
  2. the proliferation
  3. the histological differentiation
  4. the morphological differentiation

B. the calcareous apposition and calcification (maturation)

C. eruption of the teeth.

The same phases for all the teeth do not take place all at the same time and at the same stages of development.

The Initiation Stage

In the second month of i.u. life, from the basal lamina there appears: vestibular lamina outward (which forms the vestibular ditch through vacuolization) and primal dental lamina inward (from the proliferation of epithelial cells from basal lamina and from rounding mesodermal tissue).The cells in the basal layer of the oral epithelium undergo proliferation faster than the adjacent cells; the result is an epithelial thickening in the region of a future dental arch that extends along the entire free margin of the

jaw.Between 6-10th week i.u. life from primal dental lamina on its free side 20 digitations are formed – 20 dental buds for primary teeth – 10 round or ovoid swellings occur in each jaw maxillary in the symmetrical disposition. This is the bud stage: 6th week i.u. – incisor and canine; 8th week i.u. – first molar; 10th week i.u. – second molar.In the 4-5th months i.u., at the level of deciduous (primary, temporary) teeth buds pediculus, of an internal face of primal dental lamina the secondary dental lamina is formed out of which 20 buds for the following permanent teeth will be formed: 5th month i.u. – central and lateral incisors; in the first 2 years after birth the first and the second premolar, which appears behind the primary molars buds, towards the oral.Between the 4-5th months i.u. and 4-5 years after birth, is formed dorso-distal section of secondary dental lamina out of the tertial dental lamina for 12 completing permanent teeth buds (and it advances posterior into the mesenchymal tissue of maxillary): 4th month i.u. – first molar; 8th month i.u. – second molar; 4-5 years after birth – third molar.

Determinate regulator factor of this stage is genetic induction.

Perturbations: dental agenesis (oligodontia and anodontia); supplementary teeth (pleiodontia, supernumerary).

The Proliferation Stage

By the unequal development and multiplication of a dental bud the epithelial capsular stage is achieved – cap stage with: 2 epithelial layers and stellate reticulum.The mesodermal proliferated tissue and subsequent invaginated and incorporated raises the dental papilla.

By Orban quoted by Pinkham, the tooth germ during this stage has all the necessary formative tissue to embrace the development of a tooth and its periodontal ligament. The tooth germ is composed of: dental organ, dental papilla and dental sack.

Excessive proliferation of cells may generate: epithelial rests – cysts developing, odontoma and supernumerary teeth.

Deficiency of cells proliferation may generate: oligodondia and anodontia.

The Hystodifferentiation Stage

The characteristics of the stage: cellular differentiation; morphological specialization, both with alternative, inductive and receptive role.

Due to the invagination of mesenchymal tissue in the finishing part of the tooth germ, it takes the shape of a bell – bell stage, when dental follicles appear, and it is composed by: enamel organ (ectodermal origin), dental papilla (mesodermal origin) which gives rise to: dentine, dental pulp, alveolar bone, periodontal ligament.

Enamel organ is made up of: external enamelar epithelium: cubical cells with protective role; stellate reticulum (the stellate cells of the enamel pulp) with nutritive and protective role; intermediate stratum more layers of small cubical cells with big nucleus; internal enamelar epithelium high hexagonal cells – pre-ameloblasts which becomes ameloblasts – surrounds the dental papilla with a role in the: formation of enamelar matrix, induction of: - appearance of odontoblasts, differentiation of mesenchymal cells for the appearance of dental sack: fibroblasts, cementoblasts, osteoblasts; definitivation of the future tooth dimensions by entering the epithelial root sheath of Hertwig von Brun composition; basic membrane separates ameloblasts from dental papilla – future amelodentinar junction.

Dental papilla is a condensed mesenchymal tissue with a precocious vascularization. There are two zones: central one – future pulpal zone; peripheral one – future dentinogenesis zone. From the interaction of epithelium with mesenchymal tissue – fibroblasts differentiate into odontoblasts (with a role in the formation of dentine) disposed in palisade and sending to enamel Tomes cytoplasmatic fibers.

Follicular sack is due to the condensing mesenchymal tissue

around dental bud. There appears: cementoblasts - cement, osteoblasts - alveolar bone, fibroblasts - periodontium (alveolo-dental ligaments).On the mesenchymal tissue we can se two vascular nets: intradental and peridental.

Disturbance of this stage: aplasia – anodontia (not generating the follicular sack); hyperplasia – pleiodontia; proliferation of undifferentiated cells: odontoma, adamantinoma; imperfect amelogenesis – as a consequence of cells lack of specialization; imperfect dentinogenesis; congenital mesodermal dysplasia: disoriented dentine, absence of dental pulp, advanced destruction of the coronary structure; tumoral forms with undifferentiated cells.

The Morphodifferentiation Stage

The morphodifferentiation stage, as its name implies, is a stage at which the cells find an arrangement that ultimately dictates the final size and shape of a tooth. In this stage, ameloblasts and dentinoblasts form an organic matrix of enamel and dentine, which gives the shape, the volume and the morphology of the tooth. We find two different processes: dentinogenesis – which precedes and follows what comes next, that is amelogenesis.

Dentinogenesis is the result of two stages:

- the cellular stage: the odontoblasts release the primary forms of organic matrix under the influence of acid-phosphatase and ameloblasts;
- the extracellular stage: biochemical and enzymatic changes.

Amelogenesis: under the influence of a forming of the first dentine layer and the acid-phosphatase, the preameloblast becomes functional ameloblast, which releases an organic matrix (proline and oxiproline).The release of enamel and dentine structure form layers till we get to the final form of the tooth.

Disturbance of this stage: modification of the form, volume; supplementary tubers, cuspids.

The Apposition and Calcification (Maturation)

The appositional stage occurs when a network or tissue matrix of the tooth is formed. The cells having the potential for the deposition of the extracellular matrix fulfill the plan of the tooth germ established by previous stages. The growth is appositional, additive, and regular. This accounts for the layered – just as appearance of enamel and dentine. Under the influence of phosphorylase and alkaline phosphatase calcium impregnation starts and this means the formation of mineral fraction (apposition). The binding of calcospherix through an homogenous calcious substance with programmer, length and rate genetically determined – results in enamel maturation which begins before birth and continues after birth – several months for primary teeth and 2-3 years for permanent teeth (calcification – maturation). The mineralization of the dentine begins in the 5^{th} month i.u. for deciduous teeth and towards the end of the 8^{th} i.u. month for the permanent first molar. For the other permanent teeth it begins at certain birth intervals and ends at 1 – 1,5 – 2 years for primary teeth and for the permanent teeth it lasts till the third molar development. The process goes on at a certain rate in two stages: slow apposition of calcium; release of water, minerals. After the mineralization of the dentine layer, a new layer of predentine is formed. By the gradual thickness of the dentine layer, the odontoblasts are retreating centripetally, leaving the tomes fibers in dentinal canals. These fibers have a nourishing and sensitive role. The mineralization is the end of complex process in which the organic matrix is gradually impregnated with calcium phosphate crystallized – in hydroxyapatite. The proper dentine contains 70% minerals, 18% organic fraction, 12% water. The succession of intense dentinogenesis events and rest intervals results in Owen lines. The apposition takes place at the same time with the mineralization process which has as result the growth lines von Ebner. Before birth mineralization is qualitatively

higher than a post birth mineralization and it is separated by neonatal line Schoor-Massler-Orban.

Structurally and Topographically:

- peripheral dentine – under the enamel and the cement, has no canals and has lower mineralization
- peripulpal dentine – formed by the overlap of dentinal metabolic units (Tomas fibers, pericytoplasmatic space, dentinal canal, pericanalar dentine), is orientated in right angle to pulpal chamber ceiling
- intercanalar dentine between two metabolically units
- the crown dentine has incomplete mineralized areas, named Czermarck spaces.

Related to formative process and tooth age, we differentiate:

- primary dentine
- secondary dentine
- tertiary dentine (irritation dentine)
- sclerotic dentine (in chronic caries, aged persons).

Enamel mineralization is achieved by continuously successive deposits of minerals in the organic matrix – both in the releasing phase and in the maturation phase under the phosphatase control. Initially mineral fraction is 30%. During the maturation phase, the protein release falls and the mineral fraction gets to 92-93% for primary teeth and 95-96% for permanent teeth. Intra- and inter-prismatic crystals form a 45-60 degrees angle at the end of the mineralization (Schreger strips). The succession of the enamel growth process results in Retzius lines from the junction to the enamel surface. The enamel mineralization starts from the tops of the cuspids to the cervical loop and forms the depth to the surface, centrifugal. The crystals contain 98% OH-apatytes, ions of: Mg, N, Cu, Zn, Mn, F (more concentrated towards the depth) – solubility factor. The fluoridation is indicated in the last quarter of pregnancy (F – passes through the

placenta). A more intense calcium apposition activity is at the enamelar growth centers.

Disturbance of this stage: 1) by A and D avitaminosis in: calcium apposition phase - hypoplasies, hypo-calcifications; calcium maturation phase: late hypo-calcifications cause caries susceptibility (F – administration is indicated); 2) dental dystrophies – by the disappearance of prisms during the growth crisis; 3) maternal metabolically troubles cause calcification trouble for primary incisors; 4) obstetrical trauma during birth cause the circular stein caries (at primary upper incisors and molars); 5) during childhood periods the variable hypo-calcifications.

Dental pulp is formed in the 8th week i.u. gradually as follows: one artery; 2-3 veins capillaries around the odontoblast; lymphatic vessels surrounded by amyelinic fibers; myelinic fibers in the center which go into the odontoblast near the core and surround it. Pulp chamber has un-extendable walls.

Roots formation – internal enamelar epithelium has an inductive role. After enamel formation the stellate cells of the enamel pulp are resorpted and remain the primary enamel membrane, which provides the final dimension of the crown. When the enamel-dentine layer is thick enough, a new epithelial process starts, releasing a root organogenesis. At the cervical loop the internal and external enamelar epithelium bind and form the tissue muff, this gets into the mesenchymal papilla, limiting a pulp proper territory. This territory is isolated from the follicular sack by the epithelial root sheath Herwing von Brun, which induces the root formation. During the depth advancing of the epithelial diaphragm, the root odontoblasts are differentiated producing the root pre-dentine matrix, which will become the root dentine that continues the crown dentine. The multiple roots teeth have one epithelial sheath for each root. From the sheath there may remain epithelial rests called Mallasez rests.

The terms of complete formation of temporary teeth roots are presented in table 1.1, and for permanent teeth roots in table 1.2.

Table 1.1.

The terms of complete formation of temporary teeth roots

Temporary tooth	**Term,** years of life
Central incisor	1,5
Lateral incisor	2
First molar	4
Canine	5
Second molar	4

Table 1.2.

The terms of complete formation of permanent teeth roots

Permanent tooth	**Term,** years of life
First molar	9-10
Central incisor	9-10
Lateral incisor	9-10
First premolar	12-13
Canine	12-15
Second premolar	12-14
Second molar	15

For formation and maturation of radicular apex after eruption, the temporary teeth necessity 9-18 months, but the permanent teeth necessity 1-4 years. The longest period for root formation is necessary to the first and third molars (3-4 years), but the shortest – to the second premolar (1-2 years).

In process of radicular apex formation radiologically has been

distinguish some stages: 1) root with incomplete formed apex; 2) root with formed apex and open canal orifice; 3) root with formed apex and closed canal orifice.

In *I stage*, the root length reaches to the normal dimensions, its walls are parallels, makes thin to the radicular apex; the radicular cannel is large, is funnel-shape in apical region, what passes in the growth zone – a focus of osteoporosis delimited on the outskirts at cortical of bone. The periodontal space can be observed only on the lateral walls of root, but in apical region it is missing.

In *II stage* the radicular apex is formed; the growth zone is missing; the walls of radicular cannel are formed, in apical region are rounded and to approach one to another; the radicular cannel is large, but the diameter in apical region is smaller; the periodontal space is large, more in apical region.

The *III stage* is the stage of apex maturation, owing of apical orifice closing with cement radiological the radicular canal don't communicate with apical periodontium.

Root calcification complete in first dentition 1 to 1,5 years after eruption.

Root calcification complete in second dentition 2 to 3 years after eruption.

Cementogenesis is initiated in the 8^{th} week i.u. from the mesenchymal tissue of the follicular sack. By the disappearance of Hertwig sheath, the root surface remains uncovered, inducing the mesenchymal fibroblasts. They form the thin granulated matrix – cell free cement, which is thinner in the upper root third and gradually grows to the apex. To the end of the apex formation the cementoblasts are included in their own collagenical matrix, forming the cell cement. The root is getting thinner to the apex, which makes room for the vessels and nervous – apex foramen. Some cementoblasts maturate and include themselves into the cement – these are cementocits (with a nourishing role). The most

of the cementoblasts aligned at the edge of the mineralized layer, producing successive layers of cement during the whole life. The forming rate of cement in the eruptive time contributes to the establishment of the root length. The cement covers the root dentine in successive layers and steps at the cervical loop: in direct contact with the enamel (30%); covered by the enamel (60%); space between enamel and the cement (10%). From a histological point of view, the cement belongs to the tooth; functional and pathological cements belong to the periodontium. The cement is vessels free, that is why it resists to the resorption. Like the dentine and unlike the enamel, the cement remodels and after the end of the formative stage by apposition.

Periodontium development – takes space at the same time with cementogenesis: by the initiation of clinical eruption, the coronary fraction of the follicular sack disappears and bends at the cervical loop level, forming gingival sulcus; the bunches of collagen fibers from the root follicular sack form the periodontal ligament with an extremity on the cementoid matrix and the other extremity on the developing alveolar wall (Scharpey fibers).

The alveolar base – bone organic matrix formed by the precipitation of phosphates and carbonated salts around the collagen fibers. The precipitation does not occur where the collagen fibers continue from the alveolar process to the cement. The bone keeps in touch with the formative stage to remodeling permanently by the resorption-apposition process.

The tooth eruption

It represents the last stage of odontogenesis.

Tooth eruption describes the changes in tooth position from the earliest time of development through successive stages to emergence into the oral cavity. It includes, as well, the achievement of occlusal contact and subsequent changes caused by growth of facial skeleton and attrition.

According to the same author, the eruption of a tooth in this

context is the movement towards occlusion: the emergence is the perforation of the mucosa and the tooth into the mouth.

Stages

There are three different phases:

I. pre-eruptive phase
II. eruptive phase (pre-functional)
III. eruptive phase (functional)

The speed and direction of the tooth movement in eruption is not constant, it is differentiated according to each phase.

I. The pre-eruptive phase:

The tooth root begins its formation and begins to move from its bony vault, towards the surface of the oral cavity. This phase consists of that period of development in the tooth root through gingival emergence. Initially, the movement is in transversal direction and then – axially. The tooth movement is not constant – a slow movement in the direction of the occlusal plan is followed by a quick and slow movement. In the first movement of slow movement the displacement corresponds to the increase in root length.

Theories about the eruption movements of the tooth in this phase:

- growth of the root
- growth of the dentine and pulpal tissue
- growth of the alveolar bone
- pressure from:
  - cellular proliferation of the epithelial root sheath
  - the pulp tissue
  - the adjacent muscles
- vascular expansion

II. The eruptive phase (pre-functional stage) is the period of root development through gingival emergence and it is

characterised by: tooth root is usually approximated on 1/2 - 2/3 on its final length at the time of gingival emergence; periodontal elements formation; tooth movement is prevalently axial, accompanied by transversal and mesial movements; the root continues its growth (till it reaches the normal length); the factors than influence the eruption mechanism in this phase are: changes in the alveolar bone; the adjacent oral-facial muscles and the tongue; movement direction is influenced by functional matrix.

III. The functional phase begins after the tooth erupted into the oral cavity and meets its antagonist (opposite tooth in the opposite arch). The teeth will continue to move and erupt as long as the body continues to change along life. The factors that influence the eruption mechanism in this phase are: general, local and functional.

According to modern theories, the whole dental eruption represents the result of the interaction installed between root growth and alveolar septum development; the mediator factors are represented by viscosity and maturation modification, which take place in periodontal ligament.

The sequence of tooth eruption is influenced by: geographic factor, racial factor, ethnic factor, constitutional factor, genetic factor, general factors and systemic disorder, local factor, functional factor, birth weight of the child: small – permanent teeth delayed emergence, increased – early eruption; nutrition; mother's age at birth and number of birth: young mother – precocious tooth eruption of the child, advanced age – delayed eruption; the socio-economic level; environmental factors; sex: for girls - early eruption, for boys - delayed eruption.

Sequence of the temporary (primary) teeth eruption:
Period: 6 - 30/32 months on dental group.
Intervals: at each 6 months with variability of +/- 2-3 months

in the crisis of growth and +/- 6-8 months between the crisis periods.

Order of teeth eruption: 1, 2, 4, 3, 5 (first mandibular, second maxillary, with the exception of lateral incisors).

Terms of temporary teeth eruption:
Central incisor - 6 - 8 months of life;
Lateral incisor - 8 - 12 months of life;
First molar - 12 - 16 months of life;
Canine - 16 - 20 months of life;
Second molar - 20 - 24 months of life.

Sequence of the permanent teeth eruption:

Period: 6 - 18/25 years – on the dental groups.

Intervals: every 1 year – for substituted (successors) teeth (1, 2, 3, 4, 5) and 6 years for teeth of completion (M1, M2, M3) with a normal variability: +/- 4-6 months in the crisis of growth and about +/- 1 year between the crisis periods.

Sequence may be: prevalent molars, prevalent incisors, and incisors – molars.

Order of teeth eruption: for the maxillary:

- 6, 1, 2, 4, 5, 3, 7, 8
- 6, 1, 2, 4, 3, 5, 7, 8

for the mandible:

- 6, 1, 2, 3, 4, 5, 7, 8
- 6, 1, 2, 4, 3, 5, 7, 8
- 1, 6, 2, 3, 4, 5, 7, 8

The eruption of the permanent teeth situated mesial to the first permanent molars is associated with the physiological resorption (risalisa) of the root of the temporary predecessors, their exfoliating and their investing alveolar bone. The way in which the risalisa occurs is more or less related to the morphology of the approaching crown of the successors.

The eruption and emergence of the first, second, and third molars follow different pattern from the rest of the permanent

teeth (they have no primary teeth preceding them). Their eruption depends on the spatial condition of: the jaw, enlargement of the posterior section of the apical area, and dental arch.

At the moment of the clinical eruption completion of M1 and M2 permanent, the second and the third physiological rising of occlusion are produced (from 2/3 to 1/2, to 1/3) and simultaneously a new mesial sliding of mandible, takes place all along with: maturation of mandibular condyles; centric and usual relation of occlusion; individual functional envelope.

Terms of permanent teeth eruption:
First molar – 6 years of life;
Central incisor - 6 - 8 years of life;
Lateral incisor - 8 - 9 years of life;
First premolar - 9 - 10 years of life;
Canine - 10 – 11years of life;
Second premolar - 11 – 12 years of life;
Second molar - 12 – 13 years of life.

Variations in the sequence of teeth eruption

The mandibular first permanent molar is often the first tooth to erupt, followed quickly by the mandibular central incisors (it is a prevalent molars eruption). For teeth templates – 3, 4, and 5 – there are differences in order of eruption, depending of sex and jaw (maxillary or mandible). In the mandible, the most favorable sequence of eruption of permanent teeth is: M1, IC, IL, C, P1, P2, M2; in the maxillary, the most favorable sequence is: M1, IC, IL, P1, P2, C, M2 (Moyers).

With girls, the mandibular canine erupted before the maxillary and mandibular premolar. With boys, frequently, the maxillary and mandibular first premolar erupted before the mandibular canine.

The clinical situation is desirable when the mandibular canine erupts before the first and second premolars, thus being maintained an adequate arch length preventing the lingual tipping

of the incisors (Mc Donald). When second permanent molar erupts before the second premolar, it is an undesirable situation, because, it exerts a strong force on the first permanent molar, causing the mesialisation of the M1 and the closing of space for the second premolar.

In the maxillary, ideally, the first premolar erupts before the second premolar, followed by the canine. When the P2 erupts before the P1, and especially, when the M2 erupts before the premolars, it produces the loss space for upper canine.

Pathological variability of the tooth eruption manifests itself through: local, regional and general (systemic).

For temporary teeth

Local influences refer to period, sequence and order of the eruption: increased delay of eruption (in: rachitis, congenital syphilis, antenatal rubeola, TBC of pregnant woman, chromosome anomalies – Langdon-Down syndrome, cleido-cranial dysostosis, hypophysar and thyroidal disendocrinia. Premature eruption: birth (natal) and co-birth teeth – one or more teeth, usually members of the normal series or supernumerary elements – present at birth; neonatal teeth – appearance after the first 30 days from birth; Cap de Pont ejected follicles. Disorders concerned: the position (oral, lingual, overlapping, round, the place and structure.

Inflammatory local and regional complication: flushed pericoronaritis, suppurated pericoronaritis, gingival abscess, eruptive gingivo-stomatitis (Riga), Cap de Pont ejected follicles, eruption cyst, and regional vasomotor and secretor reactions: hypersalivation, hyperemia and conjunctivae congestion with epiphora and photophobia, rhino-rhea, circum-oral rash, over added infection: facial eczema, impetigo, herpes, and conjunctivitis.

General (systemic) complications: general irritability, crying, excitement, convulsions, sleeplessness; digestive disturbance:

reduced appetite, loss of appetite, increased thirst; breathing disturbance; urinary disturbance; troubles of growth in weight and height.

Treatment regards: *local:* teething toys, teething foods, topical drugs and surgical treatment; *systemic:* analgesics, hypnotic and sedatives, vitamin-therapy and antibiotics.

For permanent teeth

Local influences concerned: *period of eruption:* delayed eruption by reinclusion of temporary predecessor tooth, temporary tooth trauma, radicular-alveolar sclerosis, alveolar hyperostosis (in deep bite), dilaceration, movements of limitrophe teeth, osteopetrosis, osteosclerosis secondary to the infectious processes, Duchateau syndrome, post-traumatic osteosclerosis, premature loss or extraction of temporary tooth, disturbance of risalisa of temporary teeth, pulp necrosis, periodontal affection of temporary tooth, supernumerary teeth, praevia formation, rachitis, D-hypovitaminosis, nutrition disturbance (chronic), hypofunction of tyroid and hypophysis, hypogonadism, genetic factor (Down syndrome) ; premature eruption: facial hemangiomas, Turner dental syndrome (P2), premature loss and extraction of temporary teeth. *Sequence and rhythm of eruption:* asymmetries and inversions – as a consequence of general and local factors. *Modification (anomalies) of:* possession: oral, lingual, distal, mesial, round; place: ectopic eruption, transposition, dental inclusion, dental heterotopia; *anomalies of the developing dentition* (by J.R.Pinkham): anomalies of number: hyperdontia, hypodontia, oligodontia, anodontia; anomalies of size: microdontia, macrodontia, fusion, germination; anomalies of shape; anomalies of structure: *enamel*: imperfect amelogenesis, environmental enamel hypoplasia, localised enamel hypoplasia; *dentine*: imperfect dentinogenesis, dentine dysplasia, odontodysplasia; *cement*; anomalies of colour.

Inflammatory local and regional complications: compared to temporary teeth they are more reduced. Their clinical forms are M1 and M3 pericoronaritis.

***General complications*:** nervous, digestive and somatic growth.

The root physiological resorption (risalisa)

It is specific only for the temporary teeth. It begins after one year from the eruption of the temporary tooth or two years before the replacement, with a variable chronology depending on: group, age, sex, race, local factors (infection, inflammation, trauma, isolated dental anomalies, dento-alveolar pre-existent anomalies).

Mechanism: two factors are incriminated:

✓ **the main factor** is represented by the permanent following tooth, pressing the mesodermal tissue around the crown and transforming the cells in cementoclasts and osteclasts which provoke the resorption of the nearest tissue, including a temporary tooth root

✓ **the second factor** is represented by the dental pulp of temporary tooth that releases a self resorption process.

The radicular resorption starts on the contact surface with the permanent tooth, and the root disappears completely (gradually). The crown is maintained only by bunches of dental-gum fibbers.

The permanent incisors had an oral position, compared with the deciduous incisors, and they move forward and downwards (at maxilla) and forward and upward (mandibular), which provide an oblique resorption from oral to vestibular of temporary tooth root.

At the moment of the clinical eruption the permanent teeth are situated in vestibular position from temporary teeth widen the arch. When the resorption does not take place, the permanent teeth erupt in oral or vestibular position, achieving a double occlusion. The premolar buds are situated between the roots of deciduous molars where the resorption starts. The vestibular roots disappear faster. The resorption rhythm is discontinuous.

The terms of beginning of the temporary teeth root physiological resorption:

Central incisor – 4 years of life;
Lateral incisive – 5 years of life;
Canine – 8 years of life;
First molar – 6 years of life;
Second molar – 7 years of life.

Pathologic aspect of radicular resorption

- The premature resorption and atypical pattern of resorption of primary teeth (in case of pulp septic necrosis with periodontal affection, repeated acute apical osteitis, dental-alveolar incongruence through macrodontia, trauma and iatrogenic procedures.
- Less frequently we can meet a slowness and stopping of this process, or as already mentioned the intact root. For the permanent teeth, the radicular resorption is exclusively pathological in the case of: sever incongruence; after: trauma, periapical infections, periodontal disorders, iatrogenic paedo-orthodontical procedures; unknown cause.

The morphostructural, biological and functional particularities of the temporary teeth

- number – 20
- beginning and ontogenetic evolution prevalently prenatively
- rapid maturation
- unstable limited existence (5,5 – 8,5 years), with three physiological stages:

I. evolution period (young temporary teeth)
II. stability period (mature temporary teeth)
III. instability period (period of physiological exfoliation)

Colour: whitish, with blue-grey reflections, with less transparency lighter in colour than permanent teeth

Size, volume: frontal group is smaller than permanent frontal group (temporary incisor sum is smaller than permanent incisors sum with 4,4mm); temporary M1, M2 are bigger than premolars 1, 2 with 2,2mm (generating the "leeway space")

Form: on the whole – like the anthropoids permanent teeth crowns forms is more rounded, wider mesiodistal, relatively shorter, globular (adapted to wider infantile neuro- and viscero-cranium), more constricted at the cervical level. Vestibular and oral faces are slopes like very inclined to the occlusal face which is thus reduced in size. Cone-trunk shape (especially at the molars), with the small base to the occlusal and the big base to the cervical, where the vestibular and oral faces suddenly stop like a veritable threshold, the cement-enamel limit separating more clearly the crown from the root (compared to the permanent teeth). In 30% cases the cervical dentine is uncovered, the enamel being at the certain distance from the cement. At cervical level we remark a crest (ridge) formed by increased enamel layer, more prominent labial and lingual at anterior teeth, and oral at maxillary and mandibular first molar. The occlusal surface is smaller, the cuspids are reduced, cuspid planes are less emphasized

Structure: enamel and dentine thickness is reduced with 59% compared to permanent teeth. Enamel resistance is – 3 times smaller than in permanent teeth case. Enamel has a uniform thickness on the crown surface, excepting the cervical loop (where a threshold, crest, ridge are present). Enamel prisms – in the cervical third are oriented to the occlusal surface (at permanent teeth towards apical). Enamel prisms at cervical crest level stop suddenly, forming an abrupt slope to the anatomical cervical loop (the limit between crown and root is strangled – the neonatal line Schoor-Massler).

Roots: thinner, sharper than permanent teeth root; compared to the crown size they are bigger than the permanent teeth case.

Pulpar chamber: compared to the crown is bigger. Dentine layer is thinner, pulpar horns advance in the cuspids more than in the permanent teeth (accidental open risk during the treating caries, particularly, the mesial ones).

Root canals: thinner, narrower (with aberrant canals), more divergent at the molars (difficult for canal treatment).

There is a considerable individual variation in the size and morphology of the pulp chamber and pulp canals of the primary teeth: immediately after eruption, the pulp chamber is large, and in general, they follow the outline of the crown; the pulp chamber will decrease in size with an increase in age and under the influence of function and of abrasion of the occlusal and incisal surface of the teeth.

Biological: the pulpal organ has a regressive potential accentuated in the third physiological stage of existence.

Bio-physiological: the temporary teeth are particularized by: attrition, risalisa and physiological exfoliation.

The morphostructural, biological and functional particularities of young permanent teeth

- number – 32
- very slow maturation, especially post-natal
- persist in time
- colours: white, yellowish, grey
- morphological: bigger size than temporary teeth (excepting P1, P2)

Crown: clinical crown, after the finalising of clinical eruption is bigger than temporary teeth (in the clinical eruption period – I-II degree, the clinical crown of the permanent teeth is smaller than anatomical crown, because the epithelial insertion has not reached at the enamel-cement limit yet); hard tissues are reduced in thickness; structurally – dentinal canals are wide.

Dental surfaces present distinct morpho-functional particularities on dental templates: frontal group: IC, IL, C; lateral group: P1, P2, M1, M2, M3.

Vestibular and oral surfaces have a trapezium form, with the big base to the occlusal surface and the little base to the cervical loop allowing a better implantation of the roots in the alveolar process, which thus may present narrower zones of osseous tissue in the interdental portion and enough space vascular-nervous bundles.

Approximal surfaces - triangle like, with the base orientated cervical at the frontal group, and trapezium like, with the little base cervical orientated at the upper lateral group, and rhomb like at the lower lateral group, providing the adaptation to food cutting, and biting for frontal teeth, and a uniform distribution of the mastication forces on these surfaces for lateral teeth and simultaneously self-cleaning by the tongue and cheeks movement as well a salivary secretion.

At first molar (mesio-palatal) Carabelli tuberculum may be present (inconstantly) – factor of risk for caries. Occlusal relief is emphasised and differentiated by group functionality. Interdental contact points getting down – by slow blunting (more obvious after the age of 18-20) is changed in contact surfaces. Mesial and distal interdental areas are prefigured (except for the third molar) allowing the individual physiological dental mobility during the functions, protecting the neighboring teeth and the interdental papillae. Interdental contact area is closer to occlusal zone at frontal teeth and progressively gets closer to cervical zone at lateral teeth.

The pulp chamber is big, with a reactive biological potential.

The root: initially short, with very wide root canal (with walls diverging apically, open in funnel shape). In the apical side, root pulp has an accentuated biological formative potential

significant in apexo-genesis (the process of apex building). At many roots teeth (pluri-radicular) the roots form a common trunk and then are individualised.

Periodontium are protected and stimulated during the function of the dento-maxillary apparatus by embrasures (free interdental spaces above and below contact points). In the mixed dentition period (when temporary and young permanent teeth coexist), the periodontium is particularised through the existence of four distinct zones:

1. zones of stable lacteal periodontium
2. zones of temporary periodontium in reshuffling with:
   - risalisa of temporary tooth
   - pre-eruptive stage of permanent tooth
3. zones of eruptive periodontium
4. zones of young permanent periodontium (the tooth contacts the occlusal plan).

Control questions and topics:

1. What are the age periods children?
2. What are the development stages of prenatal period?
3. What are the critical periods of intrauterine development?
4. What are the development particularities of neonatal period?
5. Enumerate the development particularities of suckers.
6. Enumerate the development particularities of early pre-school age children.
7. Enumerate the development particularities of pre-school age children.
8. What are the development particularities of school age children?
9. How many conventional stages of teeth development one can distinguish?

10. Enumerate the intrauterine and intramaxillary teeth development stages.
11. Characterize the stage foundation and formation of dental germs – proliferation.
12. What are the main components of dental follicle?
13. Characterize the stage of dental germs differentiation – histo-differentiation.
14. Enumerate the components of enamel organ.
15. Characterize the histogenesis of dental tissues – morpho-differentiation.
16. Describe the calcification stage of solid dental tissues.
17. What is dental eruption?
18. What are the characteristic stages of dental eruption?
19. Enumerate the theories explaining the mechanism of dental eruption.
20. Enumerate the general factors of major importance for the process of dental eruption.
21. What are the signs of physiological dental eruption?
22. Describe the order of temporary teeth eruption.
23. What are the periods of temporary teeth eruption?
24. What factors affect the temporary teeth eruption?
25. What are the pathological varieties of temporary teeth eruption?
26. Describe the dental root formation process.
27. What are the particularities of root formation at pluriradicular teeth?
28. What are the terms of complete root formation at temporary teeth?
29. Describe the radiological stages in the formation of root apex.
30. What is the root growth zone?
31. What are the variants of radicular resorption of pluriradicular temporary teeth?
32. What are the terms of the beginning of resorption of

temporary teeth roots?
33. Enumerate the theories explaining the process of temporary teeth resorption.
34. What are the causes of delays in radicular resorption?
35. What are the causes of acceleration in radicular resorption?
36. Describe the order of permanent teeth eruption.
37. What are the most frequent types of permanent teeth eruption sequences?
38. What are the terms of permanent teeth eruption?
39. Describe the factors affecting the individual variations of permanent teeth eruption.
40. What are the general pathological factors that affect the permanent teeth eruption?
41. What are the local pathological factors that affect the permanent teeth eruption?
42. What are the terms of complete root formation of permanent teeth?
43. Describe the general particularities of temporary teeth.
44. Describe the morphological particularities of temporary teeth.
45. Characterize the structural particularities of temporary teeth.
46. Indicate the localization of Zuckerkandl and Carabelli tubercules.

CONTROL TESTS

Simple compartment

1. The enamel is formed from the following tissues of dental germ:

A. mesenchymal cells of the dental sack;
B. mesenchymal cells of dental papilla;
C. epithelial cells of the external stratum of enamel organ;
D. epithelial cells of the internal stratum of enamel organ;
E. intermediary stratum of enamel organ and stellate

reticulum.

2. Dentine is formed by the following tissues of dental germ:
   A. mesenchymal cells of the dental sack;
   B. mesenchymal cells of dental papilla;
   C. epithelial cells of the external stratum of enamel organ;
   D. epithelial cells of the internal stratum of enamel organ;
   E. dental plaque.

3. Pulp is formed by the following tissues of dental germ:
   A. mesenchymal cells of the dental sack;
   B. mesenchymal cells of dental papilla;
   C. epithelial cells of the external stratum of enamel organ;
   D. epithelial cells of the internal stratum of enamel organ;
   E. dental plaque.

4. Cement is formed by the following tissues of dental germ:
   A. mesenchymal cells of the dental sack;
   B. mesenchymal cells of dental papilla;
   C. epithelial cells of the internal stratum of enamel organ;
   D. anterior sector of dental plate;
   E. Hertwig epithelial sheath.

5. Periodontium is formed by the following tissues of dental germ:
   A. mesenchymal cells of the dental sack;
   B. mesenchymal cells of dental papilla;
   C. epithelial cells of the internal stratum of enamel organ;
   D. anterior sector of dental plate;
   E. Hertwig epithelial sheath.

6. The alveolar bone is formed by the following tissues of dental germ:
   A. mesenchymal cells of the dental sack;
   B. mesenchymal cells of dental papilla;

C. epithelial cells of the internal and external stratums of enamel organ;
D. anterior sector of dental plate;
E. Hertwig epithelial sheath.

7. The Nasmyth membrane is formed by the following tissues of dental germ:
A. mesenchymal cells of the dental sack;
B. mesenchymal cells of dental papilla;
C. epithelial cells of the internal stratum of enamel organ;
D. epithelial cells of the external stratum of enamel organ;
E. anterior sector of dental plate;
F. Hertwig epithelial sheath.

8. The vestibule of buccal cavity is formed by the following tissues of dental germ:
A. mesenchymal cells of the dental sack;
B. epithelial cells of the internal and external stratums of enamel organ;
C. anterior sector of dental plate;
D. Hertwig epithelial sheath;
E. posterior sector of dental plate.

9. The Bell phase is observed during the following stage of tooth development:
A. proliferation;
B. histo-differentiation;
C. morpho-differentiation;
D. calcification;
E. eruption.

10. Mineralization of temporary incisors begins:
A. on the 6^{th} week of intrauterine development;
B. on the 10^{th} week of intrauterine development;
C. on the 16^{th} -17^{th} week of intrauterine development;

D. on the 24^{th} -25^{th} week of intrauterine development;
E. on the 35^{th} -36^{th} week of intrauterine development.

11. Mineralization of temporary molars begins:
A. on the 10^{th} week of intrauterine development;
B. on the 16^{th} -17^{th} week of intrauterine development;
C. on the 24^{th} -25^{th} week of intrauterine development;
D. on the 27^{th} -28^{th} week of intrauterine development;
E. on the 2^{nd} to 4^{th} week after birth.

12. Eruption of central temporary incisors begins:
A. at the age of 4-5 months from birth;
B. at the age of 5-6 months from birth;
C. at the age of 6-8 months from birth;
D. at the age of 9-10 months from birth;
E. at the age of 11-12 months from birth.

13. Eruption of lateral temporary incisors begins:
A. at the age of 4-5 months from birth;
B. at the age of 5-6 months from birth;
C. at the age of 6-8 months from birth;
D. at the age of 9-10 months from birth;
E. at the age of 11-12 months from birth.

14. Eruption of the temporary canines begins:
A. at the age of 6-8 months from birth;
B. at the age of 9-12 months from birth;
C. at the age of 12-16 months from birth;
D. at the age of 16-20 months from birth;
E. at the age of 20-30 months from birth.

15. Eruption of IV temporary teeth begins:
A. at the age of 8-12 months from birth;
B. at the age of 12-16 months from birth;
C. at the age of 16-20 months from birth;

D. at the age of 20-30 months from birth;
E. at the age of 30-36 months from birth.

16. Eruption of V temporary teeth begins:
A. at the age of 8-12 months from birth;
B. at the age of 12-16 months from birth;
C. at the age of 16-20 months from birth;
D. at the age of 20-30 months from birth;
E. at the age of 30-36 months from birth.

17. Formation of roots temporary tooth I end:
A. at the age of 1,5 years;
B. at the age of 2,5 years;
C. at the age of 3 years;
D. at the age of 4 years;
E. at the age of 5 years.

18. Formation of roots temporary tooth II ends:
A. at the age of 2 years;
B. at the age of 3 years;
C. at the age of 4 years;
D. at the age of 5 years;
E. at the age of 6 years.

19. Formation of roots temporary tooth III ends:
A. at the age of 2-3 years;
B. at the age of 4 years;
C. at the age of 5 years;
D. at the age of 6 years;
E. at the age of 7 years.

20. Formation of roots temporary tooth IV ends:
A. at the age of 2-3 years;
B. at the age of 4 years;
C. at the age of 5 years;

D. at the age of 6 years;
E. at the age of 7 years.

21. Formation of roots temporary tooth V ends:
A. at the age of 3 years;
B. at the age of 4 years;
C. at the age of 5 years;
D. at the age of 6 years;
E. at the age of 7 years.

22. What type of radicular resorption is physiological:
A. Uniform resorption of all roots ;
B. Predominant resorption of a region of one root;
C. Predominant resorption in the region of roots bifurcation ;
D. Resorption after a chronic inflammatory process;
E. Types I, II and III of radicular resorption.

23. What type of radicular resorption of temporary teeth is produced in case of an apical chronic inflammatory process:
A. Type I of radicular resorption;
B. Type II of radicular resorption;
C. Type III of radicular resorption;
D. Pathological resorption;
E. Physiological resorption.

24. Foundation of teeth 6 buds begins by:
A. Week IV of intrauterine development;
B. Week X of intrauterine development;
C. Month 4 of intrauterine development;
D. Month 8 of intrauterine development;
E. Month 6 after birth.

25. Foundation of permanent incisor buds begins by:
A. Week X of intrauterine development;
B. Month 5 of intrauterine development;

C. Month 8 of intrauterine development;
D. Month 6 after birth;
E. Month 12 after birth.

26. Foundation of permanent teeth 4 buds begins by:
A. 6^{th} month of life;
B. At 1 year;
C. At 2 years;
D. At 3 years;
E. At 4 years.

27. Foundation of permanent teeth 5 buds begins by:
A. At 3 years
B. At 4 years;
C. At 5 years;
D. At 6 years;
E. At 7 years.

28. Foundation of permanent teeth 7 buds begins by:
A. At 3 years
B. At 4 years;
C. At 5 years;
D. At 6 years;
E. At 7 years.

29. Foundation of permanent teeth 8 buds begins by:
A. After 4 years;
B. After 5 years;
C. After 6 years;
D. After 7 years;
E. After 8 years.

30. Mineralization of permanent incisors begins by:
A. Month 6 of intrauterine development;
B. Month 9 of intrauterine development;

C. Month 2 of life;
D. Month 6 of life;
E. Month 12 of life.

31. Mineralization of teeth 6 begins by:
A. Month 5 of intrauterine development;
B. Month 9 of intrauterine development;
C. Months 3-4 of life;
D. 12 months of life;
E. The age of 2 years.

32. Mineralization of permanent teeth 4 begins by:
A. Month 5 of intrauterine development;
B. Month 9 of intrauterine development;
C. Month 12 of life;
D. The age of 2 years;
E. The age of 2.5 years.

33. Mineralization of permanent teeth 5 begins by:
A. Month 9 of intrauterine development;
B. Month 6 of life;
C. Month 12 of life;
D. The age of 2.5 years;
E. The age of 3.5 years.

34. Mineralization of permanent teeth 7 begins by:
A. Month 9 of intrauterine development;
B. Month 12 of life;
C. The age of 2 years;
D. The age of 3.5 years;
E. The age of 4.5 years.

35. Eruption of central permanent incisors occurs by:
A. 5-6 years;
B. 6-8 years;

C. 8 years;
D. 9 years.

36. Eruption of lateral permanent incisors occurs by:
A. 5-6 years;
B. 6-8 years;
C. 8-9 years;
D. 7-9 years;
E. 9-10 years.

37. Eruption of permanent canines occurs by:
A. 7-8 years;
B. 8-9 years;
C. 9-10 years;
D. 9-12 years.

38. Eruption of permanent teeth 4 occurs by:
A. 7-8 years;
B. 8-9 years;
C. 9-10 years;
D. 10-11 years;
E. 11-12 years.

39. Eruption of permanent teeth 5 occurs by:
A. 8-9 years;
B. 9-10 years;
C. 10-11 years;
D. 11-12 years;
E. 12-13 years.

40. Eruption of permanent teeth 6 occurs by:
A. 5-6 years;
B. 6-8 years;
C. 8-9 years;
D. 9-10 years;

E. 10-11 years.

41. Eruption of permanent teeth 7 occurs by:
    A. 14-15 years;
    B. 8-9 years;
    C. 9-10 years;
    D. 11-12 years;
    E. 12-13 years.

42. Root formation of central permanent incisors ends by:
    A. 6-8 years;
    B. 8-9 years;
    C. 9-10 years;
    D. 10-12 years;
    E. 11-13 years.

43. Root formation of lateral permanent incisors ends by:
    A. 6-8 years;
    B. 8-9 years;
    C. 9-10 years;
    D. 10-12 years;
    E. 11-13 years.

44. Root formation of permanent canines ends by:
    A. 6-9 years;
    B. 9-10 years;
    C. 10-12 years;
    D. 12-15 years;
    E. 14-15 years.

45. Root formation of permanent teeth 4 ends by:
    A. 8-9 years;
    B. 9-10 years;
    C. 11-12 years;
    D. 12-13 years;

E. 13-15 years.

46. Root formation of permanent teeth 5 ends by:
A. 8-9 years;
B. 9-10 years;
C. 10-12 years;
D. 11-12 years;
E. 12-14 years.

47. Root formation of permanent teeth 6 ends by:
A. 8-10 years;
B. 7-9 years;
C. 10-11;
D. 12-13;
E. 13-15.

48. Root formation of permanent teeth 7 ends by:
A. 9-10 years;
B. 10-11 years;
C. 11-12 years;
D. 12-13 years;
E. 14-15 years.

49. Usually, the teeth 75, 85 have the following morphological characteristics:
A. Occlusal surface of biggest diameter and vestibular -oral orientation;
B. Three roots;
C. Two roots;
D. 2 channels: medial and distal;
E. 3 channels: 1 medial and 2 distal.

50. The process of root formation involves:
A. The epithelial cells of internal stratum;
B. The intermediary stratum of enamel organ;

C. The mesenchymal cells of dental sack;
D. The epithelial diaphragm;
E. Dental papilla.

51. The sequence of temporary teeth eruption is the following:
A. Central incisor, lateral incisor, canine, first molar, second molar;
B. Central incisor, lateral incisor, first molar, canine, second molar;
C. Central incisor, canine, lateral incisor, first molar, second molar;
D. Central incisor, first molar, lateral incisor, canine, second molar;
E. Central incisor, lateral incisor, first molar, second molar. canine.

52. Resorption of temporary teeth roots begins:
A. Immediately after the root formation;
B. In 3 years after the root formation;
C. With one year before the physiological change of tooth;
D. With 3-4 years before the physiological change of tooth;
E. Immediately after the eruption of tooth 6.

53. The Zuckerkandel tubercle is most often localized on the surface:
A. Palatal 55, 65;
B. Palatal 54, 64;
C. Jugal 16, 26, 36, 46;
D. Lingual 75, 85;
E. Jugal 74, 84.

54. The Carabelli tubercle is localized on the surface:
A. Jugal 37, 47;
B. Lingual 74, 84;
C. Palatal 16, 26;

D. Lingual 75, 85;
E. Jugal 36, 46.

55. The Bolk tubercle is localized on the surface:
A. Jugal 16, 26;
B. Jugal 35, 45;
C. Palatal 16, 26;
D. Lingual 36, 46;
E. Lingual 37, 47.

56. The microscopy of transversal section of dentine shows:
A. The Retzius bands;
B. The Ebner fibers;
C. The Tomes fibers;
D. The Schapey fibers;
E. The Sicher fibers.

57. The dentinary canalicules contain the fibers:
A. Schapey;
B. Tomes;
C. Reticular fibers;
D. Ebner;
E. Sicher.

58. The Owen lines represent:
A. Zones of reduced mineralization due to development reduction periods;
B. Zones of non-uniform, rhythmic mineralization;
C. Results of metabolic disturbances during the neonatal period;
D. Zones of hyper-mineralized dentine;
F. A transparency sector.

59. In result of disturbances in calcispher unification process appear:

A. The Owen lines;
B. The Czernac interglobular spaces ;
C. The Tomes interglobular dentine;
D. The Tomes globular stratum.

60. At the capsule stage the dental bud is composed of:
A. Epithelial cells of different dimensions;
B. Internal and external epithelial cells;
C. Intermediary and reticular strata;
D. Internal and external epithelial strata on the basal membrane;
E. Epithelial cells of the same dimensions.

61. At the age of 17 months the children's buccal cavity must contain the following temporary teeth:
A. 52, 51, 61, 62, 71, 81;
B. All incisors;
C. Incisors and inferior canines;
D. Incisors, canines and firs molars;
E. Incisors and first molars.

62. Predentine is located in the direct vicinity of:
A. Cement;
B. Enamel;
C. Enamel-dentine junction;
D. Enamel-cement junction;
E. Dental pulp.

63. The origin of cement is:
A. mesenchymal;
B. Ectodermal;
C. Endodermial;
D. Second branchial arc;
E. From stomadeum.

64. The pulp of young permanent teeth is characterized by:
A. Abundance of fibrocytes;
B. Low level of base substance hydration;
C. Incapacity to resist against caries ;
D. Increase of intra-pulp pressure in cases of inflammatory processes;
E. Apical region rich in cells with high resistance potential.

65. Cellular cement is localized mainly:
A. In the apical region;
B. In the region of enamel-dentine junction;
C. On the entire length of root;
D. In the medium sector of root;
E. In the region of radicular bi- and trifurcation.

66. Natal teeth are characterized by the following:
A. They erupt during the first 30 days from birth;
B. The child is born with these teeth already erupted in the oral cavity;
C. They are supernumerary;
D. They have superficial insertion;
E. They can undergo structural changes.

67. Closure of the apical radicular hole of permanent teeth occurs by:
A. 1 year after eruption of tooth;
B. 1-2 years after the eruption of tooth;
C. 1 year after the formation of dental root;
D. 2-3 year after the eruption of tooth;
E. 2 years after the formation of dental root.

68. The surface of dental corona in eruption is covered with:
A. Tomes fibers;
B. Nasmyth membrane;
C. Enamel prisms;

D. Secondary cuticle;
E. Scharpey fibers.

69. Foramen caecum is on:
A. Masticator surface of molars;
B. Masticator surface of premolars;
C. Vestibular surfaces of molars and premolars;
D. Oral surface of frontal teeth;
E. Approximal surfaces of frontal teeth.

70. Enamel contains:
A. 75 % mineral elements
B. 87 % mineral elements;
C. 91 % mineral elements;
D. 95 % mineral elements;
E. 85 % mineral elements.

71. At the age of 19 months at children in buccal cavity the following teeth are already erupted:
A. Central upper and lower incisors, upper lateral incisors;
B. All incisors;
C. Lower incisors and canines;
D. Incisors, canines and first molars;
E. Incisors and first molars.

72. During the antenatal period begins the formation of following permanent teeth buds:
A. First molars;
B. First molars and central incisors;
C. First molars, central upper and lower incisors, lateral lower incisors;
D. First molars and incisors;
E. First molars, incisors and canines.

73. The intermediary stratum is characterized by:

A. Location between the stellar stratum and internal epithelium of enamel organ;
B. 2-3 rows of high cells;
C. Contain the most alkaline phosphatase;
D. Induction of enamel formation;
E. Induction of root formation.

74. Structural units of enamel are represented by:
A. Tomes Fibers;
B. Scharpey Fibers;
C. Retzius lines;
D. Prisms;
E. Enamel fibers.

75. The Retzius lines are distributed:
A. Vertically;
B. Circularly;
C. Transversally;
D. In Undulated manner;
E. From the incisal edge in direction of radicular apex.

76. The weak mineralization zones represent:
A. Tops of cuspids;
B. Plates;
C. Parazones;
D. Fascicles;
E. Cracks.

77. The dentine contains:
A. 95 % mineral substances;
B. 85% mineral substances;
C. 93 % mineral substances;
D. 80 % mineral substances;
E. 69 % mineral substances.

78. The neonatal period includes:
    A. The first 4 weeks;
    B. The first 2 weeks;
    C. The first 6 weeks;
    D. The Month IX of intrauterine development;
    E. Determined individually.

79. The pre-school period is the age of:
    A. 3-6 years;
    B. 1-3 years;
    C. 2-5 years;
    D. 0.5 – 3.5 years;
    E. 1.5 - 4.5 years.

80. The theories of radicular resorption of temporary teeth are the following:
    A. Genetic, radicular, pulpal, endocrine;
    B. Radicular, endocrine, genetic, "aging";
    C. "Aging", endocrine, genetic;
    D. "Aging", vascular, endocrine.

81. During the process of histogenesis first is formed:
    A. The enamel;
    B. The dentine;
    C. The cement.

82. In the formation of enamel participate:
    A. Cementoblasts;
    B. Odontoblasts;
    C. Fibroblasts;
    D. Enameloblasts;
    E. Osteoblasts.

83. In the formation of dentine participate:
    A. Cementoblasts;

B. Odontoblasts;
C. Fibroblasts;
D. Enameloblasts;
E. Osteoblasts.

84. In the formation of cement participate:
A. Cementoblasts;
B. Odontoblasts;
C. Fibroblasts;
D. Enameloblasts;
E. Osteoblasts.

85. Reduction of calcium content is characteristic for:
A. Interglobular dentine;
B. Globular dentine;
C. Transparent dentine;
D. Replacement (substitution) dentine.

86. The relatively thinner dentine stratum of temporary teeth is explained by:
A. Structure of dentine;
B. Reduction of dentinogenesis in the period of morphogenesis;
C. Relatively bigger pulpal cavity;
D. Reduced calcification of dentine.

87. The periodontium of newborn children is represented by:
A. Mucous tissues of gum;
B. Gingival wave;
C. Gingival wave, alveolar processes.

88. The alveolar process begins to grow in height:
A. At the age of 6-7 months, simultaneously with the eruption of first teeth;
B. In the temporary teeth formation period;

C. In the permanent teeth formation period.

89. Formation of alveolar process begins:
A. During the intrauterine development period;
B. During the first half of the first year of child's life;
C. During the temporary teeth formation period ;
D. During the second half of the first year of child's life;
E. During the neonatal period.

90. Formation of periodontium tissues at children ends:
A. Simultaneously with the complete formation of root;
B. In one year after the tooth eruption;
C. In 6-12 months after complete root formation;
D. Simultaneously with tooth eruption;
E. In 2 years after the tooth eruption.

Multiple compartment

91. The enamel of temporary teeth has the following characteristics:
A. It is thinner compared to the one of permanent teeth;
B. It is of a whiter color;
C. It is less permeable compared to the permanent teeth;
D. A-prismatic sector predominate on the surface of enamel;
E. The zone of entering the cement is frequently covered with cement.

92. Calcification of dentine is characterized by:
A. Triggering after the beginning of enamel mineralization;
B. Triggering after the complete formation of enamel organic tram;
C. Rhythm with alternation of activity and pause periods;
D. Mineralization from surface to depth;
E. Ending when root formation begins.

93. Eruption of first teeth in the oral cavity is accompanied by:

A. Hypersalivation;
B. Pruritus of mucous alveolar process;
C. Hyperthermia ;
D. Reduction of appetite;
E. Pains and inflammation of mucous tissues in the region of tooth eruption.

94. Delayed eruption of temporary teeth is possible in the following cases:
A. Certain genetic diseases;
B. Hypofunction of thyroid gland;
D. Insufficiency of vitamin C;
E. Rachitis;
F. Hyper function of thyroid gland.

95. Physiological resorption of temporary teeth roots:
A. Is provoked by the pressure of permanent teeth;
B. Begins by 2 years before the average physiological tooth alternation term;
C. Is done by osteoclasts;
D. Begins from the tooth cavity to the surface;
E. Organic tram is decomposed before the collagen due to the phenomenon of endocytolisis.

96. From the dental papilla is formed:
A. The radicular dentine ;
B. The dental-alveolar ligament;
C. The radicular cement;
D. The Hertwig sheath;
E. The dental pulp.

97. The embryonic period includes the following critical periods:
A. 1-12 days;
B. 12-24 days;
C. 1-3 weeks;

D. 3-6 months;
E. 6-9 weeks.

98. Permanent teeth differ from the temporary ones by the following:
A. Shape of dental corona;
B. Curve of dental corona;
C. Incisive edge;
D. Angle of dental corona;
E. Size of dental corona;
F. Color of dental corona.

99. With aging in enamel take place the following changes:
A. Reduction of permeability;
B. Reduction of quantity of microelements;
C. Increase of permeability;
D. Condensation of crystalline lattice;
E. Increase of quantity of microelements;
F. The crystalline lattice doesn't change.

100. The neonatal line is observed on the following teeth:
A. Temporary incisors and canines;
B. Temporary incisors, canines and first molars;
C. All temporary teeth;
D. Permanent incisors and first molars;
E. Permanent incisors, canines and first molars.

101. The placental period includes the following critical phases:
A. Weeks 6-9;
B. Month III;
C. Months IV-V;
D. Months V-VI;
E. Months VI-VII.

102. The enamel organ is composed of:

A. Internal and external epithelial strata;
B. Pulp;
C. Intermediary stratum;
D. Reticular stratum;
E. Basal membrane.

103. Administration of tetracycline to 4- months aged child can result in the affection of solid dental tissues:
A. In the central region of dental coronas 16, 26, 26, 46;
B. In the region of corona collars 17, 27, 37, 47;
C. Cutting edges of teeth 11, 21;
D. In the region of coronas 55, 65, 75, 85;
E. In the region 18, 28, 38, 48.

104. Morphologically cement is composed of:
A. Primary cement;
B. Secondary cement;
C. Cellular cement;
D. Plate cement;
E. Reactive cement.

105. The structural elements of dental bud include:
A. Stellar reticule;
B. Dental papilla;
C. Dental sack;
D. Intermediary stratum;
E. Hertwig sheath;
F. Internal and external epithelial strata;
G. Enamel organ.

106. Pathological factors that may cause delays in the dental eruption include:
A. Congenital syphilis;
B. Prenatal rubeola;
C. Acute herpetic stomatitis;

D. Rise of body temperature during dental eruption;
E. Trisomia 21.

107. Disturbances at the stage of morphological differentiation can lead to:
A. Hypodontia;
B. Premature eruption;
C. Microdontia;
D. Delayed eruption;
E. Macrodontia.

108. Disturbances on the stage of determine the following pathologies:
A. Adamantinoma;
B. Microdontia;
C. Hutchinson teeth;
D. Adontia;
E. Supernumerary teeth.

109. Disturbances at the stage of morphological differentiation can lead to:
A. Adamantinoma;
B. Hutchinson teeth;
C. Supernumerary teeth;
D. Follicular cyst;
E. Microdontia.

Correct Answers

1.D
2. B
3. B.
4. A.
5. A
6. A
7. D
8. C
9.A
10. C
11. D
12. C
13. D
14. D
15. B
16. D
17. A
18. A
19. C
20. B
21. B
22. E
23. D
24. C
25. C
26. C
27.A
28. A
29. B
30. D
31. B
32. E
33. E
34. D
35. B
36. C
37. D
38.C
39. D
40. A.
41. E
42. C
43. C
44.D
45. D
46. E
47. A
48. E
49. C
50. D
51. B
52. B
53. E
54. C
55. E
56. B
57. B
58. B
59. B
60. E
61. D
62. E
63. A
64. E
65. E
66. B
67. C
68. B
69. D
70. D
71. D
72. D
73. A
74. D
75. B
76. E
77. E
78. A
79. A
80. C
81. B
82. D
83. B
84. A
85.A
86.C
87.B
88.A
89.A
90.C
91.A, B, E
92. C, D
93. A, B, C, D.
94. A, B, D.
95. A, B, C
96.A, E,
97. A, D.
98. A, C, E, F.
99. A, D, E.
100. C, D.
101. B, D.
102. A, C, D, E.
103. A, C,
104. A, B, C
105. B, C, E, G.
106. A, B.
107. C, E,
108. A, B.
109. A, C, D.

2 BEHAVIORAL MANAGEMENT IN PEDIATRIC DENTISTRY

Interpersonal relations

Stomatological treatment of children implies an interpersonal relation between the doctor, child and his family, this relation may be defined as the "triangle of pedodontic treatment" (G. Z. Wright, 1988).

The child is denoted as the upper angle of the triangle and center of attention of both family and stomatological team. (Fig.2.1). The triad parent- child- dentist, if it works properly, will result in creation of certain beneficial relations between the doctor and the child (Maxim A., Balan A, Pasareanu M, Nica M., 1998).

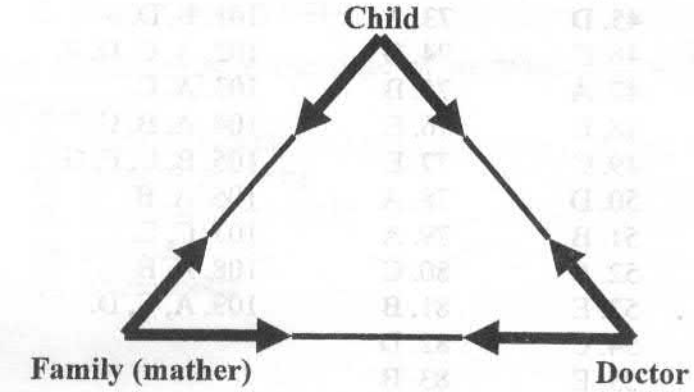


Figure 2.1. **Triangle of pedodontic treatment (by G. Wright)**

Beginning already with the ante-preschool period to the children's attitude is added the influence of other persons (teachers, professors, friends, etc.) – the element of surrounding environment (Fig. 2.2). During the school age, especially during

the pre- and pubertal period the role of family (parents) decreases and the role of environment grow greater in these interpersonal relations.

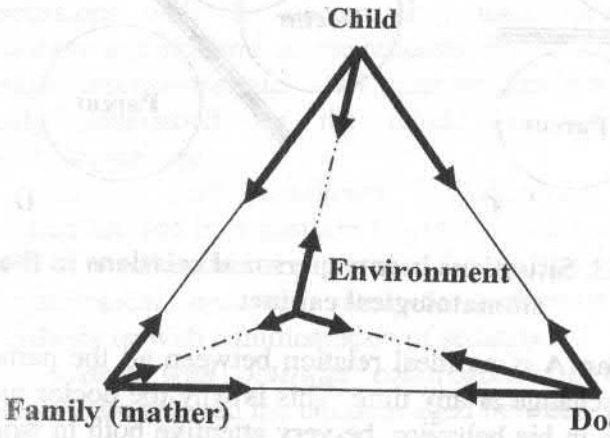
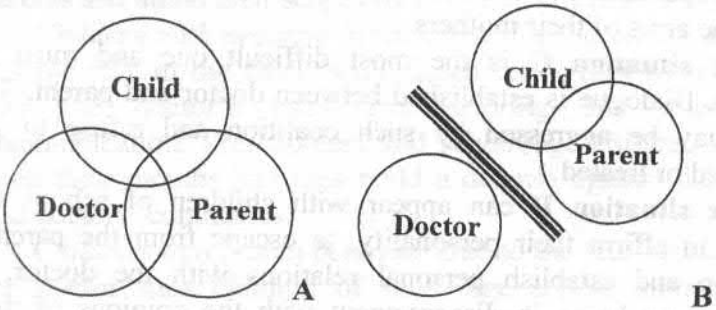


Figure 2.2. **Interpersonal relations in stomatological treatment**

In the stomatological cabinet can appear the following kinds of interpersonal relations (Fig. 2.3).



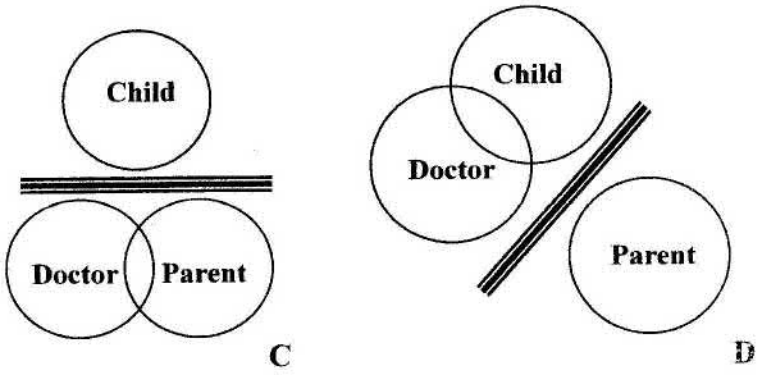


Figure 2.3. **Situations in interpersonal relations in the stomatological cabinet**

The situation A is an ideal relation between all the parties, however, it can change at any time. This is why the doctor must be very cautious in his behavior, be very attentive both in words and actions.

The situation B is characteristic for ante-preschool age children that are still mother-dependent, as mothers represent for them the source of security and a guide of communication. The dialogue must be implemented via mothers, while all procedures and manipulations with such children are performed when they are in the arms of their mothers.

The situation C is the most difficult one and must be avoided. Dialogue is established between doctor and parent. The child may be aggressed by such coalition and refuse to get examined or treated.

The situation D can appear with children of school age willing to affirm their personality, to escape from the parental tutorship and establish personal relations with the doctor, in certain cases being in disagreement with the opinions of their parents.

Communication between doctor and uncontrollable

(unwilling to obey) children can be successful only in the case the doctor can interpret correctly the different aspects of their behavior. There are many variants in the behavior of children.

Children agreeing to collaborate Such children maintain discussions with the doctor; they understand the need for treatment and perform all the requests. Preparation for a visit to a dentist, discussions, etc. – all must be conducted in a language easily understood for the child and accessible for his psychological age.

Children with insufficient collaboration capacity Such children are not in a position to enter into a verbal contact with the doctor and understand the need for such a visit. In such cases stomatological treatment must be performed under general anesthesia or with administration of sedatives.

Children with potential collaboration incapacity To this category are referred the children aged between 3 and 6. Children with negative stomatological experience or the ones threatened by the stories of their parents, friends, etc. are afraid of doctors and require special approach.

Children with hysterical or uncontrolled behavior Such children scream, cry, make uncontrolled movements, etc. This type of behavior is classical for the children that manipulate their parents and attain their scopes by hysteric behavior.

Children with negative behavior Such children are tensioned, passive stay in the chair, with closed teeth; they avoid looking into the doctor's eyes and ignoring any attempts of communication. Preschoolers and school-age children refuse to open their mouths and thus build a defense against the parents' and doctors' commands.

Children with feared behavior Due to the fear before visiting the doctor such children of small age appear threatened and feared. In order to win their trust the doctor must explain them calmly the scopes of treatment.

Children with frustration behavior but accepting collaboration Such a child is tensioned, follows every movement of medical personnel. The child accepts the treatment but often cries. The doctor controls the situation, as the child is willing to collaborate.

Children with pathetic behavior In this case crying is a compensatory reaction on fear. Treatment is accompanied by uninterrupted crying regardless of perfect anesthesia. Only by wining the trust the doctor can have success.

Children with stoic behavior Such children withstand the treatment passively and calmly, but are recessed and sorrowful. It is not a typical behavior for children and can be a consequence of a punishment applied before treatment.

Children are patients with special behaviors marked by two types of reaction: frustration and fear. The frustration feeling is generated by the fact that for any child medical treatment is an event decided by the adults. Simultaneously stomatological treatment at them is associated with fear, especially at its beginning; it is present at all children but has different manifestations, varying depending on constitutional factors, on family environment, on education and previous experiences.

Fear is a straight-forward reaction caused by the low appreciation of own forces compared to the ones of the hostile factor. It can be native and associated with a complex of unsafety; it can be a result of previous unpleasant experiences.

Timid children refuse to enter the consulting room, to sit in the chair; they stay tensioned without their company. They watch the doctor, stay away from him; often they do not hear and do not want to understand the doctor's sayings, without concentrating attention on his words, so any attempt to establish a good dialogue will be left without success. A warm glance, an amicable gesture will reduce fear, preparing the child for a good cooperation. We need to convince the child that there is no

aggression in stomatological manipulations, how the equipment works, etc. using various psychotherapeutic techniques.

Uncontrolled attitudes can appear especially during the first visit to the doctor when the child has accesses of fear, cries, hits, bites, and comes down to a more infantile behavioral level compared to the real one of his age. Such children strongly oppose to stomatological examination: they shut their lips and teeth, get agitated. Generally, there exist capricious and spoilt children that sometimes try to establish relations in the stomatological room similar to the relations they've established at their homes. It is important to communicate with them without constricting, but the doctor must take a very firm position so that they understand that the home-like behavior here is disallowed.

Timid attitude is usually observed at children with limited social experience and emotional lability. Such children feel insecure; they manifest deficiencies in actions and communication and try to consolidate on defensive positions. If we do not approach them the right way their behavior can fall out of control. Such children must be stimulated for cooperation during the next sessions by commendations and acknowledgement of progress.

The attitude of distrust is much more frequent, so the children don not let themselves caught in dialogue but accept the treatment. One must be very prudent with them as their hidden distrust can alter their will to cooperate.

Absence of will to cooperate can be present at children with important psychic disturbances, at mentally handicapping children. They require special attention and their treatment in certain cases should be performed in conditions of hospital under general anesthesia.

The above-mentioned types of reaction are frequently associated.

Depending on the diagnosed psychological type the doctor's

tactics must be applied accordingly. However, it must always be calm, friendly and warm but firm in order to encourage the good inclinations of children and eliminate the negative ones straight from the beginning.

The first consultation is the most important one from the psychological point of view. One must try to give an appreciation to the real capacities of a particular child and to the cooperation level of parents. Friendly words, warm glance, amicable smiles will favor the first contact and the child will accept the examination. In such case the doctor must show his tools to the child and explain (in terms adequate to age) their application. It is recommended to use the first visit only for familiarization with the cabinet, with the stomatological chair, with the modality of examining the buccal cavity. If the first visit is associated with pain, it must be removed as soon as practically possible, with warmness but firmness. If the child is capable to cooperate he must understand that the doctor is willing to help him and remove the pain, thus raising the motivation to collaborate.

The first visit to the doctor must by prepared in advance in the family, in the kindergarten, etc. Calm environment in the cabinet is of great importance. It is also useful to demonstrate other children that accept and support well the treatment.

Frequently the doctors distract the children's attention with music, demonstration of drawings, toys, slides, etc.

Medicamental preparation

Psychological preparation is not sufficient for a child. On the contrary, it can raise the alarm state and cause and agitation crisis. For this reason it is recommended to administer tranquilizers and sedatives to such children in advance. For example: Amyzil, Elenium, Oxilidine, Trioxasine, Hexobarbital, etc. These medicines are administered 30-40 minutes before the treatment, or during 5-7 days.

Most efficient are the following combinations of sedatives

with analgesics:

1. Oxilidine + Anagine + Amyzil;
2. Meprobamate + Amyzil +Amidopyrine + Diprazine;
3. Meprobamate + Phenobarbital + Aminazine;
4. Acetylsalicylic acid + Tavegil;
5. Diazepam + Nitrazepam + Mephenaminic acid + Tavegil.

Dosing:

3 years: 1/3 of dose for adults;
7 years: 1/2 of dose for adults;
10 -12 years: 2/3 of dose for adults.

Such priory treatment stabilizes vegetative reactions, produces an emotional indifference, reduces fear and creates a psychomotor inhibition.

In order not to provoke painful sensations local anesthesia can be applied:

1. Terminal anesthesia without injections with the following methods:

a) physical methods (hypothermia, use of electric current);
b) chemical methods (application, instillation);
c) combined methods (electrophoresis of anesthetics).

For the scopes of local anesthesia of children can be used:

Pyromecain – unguent 5% - for mucous tissues and open pulp in the buccal cavity;

Anestesine - emulsion or unguent 5-10% for the anesthesia of buccal mucous tissues.

Xylocaine, Lydocaine - solution 1-2% for local, infiltrative, trunk anesthesia, ligament anesthesia or in the form of aerosol for mucous tissues, gums, etc.

Dicaine 0.5 –1% is for local anesthesia. However, it is 10 times more toxic than novocaine and due to rapid resorption properties it is not administered to children less than 10 years.

Trimecaine - solution 1-2% is used for infiltrative and trunk anesthesia.

2. Anesthesia by injection:

a) infiltrative ;
b) regional.

Infiltrative anesthesia is used act children due to the high absorption capacity of upper maxillary and on mandible - in the frontal region, before the complete formation of roots or during the resorption period.

Regional anesthesia assures the removal of pain on a considerable portion of maxillaries. On the upper maxillary anesthesia is used for the nasopalatal nerve in incisive orifice when treating caries and its complications on upper frontal incisors. For lateral teeth is used tuberal and palatal anesthesia. On mandible direct anesthesia is used. When treating pulpitis it is recommended to add some adrenaline into anesthetics. In some cases amplification of anesthetic action is achieved by intra-pulpal injection after the opening of pulpal chamber.

When local anesthesia is impossible (child's age, disturbances of CNS, etc.) and the analgesic-sedative treatment is inefficient, one may resort to general anesthesia in stationary conditions.

Control questions and topics:

1. What is the triangle of stomatological treatment?
2. What are the interpersonal relations in the stomatological cabinet?
3. What children's behavior variants in the stomatological cabinet do you know?
4. First visit to the dentist.
5. Sedation of child for stomatological treatment.
6. Types of anesthesia at children.
7. Local anesthetics in the pedodontic practice.

3 METHODS OF STOMATOLOGICAL EXAMINATION OF CHILDREN

Stomatological examination is aimed at obtaining of better and more complex data on the diagnosis of dental, periodontal affections and affections of dental-maxillary apparatus, their evolution, possibilities of treatment and prophylaxis.

The doctor's attention must cover all elements of the dental-maxillary apparatus and the state of all of them must be registered in the clinical pedodontic card.

The first part of the card comprises:

1. *Personal data* (Name and Surname, date, month and day of birth, place of birth, address, phone number, occupations of parents, number of children in family, etc.).

2. *Motives of appeal, accuses.* Children or parents may usually present the following accuses: pains, physiognomic disturbances, phonetic disturbances, disturbances of mastication, inflammatory processes, abnormal formations, disturbances of dental eruption, etc.

If the dominant symptom is the pain, one must clarify: localization, character (spontaneous or provoked), triggering or aggravating factors, intensity (ache, hard pain, moderated pain), form of pain (striking, tension, lancinating pain), irradiation, duration (intermittent, crises intermittent with periods of continuous anxiety), associated disturbances (lacrimation, hypersalivation, facial congestion, hypostases, etc); physical and thermal factors (cold, hot, warm) or medicaments that reduce the pain.

3. *History of affection* The gathered data shall refer to the

very moment of appearance of the disease as manifested by some of the more important symptoms: pain, hypostases, disfunction, character of debut: sudden, long-lasting; previous treatment and its effects.

In cases of abnormal formations and morphological elements we will figure out the circumstances in which they appeared, duration, development history, as well as the functional disturbances provoked (physiognomic, masticating, phonetic), general associated disturbances (absence of appetite, fever, asthenia, etc.).

4. *Hereditary-collateral circumstances* One should clarify the general factors (genetic, neural-endocrine, metabolic, environmental factors and chronic infections) present in family, as well as the affections suffered by the mother during the period of pregnancy:

- acute infections: viral affections etc.;
- chronic maladies : a) endocrinopathies: sugar diabetes, thyreotoxicosis, etc.; b) nephropathies: nephritis, chronic glomerulonephritis, etc.; c) diseases of digestive tract: gastritis, stomach and duodenal ulcer, colitis, etc.; d) diseases of liver and biliary bladder: hepatitis, cholecystitis, etc.; e) diseases of blood: anemia, etc.;
- pathology of pregnancy and birth: toxicosis of first pregnancy period; toxicosis of second pregnancy period, anemia, birth complications, etc.;
- medical drugs administered to the expectant mother (antibiotics, hormones, sulfanilamides, barbiturates, etc.);
- working conditions of the expectant mother: chemical intoxication, etc.;
- pernicious habits of the parents (smoking, alcohol, etc.) that could affect the development of the child and of his dental-maxillary apparatus.

5. *Personal history*

A. Physiological: we will clarify the moment of birth (timed or premature delivery), eutopic or distopic delivery; height and weight upon birth, nutrition (natural, mixed, artificial) during the first year of life;

- age of dental eruption;
- age of puberty.

B. Pathological: presence of congenital malformations, traumatism or surgical interventions in the cervical-facial region; nutrition distortions during the first age of life, rachitis, infectious diseases; dysendocrinia, cardiac affections, renal and hepatic affections, nervous disturbances, epilepsy, sanguine dyscrasia, bronchial asthma, allergies, anemia, tuberculosis, rheumatism, etc.

One must establish if the child previously followed or is currently following medical treatment, if had any reactions on pharmaceutical treatment, followed a course of local fluoridation, etc. One must pay particular attention to nasal-pharyngeal affections (vegetation, recurrent amigdalitis, sept deviations) and note if any surgical intervention was performed with indication of its results, if any. It is also important to check the pernicious habits: finger sucking, tearing of lips, tongue, putting objects between the dental arcs, etc.; if the child is at the doctor for the second time it will be also necessary to check how did his behavior change compared to the previous visit. Data on psychic development can be obtained from parents, as they should know the level of kid's intellectual level or the problems of mental retard, if any.

6. *Objective examination* includes inspection, percussion, palpation and a series of additional manipulations and investigations.

Inspection of oral cavity organs and tissues schematically includes the general examination of patient and the general examination of buccal cavity at sufficient natural or artificial

illumination.

During the general examination it is necessary to evaluate the somatic development of the child according to his sex, age and constitutional type. Depending on the obtained data we can divide the children into three categories: hyposoms, normosoms and hypersoms.

Local-regional examination of lymphatic system must be performed with attention, as it is especially reactive to stomatological affections.

Facial examination is effected from frontal and lateral sides. During inspection one must note:

- shape of face (oval, round, triangular, square);
- symmetrical or asymmetrical face with precision of zone and cause, if possible (deviation of mentone, nasal pyramid, presence of cicatrices, inflammatory and tumor processes);
- facial grooves (blurry or accentuated), appearance of teguments (fine, rigid, dry, humid), modifications of color (congestion, pale), integrity of teguments, redness of lips and dermal portion (high, medium and short), labial convexity (open, close), proportionality of lips and facial stories.

Examination of temporal-mandibular articulation: mouth opening (within normal limits, blocked or limited); excursion of mentone and inferior interincisive line at opening or closing of mouth. Trajectory of mouth opening can be arc-shaped (normal) or saccadic (in cases of ATM dysfunction). Mentone can deviate to the right or to the left, evidencing affections limiting the movement of one of the two articulations.

Excursion of condyles is evaluated by the introduction of indices into the external auditive channels and application of polices onto the pre-auricular region. Asking the patient to close and open the mouth, the doctor observes the symmetry and amplitude of movements, presence of any crepitation, cracking sounds, articulation jumps, pains, and deformations.

Examination of buccal cavity

Inspection of buccal cavity starts from vestibule with shut dental arcades and open lips by lifting the upper lip and retraction of lower lip or of the cheek with the stomatological mirror. First of all is inspected the red edges of lips and labial commissures. It is necessary to take into consideration the color, presence of squame, crust. On the internal surface of lips one may observe a slight roughness explained by the presence of small salivary glands.

Appreciation of buccal cavity vestibule depth is effected with the help of a gradated probe: one has to measure the distance from the edge of gum to the horizontal level of transitory plate. If the buccal vestibule is less than 5 mm deep – it is considered superficial, if between 5 and 10 mm - medium, if greater than 10 mm – profound.

After that, are examined the labial frenum (length and fixing levels) and the frenum placed in the stroma of transition plate. Pakalns distinguishes three types of frenum: weak – that during extensions do not affect the position of papillas and gingival margins, medium – fixed at 1.5 mm from the edge of gingival papilla and strong – fixed on gingival papillas and displaced during movements.

In continuation, with the help of mirrors is performed the inspection of jugal mucous tissues, considering their color and degree of humidity. On the line of teeth closure in the posterior portion are located the sebaceous glands (Fordyce) that sometimes are visible only after the tensioning of mucous tunic. At the level of molars II one may see the papillas were are opened the orifices of parotid salivary glands.

Inspection of teeth

Examination of teeth starts from the upper arcade from right to left and then is continued by the lower arcade from left to right.

Dental formula can be denoted in different ways – according

to different systems. Till now the dental formula depending on the present teeth denoted: with roman figures – temporary teeth and with Arabic figures - the permanent ones (by Zigmondy, 1861) (figure 3.1).

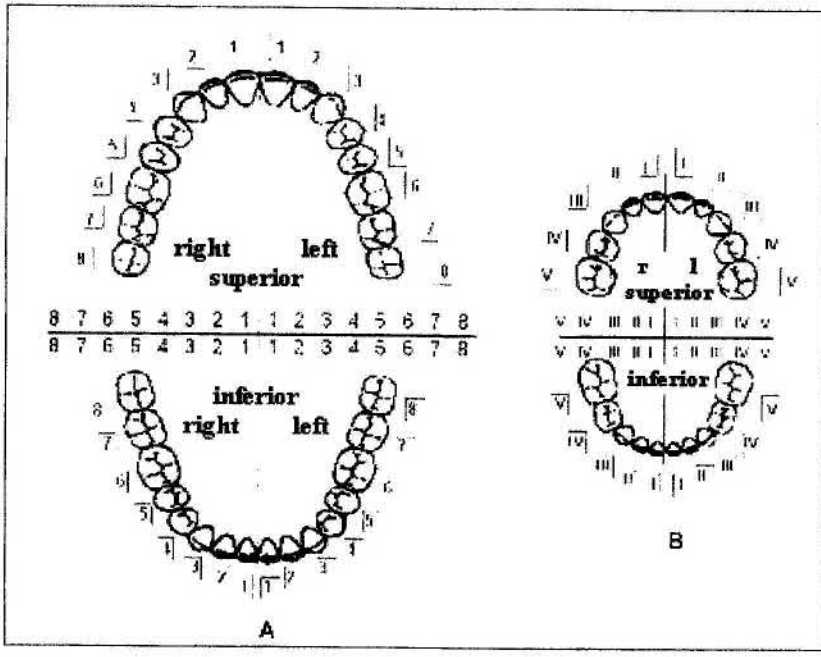


Figure 3.1. **Traditional dental formula (by Zigmondy): A - permanent dentition; B – temporary dentition**

Now in order to unify the teeth denoting formulae the WHO and FDI recommend the binary denotation system with use of prefixes 1, 2, 3 and 4 for permanent teeth and 5, 6, 7 and 8 for temporary ones (see Figures 3.2, 3.3).

According to the dental formula one can determine the dentition; temporary, mixed, definite.

The evolution and formation process is also called dentition or dental age that can be: commensurable with age, premature or

delayed.

After registration of data in dental formula one has to determine the dental occlusion (physiological or pathological).

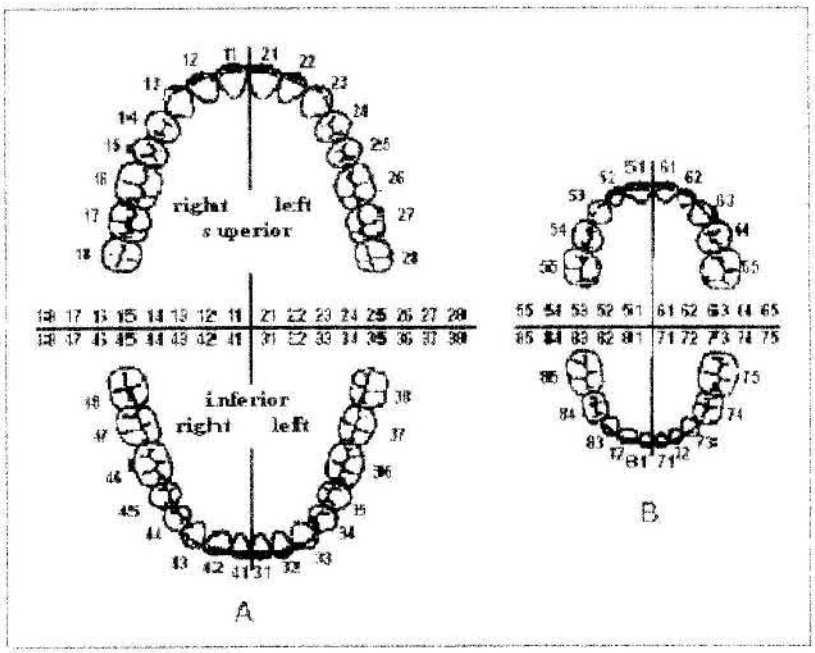


Figure 3.2. **FDI dental formula:**
A - permanent dentition; B – temporary dentition

During the examination of each tooth the following aspects must be determined: dimension, shape, color of corona, shine of enamel, etc. By inspection and probing each dental surface is examined in order to identify any dysplasia of solid tissues, caries or obturation, traumatic lesions, etc. Special attention must be paid to the places vulnerable to caries (holes, cracks, grooves), to topography and size of caries lesions (acute, chronic, superficial, medium or profound, non-penetrating or penetrating); degree of affection of pulp and periodontal apical tissues. If the clinical

examination doesn't allow determining the state of pulp, tooth vitality tests shall be performed.

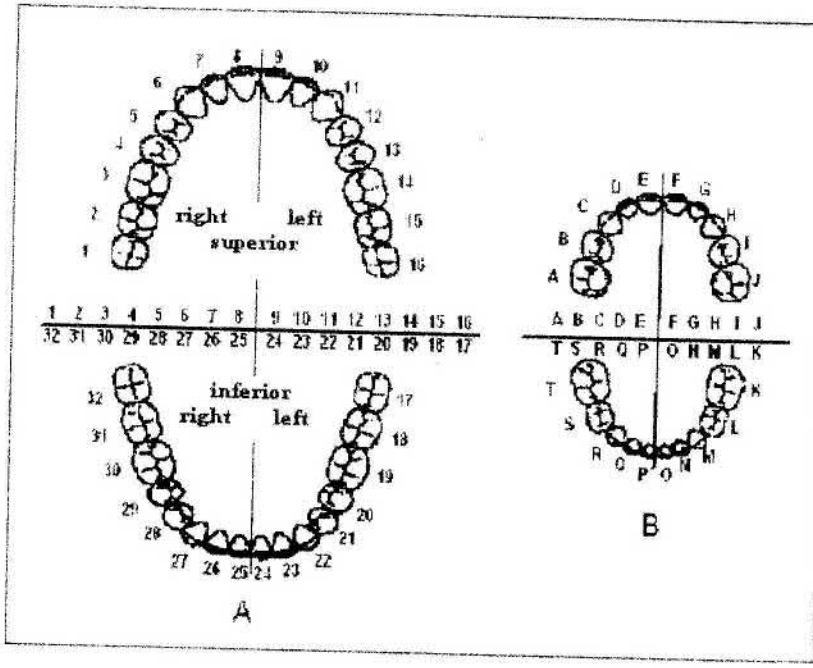


Figure 3.3. **Dental formula by ADA:**
A – permanent dentition, B - temporary dentition

In order to denote the state of teeth the following letters are used; C - caries, O – obturation, A- absence (anodontism), E- extraction, P- pulpitis, Pt – apical periodontitis, T- dental trauma, H- dental hypoplasia, F- dental fluorosis.

In the dental formula "0" means the absence of a temporary tooth in the age close to the period of physiological replacement and "X" – premature extraction of a temporary or permanent tooth.

Tooth mobility is characteristic for the period of physiological replacement of temporary teeth. There are three

degrees of tooth mobility: I – in vestibular-oral direction, II - in vestibular-oral and lateral directions and III – vertical mobility.

In some cases caries is denoted depending on the localization of defects on the dental surfaces:

O – occlusal (masticatory); I- incisal; M – mesial; D- distal; F - vestibular (frontal, labial); B- buccal, L – lingual.

Combined carious defects localized on several dental surfaces are denoted by several respective letters, for example: MO – mesio-occlusal cavity, MOD – mesio-occlusal-distal; MI – mesio –incisal, FO - vestibular-occlusal, LO – lingual-occlusal, etc.

In order to evaluate the activity of carious process the caries intensity index is used. This index represents the total number of teeth affected by caries and its complications, the number of decayed, missing, and filled permanent teeth (DMFT); decayed, missing, and filled deciduous teeth (dmft); decayed exfoliated, and filled deciduous teeth (deft); decayed and filled deciduous teeth (dft); and decayed, missing, and filled surfaces in permanent teeth (DMFS).

The caries rate represents another index of gravidity expressed by the average number of affected surfaces or teeth during a fixed time period (6 months, 1 year, etc.).

The caries incidence index represents the percentage of persons affected within a population:

$$IF = \frac{\text{Number of children affected by caries within a group}}{\text{Total number of children in the group}} \times 100\%$$

By axial and paraxial percussion one has to determine the sensitivity of apical and marginal periodontium. Percussion starts from evidently intact teeth in order not to cause a brutal pain and let the child compare the sensations between a healthy and an affected tooth.

Inspection of buccal cavity continues with the examination of

gums: color, state of gingival papilla, deepness of dental-gingival groove, presence of dental tartar – using a periodontal probe (angular, gradated and bulbous-end probe).

In order to evaluate the state of periodontal tissues and need for treatment the **CPITN** ***(Community Periodontal Index of Treatment Needs)*** index is used (Fig. 3.4.).

Author - Ainamo (1982)

Measures – periodontal status and treatment needs

Procedure for use

Examination of periodontal tissues is performed with the help of a gradated periodontal probe marked at 3.5, 2.0, and 3.0mm intervals from the tip with colour-coding between 3.5 and 5.5mm and a ball 0.5mm in diameter at the working tip. The force exerted onto the probe during examination must not exceed 25 g. Probing can be divided into operative component (in order to determine the depth of recess) and sensitive component (in order to detect the presence of sub gingival tartar). Sub-gingival tartar is determined not only when it is abundant, but also when a roughness is felt when moving the probe along the dental root with observation of anatomic configuration. If after the examination and probing of teeth no changes are identified, the same tooth must be examined with the mirror in order to localize gingival hemorrhage that may appear in 30-40 seconds. For adults (20 years and older), divide the dentition into sextants. Evaluate all teeth except third molars. For the children and adolescents (7-19 years of age) divide the dentition into sextants but evaluate only the first molars in posterior, right central incisor in maxilla, and the left central incisor in mandibular anterior. Are examined the sextants in the region of the following groups of teeth: 16/17, 11, 26/27, 36/37, 31, 46/47. Examining the teeth one must note the codes depending on the gravidity. If one of the mentioned teeth is absent, the next neighboring tooth in the respective sextant is examined.

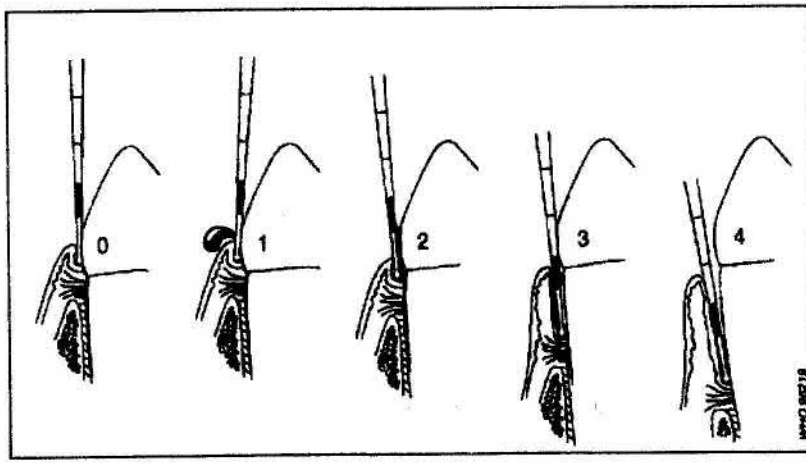


Figure 3.4. **Determination of CPITN index**

Use the periodontal probe Score according to the following criteria:

Code 0 = Healthy periodontal tissues

Code 1 = Bleeding after gentle probing

Code 2 = Supra- or sub gingival calculus or defective margin of filling or crown

Code 3 = 4- or 5- mm pocket

Code 4 = 6 mm or deeper pathological pocket

Mark one score to represent each sextant. Record only the highest code that corresponds with the most severe condition. Patients are classified (0, I, II, III) into treatment needs according to the highest coded score recorded during the examination. Criteria for classification are:

0 = No need for treatment (code 0)

I = Oral hygiene instruction (code 1)

II = Oral hygiene instruction plus scaling and root planing, including elimination of the plaque retentive margins of fillings and crowns (codes 2 and 3)

III = I + II + complex periodontal therapy that may include surgical intervention and/or deep scaling and root planing with local anesthesia (code 4)

Rating score

To identify high and low priorities for treatment in a community, calculations of the number and percentage of individuals with the following can be made:

a. No sextant scoring each code
b. 1 to 2 sextants scoring Code 1, 2, 3, or 4
c. 3 to 4 sextants scoring Code 1, 2, 3, or 4
d. 5 to 6 sextants scoring Code 1, 2, 3, or 4

Papillar – marginal –alveolar Index (PMA) Parma

Measures - denoting the localization and intensity of gingival inflammatory processes.

Procedure for use

The gingival papillas are plugged; the marginal and alveolar gums are treated with a solution containing iodine (Lugol). Inflammation of papilla (P) in the region of a tooth is given 1 point, inflammation of marginal gum (M) – 2 points and of alveolar gum (A) – 3 points.

$$PMA = \frac{\text{Number of points}}{3 \times \text{numbers of teeth}} \times 100$$

Rating score

In case of generalized gingivitis the value of index is:

- under 30 % - slight gingivitis;
- between 30 and 60 % - medium degree gingivitis;
- greater than 60 % - severe gingivitis.

Simplified Oral Hygiene Index (OHI-S)

Author – Greene and Vermillion (1964)

Measures – oral debris and calculus

Procedure for use – Divide the dentition into sextants.

Estimate oral debris, and supragingival and subgingival calculus on the facial and lingual surfaces of the teeth using the side of the tip of the periodontal probe or explorer. Select one tooth from each quadrant with the greatest amount of debris or calculus and score the facial and lingual surfaces using the following criteria:

Oral debris index

0 = No debris or stain present

1 = Soft debris covering not more than one-third of the tooth surface being examined, or the presence of extrinsic stain without debris, regardless of surface are covered

2 = Soft debris covering more than one-third but not more than two-thirds of the exposed tooth surface

3 = soft debris covering more than two-thirds of the exposed tooth surface

Calculus index

0 = No calculus present

1 = Supragingival calculus covering not more than one-third of the exposed tooth surface being examined

2 = Supragingival calculus covering more than one-third but not more than two-thirds of the exposed tooth surface, or the presence of individual flecks of subgingival calculus around the cervical portion of the tooth

3 = Supragingival calculus covering more than two-thirds of the exposed tooth surface or a continuous heavy band of subgingival calculus around the cervical portion of the tooth

Separately determine the Debris index (DI) and Calculus index (CI) by totaling the scores and dividing the total by the number of sextants. Add the DI and CI to determine the OHI-S.

Rating score

OHI-S	***DI-S or CI-S***
0,0 - 1,2 Good oral hygiene	0,0 – 0,6 Good oral hygiene
1,3 - 3,0 Fair oral hygiene	0,7 - 1,8 Fair oral hygiene
3,1 - 6,0 Poor oral hygiene	1,9 - 3,0 Poor oral hygiene

From here one may proceed to the inspection of buccal cavity. First it is necessary to perform a general inspection paying particular attention to the color of mucous tissues and degree of their uniformity.

Examination of tongue starts from the evaluation of papilla state, taking into account the fact that the tongue root to the right and to the left has a lymphoid tissue of pink or pink-cyan color that often is wrongly thought to be a pathological sign. Inspection of tongue includes evaluation of its dimensions, configuration and position.

During the inspection of buccal floor attention must be given to the state of mucous tissues. They are characterized by resilience, presence of folds and orifices of sub-mandibular and sublingual salivary glands. Special attention must be paid to the lingual frenum (length, fixation level).

The mucous tissue of solid palate is compact, in its frontal portion it may have transversal folds, and one may notice the point-shaped orifices of salivary channels and sometimes drops of their secretion.

The buccal mucous tissues may bear signs of primary and secondary lesions. Primary elements include: maculae, nodules, tubercles, vesicles, pustules, cysts, micro abscesses, and urticariotic papules. Secondary elements appear on the basis of primary ones and represent erosions, aphthe, ulceration, fissures, crusts, scum, cicatrices, and pigmentation.

Macula is a limited portion of mucous tissue with modified color. There may be maculae caused by inflammation and catarrhal maculae (non inflammatory ones). Inflammatory maculae that have diameter under 1.5 cm and are called roseola, while the ones with diameter above 1.5 mm are called erythemas. Catarrhal maculae originate from the melanin pigment and others, they can be congenital or acquired due to administration of certain medicines.

Papule is a non-cavity element of inflammatory origin with dimensions less than 5 mm that projects out of the level of the mucous tissue, comprises the epithelium and superficial strata of the mucous tissue itself.

Nodule differs from papule by the greater dimensions and by involvement of all mucous strata into the inflammatory process. During palpation one may detect a slightly painful infiltrate.

Tubercle is an element of inflammatory origin; it involves all the strata of mucous tunic. It has a diameter of 5-7 mm. Solid, slightly painful at palpation. The affected mucous tissue is hyperemic and edematous.

Rash is a limited and pronounced element of own mucous tunic observed in allergic reactions.

Vesicle constitutes a cavity formation of oval shape (under 5 mm in diameter) that projects out of the level of mucous tissue, being gilled with serous or hemorrhagic exudation. Located inside the epithelium and is easily torn.

Pustule is similar to the vesicle, but is filled with purulent exudation.

Bulla differs from vesicle by greater size, can be localized in the epithelium or under it.

Cyst is a cavity formation with epithelial stratum a membrane of conjunctive tissue.

Erosion is a lesion of mucous that doesn't go beyond the limits of epithelium and appears after the rupture of a vesicle, bulla or appears in the place of papule, or wound in result of a traumatic lesion. It gets epithelized without formation of cicatrices.

Aphthe is an erosion of oval shape covered with a fibrous membrane, surrounded by a circle of hyperemia.

Ulcer is a defect that comprises all the strata of buccal mucous tissue. An ulcer has walls and a bottom. After the healing of an ulcer remains a cicatrice.

Fissure is a linear defect that appears in a tissue due to loss of elasticity.

Scum is an epithelial formation that appears due to the disturbances of desquamation processes.

Crust constitutes a dry exudation that appears in the place of fissures and erosions.

Cicatrix is formed in result of substitution of a defect of mucous tissue by a conjunctive tissue.

Pigmentation is a modification of the mucous color or skin at the level of pathologic process due to deposition of melanin or other pigment.

Palpation is used for the determination of intumescences, induration, and mobility of organs or tissues of buccal cavity. The method of palpation depends on the localization and topography of pathologic nidus. Sometimes it is affected only with one forefinger, in other cases the mucous tunic is folded between two fingers. When palpating jugal tissues we use the forefingers of right and left hands. One forefinger is used for internal palpation, another one- for external palpation on the cheek. It is recommended to start from the palpation of evidently intact area of the mucous tissues with gradual nearing to the pathological nidus. Thus we can determine with a greater precision the limits of painful sensations and the limits of induration.

Supplementary investigation methods

Thermo diagnosis Reaction of teeth to the action of thermal agents is one of the oldest physical investigation methods used for the determination of the state of pulp. The excitation agent that may be used for children is hot and cold water introduced on a impregnated tampon into the carious cavity or applied to the tooth surface. The approximate indifference temperature for teeth is 30^0C. The temperature of 50-52 ^{0}C provokes the reaction on hot and the temperature of 17-22 ^{0}C – on cold excitement.

During the period of physiological resorption of temporary

teeth roots when takes place the pulp involution process, its sensitivity to thermal excitations is reduced.

Electroodontodiagnosis Electroodontometry gives an ampler image of the state of pulp and periodontal tissues. In órder to determine the electric excitability of pulp (EEP) are used the devices EOM-3 (for AC) and EOM-1 (for DC), as well as other devices that allow to determine precisely the linear tension. of electric current (minimum force of the exciting agent that may trigger the excitation). EEP is measured starting from the age of 6-7 years.

Intact teeth with already formed roots react to the currents of 2-6 mA. At the incipient stages of dental caries the sensitivity of teeth to the action of electric currents doesn't change significantly. A higher EEP is observed on carious teeth, on teeth with cracks, superficial or medium caries. In cases of profound caries EEP depends on the state of pulp.

EEP varies depending on the root development degree: during the period of physiological dental eruption EEP is reduced, while during the formation of roots it reaches the normal values. Should the necessity to determine the EEP appear during the root development period, first one must measure the EEP of the tooth from opposed semi-arcade and only thereafter the EEP of the affected tooth; the obtained results are compared. It is not recommended to measure the EEP of temporary teeth as children do not react adequately on excitation (reaction depends on the type of nervous system, etc.) and do not interpret objectively their sensations. During the period of formation and resorption of dental roots the EEP values of intact teeth are comparatively smaller - it depends on the degree of root formation.

Radiological investigations have become especially frequent in dentistry as in some cases they are practically the only way to identify the modifications that take place in tissues.

However, due to the harmful influence of Roentgen's rays on

human organism, especially during the period of growth, it is necessary to establish strict requirements for the performance radiography. In order to protect the growing organism against the action of Roentgen's rays lead-rubber aprons and sensitive films are used in order to reduce the emission time. In order to reduce the doses of Roentgen's rays are used the fixed apparatuses with greater focus length (1 m and more) with amplifier screens. In order to reduce the irradiated field one may use diaphragms and filters of different thickness.

In order to reduce the cumulative effect of ionizing radiation repeated investigation is allowed only after 3 weeks, while in cases of simultaneous irradiation the minimum repeat period is 5 weeks. Panoramic radiography at children is allowed only once a year.

Pedodontic practice makes use of all existing types of radiography that may be divided into 2 groups: 1) basic methods (endo- and exo-buccal radiography) and 2) supplementary methods.

Basic methods of radiography The most frequently used method is endobuccal radiography that allows obtaining images of 3-4 teeth and of a portion of alveolar process. Endobuccal radiography allows us to determine the state of solid dental tissues, degrees of root and channel formation, the teeth in eruption and the dental buds, foreign bodies in radicular channels, the degrees of permeability or obturation of radicular channels, state of periapical tissues, state of bone tissues and interdental septum, etc. Presently in the dentistry practice is used the technique name radiovisiography based on the digital technologies with multiple advantages - it allows to obtain high quality images, perform various measurements and calculations, store the information in a PC, etc.

Exobuccal radiography is used for the investigation of facial bones, maxillaries and teeth when endobuccal radiography is

possible.

Supplementary methods of radiography

Panoramic radiography is characterized by the production of simultaneous image of all teeth and bone tissues of maxillaries and mandible. Panoramic radiograms enlarge the image 1.5-2 times and perfectly show the structure of bone tissues. For this reasons they are used for the appreciation of general state of the dental-maxillary system and state of periodontal tissues.

Orthopantomography allows obtaining enlarged images without extension in plane for both mandible and maxillary on a single film. This allows comparing the state of bone tissues of different sectors.

Tomography allows obtaining radiological images for each stratum of bone located in the depth of the tissue. This technology allows obtaining information on the state of each stratum of tissue. It is used to identify the alteration centers of limited dimensions in the profound strata.

Computerized tomography allows determining precisely the location of affected tissues, identifying any incipient changes at different levels of tissues of the dental-maxillary apparatus. Nowadays it is the only methodology of obtaining simultaneous images of soft tissues and bones. Tomography is also used for the identification of temporal-mandibular articulation pathologies, congenital and acquired deformations, fractures, cysts, tumors, systemic maladies, etc.

Contrast radiography is used for the investigation of salivary glands (sialography), sanguine vessels (angiography), lymphatic ganglions (lymphography), of temporal-mandibular articulation (arthrography). This method involves administration of radio-contrasting substances (iodolipol, urographine, dianosil, maiodil, iliiodol, ultra-liquid lipoiodol, etc.).

Particularities of radio anatomy of dental- maxillary apparatus of children at different ages

The process of dental-maxillary system development includes 3 periods:

1. First period - it lasts till the eruption of permanent teeth - from the very birth and till the age of 5-6 years.
2. Second period - period of mixed dentition - begins at the age of 6-7 years and lasts till the age of 12-13 years.
3. Third period – period of permanent dentition, it begins at the age of 12-13 years.

First period At newborns each maxillary contains 18 dental follicles (10 follicles for temporary teeth and 8 - for permanent). Dental follicle on the radiogram represents a transparency of bone tissue of oval shape surrounded by the cortical blade. The outlines of the future tooth become visible only after the beginning of mineralization, when appear the calcification centers (in the incisive and cuspidal regions) that afterwards unify and form the contour of crown. At newborns radiograms of maxillaries display the mineralization of 1/6 of the crowns of central incisors, 2/3 of crowns of lateral incisors and in the region of cuspids of temporary molars. Radiogram illustrates the complete calcification of temporary teeth crowns in the following age: -

- central incisor - 1-2 months of life;
- lateral incisor - 3-4 months of life;
- canine - 9 months of life;
- molar I – 6 months of life;
- molar II - 12 months of life.

Root formation takes place partially during the period of follicular development and continues in the period of dental eruption and after it. During the period of temporary teeth eruption the adjacent bone tissue of the incisive margin or masticatory surface of dental crown is rhizalyzed. Simultaneously with the dental eruption takes place the rhizalysis of bone tissues

that surround the follicle while with the beginning of root development starts the formation of inter-alveolar septum.

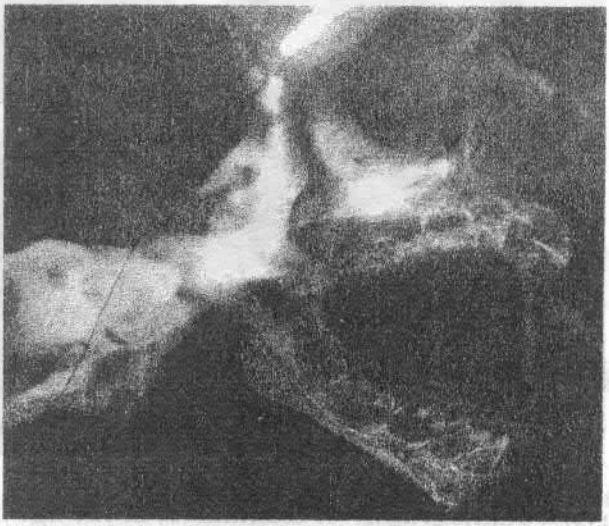


Fig.3.5. **Radiography of maxillaries at newborn**

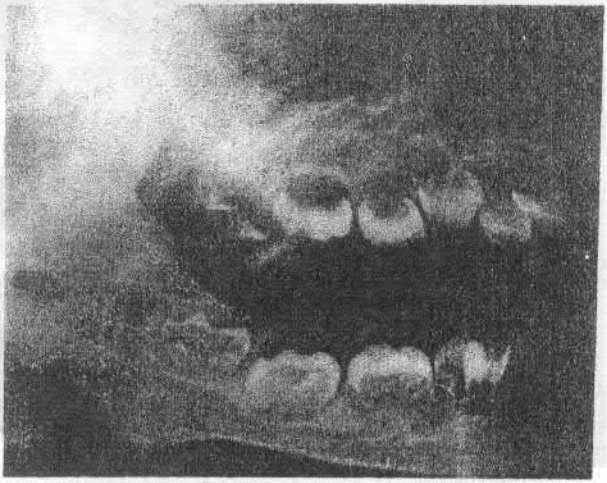


Fig.3.6. **Radiography of maxillaries at the age of 6 months**

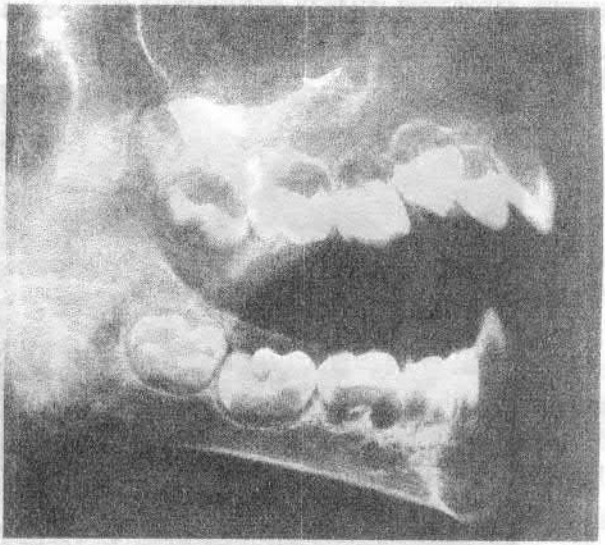


Fig.3.7. **Radiography of maxillaries at the age of 9 months**

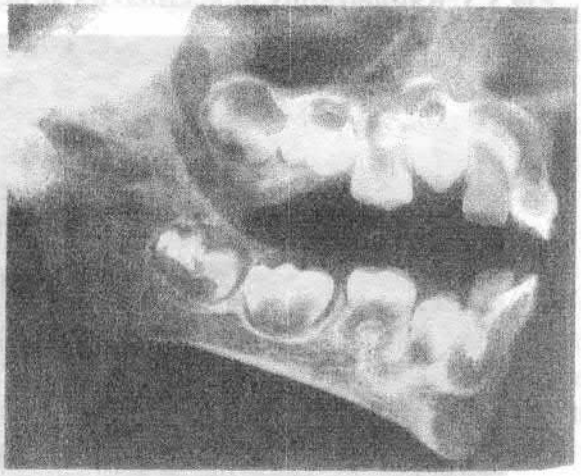


Fig.3.8. **Radiography of maxillaries at the age of 1 year**

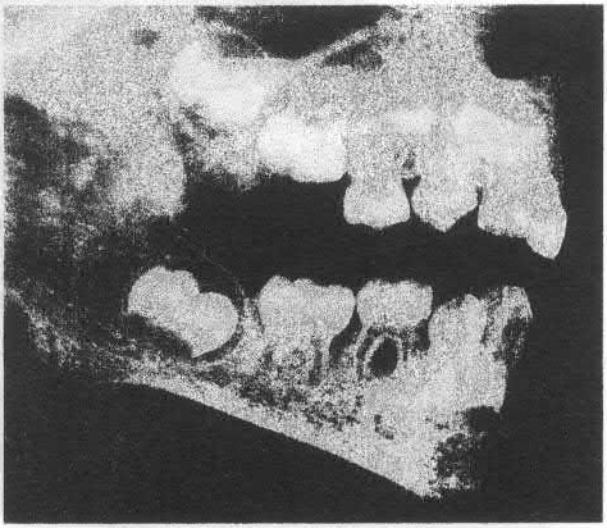


Fig.3.9. **Radiography of maxillaries at the age of 2 years**

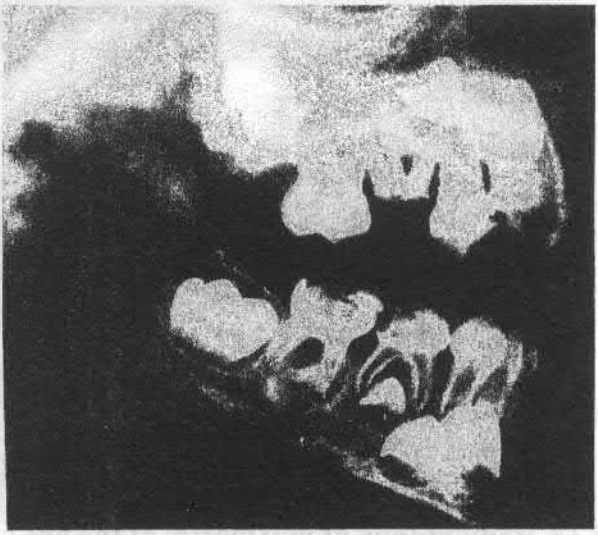


Fig.3.10. **Radiography of maxillaries at the age of 4 years**

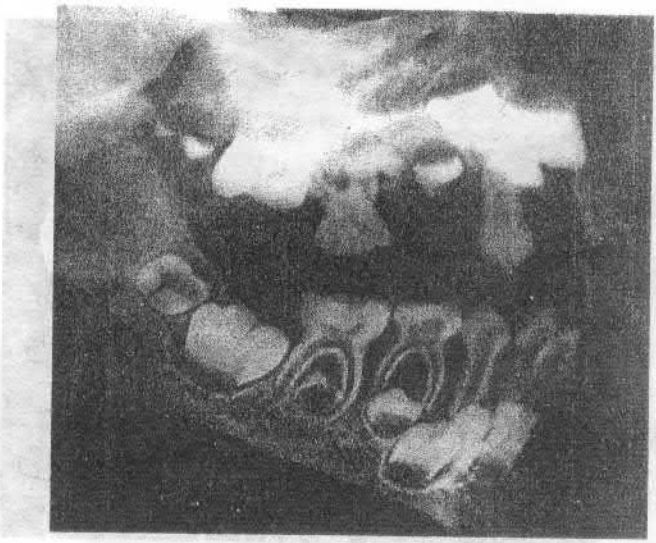


Fig.3.11. **Radiography of maxillaries at the age of 5 years**

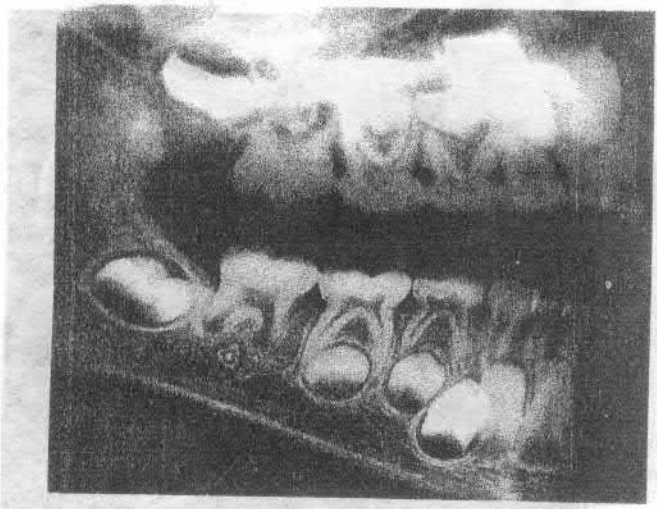


Fig.3.12. **Radiography of maxillaries at the age of 6 years**

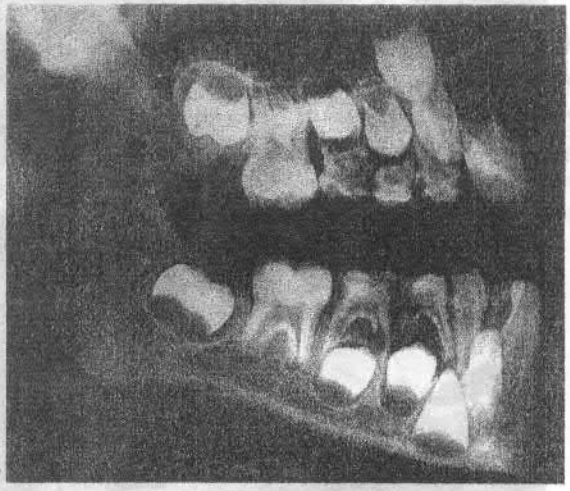


Fig.3.13. **Radiography of maxillaries at the age of 7 years**

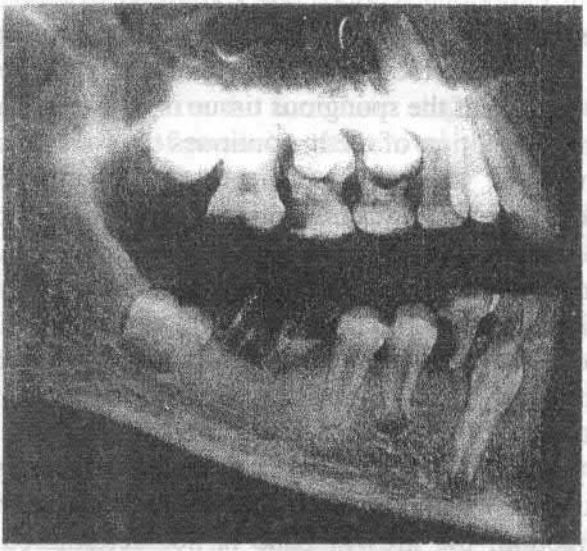


Fig.3.14. **Radiography of maxillaries at the age of 9 years**

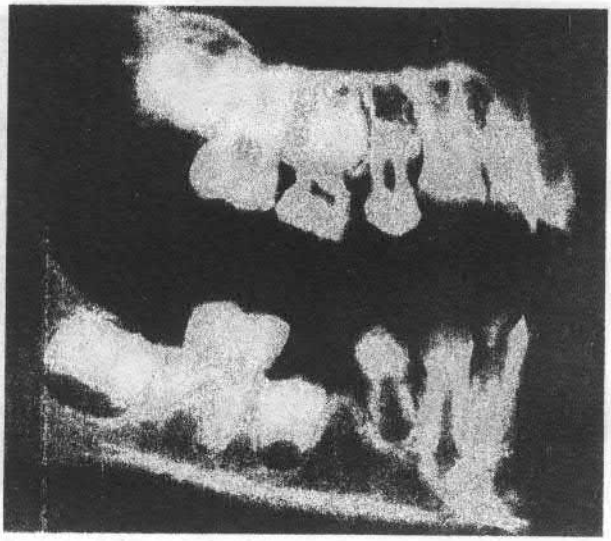


Fig.3.15. **Radiography of maxillaries at the age of 10 years**

Upper edges of the inter-dental septum appear leveled in the direction of erupting tooth; the compact blade is thickened a little, while the texture of the spongious tissue is not well pronounced.

After the eruption of teeth, continues the root formation from collum to the apex. In the process of root formation, radiologically we distinguish 2 periods: period of incomplete apex and period of complete apex.

During the period of incomplete apex radiograms display a wide radicular channel that in the apical region has a funnel-like shape and is surrounded by a zone of transparent bone tissue of oval shape - the "growth zone", well delimited from the cortical blade of alveole.

The period of complete apex includes two stages. On the stage of open apex the radicular channel becomes narrower in the apical direction, the growth zone is not determined any more, while the periodontal zone in the apical region becomes enlarged,

and the wide apical orifice is well seen. On the stage of close apex the radicular channel doesn't "communicate" with periodontium.

On the radiological images temporary teeth display the following characteristic features: their crowns and roots have smaller dimensions, the radicular channels and pulp cavities are wide, while the roots are more furcated.

Primary mineralization of permanent teeth crowns begins in the following ages:

- First molar - at newborns;
- Central incisor - 6 months;
- Lateral incisor - 6 months;
- First premolar – 2,5 years;
- Canine- 9 months;
- Secondary premolar- 3,5 years;
- Secondary molar – 2,5-3,5 years.

During this period special attention must be paid to the report between the buds of premolars and roots of temporary molars. At the age between 2 and 3 years takes place the calcification of crowns of premolars whose follicles are located between the roots of the temporary molars on the formation stage. The periodontal zone is clear. Follicles of premolars have oval shape and are well delimited. Between the cortical llama of the follicle and the alveole of the temporary tooth in the region of root bifurcation some bone tissue with alveolar texture is identified. Simultaneously with the growth of maxillaries and calcification of crown the follicle of the permanent tooth migrates to the edge of maxillary. During the period when the roots of temporary teeth are completely formed, the periodontal zone has clearer limits on the external surface of roots. On the internal surface of roots the periodontal zone is unclear, narrowed, especially in the region of root bifurcation.

The following stage is characterized by the growth of permanent teeth roots, resorption of temporary teeth roots and

migration of follicles towards the alveolar process. The follicles of permanent teeth have clear contours and are located in the immediate vicinity of temporary teeth roots. Before the alternation of teeth the roots of temporary teeth come in direct contact with the follicles of permanent teeth.

Second period This period begins in the age of 5-6 years and is characterized by the alternation of temporary teeth with the permanent ones. From complete formation of crown and till the eruption of tooth it takes approximately 5 years. Eruption of permanent teeth is anticipated by the resorption of temporary teeth roots.

Simultaneously with the resorption of temporary teeth roots take place the bone tissue formation processes. Due to this fact the resorpted roots are surrounded by bone tissues.

Resorption of temporary dental roots doesn't occur identically and uniformly at all teeth and is determined by the positioning of permanent tooth follicle in relation to the temporary teeth roots. Radiologically one can distinguish 3 types of physiological resorption of temporary teeth roots:

I - uniform resorption of all roots in the apical region is produced in vertical direction, reducing the roots in length. The radicular resorption predominates, while the resorption in the region of bifurcation is minimal.

II – simultaneously with the partial resorption of roots in the region of radicular bifurcation predominates the resorption of a root, namely – of the one located in the vicinity of the permanent tooth bud. More intense is the resorption of jugal roots, especially of the posterior jugal root at superior molars and distal roots of the inferior molars.

III – the resorption predominates in the region of root bifurcation. In some cases the morphological integrity of the apical region of root is preserved, while in the region of bifurcation resorption is so extensive that communication with the

coronary pulp is established.

Resorption of incisor and canine roots is produced according to the type I, in some cases predominates the resorption of the lingual surface of incisor roots and medial surface of canine roots.

During the second period the teeth on the orthopantomograma are presented in three rows on each maxillary. The first row forms the temporary teeth located in the dental arcade, the second one form the buds of permanent teeth - at different development stages, while the third one constitutes the canine buds: on maxillary they are located under the infraorbital edge, while on the mandible they are above the cortical stratum of the inferior margin.

Third period The third period begins at the age of 12-13 years and is characterized by the presence of permanent teeth only with roots at different development stages.

Methods of laboratory investigations

Microscopic investigations are performed in order to determine the cellular state of wound surface and identify qualitative modifications of the cells forming the mucous tunic, as well as to determine the bacteria on the surface of the wound or mucous. Depending on the scopes we can distinguish: cytological method, biopsy and bacteriological investigations.

Cytological method is used for the diagnosis of viral infections, acantolytic pemphigus, tuberculosis ulcers, and tumors and in order to evaluate the efficiency of administered treatment.

Bacteriological investigation involves bacterioscopy of materials obtained from the surfaces of mucous tissues of buccal cavity, erosions and ulcers. Used for the determination of mycosis, syphilis, etc.

Clinical and biochemical analysis of blood is done for all children with generalized affections of periodontium and mucous tissues of buccal cavity, with systemic maladies and in cases of individual indications.

Diagnostics of bacterial and medicamentous allergy

In order to determine the immediate allergic reactions are used the cutaneous samples with bacterial allergens, leuko- and thrombocytopenic tests, hemagglutination test, etc.

Prolonged allergic reactions are identified with the help of cutaneous samples, leukocytosis reactions, lymphocyte blast transformation test, etc.

Luminescent diagnosing

One of the methods of luminescent diagnosing is the investigation in the Wood's rays that is used for the differentiation of some affection of mucous tissues of buccal cavity and isolated lesions of the red margin of lips.

All the data of clinical examination are introduced into the patient's record file. After the objective and subjective examination, including the supplementary investigation methods the disease diagnosis is formulated. In complicated cases a preliminary diagnosis is established while the definite diagnosis is made only after the complete performance of special diagnostic methods or after some preventive treatment.

On the basis of established diagnosis and individual particularities of the patient the treatment plan is produced. The treatment plan allows undertaking complex therapeutic actions taking into account all the specific elements.

Nowadays the data on patients and the methods of their treatment in some medical institutions (including dental clinics) are introduced into personal computers. This allows increasing considerably the efficiency of registration and archiving of data: data can be stored, periodically updated, consulted immediately, analyzed, etc.

Control questions and topics:

1. What is the OMS dental formula?
2. What is the dental formula by Zigmondy?
3. What is the dental formula by ADA?
4. What are the main elements of the patient's observation card?
5. Degrees of tooth mobility.
6. Dental caries incidence index.
7. Dental caries intensity index.
8. The CPITN index.
9. The Papillar-Marginal-Alveolar (PMA) Parma index.
10. Oral Hygiene Index – Simplified (OHI-S).
11. Affections of mucous by primary and secondary lesion elements.
12. Supplementary methods of radiography.
13. Mineralization of temporary teeth crowns.
14. Primary mineralization of permanent teeth crowns.
15. Types of physiological resorption of temporary teeth roots.
16. Luminescent diagnostics.

4 STRUCTURAL AND DEVELOPMENTAL ANOMALIES OF TEETH

They represent numerous groups of dental tissue affections that can be manifested in an isolated manner or be combined with anomalies of structure and development vices of organs and systems of child's organism.

Dental pathology (by number, shape, volume, structure, etc.) represents a big number of clinical forms described by the specialty literature, most of them are described by different authors in different chapters, such as "Non-carious affections of solid dental tissues" (by Патрикеев В. К., 1973, Колесов А.А., 1991, Курякина Н.В., 2001), "Dental hypoplasia" (by Агапов Н.И., 1929, Вайс С.И., 1965, etc.), "Dental Dystrophy" (Zarnea L., 1993, Cocarla E., 2000), "Disturbances of tooth development" (Cura E. 2000), "Dental Anomalies" (J. P. Fortier, 1987). We consider it rationally to study this pathology in the chapter of structural and developmental anomalies of teeth.

Dental anomalies can be manifested in an isolated manner; however, more often they are combined with other anomalies of structure and development vices of maxillaries or face and with anomalies and vices of organs and systems of child's organism.

Dental tissues have a mixed origin: the enamel has ectodermal origin, while the dentine, cement and pulp - mesodermal origin. Due to these reasons the structural dental anomalies can be both isolated and combined. The tooth development process passes through several stages: proliferation, formation and mineralization of enamel, formation and mineralization of coronary dentine, tooth eruption, formation of dentine and radicular cement, resorption of root (temporary

tooth), maturing of dental tissues after eruption. At each of these stages pathological factors of both hereditary and acquired origin can intervene.

Hereditary affections are provoked by changes in the genetic code – mutations induced by the action of certain external factors (radiation, some chemical compounds, etc.) or internal factors on cells and on the organism in totality.

Besides these, they are formed on the basis of system pathology (hereditary, congenital and acquired), endocrine disturbances or maladies, infectious diseases, etc. at mothers during pregnancy or at children during the first years of life.

Among the harmful factors able to provoke dental anomalies must be mentioned: intoxication (industrial, alcoholism, smoking, drugs, etc.) and irradiation of mother during pregnancy or of the child during the first years of life.

The local factors able to provoke dental anomalies are: traumas of maxillaries and dental buds, inflammatory processes and tumors of maxillary-facial region at children.

As a well, dental anomalies and dental development vices can be results of disturbances in the enamel formation mechanism and dentine of dental crown, dentine and radicular cement, of the dental eruption mechanism and radicular resorption, disturbance of the tooth maturing process after eruption, etc.

Anomalies and vices of tooth development that appear after the systemic maladies of organism are characterized by the following signs:

1. Affected are groups of teeth with the same terms of foundation and development, or all teeth are affected;
2. Simultaneously is observed the appearance of several types of anomalies;
3. Distortions of maxillaries or occlusion are observed;
4. Some specific disturbances are typical for certain pathologies: Hutchinson teeth, Moser syndrome, etc.

The harmful factors can provoke the same or different anomalies and development vices of tooth depending on the tooth development period. Various negative factors, acting on the development of dental-maxillary system during the same development periods can provoke the same vices and anomalies. However, the same factor acting during different development periods can provoke different types of anomalies and vices. Of special importance are the intensity and duration of harmful action.

There are many classifications for this group of dental pathologies.

The classifications of American authors (Cohen, Pindburg and Sewetin, McDonald) consist in the systematization of the total modifications in the dental tissues (by number, by shape and dimension). These very extensive classifications, however, are too sophisticated for being used in practice. The classifications of French authors (Gilbert, Lebourg, Magitot and Dechaume) categorize dental anomalies depending on the development periods during which the etiologic factors applied. These classifications are more accessible for practical application. Nevertheless, not all the mechanisms invoked therein comply with reality.

Magitot distinguishes the dental dystrophy in the following manner:

1. Primary dystrophy caused by impacts during the tooth formation period:

a) stable dystrophy (cicatrices): number, volume, shape and structural modifications, hypoplasia;

b) progressive dystrophy (evolutive): amelogenesis imperfecta, dentiongenesis imperfecta, enamel and dentine aplasia.

2. Secondary dystrophy that appears after some period of normal state of teeth: poly caries, Dubreuil syndrome, circular caries.

Classification of Dechaume
(completed by Burlui et. al., 2000)

Class I – Anomalies of location, volume, shape and direction.

Class II – congenital coronary dental lesions with hereditary specifics:

- amelogenesis imperfecta;
- hereditary opalescent dentine:
  a. Capdepont dysplasia;
  b. Hodje opalescent dentine;
  c. dentinogenesis imperfecta;
  d. dental dysplasia;
  e. Phantom teeth.

Class III –coronary dental lesions acquired before dental eruption:

- simple enamel hypoplasia;
- complex hypoplasia – Hutchinson teeth;
- irradiation-caused anomalies – Moser teeth.

Class IV - coronary dental lesions acquired after the eruption period:

1. on temporary teeth:
   a. Beltrami and Romieux melanodentism;
   b. Dental vulnerability - multiple caries with evolution on surface and in depth;
   c. Caries, abrasions and traumas.
2. on permanent teeth:
   a. Dubreuill Chambardel syndrome;
   b. Dental lesions caused by consecutive medicamentous intoxication (morphine) - present in the form of carious lesions with localization on the dental collar;
   c. rampant caries;
   d. dental lesions caused by prolonged irradiation;
   e. dental dyschromia:

- of internal origin - Rh incompatibility;
- wrongly executed pulpectomy.

f. dental wearing:

- physiological (attrition);
- pathological (dental abrasion).

g. dental erosion:

- chemical
- idiopathic.

h. dental lesions caused by dental caries.

Pathological factors can affect the genotype; provoke various clinical forms of anomalies – from simple to composite. It is quite difficult to differentiate the etiology of number anomalies from the etiology of structural anomalies. Due to these considerations it is more convenient to use a simpler ***classification proposed by Fortier:***

- anomalies of morphology and number;
- structural anomalies.

В.К.Патрикеев (1968) recommends the following classification of non-carious affections of teeth:

1. Dental affections appearing during the period of formation and development of dental tissues, i.e. before eruption:

a) hypoplasia; b) hyperplasia; c) endemic fluorosis; d) development and eruption anomalies; modification of tooth color; e) hereditary modifications of tooth development (amelogenesis imperfecta, dentinogenesis imperfecta, Stainton-Capdepont dysplasia, imperfect osteogenesis, marble-bone [Albers-Schonberg] disease, hypophosphatosis, etc.).

2. Dental affections appearing after eruption: a) pigmentation and dental deposits; b) abrasion; c) cuneiform defect; d) necrosis (erosion) of solid dental tissues; e) dental trauma; f) hyperesthesia.

Classification of strcutural anomalies and development vices of solid dental tissues *(by Т.Ф. Виноградова, 1987)*

1. Structural anomalies of teeth with hereditary transmission: amelogenesis imperfecta, dentinogenesis imperfecta, Stainton-Capdepont syndrome;

2. Anomalies of number, volume and shape with hereditary etiology;

3. Structural and developmental anomalies of dental tissues caused by systemic pathology (hereditary, congenital and acquired): hereditary dysplasia (adontia, hyperdontia, conical teeth, hypophysary nanism (microdontism), osteogenesis imperfecta (amber teeth), syphilis (Hutchinson teeth), hemolytic syndrome and hemolytic icterus (gray teeth, brown teeth, etc.).

4. Structural and developmental anomalies of dental tissues provoked by the action of external factors: fluorosis, "tetracycline teeth", systemic hypoplasia, local hypoplasia.

DENTAL ANOMALIES OF NUMBER

Represents reduction or increase of number of teeth compared to the age norm.

Total absence of teeth and dental buds is named ***anodontia*** (*primary total adontia*). ***Partial anodontia*** (*partial adontia*) is manifested by the reduction of teeth number: ***hypodontia*** - if at least 4 dental buds are absent; ***oligodontia*** - of more than 5 dental buds are absent.

Adontia can be hereditary or caused by the influence of pathologic factors on the VI-VIIth weeks of embryonic development when the dental plate is being formed.

Partial adontia of temporary teeth can appear during the period of VII-X weeks of intrauterine development, while that of permanent teeth – during the XVII week (see color insert fig. 4.1.).

Total adontia is an extremely rare phenomenon caused by ectoderm dysplasia (severe congenital disease characterized by disturbances of ectoderm development).

Primary partial adontia is usually observed on the maxillaries and is characterized by the absence of lateral incisors. On the mandible can be absent the premolars, molars III can often be absent on both maxillaries due to the involution of the dental-maxillary system in the process of phylogenesis.

Hyperdontia is the excessive size of teeth manifested by the appearance of supernumerary teeth. In cases of hyperdontia the number of temporary teeth can exceed 20, while the number of permanent teeth can exceed 32. Frequent localization of the supernumerary teeth is found in the region of superior teeth (in 90% of cases by P. Firu and M. Rusu).

The over-numbered teeth can appear in the region of superior and inferior incisors: *mesiodens* – between the teeth 11, 21; between the central and lateral incisors (fig. 4.2.); *peridens* – than the tooth appear out arcades; rarely: in the molar region – the fourth permanent molar localized in distal area from the third one in the region of premolars - three premolars.

Fig.4.2. **Mesiodens**

These teeth, according to the opinions of some authors, appear due to the excessive development of dental lamina, and its division under the influence of certain hereditary factors. Other authors state that the appearance of supernumerary teeth by the division of dental follicle or by the manifestation of atavism as

the primates had 6 incisors – central incisors have disappeared in the process of phylogenesis.

Supernumerary teeth can erupt in the nasal cavity, at the edge of orbit and can provoke various disturbances. Crowns of supernumerary teeth can have atypical shapes and dimensions, often their formation periods do not coincide with the average foundation periods of normal teeth.

Their shape can be similar or totally different than that one of neighboring teeth: they can erupt on the arcades or remain narrow, provoking various disturbances. The presence of supernumerary teeth can be associated with certain general order anomalies: oro-digital-facial (syndactily, bone anomalies, multiple oral frenulum, and lobular tongue), cleido-cranial disostosis (aplasia of clavicles), etc.

ANOMALIES OF TOOTH SHAPE

Anomalies of tooth shape in the majority of cases have hereditary causes; however, they can appear also in the consequence of local action factors. Disturbances appear during the period of dental tissue morphogenesis. Clinically we can distinguish partial or total anomalies of dental shapes. Anomalies of shape are determined in the permanent dentition, especially in the cases of supernumerary teeth.

Partial disturbance of crowns are less often met. Most frequently are observed the supernumerary tubercles:

- Carabelli tubercles – on the palatal surface of first superior permanent molars;
- Bolk tubercles – on the vestibular surfaces of molars I and II (16, 17, 26, 27);
- Zuckerkandel tubercles – on the vestibular surfaces of the second temporary molars.

Supernumerary tubercles appear as result of increase of activity of some of the growth centers of molars during the period

of morphogenesis.

Knowledge of the anomalies of shape and number of roots is of special importance for the endodontic treatment and dental extraction. These radicular anomalies can be observed on:

- Inferior canines - they can display bifid roots (33 and 34);
- First superior premolar (14 and 24): their roots can be unified or curved, they can have 3 roots;
- First inferior premolar (34 and 44): can have 2 roots;
- First superior molars (16 and 26): can have 4 roots or fusion vestibular roots;
- Second and third inferior molars (37 and 47; 18, 28, 38 and 48) can have joined roots, convergent, divergent roots, etc.

Total anomalies constitute the group of amorphisms and represent fusion by concrescence or gemination, syndesmo-coronary anomalies.

Amorphisms include the teeth that have distorted dental crown: fish-shaped teeth, narrow teeth, "piano key" teeth, triangular teeth, torsion teeth, "rye-seed" teeth, "nail-shaped" teeth, "screwdriver" teeth, Hutchinson teeth, Fournier teeth, Pfluger teeth, etc.

Hutchinson teeth - central superior incisors have the shape of screwdriver or of spindle at dental crown, their dimensions in the collar region exceeding the dimension of incisive margin, while the incisive margin has a crescent shape.

The Fournier teeth are the central incisors with screwdriver-like shape; however they do not have a crescent incision.

The Hutchinson and the Fournier teeth were previously considered as symptoms of congenital syphilis, or one of the signs of triad: parenchymatous keratitis, congenital deafness and anomalies of shape of incisors. However, it was found that this anomaly is observed in other cases: rachitis, etc.

Pfluger teeth: first molars are cone-like, while the crown dimensions at the collar exceed the dimensions of masticatory

surface.

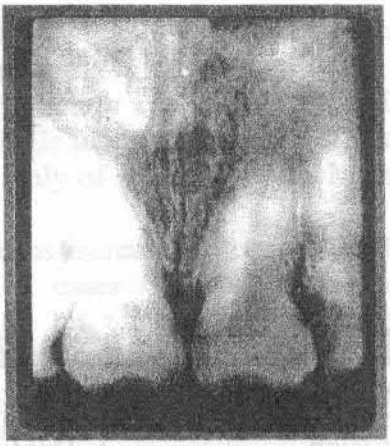


Fig.4.3. **Hutchinson teeth**

Turner teeth: premolars have maculae of white-yellow-brown color with partial or total defects of enamel and dentine. Such teeth can be the result of ameloblast function disturbances, sometimes they are caused by disturbances of odontoblasts due to the extension of the inflammatory process of apical periodontal tissues on the dental follicles or in cases of traumas of temporary teeth with affections of permanent tooth buds.

Fusion by concrescence or germination Dental fusion (fig. 4.5.) is characterized by the fusion of all tissues of two or more teeth (see color insert fig. 4.4.).

Concrescence is characterized by fusion at the level of roots. Gemination (fig. 4.6.) is an anomaly of shape and volume, resulting from the incomplete inclination of a dental bud during morphogenesis causing a bifid crown with a single root to appear. Gemination is favored by the immediate proximity of follicles that have to determine it, by the simultaneity of calcification, convergence and local traumas or inflammation. Most often it is met in the permanent dentition in the region of incisors.

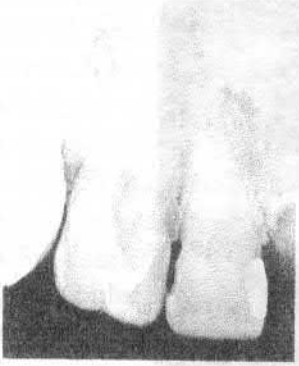


Fig. 4.5. **Radiography: Dental fusion**

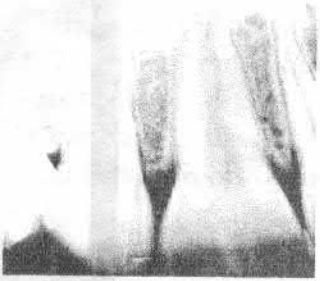


Fig.4.6. **Radiography: Dental gemination**

Dental invagination *(dens in dente)* (fig.4.7.) It is a development anomaly caused by the invagination of internal adamantine epithelium in the dental papilla causing the appearance of a tooth inside another tooth. Most often such "teeth" are found radiologically in the pulp chamber of permanent lateral incisors.

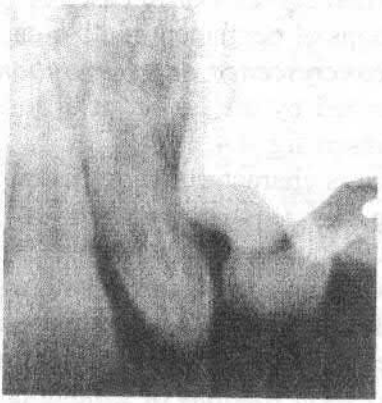


Fig.4.7. **Dens in dente**

ANOMALIES OF TOOTH VOLUME

Anomalies of tooth volume are consequences of genetic or constitution disturbances.

Macro- and *microdontia* can affect the entire dentition, a group of teeth or a single tooth. Separate modifications of a tooth can be identified, or only of crown or roots. Most frequently are affected the incisors.

Macrodontia means increase of tooth sizes. Macrodontia of all teeth is found in cases of neurofibromatosis (congenital deformation followed by tumoral growth of tissues), while gigantism of all teeth is provoked by the pathology of hypophysis.

Solitary gigantic teeth appear due to the fusion of normal tooth with the supernumerary one:

- In case of complete fusion the gigantic crown has an incision on the incisive part that indicates the limit between the two fusion teeth. Radiography denotes a gigantic pulp cavity and a single radicular channel;
- If only crowns are in fusion – the radiogram shows only a big pulp cavity and two radicular channels;
- If only roots are in fusion – only one radicular channel is displayed with two pulp chambers.

Increase of dental root volume is known as *rhizomegaly*. Most often is observed the rhizomegaly of superior canines that in length can exceed 40 mm.

Microdontia is reduction of tooth size. Total microdontia has a hereditary character. However, sometimes it can be provoked by endocrine disturbances (hypophysary nanism). Reduction of tooth size can be caused by ectodermal dysplasia.

Microdontia of lateral superior incisors and third molars (fig.4.8.) is considered to be a manifestation of phylogenetic teeth reduction process. Reduction of solitary teeth sizes can be caused by the action of local factors: inflammatory processes, traumas and tumors.

Fig. 4.8. **Microdontia of the third molar**

Reduction of root volume (*radicular nanism* or *rhizomicria*) is characteristic for lateral incisors, premolars and third molars.

STRUCTURAL ANOMALIES OF TEETH

Structural anomalies of teeth are development disturbances appearing in the process of odontogenesis on the stages of histological and morphological differentiation and mineralization. For this reasons anomalies of structure affect all the dental tissues (enamel, dentine, cement) in separate or integrally – of both temporary and permanent teeth.

Depending on the period of odontogenesis when the pathological factors occurred, on the intensity and duration of pathological influence one can determine the type, localization, degrees and clinical evolution of structural anomalies that are frequently combined with other kinds of anomalies.

Systemic hypoplasia is a development vice manifested by the disturbances of structure and mineralization of dental tissues caused by the influence of harmful factors on the dental follicle during the period of development and mineralization. These factors lead to disturbances in the embryogenesis of teeth and are manifested by non-specific disturbances of the formation and

mineralization of dental tissues. The vices appear due to the disturbances in the functions of ameloblasts, while in severe cases – of the odontoblasts. Affected are the teeth that got formed and mineralized during the period of action of harmful factors.

Some researchers consider that hypoplasia implies disturbances in the formation of solid tissues due to modifications in ameloblasts (Абрикосов А.И, 1914, etc.).

Other authors consider hypoplasia to be a consequence of disturbances in the process of mineralization of solid dental tissues (etc.).

Other authors consider hypoplasia to be a consequence of disturbances in the process of mineralization of solid dental tissues (Лукомский И.Г., 1953, Вайс С.И. 1965, etc.).

И.О. Новик (1961) and А.И. Рыбаков (1966) consider that these two processes are indivisible and have a reciprocal relation. To their opinion, hypoplasia is the result of disturbances in the enamel formation process of ameloblasts associated with disturbances of mineralization processes.

To the opinion of В. К. Патрикеев (1967), in the case of dental hypoplasia are affected not only the processes of mineralization but also there are organic traumas of enamel due to the delayed or insufficient functioning of ameloblasts.

In experimental hypoplasia biochemical processes in teeth are preceded by morphological changes. In experiments on animals with use of radioactive isotopes it was determined that hypoplasia implies disturbances of mineral and protein metabolism. The role of protein metabolism was demonstrated by the fact that the enamel matrix and the matrix of dentine are formed and get mineralized simultaneously, in parallel with other processes of tooth formation.

Hypoplasia can affect both temporary and permanent teeth. Temporary teeth are affected due to the metabolic disturbances at pregnancy (general diseases, pregnancy toxicosis, insufficient or

irrational nutrition, intoxication, administration of medicines, etc.) leading to hypoxia. It appears at children that during the first months of life suffered from CNS diseases, hemolytic icterus, rachitis, nephropathy, at children with disturbances of gastrointestinal system, infectious diseases, etc.

Frequently it is observed at prematurely born children and artificially-fed children (see color insert fig. 4.9.).

Affections of permanent teeth can be a consequence of disturbances of physiological state of child's organism in cases of general diseases (diseases of gastrointestinal tract, gastroduodenitis, colitis, hepatitis, etc., affections of respiratory system: bronchitis, pneumonia, acute viral respiratory diseases, and infectious diseases: dysentery, etc.).

If the pathology occurs during the first year of life, usually the incisive margin of permanent incisors and the cuspids of molars of 6 years are affected (see color insert fig.4.10.).

When the action of harmful factors occurs on the second-third years of life, the modifications will be found in the central region or pre-gingival region of crown on the same teeth. Dental hypoplasia is frequently associated with open and mesial occlusion.

Manifestation forms of systemic hypoplasia and its localization depend on the intensity and duration of the affecting factor.

Localization of process on the dental crown indicates the age at which the child suffered from the respective affection, the width of defect denotes the duration, while the character and deepness of defects denote the intensity of pathological factor (see color insert 4.10.).

There exist the following clinical forms of systemic hypoplasia:

1) maculated - white maculae are observed on the surface of dental crown – they are chalky, white , have clear borders, have a

smooth and shiny surface, localized at the same level;

2) undulated - depressions, oval or round - with different dimensions and depth, on some sectors the yellow dentine can be seen;

3) furrow - depressions of different width and deepness frequently oriented parallel to the incisive margin of teeth;

4) destructive – aplasia or absence of enamel in a sector of tooth.

All the forms of systemic hypoplasia are more pronounced on the vestibular surfaces – compared to the palatal ones.

Systemic hypoplasia can be: 1) combined with a process of demineralization (maculae or carious cavities on the sectors not affected by hypoplasia); 2) complicated by caries (when a hypoplasic sector is affected by caries); 3) complicated and associated with caries and modifications of tooth color.

Treatment Hygiene of the buccal cavity is indicated. In easy cases no treatment is indicated.

Maculated form: 1) remineralization therapy; 2) application of solutions and gel with fluoride 3-4 times a year 10-15 procedures. After the remineralization therapy defects can be filled with ionomer resins, compomers or composite materials. In cases of extensive lesions the masticatory teeth are covered with pre-fabricated crowns.

Prophylaxis Health protection of mother and child (healthy style of life and work, rational nutrition, avoidance of intoxication, timely diagnosing and treatment of general diseases, etc.); prophylaxis of general diseases at children of young ages, natural breast-feeding, etc.

Tetracycline teeth Administration of tetracycline to pregnant women and children of small ages can provoke modifications of color of temporary and permanent teeth, while administration in large doses can result in hypoplasia. Administration of tetracycline-based medications to pregnant women leads to the

modification of color of temporary teeth in the incisive regions as tetracycline-based compounds can penetrate through the placenta barrier. Administration of tetracycline during the first months of life leads to the modification of pre-gingival region color of incisors and masticatory surfaces of temporary molars; at the age of 6 months it results in the coloring of temporary molars and 6-years molars, administration at the age of 2-3 years will result in the coloring of permanent frontal tooth collars, while administration of tetracycline at the age above 4 years leads to the coloring of permanent premolars and second molars (see color insert fig. 4.11.).

Tetracycline gets settles in bones and dentine of developing teeth, negatively affecting the mineralization processes. Intensity of coloring varies from open yellow to dark yellow. Under the action of light the initial color of vestibular surface goes gray or brown-gray. After the color modification the tetracycline teeth loose their fluorescent properties.

Modifications of temporary teeth color (from yellow to black) can be observed at children that suffered from hemolytic disease of newborns.

Treatment Tetracycline teeth are hardly treatable with whiteners. It is recommended to whiten such teeth beginning from radicular channel (after depulpation) with hydrogen peroxide, hydroperitis or perhydrol.

Prophylaxis Exclude the tetracycline-based medications at pregnant women and children, and administration of other antibiotics.

Local hypoplasia appears due to lesions of follicles under the action of a chronic inflammatory process in the temporary teeth. or due to tumors of maxillaries. On temporary teeth can be found in cases when temporary follicles are damaged – beginning from the fracture line of maxillary. Permanent teeth buds can be affected in cases of intrusion of temporary teeth (most frequently

– incisors). Affections of teeth buds are also possible in cases of chronic inflammatory processes, such as apical periodontitis of temporary teeth, osteomyelitis of maxillaries - most often found in the region of permanent premolars, as the buds are located between the roots of temporary molars frequently affected by caries and its complications (see color insert fig.4.12.).

Traumas and infections disturb the normal activity of ameloblasts while in severe cases the activity of odontoblasts is disturbed as well, resulting in modifications of shape and structure. Such pathologies have been named as *Turner teeth* (see color insert fig. 4.13). Slight affections are manifested by the appearance of maculae (chalky, yellow, brown, etc.).

Teeth affected by local hypoplasia require radiographic examination as the growth zone can be affected and the root can remain under-developed.

Treatment of such affections depends on their severity, localization, etc. In cases of maculated- chalky hypoplasia remineralization treatment is prescribed. If the maculae are pigmented - whitening with successive remineralization is performed. In cases of dental tissues defects – filling with composites. When the shape, dimensions or the color of teeth are affected - porcelain, metallic-ceramic, acrylic crowns are installed.

Prophylaxis consists in the timely treatment of dental caries and its complications on temporary teeth and avoidance of dental traumas.

Enamel hyperplasia is manifested by the excessive formation of dental tissues that are also named pearls (nodules, projections) of enamels. Their provenience is determined by disturbances of the process of cell differentiation in the Hertwig sheath in the ameloblasts.

Clinically they are not manifested and not detectable in examination. Pearls of enamel are most often localized in the

region of dental collar, sometimes at the bifurcation (trifurcation) of roots. They are caused by excessive production of dentine that on the outer side is covered with a thin layer of enamel. Sometimes in the center of pearl some cavity can be found filled with a tissue similar to pulp.

By localization there exist three types of pears (Cawanha A.O., 1965): radicular, collar-region and coronary ones. From the morphological point of view, there can be distinguished 5 groups of hyperplasia: veritable enamel pearls, enamel-dentine pearls; enamel-dentine pearls with pulp (frequently connected to the dental cavity); Rodrighes-Ponti pearls - nodules of enamel of small dimensions in periodontium, interdental pearls localized in the coronary or radicular dentine.

Enamel pearls in the collar region are detected when retracting the gums, while the radicular ones - by radiography or after tooth extraction. Interdental pearls (interdentinary) are most frequently found during the preparation of cavities within the dentine limits and characterized by greater hardness in a certain sector.

Treatment is prescribed only for pearls in the collar region. They must be ground off with diamond burs with subsequent remineralization and perfect hygiene in this region.

Dental fluorosis is an endemic affection caused by intoxication with fluoride (F), mostly conditioned by the consumption of water with high fluoride content. According to the opinions of some authors, fluorosis is a development vice of solid dental tissues, or acquired dental hypoplasia with specific etiology – caused by surplus of F in potable water.

According to А.П. Авцын (1991), fluorosis is a microelementosis provoked by excessive intake of F into the organism.

These pathological manifestations of solid dental tissues began to attract the attention of researchers already in the

beginning of the XXth century: J.M. Eager (1901) described pathologic changes of solid dental tissues at Italian emigrants in USA, manifested by appearance of maculae of yellow or dark brown color, sometimes associated with destruction of dental tissues. According to the data of J.N. Eager such phenomena were described by S Chiae in the year 1900 at people of Pozzuoli from the surroundings of Naples. Consequently such teeth were called "Chiae teeth" (or "Denta Chiae") in 1916 the clinical image of teeth affected by fluorosis was described in detail by G. Black and F. McKay. Simultaneously they have mentioned the endemic character of the affection and made a supposition that the disease is being caused by some unknown factor. Clinical manifestations of dental fluorosis were observed in Colorado (USA), enamel maculae on teeth there were named "brown stains of Colorado". The investigations performed by F.S. McKay, G.V. Black and H.T. Dean during the years 1934 and 1938 identified a connection between the disease and concentration of fluoride in potable water.

In 1913 A. Gautier reported increased content of F in water and in volcanic gases in the region of Naples; however, nobody did not take into consideration that it may result in pathological affections of teeth. Only in 1931 H.V. Churchill in Pennsylvania, M.C. Smith and others in Arizona and H.Velu in Morocco, almost simultaneously established a correlation between pathological dental modifications and consumption of water with excessive content of fluoride.

M.C. Smith et al. In 1931 experimentally demonstrated the dependence of pathological modifications of teeth in development and increased concentration of F in potable water. Taking into consideration the Latin name of fluoride (Fluor), such pathological changes were named endemic fluorosis. Later it was discovered that fluoride can affect not only the teeth, but also the skeleton bones and some internal organs.

It was demonstrated that teeth can be affected by fluorosis only during the development period. The enamel of already developed (mineralized) teeth can not be affected by fluorosis (Dean H.T., Elvove E., 1962, Грошиков М.И., 1985).

Pursuant to State standard 2874-82 (potable water) the quantity of F must not exceed the following limits: for climatic regions I-II: 1.5 mg/l, III – 1.2 mg/l, IV: 0.7 mg/l.

The daily content of F received with food amounts to 0.5-1.1 mg. If the content of fluoride in potable water is 0.4 mg/l then the quantity of fluoride received with water is almost equal to the one received with food. If the content of fluoride in potable water is 1 mg/l, then the quantity of fluoride received with water is 2-2.5 times greater than the quantity received with food. According to the data of M. Triller et al. (1992) dental fluorosis appears early if during the period of mineralization of solid dental tissues the quantity of received F per 24 hours exceeds 1.5 mg.

In the specialty literature are described more than a thousand of centers of endemic fluorosis in various localities of USA, Canada, Chili, Mexico, Australia, India, Sri-Lanka, China, Saudi Arabia, South Africa, Kenya, Nigeria, Tanzania, Sudan, Morocco, Italy, Russia, Ukraine, Azerbaijan, Turkmenistan, etc.

In the Republic of Moldova there also are many localities where the concentration of fluoride in potable water exceeds the normal limits: such localities exist in the Ungheni, Nisporeni, Straseni, Floresti, Falesti, Camenca, Ciadar-Lunga, Cimislia and other districts.

Besides consumption of potable water with increased fluoride content, there are some additional risk factors. For example, a particular risk can be represented by foodstuffs rich in fluoride regularly consumed by the children. Consider an example: green tea and drinks with caffeine may contain up to 3.1 mg/l of fluoride. Analysis of content of more than 532 juices and drinks showed that their fluoride content may vary from 0.02 to 2.8 mg/l.

It was also proved that such foodstuffs can provoke the appearance of dental fluorosis at the children that regularly consume such products (Kiritsy M. C., Levy S. M., Warren J. J., Guha-Chowdhury N., Heilman J. R., Marshall T., 1996).

In some localities of China, where the quantity of fluoride in potable water varies between 0.11 – 0.32 mg/l were found several forms of dental fluorosis conditioned by the national tradition to consume large quantities of green tea (with content of fluoride up to 3.69 mg/l) from the very childhood (Cao J., Zhao Y., Liu J., 1997).

The social-economic situation, insufficient nutrition, reduced content of proteins and calcium in food, especially the lack or insufficient consumption of milk are friendly factors of dental fluorosis at children.

The general state of child's organism is of particular importance in the appearance of dental fluorosis as one of the forms of dental hypoplasia. This can explain why in the localities with increased concentrations of F in potable water some of the children are not affected at all or display very slight elements of affection by dental fluorosis compared to the children suffering from chronic diseases or with weaker resistance of organism. Studying the activity of lysosomes of mixed saliva and the bacterial properties of skin, И. С. Рединов (1984) affirms that the appearance of dental fluorosis depends not only on the concentration of fluoride in potable water, but also on the non-specific resistance of organism.

Out of 60 children from village Pirlitsa, Ungheni district with signs of dental fluorosis, 22 were found to be suffering from diseases of internal organs: hepatitis – 13 cases, gastritis – 4 cases, pyelonephritis – 3 cases, affections of cardiac system – 2 cases. Also the appearance of dental fluorosis was favored by the daily consumption of 2-3 glasses of tea and by the deficit of milk products (Гнатюк П.Я., 1988).

A risk factor for dental fluorosis can also be the cleaning of teeth with toothpaste with high content of fluoride. For this reasons in USA to children of pre-school and school age it is recommended to use toothpaste with maximum content of fluoride of 400-500 ppm (Horowitz H.S., 1992). However, other publications contest the fact that various kinds of toothpaste containing F can represent a danger of dental fluorosis for children (Levine R.S., Beal J. F., Fleming C.M., 1989).

In localities with insufficient content of F fluoridation of potable water can also provoke light forms of dental fluorosis at children.

Professional fluoridation, such as mouth rinsing with fluorinated solutions and topic use of preparations with F can lead to incipient forms of dental fluorosis at children.

Administration of sodium fluoride as daily food supplement during the first year of life (correctly or incorrectly prescribed) caused the appearance of light forms of dental fluorosis at 12% children. For this reason M.A. Awad et al. (1991) doesn't recommend endogenous administration of sodium fluoride as daily food supplement due to difficulties of administration control and risk of dental fluorosis. Consumption of food supplements containing F without doctor's prescription can result in the appearance of dental fluorosis at children (Riordan P.J., 1993, Den D., 1999).

Statistical analysis of risk factors for the appearance of dental fluorosis at children demonstrates that the most significant factors are: consumption of water with fluoride content exceeding 1.5 mg/l by children aged under 12, birth of children in the months December -January and August, pathology of pregnancy, artificial feeding during the first year of life: use of mother milk substitutes during the first 6 months of life, somatic diseases during the first year of life.

Pathogenesis of endemic fluorosis is not fully studied

According to И.Г. Лукомский (1940), fluorosis appears as the result of action of F against calcium, magnesium, manganese and other elements of solid dental tissues, provoking disturbances of biological activity of these elements and respectively - destruction of enamel on formation stage.

According to И. О. Новик (1951), fluorosis is caused by the fact that the chemical activity of fluoride is greater than that of iodine and due to this reason the activity of thyroid is reduced, respectively the solid dental tissues don not develop correctly.

Some researchers observed reductions in the levels of alkaline phosphatase, cholinesterase, and other substances under the action of fluoride and made an assumption that it leads to disturbances in the process of mineralization of solid dental tissues. However, most substantiated is the theory of toxic action of fluoride on ameloblasts during the enamel organ development period leading to structural disorders (Pedersen P.O., Scott D.B., 1959; Патрикеев В.К., 1968, Matsuo A., 1998).

Pursuant to the hypothesis of А. К. Николишин (1996), during the tooth development period (amelogenesis) the excess of fluoride via the vases of follicular sack, through the intracellular spaces penetrates into the ameloblasts and makes close connections with the protein fixing the calcium of enamel in the process of formation. As result, in the process of solid tissue differentiation appears the substance named hydroxifluoroapatite. The complete substitution of hydroxyl group with fluoride can not take place as fluoride penetrates via the epithelial cells (ameloblasts) of the internal and external strata of the enamel organ in limited quantities necessary for the vital processes and maintenance of cellular metabolism. In the process of mineralization of epithelial cells the barrier function of ameloblasts (capacity to withhold excess quantities of fluoride from passing through the biological membranes) gradually decreases. Due to this reason, after the calcification of

ameloblasts and reduction of fluoride content in blood, evolution of fluorosis stops. This type of evolution is characteristic for the incipient forms of dental fluorosis. However, in cases of sufficiently high concentrations of fluoride in the organism's internal environment, under prolonged action fluoride settles on the surface of solid tissues (teeth, bones) in the form of calcium compounds insoluble in water. This process evolves in the form of epitaxy on the surface of fluoroapatite calcium fluoride is deposited. This process can last during the entire intramaxillary development period. Besides these, during the intramaxillary development of permanent dental follicles takes place the radicular resorption and resorption of alveolar bone. One may suppose that fluoride released from the alveolar bones again forms compounds with calcium and is settled on the surface of follicle enamel in the form of calcium fluoride. The most intensive calcium fluoride formation period occurs at the age of 2-4 years. It is not excluded that formation of CaF_2 on the surface of enamel can continue after dental eruption with the condition that the concentration of fluoride in potable water and buccal liquid in the nidus of dental fluorosis is sufficiently high. The connection between CaF_2, the superficial stratum of enamel and strata of fluoroapatite in cases of severe fluorosis is not strong. By structure calcium fluoride is softer and more fragile. This is why under mechanical impacts the surface of enamel can display caverns and other defects. Clinically it was proved that destruction of solid dental tissues in severe forms of dental fluorosis are especially intensively manifested during the period of enamel mineralization, immediately after the eruption of permanent teeth. The degree of enamel destruction is determined by the thickness of CaF_2 on the stratum of enamel, nutrition pattern and mineralization properties of saliva.

Pursuant to the opinions of some national and foreign scientists, appearance of light forms of dental fluorosis is possible

at 10-20 % of population when the concentration of fluoride in potable water is between 0.8 –1.0 mg/l, at 1.0-1.5 become affected 20-30% of population, at 1.5-2.5 mg/l - 30-45 %, at 2.5 mg/l and more - up to 50 % of people consuming such water become affected.

A study performed by the specialists of the chair of pediatric dentistry of State Medical and Pharmaceutical University "Nicolae Testemitanu" demonstrated that there exists a correlation between the increase of F content in potable water and increase of incidence and degree of affection by fluorosis. At children less than 6 years the prevalence of dental fluorosis is 81.77 %, at children aged under 12 – 82.57 % and ones aged about 15 - 89.87 %. The average degree of affection by fluorosis is 2.01+/- 0.33, for the children aged about 6 years it is 1.73+/- 0.33, for ones aged about 12 – 2.22+/-0.47, while for the ones aged about 15 years – 2.24 +/- 0.77.

The study of prevalence and degree of affection by fluorosis (fig.4.14.) for each tooth denoted a symmetrical character of affection: most affected are the superior and inferior teeth, as well as the teeth on the left and right semi-arcades. Most often are affected the first permanent molars, and further in descending order: second molars, central incisors, lateral incisors, premolars, canines are rarely affected.

Fluorosis first of all attacks the superior incisors and the first permanent molars, further it affects the premolars. Less often are affected the inferior incisors and the canines. This phenomenon is determined by the fact that the incisors and first permanent molars mineralize, in same time, with later years the specific consumption of water per one kilogram of body weight decreases.

Most affected are the central superior incisors, followed by first molars and further in descending order: second molars, superior lateral incisors, central inferior incisors, superior and inferior premolars, lateral inferior incisors. Less affected are the

canines (fig.4.15.).

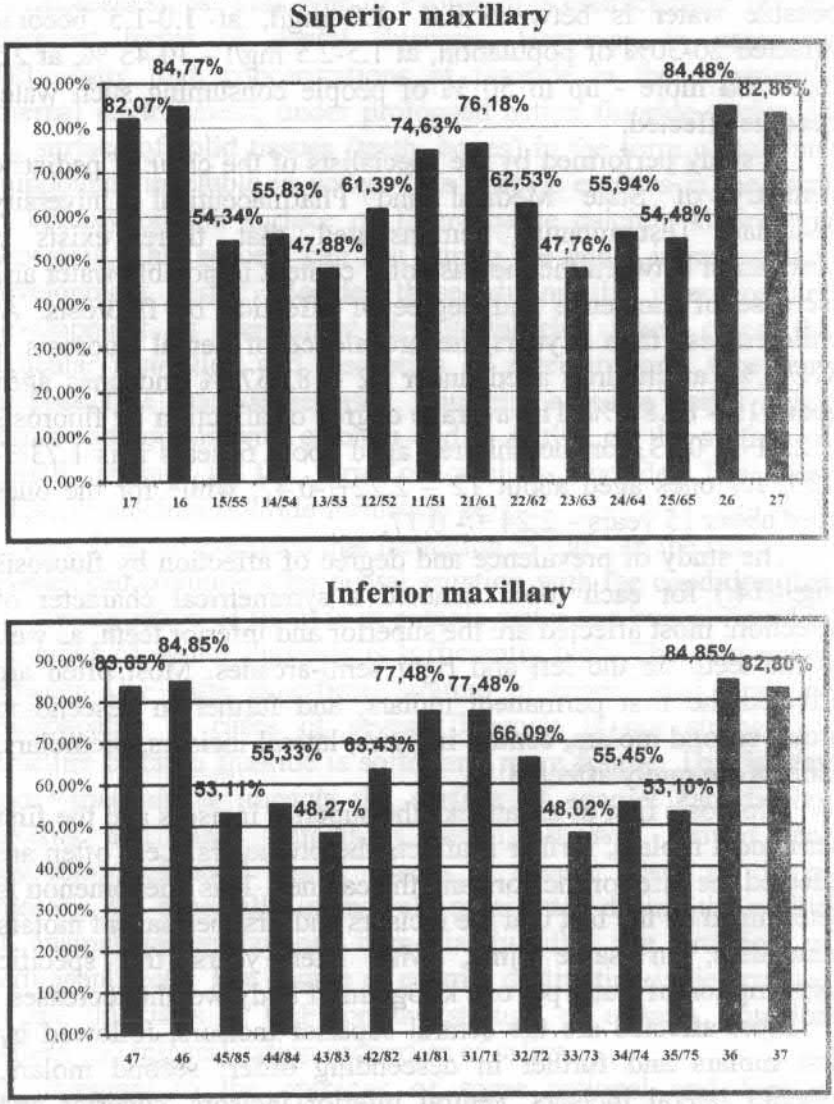


Fig.4.14. **Prevalence of affection by dental fluorosis at children**

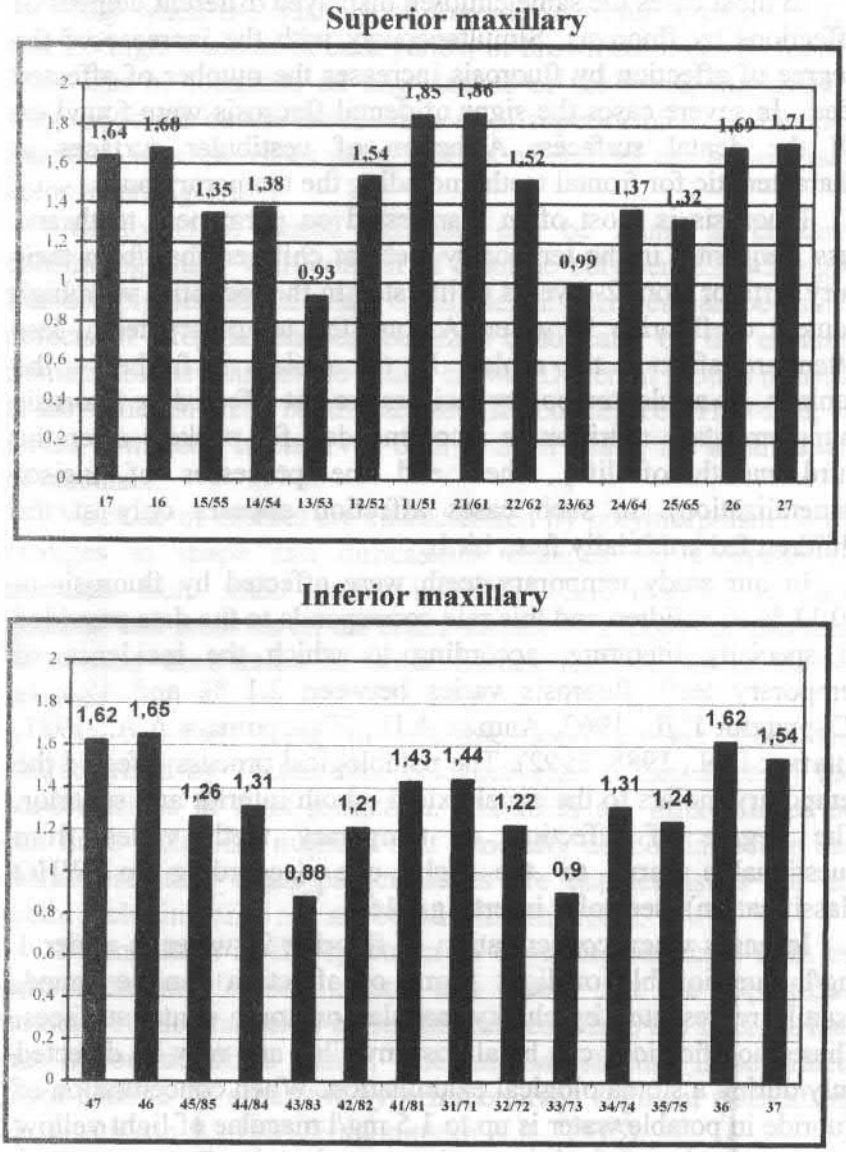


Fig.4.15. **Degree of affection by dental fluorosis at children**

In most cases the same children displayed different degrees of affections by fluorosis. Simultaneously with the increase of the degree of affection by fluorosis increases the number of affected teeth. In severe cases the signs of dental fluorosis were found on all the dental surfaces. Affection of vestibular surfaces is characteristic for frontal teeth, including the temporary ones.

Fluorosis is most often manifested on permanent teeth and less frequently in the temporary teeth at children that from their very birth or from 2-4 years of life stay in the localities with high content of fluoride in water. Among the temporary teeth, less often are affected the molars II, the molars I, further – the canines. As a rule, temporary incisors are not affected by fluorosis (supplementary nutrition is recommended for suckers after the third month of life, when end the processes of incisor mineralization). In such cases affection appears only at the children fed artificially from birth.

In our study temporary teeth were affected by fluorosis at 20.11 % of children and this rate corresponds to the data provided in specialty literature, according to which the incidence of temporary teeth fluorosis varies between 2.1 % and 49.5 % (Овруцкий Г.Д., 1962, Авцын А.П., Жаворонков А.А., 1981, Гнатюк П.Я., 1988, 1992). The pathological process affected the temporary molars to the equal extent – both inferior and superior. The degree of affection of temporary teeth varies from questionable form to the light one (according to WHO classification) (see color insert fig.4.16.).

In cases when concentration of fluoride in water is under 1 mg/l, questionable or light forms of affection can be found, usually represented by chalky maculae on some dental surfaces. These modifications can be almost invisible and may be detected only during a stomatological examination. When concentration of fluoride in potable water is up to 1.5 mg/l maculae of light yellow color can be detected, giving to the enamel a "tiger" appearance.

Only when the quantity of fluoride in water is between 1.5 and 2.5 mg/l maculae of dark yellow or brown color can be found. They can be localized on any surface of the tooth, but usually they are found on the labial surfaces of superior incisors. Besides maculae, in some cases point defects of enamel can appear (see color insert fig.4.17.).

All forms of dental fluorosis can be found at children consuming water with content of fluoride between 2.5-3.5 mg/l. Besides pigmented maculae, on all dental surfaces can be found defects of irregular shapes localized chaotically on the enamel. Sometimes they distort the dental crown. Different groups of teeth at the same child can be affected to different degrees. However, as a rule, symmetry is observed both in form and in the localization of maculae.

Maculae of enamel are characterized by polymorphism. With changes in shape and dimensions changes the character of maculae: from small almost invisible white maculae to dark maculae and erosions on the entire surface of a tooth that can lead to evident destruction of solid tissues and shapes of dental crowns.

At children living in endemic localities the teeth are characterized by whitish tone of dental enamel explained by the particularities of light refraction. The teeth are milk-colored or can have yellowish nuances that generally do not influence the dental esthetics. Such particularities are characteristics for all teeth, including ones not affected by the fluorosis.

Examination in the spectrum of daylight, polarized light or microradiographic examination can reveal that the superficial stratum of the enamel is hypermineralized while the internal one has hypomineralized bands. The internal stratum is separated from dentine by a narrow hypermineralized segment (Gustafson G., Gustafson A., 1961, Николишин А.К., 1975).

Examination under electronic microscope allow to state that

fluorosis maculae are insufficiently mineralized and that the inter-prism substance is being worn rapidly (Takuma Sh., 1955; Pedersen P.O., Scott D.B., 1959, Thylstrup A., Boyar R.M., Homen L., Bowden G. H., 1990). The electron-microscopic investigations performed by Патрикеев В. К. (1968) substantiated the fact that for severe forms of fluorosis are characteristic the loss of structural clarity of enamel due to destruction of apatite, weak adherence of crystals, while in some sectors complete destruction of enamel was stated. With the help of electronic microscope Sundstrom B.K.G. (1972) discovered focal or diffuse demineralization of enamel. In cases of pronounced modifications caused by fluorosis was observed a selective affection of enamel prisms but not affection of inter-prism substance.

The teeth affected by fluorosis at the incipient stage displayed modifications in the superficial strata of enamel: tabby texture of enamel; widening of inter-prism spaces due to prismatic resorption, zones of hypo- and hypermineralization; expressive Gunther –Schreger bands and Retzius striates; loss of clarity in the dentine-enamel junction; increase of enamel permeability and resulting pigmentation. The dentine can also display modifications specific to severe forms of fluorosis: the structure of dentine matrix is more compact, around the dentinary channels is well noticeable the hyper-calcification zone, micro-hardness of dentine increases by an average of 16%.

The mature fluoridated enamel in electronic-microscopic examination is characterized by demineralization of sub-superficial strata, simultaneous increase of permeability and relative growth of degree of affection by fluorosis. Changes in the physical properties of enamel affected by fluorosis can be quantitatively compared with the light reflection coefficient (Ciambro N., Prostak K., Denbesten P., 1995).

The superficial strata of the dental enamel may be affected

by advanced fluorosis due to greater quantities of organic substances and micro-heterogeneous character revealed by spectrometry (Николишин А.К., Кисловский Л.Д., 1991).

One of the first and most detailed classifications of dental fluorosis is the classification proposed by H. Dean, USA 1937) that comprises 7 degrees. Further, numerous researchers elaborated their own classifications of dental fluorosis: Р. Д. Габовоч (1949) – with 4 degrees of fluorosis, В.К. Патрикеев (1956) – with 5 degrees and most widely used in the former USSR; И. О. Новик and Г.Д. Овруцкий (1961) – with 3 types of fluorosis, I. Muller (1965) - with 5 degrees; П.Т. Максименко и А.К. Николишин (1976) with 2 degrees; Thylstrup and Fejerskow (1978); Horowitz (1984); Pendrys (1990) with 5 degrees.

Classification of fluorosis by В.К. Патрикеев (1956):

1) ***Hatched form*** - small chalky hatches, weakly pronounced, located in the superficial strata of enamel, sometimes detected only after the drying of tooth surface, most frequently localized on the vestibular surfaces of superior incisors.

2) ***Maculated form*** - chalky and well-pronounced maculae, localized on all the surfaces of the tooth. They can melt together, do not have well-pronounced borders, their surface is smooth and shiny. Sometimes maculae may have light –brown nuances; most often are affected the incisors, canines. Less often are affected premolars and molars.

3) ***Granular - chalky form*** – all teeth can be affected. Clinical image is variable. All dental surfaces are de-pigmented, chalky. However, the gloss can remain. In some cases light-brown or dark-brown pigmented sectors can be displayed, often with small defects, point and granular defects (with diameter up to 1.5 mm and depth up to 0.3 mm);

4) ***Erosive form*** - represents defects of different forms and dimensions, pigmentation of enamel (brown, dark brown, black).

Abrasion of enamel to the very dentine;

5) ***Destructive form*** - appears in localities where the concentration of fluoride in potable water exceeds 5 mg/l. Modifications of shape, color and dimensions of teeth in cases of enamel destruction, pigmentation of dental tissues in dark colors, pronounced abrasion. Enamel may be completely or partially absent; there can be various fractures of dental crowns.

WHO (1965) recommends the classification of fluorosis by I. Muller

1. ***Questionable form*** - the enamel surface displays hatches or weakly pronounced chalky maculae;

2. ***Very mild form*** – white maculae, chalky, occupying up to 25% of enamel surface;

3. ***Weak form*** – white non-transparent maculae of big dimensions, occupying up to 50 % of enamel surface;

4. ***Moderated form*** – affection of all surfaces of teeth, brown maculae, dental abrasion opening the enamel surface;

5. ***Severe form*** – all the surfaces of teeth are affected, large sectors of brown pigmentation, destruction of enamel.

Differentiated diagnosing of maculated fluorosis

Characteristic	Fluorosis	Caries	Hypoplasia
Appearance	Before eruption	After eruption	Before eruption
Affection of teeth	Most often, permanent teeth	Temporary and permanent teeth	Most often, permanent teeth
Localization	All surfaces	Vestibular and contact surfaces	All surfaces
Number of maculae	Numerous	Very few	Often very few
Permeability	Not increased	Pronounced	Not increased
Evolution of maculae	For life	Disappear very rarely	Never disappear
Concentration of fluoride in potable water	Increased	Low or normal	Low or normal

Differentiated diagnosing of erosive fluorosis

Characteristic	Fluorosis	Superficial caries	Hypoplasia	Cuneiform defect	Erosion
Accuses	Aesthetic defect	Enamel defect	Aesthetic defect	No	Aesthetic defect
Localization	All surfaces	In cracks, on the contact surfaces	All surfaces	Vestibular surfaces	Vestibular surfaces
Clinical signs	Enamel defect	Enamel defect	Projections into enamel	Cuneiform defect	Erosion-form defect
Permeability for colorants	Not increased	Pronounced	Not increased	Weakly pronounced	Weakly pronounced

Treatment of dental fluorosis consists in the reduction of excessive doses of fluoride received with potable water and application of various methods for the removal of pigmentation and dental defects, increase of organism's resistance, indication of a more rational diet and medicaments (first of all, with calcium, phosphorus and vitamins) with effects on mineral metabolism.

S.K. Gupta et al. (1996) consider that some forms of dental and bone fluorosis can be reversible if ascorbic acid, calcium and vitamin D_3 are administered.

Incipient forms (degrees I, II) do not require special treatment, if the patient's aesthetic requirements are met.

In cases of pigmented maculae on the frontal teeth de-pigmentation methods or "whitening" techniques are used. The specialty literature describes various methods of whitening: for vital and de-vital teeth, internal, external and combined whitening performed at domicile or in the dentist's cabinet (Murrin J.R., Barkmeier W.W. 1982; Hanosh F.N., Hanosh G.S. 1992; Cohen S., Burns R., 1998).

Vital techniques involve only vestibular applications (in their vast majority) or application of medicaments on all dental surfaces, while the de-vital techniques are implemented after endodontic treatment through the pulp chamber.

Г. Д. Овруцкий (1962) proposed acetic acid and lactic acid as preparations for the whitening of teeth affected by dental fluorosis. Besides Г. Д. Овруцкий, J.R. Murrin and W.W. Barkmeir (1982) recommend, before de-pigmentation of teeth by И.О. Новик (solution of 5 portions of H_2O_2 33 % and 1 portion of ester), to polish the superficial strata of enamel with carborundum stone or paper discs. After de-pigmentation Г. Д. Овруцкий used a paste with fluoride of 75 % applied on the surface of tooth afterwards polished with wood hone. Mechanical and chemical treatments of enamel were performed with intervals of 5-7 days and were aimed at the re-mineralization of dental surface.

В.К. Патрикеев (1958) and P.G. Colon (1980), in order to remove the dark brown pigmentation on the frontal teeth recommended to use saturated solutions of organic acids (citric and tartaric acids) neutralized after application with a paste of sodium bicarbonate. They also recommended polishing the treated teeth with pumice and wood.

In order to whiten the teeth affected by dental fluorosis A. K. Николишин (1977) applied a solution consisting of HCl 36 % and H_2O_2 33 % in proportion 1:2. Besides these, he invented a device consisting of several spoons adapted for maxillaries and containing impress treatment material and which were filled with whitening solution via two needles. For severe forms of dental fluorosis complex treatment was recommended: administration of calcium into bones and electrophoresis in order to stimulate the mineralization of solid dental tissues with subsequent whitening.

In the Republic of Moldova is presently widely used the whitening method proposed by the specialists of the department of therapeutic dentistry of the Medical University of Chisinau. This method consists in the application of a labial extender, isolation of teeth from saliva, application of vaseline on the marginal gums (or other neutral unguent - in order to prevent chemical combustion); on the vestibular surface of the affected

teeth for 5 minutes is applied a plug of cotton wool impregnated with 18% solution of HCl. After this the buccal cavity is rinsed with 2 % sodium bicarbonate solution. Further, plugs of cotton wool impregnated with 33 % solution of H_2O_2 for 5-10 minutes. This procedure is again followed by mouth rinsing with sodium bicarbonate solution. The whitened surfaces are treated with Calmecin paste for 20 minutes or 10 % solution of calcium gluconate. Are contraindicated the products containing pigments (coffee, grapes, etc.). It is advisable to the patients to clean their teeth with sorts of toothpaste not containing fluoride, such as "Jemciug", "Arbat', etc. (Гнатюк П.Я, 1984).

For the treatment of incipient forms of dental fluorosis is indicated the re-mineralization therapy with calcium preparations (calcium gluconate, calcium glicerophosphate) in the form of applications or by electrophoresis (Лебедева Г.К., Галченко В. М., 1981; Гнатюк П.Я., Бурлаку В.З., Елашко М. Л., 1984; Гнатюк П.Я., Сырбу С.В., Бурлаку В.З., 1989).

The tooth whitening method of R.J. McCloskey (1984): the teeth affected by maculated form of fluorosis and the teeth resistant to prophylactic treatment are treated with 18 % solution of HCl with mechanical friction. The procedure begins from premolars and gradually passes to the incisors.

The specialty literature contains information on methods of whitening by temperature and light that involve the release of oxygen from hydrogen peroxide. Teeth are treated with 30-35% solution of hydrogen peroxide and then exposed to the temperature of 40-57^{0}C for a maximum of 20 minutes. At least three sessions with periodic verifications are required (Murrin J.R., Barkmeier W.W., 1982).

Methods of whitening with gels are efficient; some of the gels can be applied by the patient at home. There is a wide spectrum of teeth whitening gels on the market (products of companies "American Dental Hygienics", "Bisco Dental Products", "Schofu

Dental Corporation", "Colgate Oral Pharmaceuticals", etc.) that represent the following forms: SiO_2 powder with hydrogen peroxide (applied on tooth surface for 10-20 minutes), carbamide peroxide 10-16% at pH =6.8 applied on individual spoon (for 30 minutes). Treatment can be repeated 3-4 times a day or nighttime during sleep. In these systems the efficiency of whitening depends on: type and intensity of pigmentation; contact time and quantity of active ingredient available in the whitening material.

The superficial forms of dental fluorosis can be treated by microabrasion - method proposed by Th. Croll (1991) that consists in the removal of pigmented strata of enamel with a mix of chlorohydric acid and abrasive material (pumice stone) named PREMA and produced by Premier Dental Products Company. Microabrasion involves removal of strata of 25-70 mkm. Microabrasion of enamel removes the eminent elements of enamel and draws the mineralizing substance into the depressions of enamel. The cause of improvements in the enamel appearance after microabrasion is still not completely known. Th.Croll (1991) states, that the effect of this methodology is assured by the dynamic re-mineralization process. After the removal of affected stratum of enamel the surface of tooth is covered with a paste or gel containing fluoride. Further the treated surface is polished and the fluoride gel is again applied for 4 minutes.

The methods of treating dental fluorosis can be grouped into:

1) ***Non-traumatic (non-invasive) or minimum invasion methods:***

Tooth whitening - fluorosis of II-IV degrees;

Microabrasion of enamel - fluorosis of II-IV degrees;

Whitening + restoration with composite compounds - fluorosis of III-V degrees.

2) ***Traumatic (invasive) methods:***

Adhesive facets - fluorosis of III-V degrees;

Covering crowns – fluorosis of IV-V degrees.

For children and adolescents (especially during the root formation period) with dental fluorosis it is recommended to use non-traumatic or minimum invasion methods.

In cases of pigmentation of solid dental tissues vital whitening of vestibular surfaces of frontal teeth with gels or solutions is indicated (systems: Opalescence (Ultradent); Illumine TM; Rembrandt (Den-Mat Corp); Peroxyl (Colgate-Hoyt Laboratories); White&Brite (Omni International); Natural White (Aesthete Laboratories, etc.).

In order to preserve the effect of whitening fixing agents are applied (OptiGuard (Kerr)) or composite compounds and compomers of fluid consistency: Dyract Flow (Dentsply); Revolution, Point 4 (Kerr); Filtek Flow (3M); Aeliflo (Bisco); Tetric Flow (Vivadent); PermaFlo (Ultradent), Admira Seal (Voco), etc.

However, de-pigmentation of teeth often doesn't get crowned with sufficient and stable results, especially in cases of advanced dental fluorosis. For this reason, after 2-3 sessions of de-pigmentation, in order to correct the color and sometimes in order to correct the shape of teeth affected by fluorosis composite materials can be applied – with mechanical removal of some enamel sectors with excessive pigmentation and minimum preparation within the limits of superficial strata of enamel of the vestibular surfaces of frontal teeth crowns.

In cases of profound pigmentation of enamel and dentine the whitening methods are insufficient and appear the need to apply some techniques of restoring the dental surface with composite resins or laminated facets. Defects of enamel can be removed by application of composite materials, direct adhesive facets made of micro-hybrid compounds.

In cases of advanced dental fluorosis, especially in cases with associated abrasion of teeth with already formed roots different

prosthetic methods can be applied: indirect adhesive facets or crowns with ceramic, metallic-ceramic, acrylic or combined covers, etc.

Prophylaxis of dental fluorosis at children comprises exclusion or reduction of F ions assimilation first of all with potable water with high content of F. Prophylaxis of dental fluorosis can be organized by individual or social methods.

Potable water can be de-fluorinated in centralized water supply systems involving different methods: chemical methods with sedimentation and coagulation of fluoride compounds (electrical coagulation with bipolar aluminum electrodes, electromagnetic method, ion exchange systems and adsorption systems, methods based on separation membranes and bone filters). In order to optimize the concentration of F it is recommended to mix water from different sources with different concentrations of F.

Prevention of dental fluorosis is implemented by direct individual interviews with pregnant women and parents of children. In order to reduce or exclude the consumption of fluoride, especially with potable water, individual prevention of fluorosis at children is assured by:

a) Prohibition to use the water with high content of fluoride for the feeding of children, especially in organized children's institutions;

b) Substitution of water with high fluoride content with mineral bottled water with normal levels of fluoride, with juices, milk, etc;

c) Filtering of potable water for children in the endemic zones;

d) Sending children to rest camps located in zones with low content of fluoride in water;

e) Administration of fluoride preparations to children in the form of solutions, gels, tablets, dental pastes only under the

doctor's supervision;

f) Limitation of consumption of products with high content of fluoride: sea fish, fat meat, melted butter, some kinds of tea and artificial milk, food supplements (salt, juices, mineral waters, etc.) and consumption of foodstuffs rich in proteins, mineral salts (Ca, P), vitamins (A, B, D) and dairy products;

g) Administration of calcium products (calcium gluconate, calcium glycerophosphate), poly-vitamins - depending on age;

h) Administration of sodium nucleinate in order to increase the non-specific resistance of organism;

i) Observation of buccal cavity hygiene.

Amelogenesis imperfecta represents an enamel pathology or hereditary origin caused by the pathological disorders of ectoderm with variability of clinical manifestations depending on the degree of genetic transmission and histological modifications.

The genetic or other general or local environmental factors can disturb the normal process of amelogenesis, leading to the appearance of amelogenesis imperfecta that in its vast variety is determined by genetic mutations (Luca R., 1998) (see color insert fig.4.18.).

Classification of amelogenesis imperfecta by Witkop (1989)
(Cited by Rodica Luca, 1996)

Type I - *Hypoplasic*

IA - Hypoplasic with roughness, dominant autosomal
IB - Hypoplasic localized, dominant autosomal
IC - Hypoplasic localized, autosomal recessive
ID - Hypoplasic, smooth, dominant autosomal
IE - Hypoplasic, smooth, X-linked dominant
IF - Hypoplasic, rough, dominant autosomal
IG - Agenesis of enamel, autosomal – recessive

Type II - *Hypo-matured*

IIA - Hypo-matured, pigmented, autosomal - recessive

IIB - Hypo-matured, X-linked recessive

IIC - Snow teeth dominant autosomal

Type III - *Hypo-calcificated*

IIIA - Dominant autosomal

IIIB - Autosomal –recessive

Type IV - *Hypo-maturated – hypoplasic with taurodontism* (fig.4.19.)

IVA - Hypo-maturated – hypoplasic with taurodontism, dominant autosomal

IVB - Hypoplasic – hypo-maturated with taurodontism, dominant autosomal

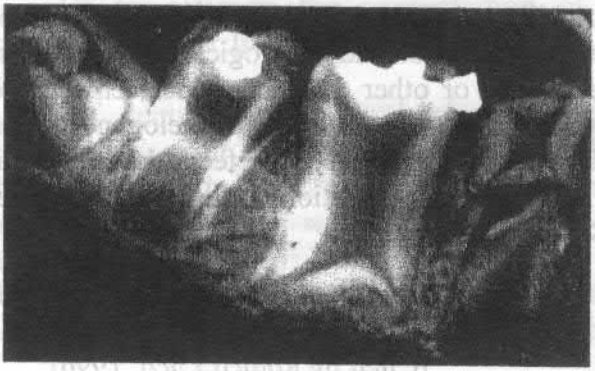


Fig. 4.19. **Amelogenesis imperfecta with taurodontism**

А.А. Колесов (1991) distinguishes four ***forms of clinical manifestations of amelogenesis imperfecta***:

Form 1. *Minor quantitative and qualitative disorders* Usually the teeth erupt in average terms; sometimes they can be of smaller volume, leading to the appearance of tremes. The enamel surface is smooth, glossy, however, of yellow or brown color.

Form 2. *Major quantitative and qualitative disorders* The

teeth erupt within the average normal terms; however their shape is conical or cylindrical. The enamel surface is rough, as the enamel is partially preserved (most often in the region of collar). Color can vary from yellow to dark brown. This form of affection is caused by structural changes and insufficient mineralization of enamel. Dentine doesn't display pathological changes.

Form 3. The teeth may have normal dimensions, shape and color, or the enamel can be thinner and with changed color. In both cases on all the surfaces of dental crowns vertically oriented grooves are seen (from incisive edge to the collar). Are affected not only groups of teeth with the same formation periods but all teeth (both temporary and permanent). Grooves are found on the entire surface of dental crown.

Form 4. Dental crowns have normal dimensions and shapes; however, the enamel has a chalky surface and is not glossy.

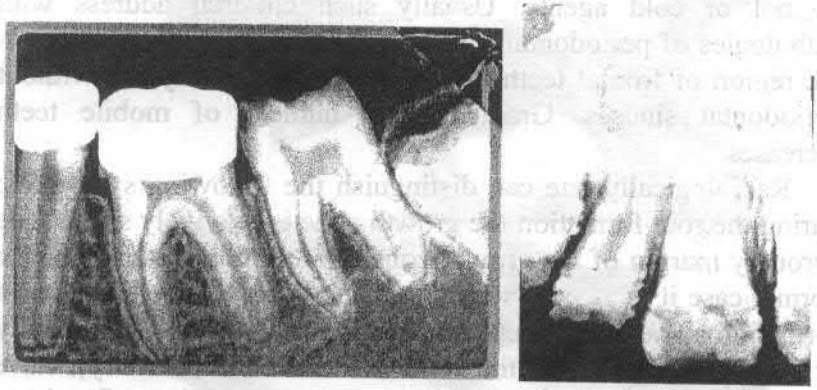


Fig.4.20. **Radiography: Amelogenesis imperfecta.**

Some authors presume that this is explained by the absence of cuticle and pellicle. The enamel is fragile and doesn't resist well to mechanical impacts. The dentine without enamel is yellow. Gradually it becomes brown (gets pigmented), often one can observe hypersensitivity to thermal irritation. Other manifestation

variants are also possible. Usually, after the eruption of teeth the process progresses, leading to changes in the color of dental crown, to fragility and abrasion of enamel, decrease of size, change of color of all temporary and permanent teeth.

In all forms of amelogenesis imperfecta the number and the shapes of roots, pulp chambers and radicular channels doesn't change. Most often are observed the forms I and IV, equally distributed among children of both sexes.

Dentinogenesis imperfecta is a hereditary disorder of dentine evolution (fig.4.21.). Clinically this pathology is not manifested till the moment of dental eruption: the teeth have normal color, dimensions and shapes. Caries is rare in such teeth and if it appears - then only on the permanent teeth of mature patients. However, children can have accusations on pain caused by hot or cold agents. Usually such children address with pathologies of periodontal tissues: catarrhal gingivitis (usually in the region of frontal teeth) associated with mobility but without periodontal sinuses. Gradually the number of mobile teeth increases.

Radiologically one can distinguish the following symptoms: during the root formation the growth zone is relatively small. The coronary margin of the growth zone represents a straight line (in normal case it is a curve with sharp edges). After the end of root formation some of the teeth practically do not have pulpal cavity or it is very small. All the roots are short (rudimentary), have sharp apexes (especially on inferior incisors). Molars often have only one plate and short root. Radicular channels are not distinguished, sometimes they are observed only in the apical region and are very narrow. The pulpal cavity can have denticles of different dimensions.

The apical region can display osteoporosis of bone tissue. The edges of inter-radicular septum are lower than the enamel- cement junctions.

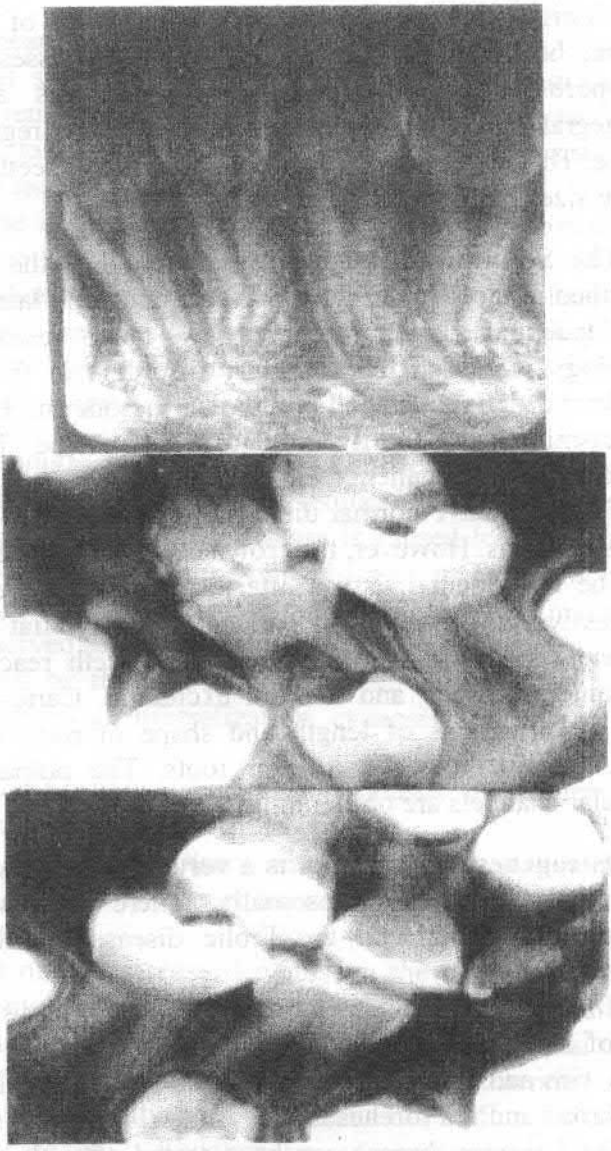


Fig.4.21. **Dentinogenesis imperfecta**

Mobility of teeth is caused by the reduction of inter-alveolar septum height and apical inflammatory processes. Focuses of osteoporosis appear due to pulpal necrosis as result of disintegration of neural-vascular tissues in the region of apical orifice. This is caused by functional over-use of teeth with normal crown sizes but with shorter roots.

The Stainton-Capdepont syndrome For the first time is described in the year 1892 by C.W. Stainton and later, in 1905 by C. Capdepont. Later, observed by other researchers. This pathology is caused by hereditary disorders in the function of ectodermal derivatives and embryonic mesoderm. This dominant is transmitted hereditarily only to a half of the children. Both temporary and permanent teeth can be affected.

The teeth have normal dimensions and shapes; they erupt in the usual terms. However, the crown color is changed.

The solid dental tissues after eruption begin to decompose progressively. The abrasion surface is glossy, flat and smooth. Dentine appears to be transparent. The teeth react weakly on mechanic, chemical and thermal excitation. Caries is observed rare. Modifications of length and shape of roots are possible: shorter roots, thinner or thicker roots. The pulpal cavity and radicular channels are obliterating.

Osteogenesis imperfecta is a very rare hereditary pathology transmitted dominant autosomally. There are two forms of osteogenesis imperfecta: the Frolic disease and the Lobstein disease.

The Frolic disease is more often found at boys on the first year of life and is characterized by fractures of long tubular bones, ribs and clavicles. Such children are of small height, and have broad and flat foreheads.

The Lobstein disease can be detected after the first year of life. The symptoms are alike. On examination the child displays

blue sclera, hearing impairment and modifications of teeth: being of usual shapes, their color varies from blue –gray to brown. Different groups of teeth may have different nuances of colors. Later takes place the abrasion of solid tissues and obliteration of radicular channels.

The disease is caused by the imperfect mineralization during the bone and teeth development period.

To these diseases are characteristic the structural changes of the bone tissues that radiologically are characterized by short diaphyses, thin cortical stratum, rare and thin bone trabecules, and porosity of spongy bone tissues.

Hypophosphatasia For the first time ever describe by Rathburu in the year 1948. It is a hereditary disease associated with metabolic disorders. The disease is caused by a congenital anomaly of synthesis of alkaline phosphatase: its reduction in the sanguine serum under normal content of calcium and phosphor. Usually is observed at birth and during the first six months of life. It is manifested by the deformation of extremities, fractures of bones, disturbances of mineralization, cyanosis, fragility of nails and hair.

Dental modifications: enamel hypoplasia, insufficiency of root formation with premature rhizalysis.

Radiological examination: insufficient development of alveolar processes, osteoporosis, premature rhizalysis of temporary teeth roots.

Treatment of this disease depends on the degrees of dental affections. Simultaneously with dental eruption or in cases of incipient forms of hereditary disturbances of solid dental tissue development re-mineralization treatment is recommended to be performed systematically with solutions of sodium fluoride, calcium gluconate, etc. Endogenous administration of fluoride and calcium preparations.

In cases of progressive destruction of solid tissues metallic,

acrylic, combined, porcelain, metallic-ceramic crowns are installed not only on the frontal teeth, but also on the posterior ones. It is very important that such structures do not increase occlusion, they must only stabilize it. It is also necessary to remove the over- contact points and to distribute uniformly the masticatory forces on teeth.

In cases of total advanced abrasion it is recommended to install mobile prosthetic devices.

Control questions and topics:

1. Classification of structural and developmental anomalies of solid dental tissues by Е. Виноградова.
2. Characterize dental anomalies of number at children.
3. Describe dental anomalies of shape.
4. Enumerate dental anomalies of volume.
5. Characterize dental anomalies of structure.
6. What are the clinical manifestations of systemic hypoplasia of solid dental tissues?
7. Local hypoplasia of solid dental tissues.
8. Tetracycline teeth at children.
9. Dental fluorosis at children.
10. Amelogenesis imperfecta.
11. Dentinogenesis imperfecta at children.
12. The Stainton-Capdepont syndrome at children.

CONTROL TESTS

Simple compartment

1. Pathology of temporary incisors can appear:
   A. Before the 17th week of pregnancy;
   B. After the 17th week of pregnancy;
   C. After the 24th week of pregnancy;
   D. In the first half of the first year of life of the child;
   E. In the second half of the first year of life of the child.

2. Pathology of temporary molars can appear:
   A. Before the 17th week of pregnancy;
   B. After the 17th week of pregnancy;
   C. After the 24th week of pregnancy;
   D. In the first half of the first year of life of the child;
   E. In the second half of the first year of life of the child.

3. Dystrophy of molars, incisors (in the middle third of the vestibular surface) and on the incisive surface of canines can appear:
   A. Before the 17th week of pregnancy;
   B. After the 17th week of pregnancy;
   C. After the 24th week of pregnancy;
   D. In the first half of the first year of life of the child;
   E. In the second half of the first year of life of the child.

4. The Hutchinson teeth have:
   A. The screwdriver or cask shape of dental crown and the incisive margin has a crescent shape;
   B. The shape screwdriver or cask shape of dental crown;
   C. First molars are cone-shaped, tubercles are insufficiently formed;
   D. Dental crown has yellow color;
   E. Spear-shaped.

5. The Fournier teeth are:
   A. The screwdriver or cask shape of dental crown and the incisive margin has a crescent shape;
   B. The shape screwdriver or cask shape of dental crown;
   C. First molars are cone-shaped, tubercles are insufficiently formed;
   D. Dental crown has yellow color;
   E. Spear-shaped.

6. The Pfluger teeth are:

A. The screwdriver or cask shape of dental crown and the incisive margin has a crescent shape;
B. The shape screwdriver or cask shape of dental crown;
C. First molars are cone-shaped, tubercles are insufficiently formed;
D. Dental crown has yellow color;
E. Spear-shaped.

7. The tetracycline teeth:
A. The screwdriver or cask shape of dental crown and the incisive margin has a crescent shape;
B. The shape screwdriver or cask shape of dental crown;
C. First molars are cone-shaped, tubercles are insufficiently formed;
D. Dental crown has yellow color;
E. Spear-shaped.

8. The dental tissues affected by hypoplasia under ultraviolet rays are:
A. Open green;
B. Gray-green;
C. Dark;
D. Light;
E. Light blue,

9. Systemic hypoplasia affects:
A. One or a group of neighboring teeth;
B. A single tooth;
C. Several symmetrical teeth on the dental arcade.

10. Focal hypoplasia affects:
A. One or a group of neighboring teeth;
B. A single tooth;
C. Several symmetrical teeth on the dental arcade.

11. Local hypoplasia affects:

A. One or a group of neighboring teeth;
B. A single tooth;
C. Several symmetrical teeth on the dental arcade.

12. The cause of local hypoplasia is:
A. A general disease of mother during pregnancy;
B. A general disease of the child during the first year of life;
C. A periapical inflammatory process of the temporary dental root, complicated luxation of temporary tooth;
D. Artificial feeding of the child;
E. A trauma, a tumor, an inflammatory process in the region of one or several roots of permanent teeth.

13. The cause of focal hypoplasia is:
A. A general disease of mother during pregnancy;
B. A general disease of the child during the first year of life;
C. A periapical inflammatory process of the temporary dental root, complicated luxation of temporary tooth;
D. Artificial feeding of the child;
E. A trauma, a tumor, an inflammatory process in the region of one or several roots of temporary teeth.

14. The dentinogenesis imperfecta is conditioned by:
A. Modification of ectodermal cells function;
B. Pathological modifications of mesoderm;
C. Toxic action of fluoride on ameloblasts;
D. Excretion of insoluble calcium and fluoride compounds from organism;
E. Action of fluoride on phosphatase;
F. Action of pathologic factors of metabolic processes in the organism of mother and child on ameloblasts;
G. Formation of organic acids as result of vital activity of microorganisms;
H. Prolonged action of a chronic inflammatory process in the

periapical tissues of temporary teeth.

15. The amelogenesis imperfecta is caused by:
    A. Modification of ectodermal cells function;
    B. Pathological modifications of mesoderm;
    C. Toxic action of fluoride on ameloblasts;
    D. Excretion of insoluble calcium and fluoride compounds from organism;
    E. Action of fluoride on phosphatase;
    F. Action of pathologic factors of metabolic processes in the organism of mother and child on ameloblasts;
    G. Formation of organic acids as result of vital activity of microorganisms;
    H. Prolonged action of a chronic inflammatory process in the periapical tissues of temporary teeth.

16. Fluorosis is provoked by:
    A. Somatic diseases of child;
    B. Diseases of mothers during pregnancy;
    C. Excessive consumption of carbohydrates;
    D. Insufficient oral hygiene;
    E. Excessive consumption of fluoride.

17. Fluorosis affects:
    A. Temporary molars;
    B. All temporary teeth;
    C. Permanent incisors and canines;
    D. All permanent teeth;
    E. All temporary and permanent teeth.

18. Specific prophylaxis of fluorosis consists of:
    A. Limiting of excessive consumption of fluoride;
    B. Administration of preparations that increase the non-specific resistance of organism;
    C. Exposure to ultraviolet radiation in the spring-summer

period for 15-20 cures, beginning with ¼ of biodose;
D. Treatment of chronic somatic diseases;
E. Administration of fluoride preparations.

19. The Stainton-Capdepont syndrome affects:
A. The enamel of temporary teeth;
B. The enamel of permanent teeth;
C. The dentine of temporary and permanent teeth;
D. The enamel and dentine of permanent teeth;
E. The enamel and dentine of temporary and permanent teeth;
F. The enamel of temporary and permanent teeth.

20. Amelogenesis imperfecta affects:
A. The enamel of temporary teeth;
B. The enamel of permanent teeth;
C. The dentine of temporary and permanent teeth;
D. The enamel and dentine of permanent teeth;
E. The enamel and dentine of temporary and permanent teeth;
F. The enamel of temporary and permanent teeth.

21. Dentinogenesis imperfecta affects:
A. The enamel of temporary teeth;
B. The enamel of permanent teeth;
C. The dentine of temporary and permanent teeth;
D. The enamel and dentine of permanent teeth;
E. The enamel and dentine of temporary and permanent teeth;
F. The enamel of temporary and permanent teeth.

22. The following clinical signs are characteristic for amelogenesis imperfecta:
A. White maculae, multiple, localized on the vestibular surface of dental crown;

B. The surface of dental crowns is rough; the enamel is present in the form of small islets;
C. The teeth have normal dimensions, the enamel is not affected, the dental roots are short, the dental channels and cavity are obliterated;
D. Pathological abrasion of teeth, enamel preserved in the form of small islets, the dental roots are short, and the channels are obliterated.

23. The following clinical signs are characteristic for dentinogenesis imperfecta:
A. White maculae, multiple, localized on the vestibular surface of dental crown;
B. The surface of dental crowns is rough; the enamel is present in the form of small islets;
C. The teeth have normal dimensions, the enamel is not affected, the dental roots are short, the dental channels and cavity are obliterated;
D. Pathological abrasion of teeth, enamel preserved in the form of small islets, the dental roots are short, and the channels are obliterated.

24. The following clinical signs are characteristic for the Stainton-Capdepont syndrome:
A. White maculae, multiple, localized on the vestibular surface of dental crown;
B. The surface of dental crowns is rough; the enamel is present in the form of small islets;
C. The teeth have normal dimensions, the enamel is not affected, the dental roots are short, the dental channels and cavity are obliterated;
D. Pathological abrasion of teeth, enamel preserved in the form of small islets, the dental roots are short, and the channels are obliterated.

25. The following investigations are required to diagnose dentinogenesis imperfecta:
A. Examination, anamnesis;
B. Auscultation, percussion;
C. Radiography;
D. Vital coloring;
E. EOD.

26. In cases of pregnancy pathology the development and mineralization of temporary teeth:
A. Is delayed;
B. Occurs faster;
C. Doesn't change.

Multiple compartment

27. Dental hypoplasia is caused by:
A. Modification of ectodermal cells function;
B. Pathological modifications of mesoderm;
C. Toxic action of fluoride on ameloblasts;
D. Excretion of insoluble calcium and fluoride compounds from organism;
E. Action of fluoride on phosphatase;
F. Action of pathologic factors of metabolic processes in the organism of mother and child on ameloblasts;
G. Formation of organic acids as result of vital activity of microorganisms.
H. Prolonged action of a chronic inflammatory process in the periapical tissues of temporary teeth.

28. Hypoplasia can simultaneously affect:
A. Premolars, molars II and III;
B. Central incisors, molars I;
C. Lateral incisors, canines;
D. All temporary teeth;

E. All molars.

29. Dental fluorosis is caused by the following phenomena:
    A. Modification of ectodermal cells function;
    B. Pathological modifications of mesoderm;
    C. Toxic action of fluoride on ameloblasts;
    D. Excretion of insoluble calcium and fluoride compounds from organism;
    E. Action of fluoride on phosphatase;
    F. Action of pathologic factors of metabolic processes in the organism of mother and child on ameloblasts;
    G. Formation of organic acids as result of vital activity of microorganisms;
    H. Prolonged action of a chronic inflammatory process in the periapical tissues of temporary teeth.

30. Non-specific (pathogenic) prophylaxis of fluorosis includes:
    A. Limiting of excessive consumption of fluoride;
    B. Administration of preparations that increase the non-specific resistance of organism;
    C. Exposure to ultraviolet radiation in the spring-summer period for 15-20 cures, beginning with ¼ of biodose;
    D. Treatment of chronic somatic diseases;
    E. Administration of fluoride preparations.

31. Exist the following congenital dental diseases:
    A. Fluorosis;
    B. The Stainton-Capdepont syndrome;
    C. Hypoplasia;
    D. The marble-bone disease;
    E. Dentinogenesis imperfecta.

Correct answers:

1. A.
2. B.
3. C.
4. A.
5. B.
6. C.
7. D.
8. B.
9. C.
10. A.
11. B.
12. C.
13. E.
14. B.
15. A.
16. E.
17. D.
18. A.
19. E.
20. F.
21. C.
22. B.
23. C.
24. D.
25. C.
26. A.
27. F, H.
28. B, C.
29. C, D, E.
30. B, C, D.
31. B, C, E, F.

5 DENTAL TRAUMATISM AT CHILDREN

Dental traumas at children can be isolated, and sometimes combined with other traumatic affections of face.

During the recent years this pathology is more and more often met at children both nationally and internationally. It was established that the incidence of frontal teeth traumas at children correlates with the level of social-economic development of the society. This factor is much connected with the popularity of such collective sport games as football, hockey, etc.

The incidence of dental traumas is studied to an insufficient degree and there are few scientists dealing with this problem.

M. Marcus (1951) found that traumas of frontal teeth are present at 16-20% of the total number of considered children. Most often were affected the superior incisors. The ratio of superior incisors to inferior incisors traumas is 3:1. Boys are twice more often affected by dental traumas.

According to the data presented by L. Zarnea, out of 17668 children of Bucharest aged between 7 and 14, dental fractures were found at 14,65 %.

Ellis and Craing (1988), have examined 4 251 pupils from Canada, found coronary fractures at 4,2% of children. Prevalence of dental traumas at boys compared to girls was 2,5: 1.

Craig (in Scotland) during the years 1966-1967, having examined 17831 children aged between 4 and 18, found fractures of permanent teeth at 5,9% of children. Among them 8,1% already underwent adequate treatment, 2,8% had satisfactory treatment, while 88,6 % did not receive any treatment at all.

O'Mollaine of Ireland (1972) established that out of 2792

children aged between 6 and 19, 17 % had traumas of permanent teeth. Out of them 0.4 % were children aged fewer than 6, while 20 % - above 12 years.

Andreasen of Denmark (1972) found traumas of temporary and permanent teeth at 46% of 487 children. From them 30% represented untreated lesions of permanent dentition.

Zadik of Jerusalem (1973), having examined 10903 children aged between 6 and 14, found that at 8,95% dental-alveolar traumas were present. The same author in 1980 reported a level of dental traumatism of 25 % of 252 children.

Mayoral, Herrero and Nuno Diaz, out of 787 patients receiving orthodontic treatment, found those at 13,21% of patients were present traumas of superior incisors. It was stated that 84,62% of children had prodentism and 58.65% had hypotonia of lip orbicular.

Raun determined that among the students of a gymnasium of Copenhagen dental traumas were found at 34.9% of boys and 23,1% of girls.

Classification of dental traumas by Ellis

Class 1. Simple coronary fracture, comprising the enamel and partially the dentine

Class 2. Long coronary fracture with opening of dentine, without involvement of pulp

Class 3. Extended coronary fracture with pulpal exposure

Class 4. Pulpal mortification with, or without coronary loss

Class 5. Complete luxation

Class 6. Radicular fracture with or without coronary loss

Class 7. Displacement of tooth without fracture

Class 8. Total coronary fracture

Class 9. Traumas of temporary teeth

Classification of permanent teeth traumas at children as recommended by WHO

Class I. Dental contusion with insignificant structural modifications (cracks in enamel)
Class II. Non-complicated fracture of dental corona
Class III. Complicated fracture of dental corona
Class IV. Total fracture of dental corona
Class V. Longitudinal coronary- radicular fracture
Class VI. Fracture of dental corona
Class VII. Dental luxation (incomplete or partial)
Class VIII. Total dental luxation

Taking into account the fact that dental traumas can occur during different tooth development periods, in some cases can be included 2-3 types of traumas:
Variant I – trauma of tooth with incompletely developed roots;
Variant II – trauma of tooth with incompletely developed apex;
Variant III- trauma of tooth with completely developed root.

Such additions allow us not only to establish a correct diagnosis of the pathology, but also to choose the optimum tactics for the treatment of dental traumas at children of different ages.

TRAUMAS OF PERMANENT TEETH AT CHILDREN

The etiology of permanent teeth traumas at children includes numerous factors. Most frequently met factors are the sport accidents, consequences of road accidents, consequences of plays, accidental falls, etc.

Many authors indicate that a favoring factor is the prodontism of superior incisors associated with hypotonia of upper lip.

A higher incidence of permanent teeth traumas in the USA is registered in the so-called "contact sports": ice hockey, American Football, basketball. However, it is interesting to mention that in some disciplines, according to a study performed by Reeves and Mendryc in 1970 on 5 890 players, only 446 cases of dental

traumas were found, representing only around 5 %.

Most often are affected the following permanent teeth:

- central superior incisors: 72 %;
- central inferior incisors: 18 %;
- lateral superior incisors: 6 %;
- lateral inferior incisors: 3 %.

Children with psychic disturbances are more exposed to dental traumatism, as they more often make protrusive movements of mandible and in some cases their coordination of movements is inadequate.

Class I.

Dental contusion with insignificant structural modifications

General characteristics In cases of strikes the force is distributed on the surface of tooth without producing significant structural lesions of solid dental tissues. The force applied on the dental tissues is insufficient to destroy them and it (the force) is transmitted completely to the periodontium. Due to these reasons, the signs of such lesions often are invisible and the visit to a dentist is postponed for some time, or the buccal cavity is examined by the doctor without a dynamic observation of the traumatized tooth. The described traumas can be characterized as a dental contusion able to affect the vascular-neural fascicle, followed by pulpal hemorrhage, insignificant ruptures of periodontal fibers. Clinically it can be manifested by insignificant pain during mastication, slight mobility, sometimes modifications in the color of dental corona.

Class I. Variant 1

Contusion of tooth with incompletely developed root

The children have accuses on insignificant pain during mastication. Sometimes there are no accuses at all.

Usually, there are no visible changes. Only under dental transillumination small cracks of enamel can be observed, they

are usually localized on the thin margins of the incisor enamel. They do not reach the limits of the enamel-dentine junction; in 50 % of cases they can be absent at all. Dental mobility of first degree may be present. The tissues around the tooth are not affected. Percussion is painful only during the first several days following the trauma.

No essential modifications to indicate any dental pathologies or pathologies of surrounding tissues are observed on the radiogram. EOM can be lower due to the incomplete development of root.

Treatment Mastication of solid foodstuffs is contraindicated during the first week following the trauma. In cases of new accuses (pains, increase of mobility, change of dental color) a repeated examination is required. In some cases selective polishing of antagonist teeth may be necessary in order to remove the occlusion discomfort.

Dispensary control of children with such traumas is of significant practical importance as it may allow to detect pathological processes at the very beginning of their manifestation and to undertake timely prophylactic actions. For this reason, it is rational to perform clinical examinations in the following periods: 1 months, 1.5 months and 6 months after the trauma. During clinical examination EOM must be determined and tooth radiography performed.

Class I. Variant 2.

Contusion of tooth with incompletely developed apex

The children have accuses on pains during mastication. Usually there are no complaints on thermal irritation.

During examination may be observed cracks on the surface of the tooth. Dental transillumination allows detecting cracks on the enamel that may cross the border of enamel-dentine junction. Tooth mobility of first degree may be present. Percussion is slightly painful. Radiographic images do not display visible

modifications, and only sometimes periodontal space can be insignificantly enlarged. Pulp reacts weakly on EOM.

Pulpal necrosis may be present in cases of lesion of neural-vascular fascicle. To the extent of radicular apex development the probability of neural-vascular fascicle rupture increases, as the striking force is propagated from the upper edge of the root to the periodontium. During the first days following the trauma EQM data are not consistent due to the commotion and pulpal edema. However, after some time (after 10-12 days) vitality of pulp may recover.

Treatment consists from protection in the process of mastication and exclusion of excessive forces. Sometimes selective polishing is indicated. Treatment is indicated if the EOM during 1.5 –2 months displays gradual necrosis of pulp. In such cases vital amputation or profound amputation of pulp may be required. Treatment must be done with observation of aseptic and antiseptic requirements with application of biologically active medicines (reparative, odontotropic preparations). Usually, affection of neural-vascular fascicle occurs in the narrowest region of the incompletely developed root, i.e. at 1-2 mm from the radicular anatomic apex. In such cases the profound amputation must be employed in order to close naturally the radicular apex with cement or calcification.

Children are of dispensary control and must follow regular control of pulp vitality and radicular apex formation. In cases of endodontic treatment one must observe the state of periapical tissues and the formation of radicular apex.

Class I. Variant 3.
Contusion of tooth with subsequent necrosis of radicular pulp with developed root

Children complain on modifications in the color of dental corona caused by pulpal necrosis. In some cases fistulas may appear.

At examination no evident modifications of the dental corona are identified. Only under dental transillumination longitudinal or transversal cracks on the entire surface of the dental corona may be detected. Dental mobility and painful percussion are observed only in the case of exacerbation of the inflammation process in the periapical region. EOM indicates necrosis of pulp.

*Treatment*If within 6-8 weeks following the trauma the pulp vitality is not recovered, endodontic treatment must be prescribed.

Dispensary control: dynamic observation of periapical tissues until complete liquidation of pathological process.

Class II

Non-complicated fracture of dental corona

This kind of dental traumas is frequently observed at children. Coronary fractures with structural lesions of enamel and dentine are very rarely observed only within the enamel region.

Dental mobility is relatively rarely observed. The coronary fracture is considered to decrease the force of strike transmitted to the roots and periodontium.

Sometimes in complicated cases dental mobility is observed. If this is the case, immobilization is required. Radiographic images rarely display modifications. Enlargement of the periodontal space is more often observed in cases of dental mobility of first or second degree. EOM during the first days remains within normal limits. There are two variants of lesions in this class.

Class II. Variant 1

Fracture of dental corona within the limits of enamel at all stages of tooth formation

At examination fracture of dental corona is detected only within the limits of the enamel or mesial or distal angles in the limits of the enamel-dentine junction. Visually the cracks of enamel are rarely observed. In dental transillumination a

multitude of cracks can be detected on the edge of the coronary fracture.

Treatment During the first week of treatment the affected tooth must be in rest. If the fracture edges are sharp, it is recommended to polish them carefully. During the first dynamic EOM is performed, and then repeated after 6-8 weeks. In case of normal pulp reaction and if the defect is not evident, with the parents' consent the neighboring tooth is polished in order to recover the aesthetic appearance of teeth. Both teeth can be polished. However, the intervals between procedures must be long: 5-6 months. The thickness of the polished stratum must not exceed 0.5 mm. Polishing must be accompanied with cooling by water jet, while polishing movements must be intermittent. Correction of edges of the affected tooth and of the healthy one lasts for around 1-1.5 years, if the fracture line is at the enamel-dentine junction or in the superficial strata of dentine. If the dental corona is fractured in the limits of enamel, polishing intervals are reduced. The polished surface is treated with fluorinated lacquer. Before each cycle of polishing and after 2-3 weeks checking EOM procedures are performed.

Dispensary control: dynamic observation of pulp until root formation.

Class II. Variant 2

Fracture of dental corona within the limits of enamel and dentine at teeth with incompletely developed roots

Children are pains on thermal irritation, usually, by cold agents.

At examination fracture of dental corona in the limits of dentine is detected. The fracture line can be of different configurations. At dental transillumination small cracks on the fracture edges can be detected. Dental mobility can be insignificant. Percussion may be slightly painful, especially in the horizontal direction.

Radiography can display absence of substance of the dental corona; however, there can be some tissue covering the pulpal cavity. The thickness of this tissue can be different. Roots can be at different development stages. In rare cases enlargement of periodontal space can be observed.

EOM is within normal limits; however, during the first visit the data may be misleading du to possible commotion of pulp caused by trauma. In such cases one must not hurry with endodontic treatment as vitality of pulp can be gradually recovered.

Treatment Urgent treatment is required in order to prevent the infection of dentine and protect the pulp from thermal irritation. The pulp can be protected by pre-fabricated coronas (metallic or plastic).

Technique:

1. The corona is adapted to the affected tooth, while the vestibular surface is perforated for the purpose of performing the EOM.
2. The tooth is treated with warm distilled water and then dried.
3. Odontotropic substance is applied to the dentine in the place of fracture (Calmecine, Daycal, Life, etc.).
4. The corona is fixed with phosphate cement, polycarboxylate, glass ionomer resins.
5. The excessive cement is removed, including the excess cement from the place of corona perforation.

After the fixation of corona it is compulsory to verify the occlusion. Dental corona will remain on the tooth till the definite formation of radicular apex. Afterwards it is removed and the shape of the tooth is recovered with the help of composite materials with or without parapulpal pivots, or by different types of dental coronas. If the corona is fixed onto a tooth with developed roots, then it shall be removed after 1-1.5 months. After the evaluation of EOM the anatomic shape and the function are recovered with the help of compomers or composite materials.

Dispensary control: dynamic observation of pulp until root formation.

Class III

Complicated fracture of dental corona

They are met quite rarely compared to the traumas of class II. According to the data of S. Celbier (1967) they amount to 20 % of the total number of frontal tooth traumas. Usually the children address to the doctor on the day of trauma or on the next day.

Fracture of a coronary part with opening of pulp in a certain point or sector is observed. In such cases children report pain from mechanical and thermal excitation. In some cases pulp can project from the zone of opening and bleed, painful at probing. If the child addresses on the next day after trauma, the pulp is covered with fibrinous deposits. Sometimes dental mobility can be observed or change of place. Percussion is painful.

Radiography displays lack of a part of dental corona with opening of pulpal chamber and different root development degrees. In all cases radicular fracture must be excluded.

EOM is performed in the case of point opening of pulpal cavity and change in the color of dental corona in the case of pulpal necrosis. EOM is necessary in order to appreciate the vitality of the pulp remaining in teeth with incompletely developed roots in order to save the growth zone. The EOM parameters during the first days following the trauma can be within normal limits, while in cases of pulpal necrosis they can be decreased. In case of necrosis the pulp may not react at all on EOM, while if a greater period of time has elapsed from the moment of trauma, the growth zone may be resorbed and this phenomenon can be confirmed radiologically.

Class III. Variant 1

Fracture of dental corona with point opening of pulpal chamber at teeth with incompletely developed roots

At examination a horizontal or oblique fracture with point opening of pulpal chamber is determined. Probing is painful. Dental mobility can be of first-second degree and insignificant displacement. Radiography displays enlargement of periodontal space. The dental roots or the radicular apex are incompletely developed.

Treatment If more than 24 hours have elapsed from the moment of trauma, in order to prevent complications vital amputation is indicated. If only several hours have elapsed, direct coating method is used. Calcium hydroxide pastes are applied. The results are considered good when the tooth is not filled and a pedodontic (orthodontic), or pre-fabricated crown (acrylic or metallic) is used. This modality allows periodically removing the crown and verifying the vitality of pulp. The dental crown is removed after 4-6 months. It is indicate to verify the thickness of tertiary dentine radiologically. If it exceeds 2 mm, it is recommended to recover the dental crown with composite materials or after the formation of radicular apex- with acrylic, ceramic or metallic-ceramic crowns. After 6-12 months from vital amputation radiography will display continuous formation of root and apex. EOM parameters can be still lower than normal.

Dispensary control: periodic verification of pulp vitality, observation of root formation process and apex genesis. Clinical observation must be continued till definite formation of root.

Class III. Variant 2

Fracture of dental corona with considerable opening of pulpal chamber and incompletely developed roots

At examination a fracture of dental corona in the limits of dentine with an opening of pulpal chamber of several millimeters is detected. The pulp may bleed, probing is painful. Horizontal

and vertical percussion are painful and intensive pains at thermal impact.

Radiography displays absence of substance in the dental corona and the root formation degree. In some cases the periodontal space is enlarged.

Treatment Direct coating is contraindicated. Most rational method is the vital amputation.

Dispensary control: observation of the periapical tissues and root formation.

Class III. Variant 3

Pulpal necrosis in result of dental coronary fracture with open pulp cavity at tooth with incompletely developed root

Dental coronary fracture with point opening or greater opening of pulpal cavity is determined. The color of dental corona often changes. At dental transillumination one may detect multiple small cracks on the edge of enamel fracture. Dental mobility can be absent or be of first degree. Percussion is susceptible. The tooth doesn't react on thermal irritation. EOM is low. Radiography allows determining the root and growth zone formation degrees. In most cases the growth zone is not affected. However, if after the trauma several months have elapsed, a chronic apical periodontitis appears that affects the root growth zone.

Treatment of this affection is identical to the one of pulpal gangrene of teeth with incompletely developed roots with preserved growth zone. The scope of treatment in this case consists in the permitting the continued growth of root. In order to fill the channel calcium hydroxide pastes are used. In case the growth zone is affected - the radicular channel is filled with endodontic material with bactericidal properties.

Dispensary control: observation of the periapical tissues and root formation after the endodontic treatment. .

Class III. Variant 4

Fracture of dental corona in the limits of enamel and dentine with considerable opening of pulpal chamber of teeth with developed roots

A dental coronary fracture with massive opening of pulp is observed at the examination. Multiple cracks at the edges of the defect can be detected under dental transillumination. The tooth requires careful examination as it can have an oblique coronary-radicular fracture. Palpation determines insignificant mobility (of first degree). Percussion is slightly painful. Radiography displays a fracture of dental corona with opening of pulpal chamber in the tooth with developed apex. In some cases an insignificant displacement of root can be observed that on the radiological image is showed as an increase of periodontal space between the root and the compact lamina of alveole.

Treatment Pulpal extirpation method is indicated with subsequent rehabilitation of the coronary fracture with composite materials or prosthetic methods.

Dispensary control: observation of periapical tissues during 1.5 – 2 years.

Class IV

Total fracture of dental corona

This variant of dental traumas is characterized by complete fracture of the dental corona at the level of gingival margin. Most often this kind of traumas is observed on teeth with completely developed roots. This is explained by the fact that if the roots are incompletely developed then in 95 % of cases a strike of such a force is sufficient for causing a partial or total luxation of tooth. This is why only one variant of such trauma is considered.

A complete fracture of dental corona at the level of gingival margin or 1 mm below it is determined at examination. In these cases a rupture of pulp is observed that can project above the fracture line. The pulp reacts on mechanical and thermal

irritation. Percussion can be painful.

Radiography displays absence of dental corona. One must analyze the root condition. Displacement of dental root is very rarely observed. It is not rational effecting EOM.

Treatment Vital extirpation method must be employed. Corona is reconstructed with the help of radicular pivots. Acrylic, porcelain or metallic-ceramic crowns are installed on the formed surface.

Dispensary control: observation of periapical tissues.

Class V

Longitudinal coronary- radicular fracture

This kind of trauma is rarely met at children and is considered as one of the severest ones. The fracture line crosses the enamel, the dentine, and the radicular cement under the gingival level on the vestibular or oral surface. Sometimes the fracture line can cross mesio-distal or distal-mesial surfaces.

Less often are the grave cases of fractures with depth of 5-6 mm, associated with the crushing of corona and dental root. In such cases restoration treatment is not rational.

This variant of dental trauma is not always visually identifiable. Often the child mentions mobility of a part of tooth. With the help of forceps one can easily determine the mobility of a part of dental corona held in place only by the soft gingival tissues. Dental corona can be of pink color. Dental cavity is open. The tooth or the fractured part can display different degrees of mobility (from I to III).

Not in all cases such a variant of trauma can be identified radiographically. If the fracture line goes in the mesio-distal direction, then it will be clearly seen. However, if it goes in the vestibular-palatal direction, it will not be possible to determine the depth of affection.

During the first several days after trauma the EOM data can be normal or lowered.

Treatment We recommend the following method:

1) anesthesia; 2) removal of the fractured fragment that is put into a hypertonic solution; 3) hemostasis; 4) endodontic treatment of channel; 5) obturation of channel and cementing of radicular pivot; 6) installation of the fractured fragment back into the place with cement or composite material.

If this method can not be implemented, after anesthesia and endodontic treatment of radicular channel it is recommended to fill the defect with zinc-eugenol paste or with an iodoform turunda. After several days the bandage is removed and a model of defect is made in order to produce a cast incrustation. The incrustation is fixed with phosphate cement, while the coronary part is recovered by a dental crown.

Dispensary control: observation of periapical tissues after endodontic treatment during 1,5 – 2 years.

Class VI
Radicular fracture

A radicular fracture can occur at any level of the root: 1/3 apical, 1/3 medium, 1/3 coronary. The fracture line can cross cement, dentine and the neural- vascular fascicle. This trauma is more often met at children of greater age, when the dental roots are already developed. Such fractures are usually observed on central superior incisors. Fractures of inferior incisors are rare.

Practically no symptoms are observed during examination. Sometimes the affected teeth are of pink color, if the fracture occurs in the apical third part of the root, but in many cases the color doesn't change at all. Dental mobility can be of any degree. Percussion is painful during the first several days following the trauma; the pain gradually reduces within 2-3 weeks.

During the first several days radiographic investigation may fail to display the fracture. In cases of doubts radiographic investigation must be repeated again in one week. During this period dental mobility may appear (beginning of reparative

processes) and insignificant extrusion of tooth from alveole. Simultaneously, the radiographic image will display the fracture line.

EOM data may be variable, but in some cases EOM parameters remain within the normal range. The pulp may preserve its vitality during a prolonged period of time.

The subsequent state of pulp will depend on the circulation disturbances. Some authors affirm that radicular fractures do not provoke affections of the neural-vascular fascicle. Others are opposed to this opinion, stating that pulp integrity is always affected but it continues to be supplied with blood due to the sanguine circulation via the vessels of periodontium.

Approximately in one week after the trauma the reparative processes begin, they evolve simultaneously with the inflammatory process that begins immediately with the fracture. During this period an increase of space between the fractured fragments can be observed.

Reparative processes can evolve on different scenarios of fragment re-unification: pseudoarthrosis, junction based on conjunctive tissue or junction of the conjunctive tissue with the bone. In case of absence of bond the granular tissue begins to grow between the root fragments. This tissue resorbs both roots and the surrounding tissues. In such case increased dental mobility can be observed. Dental mobility increases from day to day. One or several fistulas can appear on the gum.

Radiograms can display the following stages of regeneration process: rounding of fragments; unification of fragments, formation of chronic granulated process. During the first months following the trauma one must not hasten the endodontic treatment.

The success of treatment depends on the following principles: reduction of distance between the fractured fragments; sufficient stabilization of tooth; anti-inflammatory treatment; increase of

general resistance of organism.

Treatment The first visit comprises anesthesia, reduction of distance between the fragments, stabilization of tooth, liquidation of occlusion discomfort, indication of anti-inflammatory therapy. Should any other symptoms appear, the patient shall address again to the doctor. If necessary, endodontic treatment is performed.

In cases of fractures in the apical third of the root endodontic treatment of the big fragment is recommended and the small fragment is removed. When the fracture is in the limits of the middle and coronary thirds it is recommended to implement endodontic treatment with installation of pivot.

Dispensary control: verification of pulp vitality and of the state of fractured fragments. Observation periods: 1-2 weeks from the day of trauma, 1.5-2 months, and further - once per 6 months.

Class VII
Incomplete dental luxation

This kind of dental traumas is frequently observed in pedodontic practice. It is associated with traumas of classes I, II, III and IV, i.e. in cases of dental displacement and dental mobility. This kind of dental traumas implies affection of not only solid dental tissues but also of surrounding tissues: periodontium, alveole, gingival tissues. The traumatizing force in these cases is directed into the surrounding tissues of the tooth.

Dental luxation is often associated with ruptures of periodontal fibers. Clinically it is manifested by change in the position of the tooth compared to the other teeth and by mobility. Dental mobility can be of I, II and III degrees. The color of affected tooth is not changed. EOM is normal or partially absent. Radiological investigation can display increase of periodontal space in different sectors.

In this class we distinguish five variants of lesions: 1 – commotion; 2- incomplete luxation; 3- intrusive luxation; 4 –

extrusive luxation; 5 – lateral luxation.

Class VII. Variant 1
Dental commotion

By dental commotion we understand the trauma of surrounding tissues. This variant is similar to the traumas of class I. Both types of traumas miss structural lesions of teeth. However, the complications of class I trauma are focussed on the solid dental tissues and dental pulp while the ones of class VII traumas – on the surrounding tissues.

Visible modifications can be absent. Percussion may be slightly painful; mobility of first degree is possible. Pulp reaction can still be within the normal limits. Radiography may display enlargement of periodontal space in the apical region or uniform enlargement.

Tactics of treatment: 1) assuring a relative rest period of one week for the tooth; 2) removal of tooth from occlusion; 3) in case of aggravation (pains, increase, of mobility) – immediately consult the dentist.

Dispensary control: determination of pulp vitality in 1-2 years and subsequently once per 6 months.

Class VII. Variant 2
Incomplete luxation

It is characterized by the incomplete luxation of tooth without displacement. The color of tooth is not changed. Solid dental tissues are not affected. Mobility of I-II degree is present. Percussion is painful. EOM values can vary depending on the striking force and root development degree. Radiography can display uniform enlargement of periodontal space.

Treatment Observe the above recommendations (relative rest for the tooth, avoid biting food with the frontal teeth for 7-10 days). In some cases the teeth are immobilized for 2-4 weeks. Endodontic treatment is indicated in the cases of pulpal necrosis.

Dispensary control: dynamic observation of pulp vitality and root formation. Clinical observation period: 1,5-2 years.

Class VII. Variant 3
Intrusive luxation of tooth

Clinically is characterized by the intrusion of tooth into the alveole. The patient reports that after the trauma the height of the tooth became smaller than before. This trauma is more often observed at temporary teeth and less often at permanent ones. This variant is often associated with contusion of neighboring teeth.

Usually the dental corona gets submerged into the surrounding tissues to one half. However, complete submersion is also possible. Degree of intrusion depends on the applied force. The dental root is not affected.

Practically in all cases intrusive luxation is associated with pulpal necrosis. At teeth with incompletely developed roots this trauma affects the growth zone. During the first several days after the trauma the color of tooth doesn't change. Percussion is slightly painful.

Radiographic images display intrusion of tooth, alveolar fracture. Periodontal space is missing.

Variants of treatment: 1) dental extraction; 2) re-positioning of tooth with surgical clips; 3) dynamic observation of tooth – for spontaneous normalization; 4) orthodontic techniques.

After the tooth is back in its initial position, endodontic treatment is performed.

Dispensary control: observation of periapical tissues after endodontic treatment during 1,5 – 2 years.

Class VII. Variant 4
Extrusive luxation of tooth

This kind of traumas is characterized by extrusion, i.e. advancing of tooth from alveole. During the clinical examination

a relative increase of height compared to other teeth is observed. Neighboring teeth may display commotion. The root is not affected; however it may be uncovered by gum. Mark dental mobility of degrees III-IV. If the patient addresses for assistance after 4-5 days, the tooth can stick in an atypical position. In all cases occurs a rupture of the neural-vascular fascicle in the apical region. Radiographic images display evident widening of the periodontal space in the apical region.

Treatment Re-positioning of tooth in its initial position and fixing. The tooth is immobilized for 4-6 weeks. During this visit premature occlusion contacts are removed. After fixation of tooth in alveole, endodontic treatment is done after 1.5-2 months.

Dispensary control: observation of periapical tissues and of possibility for radicular resorption.

Class VII. Variant 5
Lateral luxation of tooth

It is characterized by luxation of tooth in the lateral or antero-posterior direction.

During examination a displacement of the tooth in mesial, distal, vestibular or oral direction can be observed. EOM values are variable. However, in most cases the pulp is also affected. Percussion is painful. The color of tooth is not changed, but after several weeks it turns gray due to pulpal necrosis.

Radiographic images: root not affected, alveolar fractures often observed, periodontal space widened in the apical region or on the lateral surface of the root.

Treatment Re-positioning of tooth and its immobilization is indicated. Observation of oral hygiene is essential for preventing infections. In case of pulpal necrosis endodontic treatment must be performed. If required, orthodontic treatment may be prescribed.

Class VIII

Total dental luxation

Dental traumas of this class are relatively rare compared to the traumatic affections of frontal teeth. Such traumas are usually caused by falling from height or by striking action of solid objects, road accidents, etc.

Main symptoms: falling of tooth from alveole due to exertion of a stronger force. Most often are affected the superior frontal incisors. The dental corona and the root are integer.

It is considered that if no more than 1.5 hours elapse from the moment of trauma and till treatment, the results of treatment can be positive.

Treatment consists in the re-planting of tooth: 1) the tooth is carefully processed and stored in a hypertonic solution; 2) endodontic treatment of tooth with obturation of channel (if root is incompletely developed and if less then an hour elapsed from the moment of trauma endodontic treatment is not required); 3) treatment of dental alveole; 4) re-planting of tooth; 5) immobilization of tooth for 4-6 weeks.

Dispensary control: observation of periapical tissues and of possibility for radicular resorption, inflammatory processes, etc.

Traumatic lesions of temporary teeth

Traumatic lesions of temporary teeth are quite often met in the pedodontic practice. According to Todd (1975), the incidence of frontal teeth trauma is 8 % from the total number of traumatic lesions of maxillary-facial region. They are most often observed at children aged between 1 and 3 years, i.e. when the children are very mobile but can not adequately control their movements and actions. Most frequent trauma is luxation (with displacements or not). Intrusion and extrusion, as well as total luxation of tooth dominate in this group.

Coronary fractures are very rare. In cases of coronary fractures without pulp opening fluoridation is performed (without

coronary reconstruction) with subsequent clinical observation and control of periapical tissues.

In cases of coronary fractures with opening of pulp endodontic treatment under general anesthesia is indicated.

Root fractures of temporary teeth are very rare and can affect only the teeth with developed roots.

If the fracture is in the first apical third of the root, a relative rest for the tooth is indicated with clinical observation. In cases when the fracture occurs in the more than ½ of root length – dental extraction is indicated.

Complications of temporary teeth traumas

Intrusions of temporary teeth may damage the permanent teeth buds. If the lesion of the tooth bud is insignificant, frequently appears the hypo-mineralization of enamel or the local hypoplasia of permanent tooth. In cases of significant traumatic lesions the mortification of permanent tooth bud can occur.

According to J.J. Ravn (1975), the most serious trauma of temporary teeth is the total luxation or the dental intrusion. If the total luxation occurs at a child aged less than 2 years, in 95 % of cases complications of permanent teeth will occur, if the child is aged between 2 and 4 years complications will constitute 80 % of cases. However, if such traumas occur at children aged above 5 years, the incidence of complications is minimum – 18 %. In case of intrusion of temporary teeth at children aged less than 4 years subsequently 54 % of permanent teeth will be affected.

Inflammatory processes of the periodontal tissues, appearance of fistulas are indications for the endodontic treatment of temporary tooth if there are no risks of damaging the permanent tooth bud.

Control questions and topics:

1. Classification of permanent teeth traumas at children recommended by WHO.
2. What is the prevalence of permanent teeth traumas at children?
3. Contusion of tooth with insignificant structural modifications.
4. Non-complicated fracture of dental corona.
5. Complicated fracture of dental corona.
6. Total fracture of dental corona.
7. Longitudinal coronary-radicular fracture.
8. Radicular fracture, its variants.
9. Dental commotion.
10. Types of dental luxation at children.
11. Total dental luxation.
12. Traumatic lesions of temporary teeth at children.
13. Complications of temporary teeth traumas.

6 *DENTAL CARIES AT CHILDREN*

Dental caries is a localized, multi-factor pathological process that appears after dental eruption and is characterized by demineralization and damaging of solid dental tissues with subsequent formation of cavity defects.

Being one of the most frequently met pathological processes of human organism, caries affects both permanent and temporary teeth, sometimes shortly after eruption.

The spreading of dental caries grew considerably during the last century. In several countries the affection of population by caries is 95-98%. Morbidity is now in a growing trend, especially it refers to children. Already in the age of 6-7 years 80-90 % of children may be affected by dental caries.

In order to determine the degree of affection by caries the World Health Organization (WHO) recommends the following indexes: the prevalence index, the intensity index and the intensity growth index (rate of caries or severity). These indexes require determination separately for each age group; sometimes they are determined separately depending on sex, nationality, geographic and living conditions, general health state, nutrition pattern, etc. WHO recommends performing the assessment of caries affection degree at children aged 6, 12 and 15 years. More useful can be the data of epidemiological examinations of children aged 12 and 15. Affection of teeth by caries at children aged fewer than 12 and the state of marginal periodontium at the age 15 allows determining the efficiency of prophylactic programs.

The *Prevalence Index* (P.I.) represents the percentage of

persons affected by caries out of a group of population (refers to the number of cases existing at a specific point in time per specified number of persons).

WHO recommends the use of following levels for the determination of prevalence of dental caries at children in the age of 12 years:

1. Low (0-30%);
2. Medium (31-80%);
3. High (81-100%).

The *Intensity Index* (I.I.) is determined by the average number of carious teeth (C), obturated teeth (O) and extracted teeth (E) per person. The value of COE index expresses the gravidity (activity) of the process. There are several denotations to this index: CER (C- caries, E-extraction, R- reconstitution by obturation); DMF (Decay (caries); M- missing (extraction), F – filling (obturation).

When noting the intensity index for permanent teeth one uses capital letters, while for temporary teeth - small letters (co, cr, dmf). Extracted temporary teeth due to radicular resorption before substitution with permanent ones are not noted.

The *Intensity growth index* (rate of caries, morbidity) represents the increase of COE index during a certain period of time (6 months, 12 months, 18 months, etc.).

WHO (1980) proposed the following levels of intensity of dental caries at children under 12 years:

1. Very low (0-1.1);
2. Low (1.2-2.6);
3. Medium (2.7 – 4.4);
4. High (4.5- 6.5);
5. Very high (6.6 and more);

The data on the intensity of dental caries at children in the age of 3, 6, 12 and 15 years from the rural and urban localities of the Republic of Moldova in the years 1996 and 2001 is given in table

6.1

Table 6.1

Number of carious, extracted and obturated teeth at children from rural and urban localities

Year/ age (years)	Carious teeth		Extracted teeth		Obturated teeth		COE/coe	
1996	Rural	Urban	Rural	Urban	Rural	Urban	Rural	Urban
3	0.27	0.42	0	0	0.11	0.17	0.38	0.59
6	0.14	0.22	0	0	0.15	0.22	0.29	0.44
12	0.48	0.89	0.36	0.27	2.08	2.89	2.92	4.05
15	1.19	2.04	1.42	1.56	1.41	1.87	4.02	5.4
2001	Rural	Urban	Rural	Urban	Rural	Urban	Rural	Urban
3	0.28	0.49	0	0	0.07	0.15	0.35	0.64
6	0.12	0.30	0	0	0.10	0.18	0.22	0.48
12	0.66	0.97	0.96	0.52	1.42	2.97	3.04	4.46
15	1.88	2.65	1.60	1.68	0.84	1.20	4.32	5.53

Dental caries is very low intensity in Ethiopia, China, North Nigeria, Tuva and some localities of Georgia and Armenia.

The low levels of dental caries intensity were found in Mozambique, Uganda, Sri-Lanca, Burma, Indonesia, Switzerland, Denmark, Belgium, Uzbekistan, Tajikistan, and Russia: Altai and Amur regions, Bureatia, Kolomna, Tambov, Cita.

Moderate intensity of dental caries is observed in the Republic of Moldova, Romania, Great Britain, Sweden, Austria, Czech Republic, Finland, Jordan, Argentina, and Azerbaijan.

High intensity of dental caries is registered in Germany, France, Norway, Iran, Mexico, Cuba, and Chili, Russia: in Kamchatka, Murmansk, Mahachkala, Krasnoyarsk, Novosibirsk, Ekaterinburg, Smolensk, Tver, Krasnodar, Voronezh, Moscow.

Very high intensity is observed in Japan, Canada, USA, Italy, Lithuania, Latvia, Estonia and Russia: Archangelsk, Omsk, Sochy, Nikolaevsk on Amur.

Romania is among the countries with moderate COA index (2.7-4.4) and the average value of COA index there is 3.14 in 1986 and 3.13 in 1996 for the children aged 12 - as stated by the

Collaboration Center of WHO for Children's Oral Health of Iasi (Rusu M. et al. 1986, 1996). Romanian children and adolescents displayed the following indexes of dental caries for all age groups in 1986 and 1996:

6 years (temporary teeth):

- Prevalence index 85 % - 83%;
- COA index: 4,45-4,76.

12 years:

- Prevalence index: 79-76 %;
- COA index: 3,14-3,13.

18 years:

- Prevalence index: 91 %;
- COA index: 6,4.

Affection of population by caries, especially of children, varies considerably within a country and within regions and localities of a particular country.

During the last twenty years the caries intensity has a decreasing tendency in the economically developed countries (Switzerland, Great Britain, Denmark, Japan, USA, etc.). A study performed in Denmark (Bille et. al.) aimed at the determination of intensity of dental caries at the children of 7, 11 and 13 years in 1963, 1972, and 1981 revealed a considerable reduction over a time span of 18 years. In USA the dental caries intensity index in 1981 appeared to be 36 % lower compared to the level of 1970.

There are many opinions regarding these reductions, mainly they address increase of life levels, improvements in nutrition, resistance of dental tissues, microflora, etc. Obviously, they can be explained by implementation of prophylactic programs (fluoridation of water, oral cavity hygiene, wide use of prophylactic agents, etc.).

According to the forecasts of the World Health Organization, until 2010 year will be attained a situation in which 50 % of children aged between 5 and 6 will not have caries at all and in

the age of 12 years - they will have less than three carious, filled or missing teeth.

WHO considers that every country should formulate its own priority objectives, depending on particularities, necessities and possibilities, considering the recommendations and the unique indexes for determining the oral health and the global tasks in this domain.

Pursuant to the actual concepts, dental caries is a pathological process provoked under certain conditions by a complex of pathogenic factors, i.e. in the case the favorable conditions for carious are established that in their turn are determined by the microbial factor. Dental caries appears when the intensity of carious situation in the oral cavity exceeds the resistance capacity of solid dental tissues. The interaction of carious factors leads to the appearance of caries, while the intensity of their action determines the activity of this process. However, even in the satisfactory clinical situations in the oral cavity may be present a number of risk factors for the appearance of caries.

Interaction of four most affective factors (fig.6.1.) can lead to the appearance of dental caries: susceptibility of tooth and buccal medium, bacterial plaque, substrate (food) and time (Keyes P.H., 1969). The mechanisms involved in the appearance of dental caries at children are identical with the ones causing caries at adults. However, the intensity of these factors is obviously higher in the period of growth of organism and this to a considerable extent determines the particularities of clinical evolution of dental caries at children.

Appearance of dental caries depends on certain conditions:

1. Presence of microorganisms in the dental plaque;
2. Insufficiency of specific (immune) mechanisms and non-specific ones for the protection of oral cavity against affections;
3. Nutrition derangement;

4. Time factor.

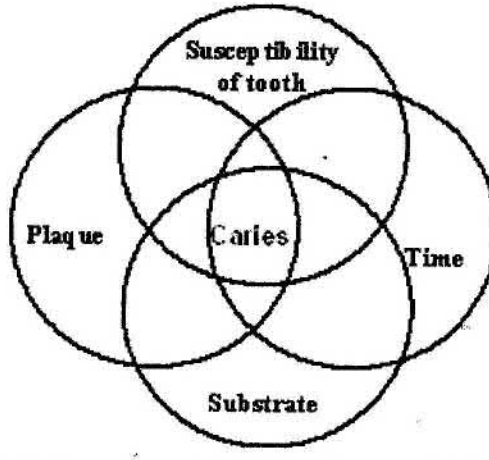


Figure 6.1. **Interaction of carious factors (by Keyes P.H.)**

Affection of temporary and permanent teeth of children and adolescents by caries depends on geographic factors, life level, age, sex, heredity, nutrition, degree of oral hygiene, state of organism, etc. At some children dental caries appears already during the eruption of temporary or permanent teeth.

Microbial flora

Experiments on animals demonstrated that bacteria play the priority role in the appearance of dental caries, especially the *Streptococcus mutans*, that initiates caries and *Dactilobacilus* that influences the progress of carious process. Such bacteria like Veillonella, Staphylococcus, Neisseria and other kinds of Streptococcus have a less important role in the evolution of dental caries. Actinomyces depend on the evolution of caries in the region of dental collar.

One of the conditions for the appearance of dental caries is the presence of nutritive substances in the dental plaque for the fermentation activity of bacillus. Experiments proved that at

animals in the presence of a big number of such germs as Str. mutans, Lactobacillus, etc. without carbohydrates caries doesn't appear. On the other hand, caries was not found at animals at which the above germs were missing (in sterile conditions) and that were fed with considerable quantities of carbohydrates. Most active carbohydrates are the mono- and bi-saccharides with approximately equal carious potential.

The high carious potential of Str. mutans is explained by:

1. Str. mutans supplementary produces the polysaccharide – sucrose from glucose. The plaque gets inserted into the surface of the tooth and Str. mutans is tied with all its layers.
2. Str. mutans supplementary synthesizes a cellular polysaccharide from fructose that is composed of sucrose and uses it as a reserve between meals.
3. Str. mutans produces organic acids (especially lactic acid) out of sucrose thus demineralizing the enamel.

Under normal conditions mechanical removal of dental plaque leads to the reduction of the number of bacteria limits their activity and development. During speech, mastication and tooth cleaning the bacteria are eliminated from oral cavity. It is most difficult to remove the germs from the hardly accessible places - from the interdental spaces, from periodontal sack, dorsal side of tongue. Dental caries can be considered as an infection process: Str. mutans can be transmitted from parents to children and from children to other children. Was established the correlation between the quantity of Str. mutans at children and their mothers. Mothers with big quantities of Str. mutans in oral cavity can transmit the germs to their children. Str. mutans can be transmitted by using the same spoons, forks, nipples, kisses in lips, etc. Early infection of children with Str. mutans can lead to the appearance of bottle caries, rampant caries, with progressing activity, to the appearance of multiple lesions able to turn into

complete destruction of dental corona, to the appearance of odontogenous infection nidus, etc. and after eruption of permanent teeth – to their affection.

One of the main conditions for the appearance of dental caries is the insufficiency or disorders of defense processes. This is confirmed by the fact that the carious process usually evolves during 1-2 years, as there are protection mechanisms inhibiting the appearance of dental caries. Such mechanisms depend on a wide variety of factors that amplify or inhibit the evolution of carious process.

The carious process can be regarded as a disbalance characterized by the demineralization of solid dental tissues. After each meal the microbial flora metabolizes carbohydrates leading to the accumulation of organic acids in the dental plaque that subsequently leads to the demineralization of enamel. The re-mineralization mechanisms of oral liquid (tampon systems, calcium and phosphates, antimicrobial agents, etc.) tend to interrupt the acid attack and reconstruct the enamel (fig. 6.2).

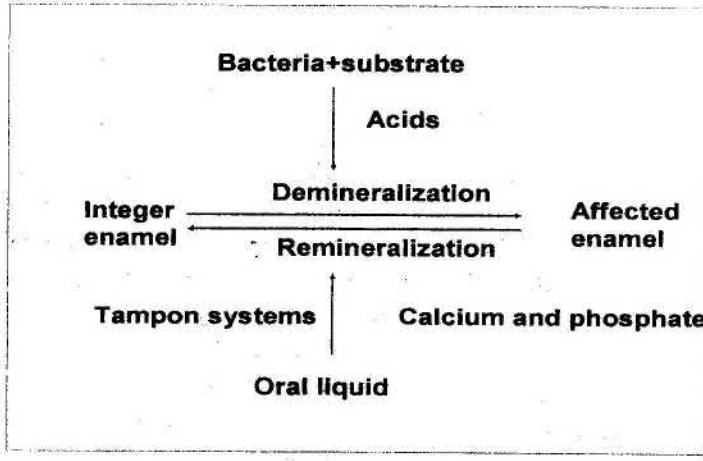


Fig. 6.2. **Re-mineralization and demineralization processes**

Duration of acid attack and the degree of demineralization depend on a wide range of factors: the greater is the number of germs in the dental plaque, the greater is the quantity of synthesized acid, the more time the food remains on the surface of teeth, the older is the bacterial plaque, the greater is the production of acids, when the quantity and quality of saliva are low the duration of acid attack is longer.

Another important factor in the appearance of dental caries is the self-cleaning capacity of oral cavity. The anatomic shape of teeth (globular shape of dental corona, with deep grooves and slots, etc.), their position on dental arcades (positioning anomalies, concentration, etc.), dental-maxillary disharmonies, quantity and quality of saliva - all these directly influence the self-cleaning capacity of oral cavity. One more protection mechanism against dental caries is the buccal liquid that contains a big number of substances with anti-carious action. They can be conventionally divided into two groups: active and passive. The active ones have bacteriostatic properties while the passive ones prevent or interrupt the formation of bacterial plaque by modification of microbial medium.

Saliva contains 1-3% immunoglobulins, the most important of them being sIgA. The buccal liquid contains different cellular protection components: neutrophils, macrophages, lymphocytes. Immunoglobulins are considered to maintain the physiological equilibrium in the oral cavity, performing control of the growth of carious germs.

At children the functional activity of local immunity against dental caries is subjected to individual fluctuations depending on age, sex, season of year, etc. At children in the age of 4-5 years no correlation was determined between the titer of antibodies against streptococcus and number of carious teeth while in the age of 9-10 years a high inverse correlation is noticed. However, in the age of 11-12 and 14-16 years the correlation reverses.

The resistance of solid dental tissues to the action of acids also determines the appearance and evolution of dental caries at children. Mineralization of solid dental tissues of temporary and some permanent teeth takes place during the second half of pregnancy, this is why their structure is very much determined by the mother's health, quality of nutrition, living and working conditions, etc. In cases of advanced chronic diseases and severe disturbances of mother's metabolism, for example, pregnancy toxicosis, endocrine diseases, etc. at children often immediately from eruption the temporary teeth become affected by multiple and progressive caries. Toxicosis during the first half of pregnancy is the cause of appearance of some incompletely developed tissues with distorted structure (Попова О., 1980). Teeth developed in such conditions have a lower resistance against caries. Toxicosis during the second half of pregnancy considerably favors the appearance of hypoplasia and dental caries. The rate of dental caries by ***co*** index at children is much lower compared to the children born by mothers that suffered from toxicosis during the first half of pregnancy while the level of hypoplasia is in inverse correlation. At children born by mothers with late pregnancy toxicosis and diseases during the neonatal period displayed a higher incidence of hypoplasia and dental caries. At children fed artificially during their first year of life and at ailing ones the intensity of dental caries exceeds the levels observed at naturally fed children and the ones that suffered from diseases less frequently.

Premature affection of teeth and their progressive destruction is often observed at prematurely born children, children that suffered from traumas during birth, different infectious diseases, etc. (dyspepsia, rachitis, etc.).

The child's organism is very sensitive to the non-observation of regime, to the nutrition patterns and living conditions. Disturbance of regime can lead to reduction of organism's

reactivity, to disharmony of physical development that are of special importance in the appearance and evolution of dental caries (Виноградова Т.Ф., 1978; Сайфуллина Х.М., 1978; Гунчев В.В., 1982; Удовицкая Е.В., 1984).

Dental caries is especially frequently detected at children with different chronic diseases, in particular, of infectious-allergic genesis (Виноградова Т.Ф. and co-authors, 1970, Удовицкая E.B. and co-authors, 1978; Белова Н.А., 1981, Jwinsik, 1973; Dymkowska, Stokowska, 1976, etc.).

At children that frequently catch colds and suffer from infectious diseases (Мясный З.П., 1976), rheumatism (Максимова О.П., 1970, Соколовская Е.П., 1972), nephritis, congenital cardiac diseases (Смоляр Е., Ковалюк И., 1973) the carious processes are often progressive and accompanied with demineralization of solid dental tissues extending into complications at the level of dental pulp. Multiple affection of teeth, 3-4 cavities per tooth, recurrent caries have been observed at children suffering from collagenosis (Гусейнова Т.Г., 1978), chronic pneumonia (Курякина Н.В., 1981), neural-psychic affections (encephalopathy, oligophrenia, epilepsy) and endocrine diseases (Rusu M., et al., 1980) and oto-rhino-laryngological diseases (Тристень К.С. et al., 1984), etc.

Children suffering from tuberculosis display an atypical localization of carious defects, affections of some groups of teeth, active evolution, intensity and higher rates of dental caries often aggravated by pulpitis and pulpal gangrene (Демнер Л.М., 1975, Просверяк Г.П., 1977).

The studies performed by Р.Г. Стародубцева (1974) have evidenced the complete destruction of coronary part at most teeth at children suffering from cerebral paralysis.

An obvious correlation was found between the demineralization and mineralization processes of enamel and the state of non-specific resistance of child's organism (Рединова

Т.Л., 1981). In cases of low non-specific resistance at children with different degrees of resistance against dental caries the solubility of enamel is inferior. The saliva of caries-prone children misses not only ions of calcium, but also organic phosphates (Сайфулина Х.М., 1980; Рединова Т.Л., 1981; Овруцкий Г.Д.), reduction of total proteins, albumin and γ-globulins in the sanguine serum and saliva (Гусев Ю.С., 1970; Гринина А.В., 1973; Сайфулина Х.М. , 1981). Reduction of lysozyme (Cisowski, 1973; Гранин А.В., 1975), and of IgA content in saliva (Everhart et Al., 1972; Oprisin et al., 1973; Legler et al., 1979; Виноградова Т.Ф. et al., 1979) were established at children with multiple caries and caries with active evolution.

The patients that followed treatment with corticosteroids that inhibit the non-specific resistance of organism displayed higher fragility of solid dental tissues, appearance of new carious cavities and recurrent caries (Варенников А. И., 1996).

Nutrition

The nutrition pattern is very important for teeth, as it directly affects the formation and development of teeth, determining the receptivity to caries or resistance against caries.

The nutritional equilibrium assumes an optimum and qualitative proportion of nutrients and biologically active substances - proteins, carbohydrates, lipids, vitamins, mineral substances.

Interrelation between metabolism in dental tissues and metabolic processes in organism has been demonstrated by multiple researches. Already from the second day the caries-favorable diet provokes disturbances in the metabolism of proteins in teeth and bones of animals. Continuation of such diet amplifies and implies different metabolic disturbances (first of all in the mineral metabolism). Caries- favorable diet provokes structural disorders in the formation of solid dental tissues. The

deficit of proteins during the development period leads to reduction of body weight and dimensions and to disorders in the solid dental tissues.

The quantity of proteins in food influences the mineral composition of teeth and maxillaries: in cases of low quantity of proteins in food accumulation of calcium and formation of apatite is insufficient. Proteins contained in meat, milk and eggs are distinguished by most favorable proportion of amine acids that assure a higher level of retention and re-synthesis of tissue proteins in the organism.

The role of carbohydrates as factor of major importance in the evolution of dental caries has been underlined in multiple studies. According to many authors, consumption of carbohydrates is now high and continues to grow. More than a half of the total number of examined persons frequently consume carbohydrates (three times a day) and in considerable quantities (sugar 3 spoons three times a day, patisserie products and bakery products at each meal) (Рединова Т.Л., Леонтиев В.Е., 1990). Consumption of carbohydrates is especially high among adolescents. The performed studies revealed that carbohydrates: can remain for a long time after ingestion in the oral cavity, adhere to the surfaces of teeth and easily penetrate into the microbial plaque, they are used by the microbial flora and lead to the reduction of pH, polysaccharides are less harmful compared to disaccharides, sticky sugars are the most dangerous ones due to prolonged sticking to the teeth: patisserie products, sweets, etc. – all these can stay on the surfaces of teeth for more than one hour (Шадиев К.К., 1983).

Consumption of carbohydrates and increase of their consumption favors the development and growth of pathogenic germs on the dental surfaces. This leads to the increase of the quantity of acids that provoke demineralization of enamel. The heaviest demineralizing action is exerted by the 3 % solution of

sucrose (micro hardness falls by 28.8 %) and 6 % solution of glucose – by 22.2 % and solution of 10 % syrup with 8 %. Further increase of concentration doesn't lead to increase of demineralization rate (Herper B.P., Arends J., 1986).

During the first years of life of special importance is the content of microelements in the nutrition of children. Deficit of microelements at suckers is compensated from the internal reserves of child's organism that accumulates a certain quantity of microelements during the period of intrauterine development. This is why rational nutrition of pregnant women is very important (Гончарова Е.И., 1986).

Microelements calcium and phosphorus - they are the components of apatite that in its turn constitute the solid dental tissues and bones. They also reduce the pH of bacterial plaque and contribute to the formation of tampon solutions (Боровский Е.В., Леус П.А., Леонтьев В.К., 1978).

The processes of enamel and dentine formation and osteogenesis are determined by the presence of fluoride (Габович Р.Д., Овруцкий Г.Д., 1969, Коваленко Л.И., 1974 et al., Hunter P.B., 1988; Marthaler T.M., 1990; Triller M., 1992;, Mathewson R.J., Primosch R.E., 1995). It was determined that the presence of fluoride during the period of formation of solid dental tissues assures a certain level of resistance against caries for many years as fluoride leads to the formation of fluoroapatite that are especially resistant against acids. The optimum quantity of fluoride for the prevention of dental caries is 1 mg/1F per day.

Favoring factors

A. Antenatal period

Negative influence of harmful factors on the pregnant woman and fetus:

- pathology of organs and systems of the pregnant woman: endocrine diseases, gastrointestinal diseases, cardiovascular ones, nephropathies, etc.;

- pathology of pregnancy: toxicosis, etc.;
- insufficient and irrational nutrition;
- deficit of fluoride in potable water;
- inadequate living and working conditions;
- intoxication;
- number of pregnancies, etc.

B. Postnatal period

During the first year of life:

- type and pattern of nutrition (natural, artificial, mixed);
- general diseases (of gastrointestinal tract, hypovitaminosis, rachitis, etc.);
- dental eruption (premature, tardy);
- hypoplasia of enamel;
- insufficient buccal hygiene, etc.
- deficit of fluoride in potable water.

At children and adolescents:

- inadequate buccal hygiene;
- deficit of fluoride in potable water;
- anomalies of position of teeth, occlusion, orthodontic devices, etc.;
- dental hypoplasia;
- dental eruption (premature, tardy);
- hyposalivation, acid medium of the buccal liquid, insufficiency of Ca, P and F ions in ferments, immunoglobulins, etc.;
- general diseases (of gastrointestinal tract, endocrine system, cardiovascular system, etc.);
- irrational nutrition: surplus of carbohydrates, insufficiency of proteins, vitamins A, B, D, C, etc. of Ca, P, F and other compounds.

Classification of dental caries

There exists a wide variety of classifications of dental caries of temporary and permanent teeth depending on the following

criteria: morphology, degree of affection, affection speed, etc.

Classification by WHO:

K.02. Dental Caries:

K.02.0. Enamel caries (incipient caries)
K.02.1. Dentine caries
K.02.2. Cement caries
K.02.3. Stabilized dental caries (stationed)
K.02.4. Odontoclastia. Infantile melanodontia
Melanodontoclasia
K.02.8. Other precise dental caries
K.02.9. Imprecise dental caries

Morphological classification by Black

Class I: cavity pits and fissures occlusal surfaces of molars and premolars, from the vestibular surfaces of molars and palatal ones of incisors.

Class II: cavity in approximal surfaces of molars and premolars.

Class III: cavity in approximal surfaces of incisors and canines, not involving incisal edge.

Class IV: cavity in approximal surfaces of incisors and canines including incisal edge.

Class V: cavity in cervical third of buccal or lingual surface of any tooth.

Class VI: affection of incisive margin, cuspidal top due to structural defects or traumatic lesions.

Classification of temporary teeth caries by L. Zarnea

- simple superficial caries : with affection of enamel and limited affection of dentine with preservation of a thick layer of integer dentine;
- simple profound caries: with extended affection of dentine layer and maintenance of a smaller layer of integer dentine.

Classification of permanent teeth caries by L. Zarnea

Degree I - limited affections of enamel;
Degree II - affections reaching the enamel-dentine junction;
Degree III - limited affections of dentine;
Degree IV – extended affection of dentine layer.

Advancing speed of caries

- acute caries;
- chronic caries.

Classification of dental caries at children by T. Виноградова:

I. Degree of activity of the carious process:
- activity degree I (reduced carious activity);
- activity degree II (medium carious activity);
- activity degree III (high carious activity).

II. Localization of carious process:
- cracks;
- approximal;
- cervical;
- circular.

III Depth of solid tissue affections:
- incipient;
- superficial;
- medium;
- profound.

IV. Succession of the appearance of carious process:
- primary caries;
- secondary caries.

V. Pathomorphologic pattern:
- maculated caries;
- caries of enamel (incipient);
- medium caries;
- medium deep caries (corresponds to clinical

manifestations of profound caries);

- profound perforate caries (corresponds to pulpitis or pulpal gangrenes).

The degree of activity of the carious process, i.e. the index of caries intensity depends on the age (table 6.2, 6.3, 6.4).

Table 6.2

Value of the caries intensity index at children with first degree of activity (reduced carious activity)

Age, years	Intensity of caries	Value
3-6	co	<2
7-10	COE+co	<5
11-14	COE+co	<4
15-18	COE	<6.

Table 6.3

Value of the caries intensity index at children with second degree of activity (medium carious activity)

Age, years	Intensity of caries	Value
3-6	co	3-6
7-10	COE+co	6-8
11-14	COE+co	5-8
15-18	COE	7-9

Table 6.4

Value of the caries intensity index at children with third degree of activity (high carious activity)

Age, years	Intensity of caries	Value
3-6	co	>7
7-10	COE+co	>9
11-14	COE+co	>9
15-18	COE	>10

General particularities of dental caries evolution at temporary teeth

Regardless of the fact that caries of temporary teeth evolves according to the same laws as the caries of permanent teeth, clinically one can distinguish several particularities determined by the following factors:

1. Structural particularities at different stages of temporary teeth development;
2. Risk factors favoring the appearance of dental caries;
3. Intensity of carious process, degree of affection of solid tissues;
4. Reaction of pulp, etc.

One must take into account the fact that at newborns the fissures and approximal slots of molars, collar regions of coronas of incisors and temporary canines are not completely mineralized. Primary and secondary mineralization of these sectors occurs during 2.5 years from birth. As till the age of 3 years caries localizes in the sectors of postnatal mineralization, i.e., on the smooth surfaces, in the region of incisor collars, after 3 years – in the pits and fissures of molars, while after the age of 4 years – on the approximal surfaces.

As the dentine and enamel of temporary teeth, compared to permanent ones, are of smaller thickness and volume their structure contains many more organic substances, the intensity of carious process in temporary teeth is higher.

First teeth affected by caries are the superior frontal incisors, especially the central incisors as this group of teeth erupts first. Thereafter are affected the first molars, second molars and canines. Most resistant are the inferior incisors.

Frequently, temporary teeth are symmetrically affected, i.e. the lesions appearing on one semi-arcade are doubled in the similar way on the teeth of the other semi-arcade. No evident differences have been found between the affections of superior

and inferior teeth at children.

Evolution of fissure caries is facilitated by the existence of fissures and pits where food remainders are retained, etc. Most frequently such caries is found at children aged between 2 - 5 years. After this age, in parallel with abrasion of solid dental tissues, the chances for the appearance of fissure and pit caries decrease, while the chances for approximal caries increase. Approximal localization for molars is frequent at the age of 4-5 years, subsequently being facilitated by intra-bone displacement of first permanent molars. Modification of relations from the level of contact points of temporary molars represents favorable conditions for retention and facilitates the appearance of approximal caries, even at children with good resistance to caries. The debut of approximal caries occurs at the level or under the contact point, this location corresponding to the Schoor neonatal line, considered as minimum resistance sector.

Cervical caries is often detected after the eruption of temporary teeth, especially superior incisors. Such caries appear in the zones of insufficient mineralization, on the surfaces with structural disorders of enamel (hypoplasia). It can also occur in the period of transition to mixed dentition when dental migration and gingival retractions open the cervical zone. They have a tendency for rapid extension on surface (see color insert fig.6.3.).

During the first years of life at children often occurs circular caries. In the debut this process appears as a white demineralized macula expanding from the enamel-cement limit on the vestibular surface of the frontal teeth. It rapidly extends in the cervical region on all surfaces of tooth and can lead to the fracture of dental corona. At the final stage brown dental spots can appear, and pulp remains vital in rare cases. Circular caries first of all is found on superior incisors, then – on canines, inferior incisors and at last – on molars, so at the age of 4-5 years all the teeth can be completely destroyed.

Acute caries is especially characteristic for the age of 2-3 years. Acute caries of temporary teeth is multiple and symmetric, rapidly progressing in depth.

Rampant caries is characterized by extended decalcification in depth and on surface. Affection of pulp usually occurs immediately after the crossing of superficial strata of dentine, as it is very thin, has short and long channels. Absence of protective reaction of the temporary teeth pulp (by formation of tertiary dentine, etc.) can be also interpreted as lack of time for the mobilization of protective mechanisms.

During the recent time a diversification of clinical forms is observed. Namely, two tendencies of caries manifestation are observed:

First form *(severe form)* is characterized by anarchic localization, with appearance on the immune surfaces, with simultaneous affection of all dental groups, without particular succession, with rapid evolution on surface and in depth, leading to rapid and premature destruction of teeth. This type of affection is described in several variants: small age caries, premature caries, box caries, violent caries, galloping caries, nursing caries, nursing bottle caries, night bottle syndrome, infantile melanodontia, rampant caries, le syndrome de biberon, etc.

The bottle caries is a specific acute form characteristic for temporary teeth, appearing at very small ages, rapidly evolving on surfaces and in depth with affection of pulp and coronary destruction, unless adequately treated. The bottle caries is the result of interaction of three factors: pathogenic microorganisms in buccal cavity, fermentable hydrocarbons and dental substrate. One of the main causes of affection is the replacement of natural breast--feeding with artificial feeding, characterized by the increase of hydrocarbon levels in the nutrition, usually associated with tardy use of nipple. Interaction of these three factors and the existence of incorrect feeding traditions, failure to observe oral

hygiene - all these contribute to the appearance and rapid evolution of dental caries. The incorrect feeding practices leading to the appearance of bottle caries are: feeding with nipple before sleep, especially during the night, feeding with liquids (milk and sweet tea, artificial milk, juices) with high concentrations of fermentable hydrocarbons, giving hydrocarbons (honey, powder sugar, syrup, etc.), prolongation of breast-feeding above recommended age (Hallonsen et al., 1995). Prevalence of nipple caries varies between 1 and 12 % (Meon, Milnes). A study performed in the Pedodontic Stomatological Clinic of Bucharest revealed that 10.85 % of children aged between 1 and 6 years displayed signs of bottle caries, boys being affected to a greater extent compared to girls (Luca R., 1998). Treatment of this form of caries is associated with difficulties.

Second form is characterized by a debut extended on surface leading to premature loss of retention zones followed by very slow evolution accompanied by the impregnation of pigment into the dentine surfaces and formation of considerable quantities of secondary dentine. Takes place the abrasion of enamel of occlusal surfaces of molars, dentine varying in color from light yellow to dark brown. It is solid, glossy and painless in probing. Finally, on the arcade are observed radicular remainders with pigmented dentine with preserved vitality. Main cause is disorders of metabolism during the first year of life conditioning the appearance of structural defects of solid dental tissues and favoring the affections of large surfaces (see color insert fig.6.4.).

Caries of temporary teeth can be localized in the sectors of hypoplasia, i.e. can develop complications or be associated with systemic hypoplasia. In such cases the carious cavities have irregular shapes without delimitation tendency, they are flat, the dentine being mottled and pigmented.

Evolution particularities of temporary teeth caries

The carious process has the following distinctive signs:

- acute evolution of caries predominates, especially in the age between 2 - 3 years;
- simultaneously with age the number of acute evolution caries decreases and the number of chronic caries increases;
- in most cases multiple caries is detected;
- localization of carious defects is symmetric for each arcade;
- first teeth affected by caries are the superior incisors (especially, the central ones), due to early eruption of this group of teeth and other carious factors. Then, in descending order, follow: first molars, second molars and canines. Inferior incisors are more resistant to caries;
- in the ages of up to 2 years most often is detected the caries of smooth surfaces, while in the age of 3 years – fissure caries, and in the age of 4 years - approximal caries. The appearance of approximal caries is characteristic at the level or immediately under the contact point corresponding to the neonatal line considered as minimum resistance sector;
- often caries is detected in the cervical region - also called circular caries. This type of affection is mostly spread on surfaces, not in depth. First appears in the form of maculae that subsequently affect the enamel-cement line in the circumference of tooth, affecting deeper and deeper, sometimes leading to the loss of dental corona;
- acute caries is spread mostly in depth while the chronic form mostly affects surfaces;
- clinical evolution is weakly pronounced, especially during the period of radicular resorption;
- profound caries is usually complicated by pathological processes in the dental pulp;
- secondary caries is detected most often;
- till the age of 3 years caries rarely develops complications while with age the incidence of complications increases.

Diagnosing temporary teeth caries

Simple caries of temporary teeth is poor in subjective signs. Subjective signs can be vague, even in cases of profound affections. This can be explained by the fact that it is not always possible to obtain precise information from small children, while at children of bigger age the involutive processes in pulp reduce the perceptibility. Sometimes children can have accuses on high perceptibility during meals as reaction on chemical or thermal excitation, especially on sweets. At children with multiple caries accumulation of perceptibility of several more teeth can lead to refuse from food, thus provoking sufferings or disturbances during meals, absence of appetite, disturbances of sleep, etc.

Inspection Diagnosis of caries of temporary teeth is simple, as the stains and carious defects are easily detected during examination. For the diagnosis of maculated caries one can successfully use the colorimetric method based on the increased permeability of enamel and demineralized dentine. In some cases it is difficult to diagnose proximal caries. However, such affections display themselves by modifications of color under the marginal crest, presence of papillae, and reduction of dental corona crest. Some forms of approximal caries can remain clinically undiagnosed and only radiological examination may permit their detection.

Probing Probing must be performed with special care and attention in order to exclude negative sensations. The probe is retained in the fissure carious holes; it easily penetrates into the damaged dentine. For this reason it is first recommended to remove detris and damaged dentine with excavator in order to appreciate the depth of affection, etc.

Percussion Vertical and horizontal percussion are negative. Only in cases of approximal caries complicated by inflammatory processes of marginal periodontium horizontal percussion can be followed by pains.

Thermal tests At small children they do not allow making any conclusions and are avoided.

Radiological examination Allow detecting and appreciating the depth of carious defect, its relation with the pulpal chamber and the state of dental roots. On radiographic images caries appears as a transparency zone with irregular edges in the corona region. Allow detecting carious defects on approximal surfaces.

Drying method of dental surface allows visualizing the carious spots that have no gloss and are of opaque color.

The silk thread sign is used for the diagnosis of approximal caries. The silk fiber passed between the teeth is defaced or torn.

Electroodontodiagnosis is practically not used on temporary teeth due to incorrect interpretation of results.

Vital colorimetry is based on the increase of permeability of the solid dental tissues. At contact with coloring solution (sol 2% blue methylene iodine, etc) during 1 minute of exposition the demineralized sector absorbs the colorant while the unaffected enamel doesn't get colored. The demineralized sectors get colored to different degrees depending on the degree of demineralization. Evaluation of coloring is made based on his standard typographic scale of 10 nuances of blue color.

Presently different detectors of dental caries are available: Caries Marker (Voco), Caries Detector (Cavex), Caries Finders (Ultradent), Cari-D-tect (Gresco Product Inc.), etc.

Transillumination (diafanoscopy) is based on the evaluation of intensity of cold visible light shadows (blue and green) that passes through the dental tissues. Carious maculae are characterized by irregular contours with different degrees of intensity, varying from light nuances to dark ones made clear on the generally light background of dental corona.

Illumination with ultraviolet rays is based on the luminescent property of solid dental tissues of light green color. This method is employed in a dark room with the help of device ОИ-18. The

carious process reduces the natural luminescence of solid dental tissues, being denoted by dark spots.

Caries of permanent teeth at children

At some children the incompletely erupted permanent teeth may be already affected by caries. The rapid evolution of caries may be explained by the fact that the enamel during the first year after eruption is insufficiently mineralized, containing less mineral elements and more organic compounds and being less resistant to caries. The rampant caries is explained by insufficient mineralization of solid dental tissues after eruption. Besides these, appearance of dental caries is also favored by the action of several factors: dental-maxillary anomalies, inadequate oral hygiene, excessive consumption of carbohydrates, caries of temporary teeth, etc.

In the dental fissures that are well determined during the first several years from eruption of permanent teeth, remainders of food can be retained, thus creating good conditions for the germs. This explains the high incidence of fissure caries shortly after eruption. Subsequently increases the prevalence of approximal caries.

Special attention must be paid to the first permanent molars that are also named "occlusion keys". The molars of 6 years are the first permanent teeth erupting under the prolonged influence of carious factors. Their secondary mineralization term is the longest. Out of permanent teeth at children most often are affected the molars of 6 years, named by Kunzel (1988) as "unmanageable child" of infantile stomatology. Incipient carious lesions are hard to detect in the deep and weakly mineralized fissures of occlusal surfaces. The masticatory surface remains vulnerable in the ages between 7 - 9 years while subsequently the localization of caries at this level decreases simultaneously with abrasion of occlusal surface. After 9 years, become vulnerable the mesial surfaces of first molars, as they are in contact with the

second temporary molars that are very often affected by caries or display physiological mobility. Besides retention of food remainders between these teeth, one must mention the insufficient degree of mineralization of enamel in the region of collar contact surfaces of first molars. Among all permanent teeth the first permanent molar most often displays pulpal affections with or without apical complications. Due to these considerations, it requires permanent supervision and prophylactic procedures, early diagnosing and obturation of carious defects.

Subsequently are affected the permanent superior incisors, usually in the age between 7 - 9 years. Localization of caries is predominantly symmetric on the contact surfaces and foramen caecum on the palatal surface. At children with inadequate oral hygiene and oral respiration often is affected the pre-gingival region of vestibular surfaces of permanent superior incisors.

After the age of 11-13 years the carious defects are detected on the occlusal surfaces, thereafter they appear on the contact surfaces, especially when the mesial surface of first molar is affected or the distal surfaces of canines.

Canines are relatively more resistant to caries. However, they can also be affected during the post-eruptive period, in the age of 13-14 years.

The occlusal surface of permanent second molar is affected at the age of 11-12 years, thereafter – the mesial and cervical regions are affected especially in cases of insufficient brushing from lateral zone of dental arcades.

In the case of caries of first degree (low carious activity) the carious cavity has pigmented dentine, its edges are rounded, and the altered dentine is dry and solid at probing. After preparation the floor and the walls of carious cavity can be pigmented, solid, and slightly painful at probing. The pigmented fissures are hardly opened during preparation. White maculae are not characteristic. The hygienic index is low, including children that do not clean

their buccal cavity regularly. The rate of caries is very low.

At children with third degree of carious activity (high carious activity): carious cavities have sharp edges with considerable quantity of damaged and clear dentine. Cavity walls even after preparation are soft and hardly dry. The pigmented fissures are misleading: after opening voluminous carious cavity with damaged dentine is opened. The following signs are characteristic: presence of chalky maculae with rough and soft surface that under preparation often lead to the appearance of defects. Multiplicity of affections is often observed, even groups of teeth display multiple affections, including the teeth considered most resistant (canines and inferior incisors). Secondary caries often appears. Evolution of caries is rapid and progressive, leading to complications (pulpitis, apical periodontitis, coronary fractures, etc.). The rate of caries is high: 4-5 teeth per annum.

Treatment of dental caries

Treatment of dental caries at children is not only a medical but also a social problem. Rational and timely treatment of dental caries assures the preservation of masticatory function and prevents the formation of odontogenous inflammatory processes, i.e. is a major element of secondary prophylaxis.

The existing concepts provide for an individual approach to the treatment of dental caries at children.

The tactics of treatment depends on:

1. Intensity of carious process (degrees I, II and III).
2. Deepness of solid tissue affections (incipient, superficial, medium and profound).
3. Localization of the affection.
4. Tooth development period.
5. Resistance of child's organism, immune status and general pathology.
6. Child's age, psychological particularities, etc.

Treatment of caries provides for the complex use of methods

for the obturation of carious cavities and methods increasing the resistance of solid dental tissues (endogenous and exogenous), hygiene of buccal cavity, rational nutrition, etc.

In case of caries of first degree (low carious activity) it is not necessary to implement the entire complex of curative measures. Treatment is limited to the obturation of carious cavities and hygiene of buccal cavity. Dispensary examination with treatment is performed once a year.

In case of caries of second degree (medium carious activity), besides obturation of carious cavities and hygiene of buccal cavity local and general prophylactic medications are prescribed. Dispensary examination with treatment is performed twice a year.

Caries of third degree (high carious activity) requires the entire complex of above described measures, while dispensary examination, treatment and prophylaxis are performed at least three times a year.

Treatment of incipient (maculated) caries of temporary and permanent teeth consists of local pathogenic therapy oriented towards the increase of resistance of solid dental tissues.

Treatment of incipient caries includes remineralization therapy as it is more efficient in the period of dental formation. The essence of remineralization therapy consists in the compensation of mineral elements lost due to demineralization. The main condition of remineralization therapy is preservation of enamel organic matrix.

Before performing remineralization therapy one must remove dental deposits, teeth must be treated – isolated and dried.

For remineralization fluoride compounds are used (sodium fluoride 1-2 %, tin fluoride 2-10%), amine fluorides (sol.Fluoramini 1%, Elmex Fluid, Elmex Fluorid Gelee, Wybert), fluoride varnishes (fluorlac, Fluor-Protector, Vivadent, Duraphat, Rhone Poulenc Rorer); calcium preparations (sol. Calcium gluconate 10%, sol. Calcium glycerophosphate 2.5 %), etc.

In order to increase the efficiency of remineralization therapy are recommended: treatment of enamel with dissolved solutions of acids before applying remineralization medications; reduction of pH of remineralization solution (reduction of concentration of the remineralization solution boosts up the exchange of ions in the hydroxyapatite crystals); increase of solution temperature (increase of temperature by 1^0C increases the precipitation of minerals by 1%), electrophoresis (amplifies the ion exchange).

Duration of remineralization treatment depends on the clinical evolution of demineralization nidus. In case of slow evolution 10 sessions are sufficient, while in case of progressive demineralization more than ten sessions will be required.

Monitoring of remineralization therapy is based on clinical methods (disappearance of carious maculae, appearance of gloss on dental surface) and paraclinical methods (colorimetry: with blue methylene 1%, transillumination).

Helium-neon laser can be used for the prophylaxis and treatment of incipient caries. This laser amplifies the action of cariostatic preparations, reduces solubility and permeability of enamel, increases the density of superficial stratum of enamel, activates enzymatic systems of pulp, etc. Laser rays are oriented between equator and dental collar for 2-3 seconds, consecutively to the oral and vestibular surfaces. Duration of one exposition is 60-90 seconds. In case of caries of first degree 5 sessions are indicated (daily), second degree - 2 times 10 sessions each, while for the third degree – 3 times 10 sessions each.

One must mention that remineralization therapy will be efficient only when the hygiene of buccal cavity is regular and satisfactory, when children endogenously receive preparations with Ca, F, P, etc., vitamins (A, group B, C, D) and when the nutrition regime is observed (more proteins, vitamins, mineral components and less carbohydrates), etc.

As a rule, superficial caries doesn't require operation

treatment, it is enough to polish the affected sector to obtain a smooth surface and apply remineralization therapy.

The method of solid dental tissue impregnation is widely used in the treatment of superficial caries, in some cases it is used for the treatment of medium caries of temporary teeth, especially during the period of radicular resorption. It consists of two stages: *polishing and impregnation of solid dental tissues.*

1. Polishing of affected surfaces (especially, of approximal and collar region surfaces) and edges of carious defects and fissures is performed with the help of stones or discs. Besides these, altered tissues can be removed with cutters or excavator.

2. Impregnation is performed after isolation of tooth, preparation and drying of affected surfaces. Some time ago was widely spread the "silvering" method with solution of silver nitrate of 4-30% that subsequently was reduced with sol. Hydrohinone 4% for 1-2 minutes (for the sedimentation of insoluble silver salts on the decalcinated surfaces of solid dental tissues). Presently for this scope are used the preparations with fluoride (varnishes or gels).

Treatment of medium caries of temporary and permanent teeth consists of preparation of carious cavity and its obturation with reinstatement of anatomic shape and functioning, etc.

Principles of carious cavity preparation

The main stages of the carious cavities prepation include:

1. Pain removal;
2. Opening of carious cavity;
3. Removal of affected dentine (necrotomy);
4. Prophylactic widening : practically not used for temporary and permanent teeth;
5. Formation of carious cavity;
6. Processing of carious cavity edges (finishing of walls).

General principles of the carious cavities preparation

During the preparation of carious cavities one must take into account that children get weary much quicker than adults, become agitated even if all the interventions are painless or performed under local anesthesia.

Use of turbine involves a risk of damaging the soft tissues if the patient moves unexpectedly. On temporary and young permanent teeth turbine is recommended only for opening the carious cavity (within the limits of enamel) in order to avoid complications (accidental opening, pulpal combustion, etc.).

Before preparing the carious cavity it is recommended to apply local anesthesia. For small and agitated children with multiple caries general anesthesia (narcosis) is recommended. Use of saliva aspirator and tongue holders considerably eases the operation. Mouth mirrors can also be used for a better comfort. In order to isolate a tooth or a group of teeth one can apply rubber dams or cofferdams, especially at children of higher age (4-5 years).

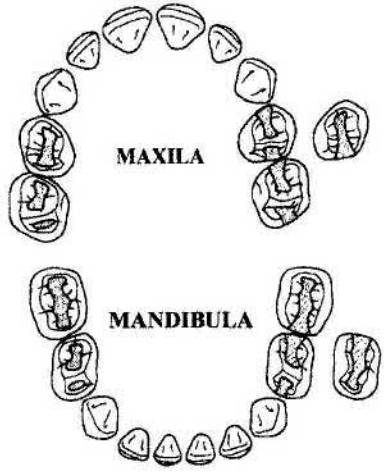


Figure 6.5. **Classical variants of preparing the cavities on temporary teeth.**

When preparing the cavities of class I by Black on the temporary and permanent teeth at children appear the need to extend the carious cavities in order to include all the fissures and holes, both carious and intact in order to prevent cariogenic situation. In such cases the carious cavity has irregular shape that assures a good fixation. In case there are sufficiently thick strata between carious cavities and masticatory surface they are prepared separately in order not to weaken the dental corona. The cavity walls must be parallel or slightly coned. The bottoms of superficial cavities must be flat, while the bottoms of profound cavities – concave (fig.6.5.).

The contour of final cavity depends on the topography and extent of carious process. Of special practical importance is the removal of carious tissues before forming the cavity (fig.6.6, 6.7).

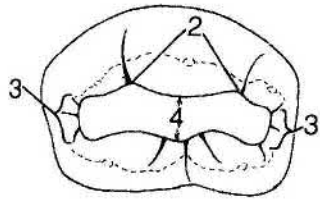


Figure 6.6. **Internal contour of class I cavity on temporary tooth (by Mathewson):**

2- preventive extension includes fissures and pits;
3 – the cavity contour is parallel to the marginal crest;
4 - widening of prepared cavity by 1/3 of the width of occlusal surface.

In particular, when preparing approximal cavities (class II Black) of temporary molars, one must create supplementary cavities on the occlusal surface in order to assure additional retention for obturation. In case the neighboring tooth is absent and sufficient quantities of tissues remain above approximal cavity when it is under equator, it is recommended to form the cavity without extension on the masticatory surface (fig.6.8.).

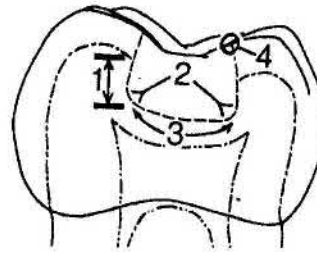


Figure 6.7. **Internal contour of class I cavity on temporary teeth (by Mathewson):**

1 - dentine is penetrated to 0.5-1 mm;
2 - internal angles are rounded, walls must be slightly convergent towards the occlusion surface;
3 – pulpal wall is slightly rounded.

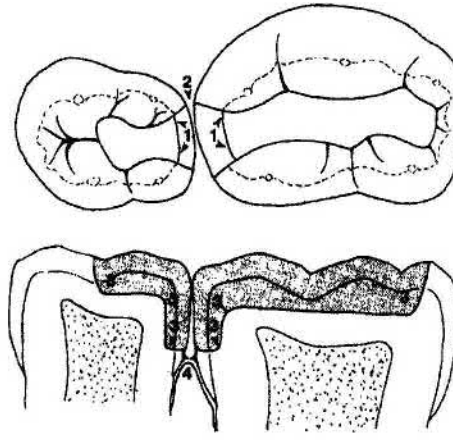


Figure 6.8. **Preparation of class II cavity on temporary teeth (by Mathewson).**

Owing to the anatomic particularities preparation of approximal cavities and pre-gingival regions of temporary frontal teeth (classes III, IV, V Black) is associated with difficulties. Compared to permanent teeth, preparation of cavities of class III

can also be performed on the vestibular surface. This improves visibility and the quality of preparation, thus allowing a better fixation of obturation that in ensemble compensates the aesthetic aspects. Cavities of class IV on temporary teeth are not prepared. In such cases it is recommended to polish the contact surfaces and apply topic fluoridation. In advanced affections covering plastic pre-fabricated coronas are recommended.

Cavities of class V are prepared classically, in some cases with formation of circular cavities. Depth of cavity must not exceed 1.5 mm. All demineralized sector must be removed.

During the preparation of carious cavities the following conditions must be observed:

1. Profound strata of damaged dentine are removed with due care in order not to allow accidental opening of pulp;
2. At profound caries the pigmented dentine can be left on the cavity floor ;
3. In cases of considerable quantities of damaged dentine remineralization therapy is performed (3-5 sessions with sodium fluoride, etc.) or tardy obturation of carious cavity: after opening and necrotomy of cavity, calcium hydroxide - based preparations are applied, or zinc-eugenol during 3-6 weeks under temporary bandage. Only after the stoppage of process (after remineralization of solid tissues and formation of tertiary dentine stratum) the cavity is shaped and definitely obturated.
4. Medicamentous treatment is done with medicamentous preparations that do not irritate the pulp (alcohol and esters, etc. are contraindicated). Choice of medications is done depending on the proximity of pulp, thin stratum of dentine with large channels compared to the less curved ones, thus creating conditions for their easy diffusion into pulp. They must not possess cytotoxic action and should be warmed up to the body temperature. Also, the air used for drying the cavities must be warm.

Hypersalivation, proximity of gums and problems with drying the operation field make the obturation of carious cavities at children especially difficult. This is why in pedodontic practice different types of aspirators, salivary screens and mini –dikes, etc. are used.

In cases of medium caries on the floor and partially on the walls of cavity it is recommended to apply a cement base:

- phosphates: phosphate cement, phosphate cement with silver, Lactodont, Visfat, Dioxivisfat, Unifas (Russia); Argil, Infantid, Adhesor (Spofa Dental), Poscal (Vovo), etc.;
- polycarboxylates: Aqualox, Carboco (Vovo); Adhesor Carbofine (Spofa Dental); Durelon (ESPE); Carboxylatzement Bayer (Bayer); Carbchem (PSP); Poly Carb (DCL); Боллокор (Стома), etc.;
- ionomers: Ionobond, Aqua Ionobond, Ionoseal (Vovo); BaseLine (Dentsply);Vitrebond (3M), etc.

In superficial medium cavities (in the proximity of enamel-dentine junction) isolation is made with resin-based varnishes (Amalgam Liner, Thermoline (Vovo), Evicrol-Varnish (Spofa Dental), etc.

It is recommended to obturate the carious cavities of temporary teeth with ionomer cements, compomer sand amalgams , and the cavities of permanent teeth - with ionomer cements, compomers and composite materials (of last generation) and less often - with amalgams.

Materials used for the obturation of carious cavities at children:

1) Cements:

- glassionomers: Ionofil, Ionofil Molar AC, Aqua Ionofil, Aqua Ionofil Plus, Argion, Argion Molar AC (Vovo); Fugi II, Fugi IX (GC), Ketac-Fil, Cheilon-Fil, Photac-Fil, Ketac-Molar (ESPE), ChemFil, ChemFil Superior, ChemFlex^{Tm} (Dentsply), Vitremer (3M), Cavitan 9Spofa Dental), etc.

2) Amalgams:

- silver : Titin Slow (Kerr), Vivacap, Amalcap Plus (Vivadent), Dispersalloy (De Try), Septalloy (Septodont), etc.;
- gallium: Галлодент (Россия), copper and gold amalgams are not produced any more.

3) Compomers: Dyract (Dentsply); Glasiosite (Vovo), Elane (Kerr), etc.

4) Plastic mass, silicate cements and composite materials are not recommended for the obturation of temporary teeth cavities as later complications appear. These materials, however, can be used for the obturation of cavities of devitalized temporary teeth.

Composite photopolymers of last generation with filled adhesive systems can be used for the obturation of cavities of temporary and permanent teeth. However, their use requires perfect isolation of operation field, strict observation of sequence stages and relatively longer time for obturation. For this reason, especially at children of preschool age the possibilities of obturating the temporary teeth with composite materials are limited.

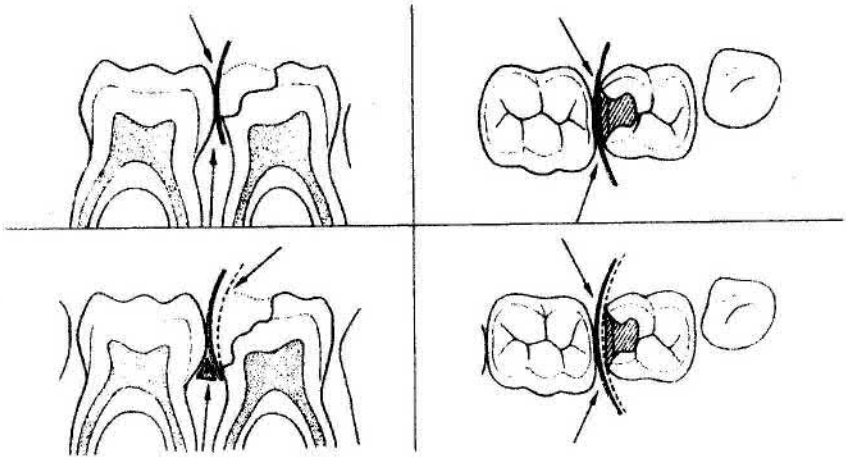


Figure 6.9. **Obturation of class II carious cavity.**

Till recently the carious cavities were prepared according to certain rules that did not take into account the action of fluoride ions. In the absence of adhesive restoration materials it was necessary to remove the entire stratum of unsupported enamel, indifferently of localization of lesion and to remove the integral dental tissues in order to make space for restoration material, thus breaking the principle of conserving the healthy dental structure.

Reproduction of initial morphology of tooth with any plastic restoration material at children is difficult, however, with the appearance of long-term enamel and dentine adhesive compounds a way was opened for the revision of cavity preparation principles. Even if the available materials are not perfect for the time being, nevertheless, they can be successfully used for the restoration of carious defects of moderate dimensions.

Reproduction of dental morphology by obturation with plastic material fully depends on the doctor's skills, while the average longevity of restorations with composite materials traditionally is 10 years. Owing to the action of fluoride ions it has become possible to limit the dimensions of cavity by restoring a part of enamel and demineralized dentine and giving a possibility for remineralization. Preservation of dental tissues increases the longevity of restorations and thus decreases the number of interventions necessary for their replacement (Mount G.J., 1999).

Changes in the understanding of the role of fluoride in the demineralization-remineralization processes and the appearance of restoration materials allowed revising the classification of carious cavities proposed by G.V. Black. Even if the principles are not fully outdated, rules of cavity preparation required revision, having become oriented towards preservation of healthy dentine structure.

Classification of dental caries (by G. J. Mount, 1999)

I. By localization

Class 1 – in pits and fissures, or defects of enamel on occlusal or other smooth surfaces

Class 2 – immediately proximally under the inter-dental contact points

Class 3 – in the cervical third of corona, or, after gingival retraction, on the naked root

This classification considers localization of caries and then their dimensioning depending on the extent of affection.

Classification is valid for both frontal and lateral teeth.

II. By dimensions

Dimension 1 – minimum affection of dentine when remineralization only is not sufficient.

Dimension 2 – moderate affection of dentine. After preparation of cavity the remaining enamel is integer, well supported by dentine. The tooth is sufficiently resistant to support restoration.

Dimension 3 – cavity enlarged. The remaining dental structure is affected, cuspids can be fractured or can cede under occlusal pressure. Cavity must be enlarged for the restoration to maintain the remaining dental structure.

Dimension 4 – extended caries with loss of a considerable portion of dental volume.

Table 6.4

Classification of cavities by G.J. Mount (1999)

Dimension	Minimum	Medium	Enlarged	Extended
Localization	1	2	3	4
Pit /fissure 1	1.1	1.2	1.3	1.4
Contact area 2	2.1	2.2	2.3	2.4
Collar 3	3.1	3.2	3.3	3.4

A cavity of dimension 1 is a new lesion, and adhesive restoration materials in such a case are the most suitable ones. A cavity of dimensions 2, 3 and 4 may be a new lesion evolved without the patient to address for treatment, or old restoration in need for replacement. In both cases the same principles of cavity preparation are recommended. The choice of the best restoration material is made depending on its properties; resistance to fractures and torsion, resistance to abrasion, etc.

- Use of composite materials is limited to their socket contraction (self-polymerrizing or photopolymerizing), presence or absence of resistant enamel that could assure a good adhesion by acid engraving;
- The main disadvantage of amalgam is its low aesthetic aspect; however, its physical properties are usually suitable for any situation;
- Glass ionomers assure a perfect adhesion both to enamel and dentine; they have good aesthetic properties but are lacking the necessary resistance for the restoration of enamel edges or occlusal surfaces (Mount G. J., 1999).

Due to the absence of adhesive restoration materials the classification of Black did not include the lesions of dimension 1 in the classes I and II. There must be made a clear distinction between the restoration of a new lesion and replacement of an old compromised restoration. In cases of new caries with active evolution the cavity must be treated in a very conservative manner making possible the remineralization of enamel and dentine. The edges must be extended only to the level of smooth surfaces capable of remineralization; "preventive extension" must be excluded. It is rational to preserve the proximal interdental contact, and the shape of cavity is determined only by the cavity itself.

In case of replacement of incorrect obturation the cavity is already prepared, being often extended excessively. For these

restorations most of the Black's principles also apply as the dental structure can not be replaced.

In order to fill a cavity, as well as to replace an incorrect restoration, one must take into consideration the limits of physical properties of the remaining permanent dental structure and of the restoration materials. A small restoration can be supported well by the remaining dental structure, especially by adhesive restoration materials. To the extent the cavity grows bigger the tooth becomes more and more unstable and straight to the point when the remaining dental structure itself has to be supported by the restoration material. In this case cavity preparation is different and another restoration material is selected (Mount G. J., Ngo H., 2001).

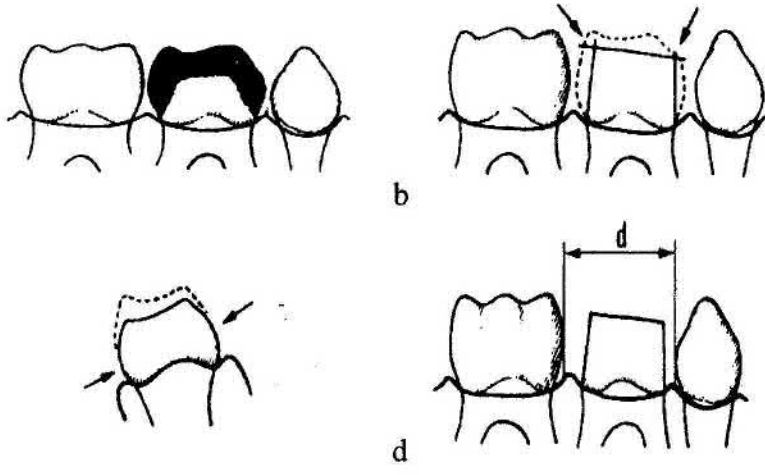
It is strongly recommended to employ the ART technique (Atraumatic Restorative Treatment), especially on temporary and young permanent teeth. The atraumatic restorative treatment consists in the removal of demineralized dental tissues exclusively by hand instruments followed by obturation of erosions and adjacent local fissures with adhesive materials. Such minimized intervention is relatively painless and has as central point the preservation of healthy solid dental tissues wherever this is possible without sacrificing the solid dental tissues for the purpose of maintaining the resistance shape for the subsequent obturation. The reconstitution of prepared lesion and obturation of fissures is performed with glass ionomers of latest generation: Fugi IX (GC), ChemFlux Tm (Dentsply), Ketac-Molar (ESPE). ART has become one of the most accessible treatment techniques and most efficient in the treatment of dental caries at children (Frencken J. et. al., 1997, 1999) (see color insert fig.6.10.).

In case of profound caries it is compulsory to apply on the floor of cavity the preparations based on calcium hydroxide or zinc-eugenol. After this, isolation material is applied and definite obturation is performed. One should apply a thin film of

preparation based on calcium hydroxide in rapid drops only on the floor of cavity in the region of pulpal horn projection. This is enough for a good curative action and will not affect the adhesion of permanent obturation.

Preparations based on calcium hydroxide used in the treatment of dental caries include: Calmecin, Calcin (Russia), Calxil (Otto); Dycal (De Trey), Life (Kerr), Reocap (Vivadent); Calcimol LC, Calcicur (Voco), etc.

Calcium hydroxide has odontotropic properties; it facilitates formation of tertiary irregular dentine. The bacteriostatic and bactericide properties of calcium hydroxide stabilize the carious process. Additionally, the calcium ions stimulate condensation of cellular pulpal membranes and reduce the excitation of nervous elements. They create an alkaline medium, reduce the permeability of capillaries, lead to the reflux of exudation and suppression of inflammatory process.



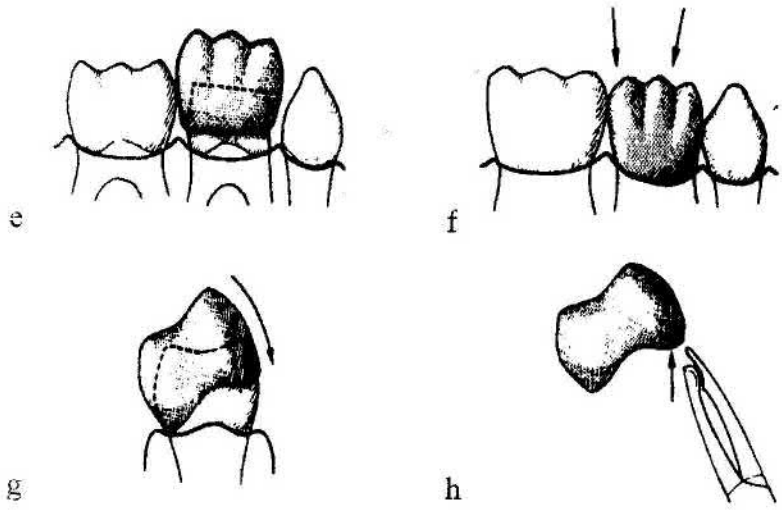


Figure 6.11. **Stages of restoring carious teeth with prefabricated coronas.**

Medications, on the basis of zinc-eugenol, act directly on pathogenic germs and their metabolism, closing them in the dentinary channels, and stopping the diffusion of toxic products, thus preventing the distribution of carious process. Preparations based on zinc-eugenol include: Cavitec (Kerr); Eugespad (SPAD); Kalsogen Plus (De Trey); Zincoxid-Eugenol Spezialpaste (Speiko), etc.

In cases of multiple or large defects in teeth it is recommended to perform reconstruction with rings, prefabricated or lab-made coronas. For lateral teeth it is recommended to use metallic prefabricated coronas with thickness of 0.1-0.2 mm, while for frontal teeth - prefabricated plastic coronas or coronas made of composite materials. Correct adaptation of such coronas on teeth requires minimum preparations of solid dental tissues: usually are processed the contact and occlusal surfaces within the

limits of enamel. Adapted coronas are fixed with ionomer cements.

General treatment of dental caries (pathogenic therapy)

General pathogenic treatment of caries presumes normalization of metabolic processes, first of all, increase of non-specific resistance of organism against the general pathogenic factors. General pathogenic treatment consists of therapy of somatic diseases that cause the progress of carious process, rational nutrition, observation of day regime, identification and removal (or suppression of actions) of factors reducing the resistance of organism, indication of individual treatment in cases of necessity.

General treatment of dental caries comprises an entire complex of measures:

1. Action on the principal causal factor (microorganisms, treatment of somatic diseases);
2. Avoidance of cariogenic situation in the organism;
3. Stimulation of reactivity (immune-biological capacity) of organism.
4. Increase of resistance of solid dental tissues against the action of cariogenic factors.

The most frequent causes of reduction of immune-biological reactivity at children are the diseases of allergic and infectious etiology, colds, gastrointestinal diseases, metabolic diseases, etc. The key point of the pathogenic therapy of caries at children consists in the prophylaxis of formation of infection nidus and prevention of intoxication, detection and timely treatment of systemic pathology by different specialists (pediatrician, endocrinologist, neurologist, etc.).

A special role in increasing the resistance of organism, metabolic regulation, recovery of functions and systems, remineralization of solid dental tissues is played by rational

nutrition. The child's organism is very sensitive to nutrition deficits. The diet must be balanced and individualized not only calorically, but also by the content of proteins, lipids, carbohydrates, mineral substances and vitamins.

The complete nutrition assumes the daily average consumption of 80-100 g proteins, 400-500 g carbohydrates, 80-100 g lipids (including 10 % vegetal oils), around 0.1 g vitamins, 20 g mineral salts (around 10 g table salt).

Dental caries is accompanied by reduction of macro- and microelements (calcium, phosphorus, iron, manganese, etc.) in the solid dental tissues. Due to this reason they must be introduced into the organism in the form of supplements. Formation of bone and dental tissues at children requires ions of calcium, phosphorus and fluoride. According to the national and international standards the nictimeral requirement of calcium is 400-1000 mg. Newborn children have considerable quantities of cartilage tissues, simultaneously in the organism they have 20-30 g of calcium acquired from mothers. The requirement of a new born child for calcium is 3 times greater compared to the requirement of fetus, while at sucker babies this requirement is even greater.

The quantity of calcium necessary for the normal activity of organism proposed by E.P. Lazzari (1968) is presented in table 6.5.

Table 6.5

Organism's calcium requirement (Lazzari E.P.)

	Children	Adolescents	Adults
Calcium	0,8 g	1,3-1,4 g	0,8 g
Equivalent in milk	3 glasses (0,9g)	4 glasses (1,2 g)	3 glasses (0,9g)

During pregnancy, lactation and adolescence the requirement for calcium increases by 0.4-0.5 g. High levels of calcium are

contained in milk and dairy products, egg yolk, vegetables, nuts, fish, beans, etc.

Phosphorus stimulates the growth and development of bones and positively affects the haemopoiesis. The daily requirement for phosphorus is 1.5-2 g. Phosphorus in relatively high levels is found in meat, fish and vegetables.

One of the endogenous methods of prophylaxis of caries consists in the fluoridation of potable water, milk, table salt, bread, etc. (when concentration of fluoride in potable water in the local water supply system is below 0.5 mg/l).

The children's ration requires proteins of animal (meat, fish, eggs, cheese) and vegetal origin (beans, peas, etc.). These products also contain sufficient quantities of amine acids. Proteins of animal origin should occupy 50-60 % of total protein quantity consumed by children of school age.

Nutrition rich in vitamins A, B_1, B_6, D, C is recommended. Products must not be subjected to prolonged thermal treatment.

Excessive content of carbohydrates in food favors the appearance of dental caries. Not only the quantity but also the frequency of their consumption is important. The more often carbohydrates are consumed, the longer they remain on the surface of teeth and the greater is the possibility of synthesis of acids leading to demineralization of enamel (Muhlmann K., 1981; Silverstone N., 1981). Consumption of sweets must be allowed only during the main meals and followed by oral hygienic procedures. Between meals sweets must be prohibited. One must limit the consumption of sticky sweets (bonbons, caramel, biscuits, etc.) and of some drinks (with pH varying between 2,0 and 6,3) that are frequently consumed in big quantities , especially by adolescents, thus provoking erosions of solid dental tissues and caries (juices with added sugar, natural apple juice, orange juice, lemon juice, Coca-Cola, mineral waters with carbon dioxide, etc.).

General (endogenous) treatment of dental caries at children presumes administration of some medications. Among *medications containing calcium*, can be recommended:

1. Calcium gluconate 0.3-0.5 g (one tablet) 3 times a day. Depending on age, may be administered: up to 1 year – 0.5 g, 2-4 years – 1 g, 5-6 years – 1.5 g, 7-9 years - 1.5-2g, 10-4 years – 2.3 g a day.
2. Calcium glycerophosphate, 0.25-0.5 g 3-5 times a day;
3. Calcium lactate 0.5-1 g 2-3 times a day.

Phosphorus preparations are administered in the following manner:

1. Calcium glycerophosphate, 3 g 3 times a day, or in granules, ½ teaspoon 2-3 times a day;
2. Phytine or geophytine, 0.25 g (one tablet) 3 times a day.

In the general treatment of dental caries at children most efficient are the preparations containing fluoride They are administrated endogenously in the form of solutions that may also contain vitamins (Videnol, Vitaflor, Vidaline), pastilles (Zymafluor, Dentaflux, Osteofluor, etc.).

Endogenous administration of fluoride preparations to children must be adjusted depending on the concentration of fluoride in potable water. L.M. Silverstone (1978) recommends the following doses of fluoride (table 6.6).

Table 6.6

Recommended fluoride supplements

Concentration of fluoride in water, mg/l	Ages of children				
	0-6 months	6-18 months	18-36 months	3-6 years	Above 6 years
0.2	0	0.25	0.5	0.75	1.0
0.2-0.4	0	0	0.25	0.5	0.75
0.4-0.6	0	0	0	0.25	0.5
0.6-0.8	0	0	0	0	0.25
0.8	0	0	0	0	0

Daily endogenous administration of fluoride preparations is recommended for 250 days under the supervision of adults, medical or didactic personnel. During the summer time administration of these preparations must be avoided as the water consumption and thus the influx of fluoride increase. To children of pre-school age administration of fluoride preparations is recommended once a day in liquid form (solutions or dissolution of tablets in tea, milk, etc.) while to the children of school age it is recommended to masticate the tablets or suck them in oral cavity.

Control questions and topics:

1. Definition of dental caries
2. Indexes recommended by WHO for the appreciation of dental caries.
3. Prevalence index of dental caries.
4. Intensity index of dental caries.
5. Rate of caries.
6. What are the major factors in the appearance of dental caries?
7. Describe the favoring factors for the appearance of dental caries during the antenatal period.
8. Describe the favoring factors for the appearance of dental caries during the first year of life.
9. Describe the favoring factors for the appearance of dental caries at children and adolescents.
10. Classification of caries of temporary and permanent teeth.
11. General evolution particularities of temporary teeth caries.
12. Bottle caries.
13. Clinical signs of slow evolution of dental caries at children.
14. Diagnosing caries of temporary teeth.
15. General particularities of evolution of caries of permanent teeth at children.
16. Particularities of diagnosing caries of permanent teeth at children.

17. Carious activity of degree I at children.
18. Carious activity of degree II at children.
19. Carious activity of degree III at children.
20. On what factors does the treatment tactics of dental caries depend?
21. Treatment of carious activity of degree I at children.
22. Treatment of carious activity of degree II at children.
23. Treatment of carious activity of degree III at children.
24. Treatment of incipient caries of temporary teeth and permanent ones at children.
25. Treatment of superficial caries of temporary and permanent teeth at children.
26. Treatment of medium caries of temporary and permanent teeth at children.
27. Treatment of profound caries of permanent teeth at children.
28. Particularities of preparing carious cavities on temporary teeth.
29. Particularities of preparing carious cavities on permanent teeth.
30. Obturation materials for medium caries cavities on temporary teeth.
31. Obturation materials for medium caries cavities on permanent teeth.
32. ART technique.
33. Pathogenic therapy of dental caries at children.
34. Treatment of bottle caries.

CONTROL TESTS

Simple compartment

1. The caries prevalence index represents:
   A. Percent of teeth affected by caries;
   B. Sum of teeth: affected by caries –C, obturated – O,

extracted – E;

C. Percentage of persons affected by caries;

D. Sum of teeth affected by caries, pulpitis and apical periodontitis;

E. Number of appearances of new carious cavity at a person during a certain time span.

2. The dental caries intensity index represents:

A. Sum of teeth affected by caries, pulpitis and apical periodontitis;

B. Sum of teeth: affected by caries –C, obturated – O, extracted – E;

C. Percentage of teeth affected by caries;

D. Percentage of persons affected by caries;

E. Number of appearances of new carious cavity at a person during a certain time span.

3. The value of COE index represents:

A. Prevalence of dental caries;

B. The gravidity and activity of process;

C. Stoppage of carious process;

D. Percent of complications of dental caries;

E. Percentage of persons affected by caries;

4. Rate of caries represents:

A. Percentage of teeth affected by caries;

B. Percentage of persons affected by caries;

C. Sum of teeth affected by caries, pulpitis and apical periodontitis;

D. Sum of teeth: affected by caries –C, obturated – O, extracted – E;

E. Dynamics of the index of dental caries intensity during a certain period of time.

5. The main cause of focal demineralization is:

A. Microorganisms in dental deposits (dental plaque);
B. Refined hydrocarbons;
C. Endotoxins of microorganisms from dental deposits (dental plaque);
D. Organic acids resulting from the vital activity of microorganisms contained in the dental deposits (dental plaque).

6. Caries of Class I, by Black, represents the lesion of fissures and pits of occlusal surfaces of:
A. Temporary molars;
B. Premolars;
C. Molars and premolars;
D. Molars, premolars, vestibular surfaces of molars and palatal surfaces of incisors;
E. Temporary and permanent molars.

7. Caries of Class II, by Black, represents the lesion of proximal surfaces of:
A. Temporary and permanent molars.
B. Molars and premolars;
C. Incisors, molars and premolars;
D. Molars, premolars and canines;
E. Temporary and permanent incisors.

8. Caries of Class III, by Black, represents the lesion of proximal surfaces of:
A. Temporary and permanent incisors.
B. Molars and premolars;
C. Incisors, with preservation of incisal edge;
D. Incisors, with loss of incisal edge;
E. Incisors, molars and premolars;

9. Caries of Class IV, by Black, represents the lesion of proximal surfaces of:

A. Incisors, molars and premolars;
B. Incisors, with preservation of incisal edge;
C. Incisors, with loss of incisal edge;
D. Temporary and permanent incisors.
E. Canines and premolars.

10. Class V of caries by Black represents the lesion of:
A. All surfaces of molars;
B. Proximal surfaces of molars and premolars;
C. Vestibular surfaces at the cervical level of all teeth;
D. Vestibular surfaces of all teeth;
E. Vestibular surfaces at the cervical level of incisors and canines.

11. The main criterion for the appreciation of carious process in classification by Виноградова T. is:
A. Localization;
B. Activity;
C. Deepness;
D. Succession of appearance;
E. Pathological morphology.

12. The criteria for the appreciation of dental caries by Виноградова T. are:
A. Localization;
B. Activity;
C. Deepness;
D. Succession of appearance;
E. Pathological morphology;
F. All answers are correct.

13. The value of dental caries intensity index at children with reduced degree of carious activity aged between 3 and 6 years, is:
A. 0;
B. 1;

C. 2;
D. <2;
E. <3.

14. The value of dental caries intensity index at children with reduced degree of carious activity aged between 7 and 10 years, is less than:
A. 2;
B. 3;
C. 4;
D. 5;
E. 6.

15. The value of dental caries intensity index at children with reduced degree of carious activity aged between 11 and 14 years, is less than:
A. 2;
B. 3;
C. 4;
D. 5;
E. 6.

16. The value of dental caries intensity index at children with reduced degree of carious activity aged between 15 and 18 years, is less than:
A. 2;
B. 3;
C. 4;
D. 5;
E. 6.

17. The value of dental caries intensity index at children with medium degree of carious activity aged between 3 and 6 years, is:
A. 1-4;
B. 2-5;

C. 3-6;
D. 4-7;
E. 5-8.

18. The value of dental caries intensity index at children with medium degree of carious activity aged between 7 and 10 years, is:

A. 3-6;
B. 4-7;
C. 5-8;
D. 6-8;
E. 7-9.

19. The value of dental caries intensity index at children with medium degree of carious activity aged between 11 and 14 years is:

A. 3-6;
B. 4-7;
C. 5-8;
D. 6-8;
E. 7-9.

20. The value of dental caries intensity index at children with medium degree of carious activity aged between 15 and 18 years is:

A. 3-6;
B. 4-7;
C. 5-8;
D. 6-8;
E. 7-9.

21. The value of dental caries intensity index at children with high degree of carious activity aged between 3 and 6 years exceeds:

A. 4;
B. 5;

C. 6;
D. 7;
E. 8.

22. The value of dental caries intensity index at children with high degree of carious activity aged between 7 and 10 years exceeds:
A. 6;
B. 7;
C. 8;
D. 9;
E. 10.

23. The value of dental caries intensity index at children with high degree of carious activity aged between 11 and 14 years exceeds:
A. 6;
B. 7;
C. 8;
D. 9;
E. 10.

24. The value of dental caries intensity index at children with high degree of carious activity aged between 15 and 18 years exceeds:
A. 6;
B. 7;
C. 8;
D. 9;
E. 10.

25. Under transillumination the demineralization nidus is marked by:
A. Light spots on the dark background of intact dental tissues;
B. Dark spots on the light background of intact dental tissues.

26. Until the age of 3 years dental caries is most often found:
A. In the pits and fissures of molars;

B. In cervical region of incisors;
C. On the proximal surfaces of molars;
D. On the proximal surfaces of incisors;
E. On all surfaces of teeth.

27. At the age of 3-4 years dental caries is most often found:
A. In the pits and fissures of molars;
B. In cervical region of incisors;
C. On the proximal surfaces of molars;
D. On the proximal surfaces of incisors;
E. On all surfaces of teeth.

28. After the age of 4 years dental caries is most often found:
A. In the pits and fissures of molars;
B. In cervical region of incisors;
C. On the proximal surfaces of molars;
D. On the proximal surfaces of incisors;
E. On all surfaces of teeth.

29. First affected by caries are the following temporary teeth:
A. Superior incisors;
B. Inferior incisors;
C. First molars;
D. Second molars;
E. Canines.

30. Among the temporary teeth most resistant to dental caries are:
A. Superior incisors;
B. Inferior incisors;
C. First molars;
D. Second molars;
E. Canines.

30. The order of affection of temporary teeth by caries is the following:

A. Inferior incisors, superior incisors, first molars, second molars, canines;
B. First molars, second molars, superior incisors, inferior incisors, canines;
C. Superior incisors, inferior incisors, first molars, second molars, canines;
D. Superior incisors, first molars, second molars, canines, inferior incisors;
E. Superior incisors, first molars, second molars, inferior incisors, canines.

32. Evolution of dental caries at children is characterized by the following particularities:
A. Multiple character of affection;
B. Symmetric character of affection;
C.Clinical evolution is weakly pronounced, especially during the radicular resorption period;
D. Circular caries;
E. All answers are correct.

33. Out of all forms of temporary teeth caries at children most often is met:
A. Profound caries;
B. Incipient (maculated)caries;
C. Medium caries;
D. Superficial caries;
E. All forms in equal proportions.

34. Profound caries of temporary teeth is complicated:
A. Frequently – by inflammatory process in pulp;
B. Rarely - by inflammatory process in pulp;
C. In all cases - by inflammatory process in pulp;
D. Inflammatory process in pulp is never attested;
E. Frequently - by inflammatory process of apical

periodontium.

35. The cervical region of temporary teeth is frequently affected by caries, as:
   A. The enamel is weakly mineralized;
   B. Retention of food remainders favors the formation of dental deposits;
   C. The enamel there is thinner.
   D. The enamel is weakly mineralized and retention of food remainders favors the formation of dental deposits;
   E. The enamel is weakly mineralized, retention of food remainders favors the formation of dental deposits, and enamel is thinner.
36. Dental fissures at children are often affected by caries due to:
   A. Incomplete mineralization;
   B. Retention of food remainders;
   C. Open fissures, incomplete mineralization, retention of food remainders;
   D. Thinner enamel;
   E. Weakly mineralized enamel and dentine, thinner enamel not resisting to pressure during mastication.

37. Florid evolution of dental caries at children is characterized by:
   A. Extended decalcification in surface and in depth;
   B. Affection of certain groups of teeth;
   C. Rapid progress;
   D. Affection of several surfaces of teeth;
   E. All answers are correct.

38. For superficial caries are characteristic the pains provoked by:
   A. Thermal excitation;
   B. Excitation by sweet and salty food;
   C. Chemical, mechanical and thermal excitation.

39. For medium caries are characteristic the pains:
    A. Short pains caused by mechanic, chemical and thermal excitation;
    B. Short pains caused by chemical excitation;
    C. Long lasting pains after removal of mechanical, chemical and thermal excitation;
    D. Spontaneous pains, caused by different excitation factors;
    E. Pains are not characteristic.

40. For profound caries are characteristic the pains:
    A. Long lasting pains caused by mechanic, chemical and thermal excitation;
    B. Short pains caused by chemical excitation;
    C. Pains caused by mechanical, chemical and thermal excitation, disappearing soon after removal of excitation factors;
    D. Spontaneous pains, caused by different excitation factors;
    E. Pains are not characteristic.

41. The treatment tactics of caries at children depends on:
    A. Intensity of carious process;
    B. Deepness and localization of carious process;
    C. Age and psychological particularities of the child, tooth development period, general pathology;
    D. Tooth development period;
    E. All above answers are correct.

42. Treatment of dental caries at children with reduced carious activity consists of:
    A. Preparation and obturation of carious cavity;
    B. Preparation and obturation of carious cavity, hygiene of buccal cavity;
    C. Remineralization therapy;
    D. Tardy obturation;

F. Endogenous therapy of dental caries.

43. Treatment of dental caries at children with medium carious activity consists of:

A. Preparation and obturation of carious cavity, hygiene of buccal cavity;
B. Remineralization therapy;
C. Tardy obturation;
D. Endogenous therapy of dental caries;
E. All answers are correct.

44. Treatment of dental caries at children with high carious activity consists of:

A. Preparation and obturation of carious cavity, hygiene of buccal cavity;
B. Remineralization therapy;
C. Tardy obturation;
D. Endogenous therapy of dental caries;
E. All answers are correct.

45. The first stage of carious cavity preparation is:

A. Opening of carious cavity;
B. Formation of cavity;
C. Processing of cavity edges;
D. Pain removal;
E. Removal of affected tissues (necrotomy).

46. The order of performing main operations for the preparation of carious cavity is the following:

A. Opening of carious cavity, removal of pain, removal of affected tissues (necrotomy), formation of cavity, processing of cavity edges;
B. Removal of pain, opening of carious cavity, removal of affected tissues (necrotomy), formation of cavity, processing of cavity edges;

C. Removal of pain, processing of cavity edges, opening of carious cavity, removal of affected tissues (necrotomy), formation of cavity;
D. Opening of carious cavity, removal of pain, formation of cavity, removal of affected tissues (necrotomy), processing of cavity edges;
E. Processing of cavity edges, opening of carious cavity, removal of pain, removal of affected tissues (necrotomy), formation of cavity.

47. The most efficient obturation of tooth 21 (class IV) at a child of 12 years is:
A. Zinc-phosphate cement, microfilament chemical composite compound;
B. Helium - composite micro-hybrid with adhesive system of generation IV or V;
C. Ionomer cement, microfilament chemical composite compound;
D. Ionomer cement, compomer with adhesive system of generation IV or V;
E. Ionomer cement.

48. The most efficient obturation of a medium acute caries of class V at a child of 14 years with a good hygienic index of oral cavity is:
A. Ionomer cement, microfilament chemical composite compound;
B. Compomer with adhesive system of generation IV or V;
C. Ionomer cement;
D. Polycarboxylate cement, hybrid helium compound.

49. The most efficient obturation of medium chronic caries of tooth 21 (class IV) at a child of 12 years with a good oral hygiene index is:

A. Zinc-phosphate cement, microfilament chemical composite compound;
B. Ionomer cement, helium - composite micro-hybrid;
C. Polycarboxylate cement, compomer;
D. Ionomer cement;
E. Helium - composite micro-hybrid with adhesive system of generation IV or V.

50. Obturation of medium carious cavity in the central fissure of the tooth 36 is better to be performed with:
A. Amalgam with packing;
B. Zinc-phosphate cement, microfilament chemical composite compound;
C. Zinc-phosphate cement, silica-phosphate cement;
D. Helium - composite micro-hybrid with adhesive system of generation IV or V;
F. Ionomer cement.

51. Obturation of a fissure with signs of enamel destruction at a child of 8 years requires:
A. Non-invasive sealing of fissure;
B. Remineralization therapy with subsequent non-invasive sealing of fissure;
C. Application of fluoride varnish;
D. Invasive sealing of fissure with helium-composite micro hybrid;
E. Application of fluoride gel.

52. The most rational method of filling a medium caries on the medial and masticatory surface that communicates between them in the tooth 36 of a child in the age of 14 years is:
A. Joining of cavities, polycarboxylate cement, and helium composite micro-hybrid;
B. Amalgam with packing of ionomer cement;

C. Tunnel obturation with packing of ionomer cement, helium composite micro-hybrid for molars;
D. Tunnel obturation with cermetic cement.

53. In the obturation of carious cavities of class V the best results are assured by:
A. Ionomer cements;
B. Composites of classes I-II;
C. Composites of class III;
D. Composites of classes IV-V;
E. Compomers.

54. Helium - composite micro-hybrids with adhesive system of generation IV or V are indicated for the obturation of medium carious cavities of permanent teeth at children with:
A. Classes I-II;
B. Class III;
C. Class IV;
D. Class V;
E. Classes I-V.

Multiple compartment

55. Medium acute caries is characterized by the following clinical signs:
A. Wide entrance into the carious cavity;
B. Solid dentine;
C. Affection of a big number of teeth;
D. Narrow entrance into the carious cavity;
E. Unitary affection (affected is a single tooth);
F. Damaged dentine;
G. Demineralization of enamel.

56. The supplementary methods of diagnosing dental caries at children include:
A. Vital coloration;

B. Федоров-Володкин hygienic index;
C. Transillumination;
D. RAS test (acid resistance of enamel);
E. Thermal diagnostics;
F. Radiography;
G. Probing;
H. Percussion.

57. For incipient caries the following symptoms are characteristic:
A. Nacreous color of macula.
B. Irregular contours of nidus;
C. Localization on tubercle or closer to the incisive edge;
D. Higher permeability for colorants;
E. Green color in the Wood's rays.
58. Etiotropic therapy of dental caries is oriented towards:
A. Increase of solid dental tissue resistance and improvement of regeneration capacities;
B. Removal of non-vital enamel and dentine;
C. Increase of organism's resistance;
D. Assurance of cavity isolation from oral environment.

59. Pathogenic therapy of dental caries is oriented towards:
A. Increase of solid dental tissue resistance and improvement of regeneration capacities;
B. Removal of non-vital enamel and dentine;
C. Increase of organism's resistance;
D. Assurance of cavity isolation from oral environment.

60. Treatment of incipient caries (chalky maculae):
A. Removal of dental plaque;
B. Preparation of affected enamel;
C. Application of fluoride varnish;
D. Electrophoresis with Ca++;
E. Application of calcium gluconate solution;

F. Application of sodium fluoride solution;
G. Application of calcium gluconate and sodium fluoride solution.

61. Treatment of profound caries of a permanent tooth requires application of curative packing with:
A. Zinc-eugenol paste;
B. Resorcin – formalin paste;
C. Calcium hydroxide based paste;
D. Not indicated;
E. Indicated only in the period of dental root formation.

62. The essence of remineralization therapy consists in:
A. Mineralization of apatite crystals of enamel;
B. Formation of fluorapatite and hydroapatite crystals;
C. Process opposite to demineralization;
D. Incorporation of ions from the buccal liquid into the network of crystals with vacant places;
E. Incorporation of ions into the network of crystals with vacant places.

63. Efficiency of remineralization therapy in the treatment of dental caries can be appreciated by:
A. Disappearance of carious maculae;
B. Appearance of gloss on the previously affected surface;
C. Colorimetric method;
D. Drying of surface;
E. Electroodontodiagnosis.

64. Remedies for the remineralization of enamel are the following:
A. Calmecine;
B. Calcine;
C. Sol. Calcium gluconate 10 %
D. Sodium fluoride 2 %;

E. Sol. Vitaftor.

65. Remedies for the remineralization of enamel are the following:
A. Varnish with fluoride;
B. Gel with fluoride;
C. Sodium chloride solution 2 %;
D. Varnish: Fluorprotector (Vivadent);
E. Aminefluoride 1%.

66. Remedies for the remineralization of enamel are the following:
A. Calcium glycerophosphate - tablets 0,5;
B. Sodium chloride 2 %
C. Sodium fluoride 2 %;
D. Chlorhexidine 0,06 %;
E. Varnish: Duraphat.

67. The pneumatic tool (turbine):
A. Can be used at all the stages of preparation of medium carious cavities of temporary teeth;
B. Can be used at all the stages of preparation of medium carious cavities of permanent teeth at children;
C. Is recommended for the opening of medium carious cavities of temporary teeth;
D. Is recommended for the opening of medium carious cavities of permanent teeth;
E. Is recommended only for the preparation of medium carious cavity walls and creation of accessory cavities.

68. In case of caries of IIIrd degree of activity is recommended:
A. Obturation of all carious cavities with amalgam in a single visit;
B. Tardy obturation of carious cavities;
C. Obturation of carious cavities after remineralization

therapy.

D. Obturation of all carious cavities with composite compound in a single visit;
E. Depulpation of teeth, as caries is frequently complicated with chronic inflammatory processes.

69. Medicamentous treatment of prepared carious cavities in temporary teeth can be performed with:

A. Alcohol, ester;
B. Proteolytic ferments;
C. Chlorhexidine 0,06 %;
D. Furacilline 1:1000;
E. Antibiotics, sulfanilamide.

70. Materials used for the definite obturation of carious cavities of temporary teeth are the following:

A. Silicine;
B. Silidont;
C. Fugi IX;
D. Noracryl;
E. Concise.

71. Materials used for the definite obturation of carious cavities of temporary teeth are the following:

A. Silver amalgam;
B. Lactodont;
C. Dycal;
D. Evicrol;
E. Glass-ionomer cement: Fugi IX.

72. Materials used for the definite obturation of carious cavities of temporary teeth are the following:

A. Infantid
B. Acryloxide;
C. Copper amalgam;

D. Life;
E. Galodent.

73. Materials used for the definite obturation of carious cavities of permanent teeth are the following:
A. Silver amalgam;
B. Compomers;
C. Life (Kerr);
D. Zinc-eugenol;
E. Glass-ionomer cement: Fugi IX.

74. Materials used for the definite obturation of carious cavities of permanent teeth are the following:
A. Stomalgine;
B. Carbodent;
C. Silver amalgam;
D. Caprofer;
E. Galodent.

75. Materials used for the definite obturation of carious cavities of permanent teeth are the following:
A. Point 4;
B. Silidont;
C. Phosphate cement;
D. Dyrect;
E. Concise.

Correct answers:

1. C.
2. B.
3. B.
4. E.
5. D.
6. D.
7. B.
8. C.
9. C.
10. C.
11. B.
12. F.
13. C.
14. D.
15. D.
16. A.
17. C.
18. D.
19. C.
20. E.
21. D.
22. D.
23. D.
24. D.
25. B.
26. B.
27. A.
28. C.
29. A.
30. B.
31. D.
32. E.
33. C.
34. A.
35. E.
36. C.
37. E.
38. B.
39. A.
40. C.
41. E.
42. B.
43. E.
44. E.
45. D.
46. B.
47. B.
48. B.
49. E.
50. D.
51. D.
52. D.
53. E.
54. E.
55. C, D, F, G.
56. A, C, E, F.
57. B, D, E.
58. B, D.
59. A, C.
60. A, C, D, E, F, G.
61. A, C.
62. C, E.
63. A, B, C, D.
64. C, D.
65. A, B, D, E.
66. C, E.
67. C, D.
68. B, C.
69. C, D.
70. B, C.
71. A, B, E.
72. A, C, E.
73. A, B, E.
74. C, E.
75. A, B, D, E.

7 AFFECTIONS OF PULP AT CHILDREN

In peridontics inflammatory processes are a basic problem. It is actualy due to the fact it is determined by a rise in the number of affected persons, increased aggravation of clinical evolution, complications and difficulty of treatment.

Structural particularities of dental pulp at children

Evolution of inflammatory process in the pulp depends on the physiological and morphological characteristics of different development periods.

For the temporary teeth one can distinguish the following development periods: I – formation of functional activity of pulp (during the formation of dental root); II - functional maturing (stabilization of already formed root); III - regression of functional properties (in the period of radicular resorption).

For the permanent teeth the following development periods are characteristic:

I – formation of functional activity of pulp (during the formation of dental root);

II - functional maturing (already formed root);

III - regression of functional properties (in the period of root resorption).

The pulp of temporary teeth has the following anatomic properties, compared to the one of permanent teeth: tooth cavity and the radicular channels are larger, pulpal horns are more pronounced; the apical orifice is larger, accessory radicular channels are more frequently observed; coronary and radicular dentine channels are shorter, larger and less sinusoidal.

During the first period after the eruption of temporary teeth the pulp is lax, massive and morphologically similar to the embryonic tissue, with pronounced plastic properties, with good vascularity. The peripheral stratum of pulp contains 3-4 layers of young odontoblasts with weak differentiation. The middle layer contains numerous mesenchymatous cells with slight differentiation: fused, flat, etc. Collagen fibers are practically absent.

In the second period, the pulp of temporary teeth is characterized by a well-contoured peripheral stratum with odontoblasts on the entire surface. In the pulp of temporary teeth is formed of secondary dentine, its structure being more regular compared to the one of permanent teeth. The sub-odontoblastic stratum and the central one contain a big number of mature conjunctive cells: pulpocytes, fibroblasts, histiocytes, plasmocytes, lymphocytes and monocytes. In result of activity of fibroblasts appear the fundamental substance and the collagen fibers. The pulp is characterized by a network rich in sanguine vessels, nervous fibers and lymphatic vessels. The pulp of temporary teeth, in contrast to that one of permanent ones, contains nervous cells (Sarbu S., 1967). A big number of neural receptors are situated in the sub-odontoblastic stratum.

The apical orifice and the large radicular channels assure the tight connection of the temporary teeth pulp with the periodontium, thus facilitating the rapid transmission of inflammatory process from pulp into the periodontium.

Third period - regression of functional properties of temporary teeth pulp - it is characterized by the beginning of radicular resorption by the pulp and its gradual transformation into granular tissue. Involution processes are characteristic for the pulp: the number of cells decrease and the quantity of collagen fibers grows higher, simultaneously takes place the dehydration of fundamental substance. Dystrophic processes are characteristic

for the pulp in this stage. A part of sanguine vessels and nervous fibers disappears. Reactivity of pulp decreases; plastic and protective functions are inhibited. The capacity to produce reaction dentine is very limited. However, dentinary sclerosis (obliteration of dentinary channels) becomes more intensive.

The structure of pulp of temporary teeth at different development stages considerably influences the installation and evolution of inflammatory process mechanisms. For the inflammation of temporary and permanent teeth pulp are characteristic alteration, exudation, metabolic disorders and proliferation. First let us consider the alteration sustained also by humoral mediators: calicrein- quinine system, complementary, blood coagulation, etc. and cellular ones (leukocytes, lymphocytes, thrombocytes, labrocytes, etc.). Acute states evolve progressively with very rapid change of vessel dilation and stasis phases and appearance of exudation due to the diapedesis of neutrophils and eusinophils, leading to the formation of microabscesses in the entire pulp. To the extent of progress of radicular rhizalysis the equilibrium between the defense factors and the pathologic ones is disturbed, leading to a diffuse purulent process (phlegmon). Simultaneously radicular resorption can compensate this, as upon enlargement of apical zone (terminal) the inflammatory process moves to the periapical zone, influencing the inter-pulpal vascular reactions and leading to the appearance of a chronic inflammatory process.

The pulp of young permanent teeth with incompletely developed roots represents a lax conjunctive tissue on the stage of maturing, with a very high biological potential determined by the mesenchymal structure of embryonic character.

The odontoblastic stratum of pulp of a young permanent tooth is established of several uninterrupted rows of highly specialized cells – odontoblasts that are interlaced with nervous terminals.

The sub-odontoblastic stratum (the Weil zone) contains

intermediary cells between fibroblasts and odontoblasts that participate in the formation of collagen fibers and fundamental substance. They can differentiate in odontoblasts, but in case of lesion of odontoblastic stratum they can elaborate elements of reaction dentine matrix.

The central stratum (the cellular zone) is rich in mesenchymal non-differentiated cells. The number of fibroblasts is very big compared to the number of collagen fibers. Specialized defense cells can transform rapidly into macrophages, plasmocytes and dentinoclasts. The fundamental substance is formed of highly polymerized molecules: mucoproteins glucoproteins, and mucopolysaccharides, being also characterized by a high degree of hydration. This allows for the penetration of nutrients and rapid elimination of inflammatory process products, assuring the preservation of constant inter-pulpal pressure owing to the wide dentinary channels and radicular channel. As the radicular apex is not formed or the apical foramen is open wide, the pulp is not in the situation of a tissue concealed in a cavity with non-extensible walls. A network of divaricated sanguine vessels assures the cellular metabolism and preserves the dentine formation capacity, the anti-infectious and anti-inflammation reactions.

The pulp from the apical region is also represented by some tissue with germination properties that is responsible for the formation of root and apical region of the dental root. The growth zone is composed of non-differentiated cells and specialized ones, it has a developed capillary network that assures the multiplication and cellular transformation processes, including dentinogenesis (root formation). The cellular growth zone has a very large contact surface between the pulpal tissues and the periapical ones, while the passage zone is represented to an equal extent by the pulpal elements and periapical conjunctive tissues that can be replaced and functionally compensated. This determines some particularities of inflammatory processes in this

zone:

a) the pulpal tissue is not subjected to obvious intra-pulpal pressure during the evolution of inflammatory process, the exudation of inflammatory process in pulp can extend via the wide dentinary channels and via the terminal zone of roots into the periradicular tissues;

b) abundance of young cellular elements capable to differentiate in case of necessity assures the autonomy of this region and facilitates regeneration of tissues;

c) maintenance of cellular vitality at this level allows the continuation of root formation, even if the pulp is affected profoundly;

d) abundant vasculature at this level facilitates the metabolic processes and rapidly eliminates the degradation products and toxins.

Caries of young permanent teeth usually evolves rapidly. However, the pulp reacts by formation of a reactive region of dentinary hypermineralization and by neodentinogenesis that can close the accidental openings by means of dentine barriers 1-2 mm thick.

Functions of pulp:

1. Formation of primary and secondary dentine;
2. Nutritive function;
3. Sensibility;
4. Formation of sclerotic dentine in cases of pulp inflammation.

The pulp of permanent teeth with developed roots is identical to the mature conjunctive tissue with active physiology.

Classification of pulpitis by WHO

K.04. Pulpal maladies

K.04.0. Pulpitis

K.04.00. Hyperemia of pulp

K.04.01. Acute

K.04.02. Purulent (pulpal abscess)
K.04.03. Chronic
K.04.04. Chronic ulcerous
K.04.05. Chronic hyperplasic pulpitis (pulpal polyp)
K.04.08. Other precise pulpitis
K.04.09. Imprecise pulpitis
K.04.1. Pulpal necrosis. Pulpal gangrene
K.04.2. Pulpal degenerescense:
Pulpal calculae (concrements)
Denticulae
Pulpolytis
K.04.3. Abnormal formations of solid dental tissue in the pulp. Irregular secondary dentine

PULPITIS OF TEMPORARY TEETH

Etiologic factors

1) Toxic- bacterial factors. Untreated caries is the most frequent cause of demineralization and rapid evolution of destructive processes in the solid dental tissues of temporary teeth and infection of pulp via the wide dentinary channels.

2) Traumatic factors. Acute traumas, incorrect preparation of carious cavity without observation of security zones, dental fractures at children aged between 2 and 3 years.

3) Chemical factors. Toxic action of silica and silica-phosphate cements, of acrylic resin, composite materials or medicamentous solutions with irritant effect, in considerable concentration: oxygenated water, alcohol, esters, eugenol, etc.

4) Thermal factors. Comparatively more often met at adults, being caused by the preparation of carious cavities with turbines or continuous drilling, filling with amalgam without base material.

Classification of pulpitis of temporary teeth

In order to classify the pulpal affections of temporary teeth till now was used the classic classification proposed by Meyer:

1. Acute total purulent pulpitis;
2. Chronic open pulpitis:
   a) ulcerous;
   b) polypous.
3. Chronic close pulpitis;
4. Pulpal gangrene.

Nowadays in the European countries is used the modern classification proposed by French and English authors:

1. Sept syndrome;
2. Pulpitis (passage type);
3. Pulpal necrosis:
   a) without periodontal pathology;
   b) with periodontal pathology.
4. Pathology of furcation.

Classification of temporary teeth pulpitis by T. Виноградова:

1. Acute pulpitis:
   a) serous;
   b) purulent;
   c) with affection of periodontium and lymphatic ganglions.
2. Chronic pulpitis:
   a) simple;
   b) proliferative;
   c) gangrenous.
3. Exacerbation of chronic pulpitis.

Particularities of evolution of pulpitis at temporary teeth

- Asymptomatic evolution of inflammatory processes;
- Chronic forms are detected more often;

- Rapid transformation of clinical forms (from acute into chronic, from serous to purulent);
- Tendency to generalization of process with rapid invasion of healthy sectors or absence of peripheral delimitation under the form of capsule or cellular barrier;
- Disturbance of the general state in advanced cases (fever, insomnia, etc.);
- Affection of periodontium (edema), enlargement of regional lymphatic ganglions;
- Coexistence of inflammation and micro-abscess zones and necrosis following the pulpitis; pulpal necrosis is more often observed at children.

Clinical forms of pulpal dental pathology according to the European classification

1. Septum syndrome

It is a syndrome accompanying the proximal caries.

Subjective manifestation: live pain, exacerbation after meals, localized between the two teeth, can be extended into an entire semi-arcade.

Objective manifestation: caries of proximal surfaces of two neighboring teeth (temporary molars). The interdental papilla is hyperemic, edematous; inflammation can be extended on the marginal gum.

Radiological manifestation: proximal carious cavities, insignificant density at the level of circular ligament, absence or lesion of alveolar and bone zone integrity (depending on the existence of apical inflammatory processes of dental roots).

Treatment: filling of carious cavities or creation of conditions for self-cleaning. In purulent cases simple drainage stops the process.

2. Pulpitis

It corresponds to the clinical forms of temporary or permanent teeth pulpitis.

3. Pulpal necrosis

A. Pulpal necrosis without periodontal pathology (clinical signs correspond to chronic gangrenous pulpitis).

B. Pulpal necrosis with periodontal pathology: acute or chronic form (corresponds to the acute and chronic forms of apical periodontitis).

4. Pathology of furcation

Furcation zone is frequently affected by infectious complications of temporary teeth and a simultaneously is a zone of interference with permanent teeth in formation.

Objective manifestation: gingival abscess in the region of projection of the dentinary furcation. Pulp can be vital or necrotizing.

Radiological diagnosis: easy when disappearance of interradicular bone architecture is observed. Preliminary signs include: widening of the periodontal space in the region of bifurcation or limited loss of bone on one side of inter-radicular septum.

Treatment: very difficult, possibilities of endodontic treatment are limited even on the stages I and II of temporary tooth formation. Often dental extraction is required.

SYMPTOMATOLOGY OF PULPITIS OF TEMPORARY TEETH

Diagnosis of pulpitis at temporary teeth is difficult. Small children can not provide information on the intensity, duration, localization and character of pain. Usually such children are tired, fearful after pain at night, can not point correctly to the affected tooth, can not formulate accuses and do not adequately react on

the tests performed by the dentist.

ACUTE PULPITIS OF TEMPORARY TEETH

Acute serous pulpitis

Subjective manifestation: transitive pains provoked by chemical and thermal excitation (pain caused by cold substances and decreasing when warming up), sometimes spontaneous nighttime pains can appear, being intensive but short-lasting (usually from 30 minutes to up to one night) and recent (from several hours to a night). Usually the child can point at the troubling tooth.

Objective manifestation: a profound carious cavity is determined, the pulpal chamber is open. Probing of carious cavity floor can result in pain. Percussion of tooth can be sensitive. Acute serous pulpitis can rapidly transform into purulent form.

Acute purulent throbbing pulpitis

Subjective manifestation: intense diffuse throbbing and long-lasting pain with short remission intervals; can be localized, however, most often it is has an irradiating pattern so that the child can not point at the affected tooth. Pain can be provoked by hot substances and decreased by cold ones. At small children (aged under 4 years) often disturbances of general mood can be observed: the child is irritated, whining, refuses to eat, can have fever.

Objective manifestation: a profound carious cavity with damaged dentine is determined. The pulpal cavity is close. Probing of cavity floor can be painful or painless. Percussion can be sensitive or painful. The pain at percussion is provoked both by the affection of periodontium and congestion of edematous and inflamed pulp. Compression of tooth doesn't provoke pain. After removal of damaged dentine the pulpal cavity can be opened easily. From there several drops of pus can be eliminated, followed by bleeding and reduction of pain intensity.

Acute pulpitis with affection of periodontium and lymphatic ganglions

Subjective manifestation: at small children (aged less than 3-4 years) can evolve rapidly, with symptoms of general intoxication, rise of body temperature up to 38^0C and above, insomnia, irritation, crying, and refuse to eat. Pains are spontaneous, localized or irradiating, sometimes permanent. Analgesics remove the pain for 1-2 hours. By the end of the first or second day from the appearance of pain edema of soft maxillary-facial tissues at the trouble tooth can be observed.

Subjective manifestation: asymmetry of face at the side of affected tooth due to collateral edema. The affected tooth contains a profound carious cavity with damaged dentine. Touching the tooth can provoke pains. The tooth can be mobile. Excavation of damaged dentine leads to the opening of a pulpal cavity with several drops of pus or bleeding. Percussion can be intensively painful. The gum in the radicular apex projection region is hyperemic, edematous and painful at palpation. The regional lymphatic ganglions are enlarged and also painful at palpation.

CHRONIC PULPITIS OF TEMPORARY TEETH

General clinical particularities of chronic forms of pulpitis of temporary teeth are the following:

- Often the affection evolves without characteristic symptoms;
- Chronic forms can appear as primary chronic process;
- The most often is the simple chronic pulpitis (close) followed by gangrenous and rarely – proliferative (hypertrophic);
- At around 44 % of children pain can be caused by thermal and mechanical irritation;
- In 57% of cases chronic pulpitis is followed by destructive processes of periapical tissues that can be detected radiologically;
- Most often are observed the complications at the level of

periodontium, lymphatic ganglions and child's organism provoked by chronic gangrenous pulpitis;

- Simple forms of chronic pulpitis and gangrenous pulpitis can be detected both in closed and open cavities of teeth;
- Chronic proliferative (hypertrophic) pulpitis is detected only on the teeth with open pulpal cavity.

Simple chronic pulpitis (fibrous)

Subjective manifestation: evolves without subjective signs, is diagnosed during prophylactic examination of children. Nighttime pains can sometimes be observed from anamnesis.

Objective manifestation: the tooth displays a medium-size or profound carious cavity, dentine can be degraded and pigmented. Probing of cavity walls and floor can provoke pains. Sometimes thermal diagnostics can provoke a growing pain. Percussion is painless. Excavation of degraded dentine from the floor of carious cavity can result in the opening of carious cavity, insignificant pain and bleeding.

Chronic proliferative pulpitis (hypertrophic)

Subjective manifestation: pains and bleeding after mechanic irritation (mastication of food, dental brushing, etc.). The child avoids mastication of food on the affected side.

Objective manifestation: often the dental corona is destroyed; the pulpal chamber is wide open, filled with granules of different dimensions or with pulpal polyp. Probing leads to bleeding and insignificant pain. Dental percussion can be slightly sensitive. Often radiograms allow detecting characteristic signs of apical granulated periodontitis.

Chronic gangrenous pulpitis

Subjective manifestation: pains during mastication or spontaneous evening pains, or pains provoked by hot excitants. Past pains can be confirmed by anamnesis.

Objective manifestation: dental corona can have gray color, carious cavity being of medium size or profound. Under the damaged dentine or in the tooth with open pulpal chamber the pulp is necrotized on variable surface. Sometimes specific gangrenous smell can be felt. Painful sensations appear during profound probing in radicular channels. Percussion sound is damped and certain sensitivity is manifested.

Chronic gangrenous pulpitis in 57 % of cases (by S. Sarbu, 1976) is complicated by apical chronic granulated periodontitis that can be diagnosed radiologically. In cases of complication with apical periodontitis hyperemia and gingival edema in the projection region of affected apex is determined, often fistulas appear, and lymphatic ganglions can be increased, painful at palpation.

Exacerbation of chronic pulpitis

Subjective manifestation: the child has accuses on paroxysmal pain of spontaneous character. Live prolonged pain caused by external excitants can be present (thermal or mechanical excitants). The child doesn't masticate the food on the affected part for a long time. From anamnesis one can outline pains characteristic for one of the chronic forms of pulpitis.

Objective manifestation: the carious cavity is profound, with damaged and pigmented dentine, the pulpal chamber being close or open. Probing of pulp is painful, percussion provokes slight pains. Radiograms often display signs of chronic granulated periodontitis.

Exacerbation of chronic gangrenous pulpitis

Exacerbation of chronic gangrenous pulpitis of temporary teeth is a very grave process.

Subjective manifestation: accuses on spontaneous paroxysmal pains provoked by thermal excitants (hot ones), disturbance of general mood with intoxication syndrome.

Objective manifestation: the pulpal chamber is usually open, a bad smell is present. Profound probing of cavity and percussion are painful. The gum in the projection region of affected root apex is hyperemic, edematous, fistulas are observed often. Regional lymphatic ganglions are enlarged.

Clinical forms of temporary teeth pulpitis depend on the root development degree and respectively – on age.

Evolution of pulpitis (acute or chronic) depends on the activity of dental caries and child's state of health:

- acute forms are more often determined at healthy children and children with dental caries of degree I (compensated form);
- primary-chronic forms are met at morbid children, with reduced resistance of organism or with caries of degrees II or III (sub- and de-compensated forms).

PULPITIS OF TEMPORARY TEETH AT CHILDREN AGED UNDER 4 YEARS

Till the age of 4 years dominate the primary-chronic forms of pulpitis and their exacerbation. Morphological particularities of clinics in this period are the following: more often are affected the superior incisors and the inferior temporary molars, also are affected the teeth with hereditary dystrophy, dental roots can be on the formation stage, radicular channel being large, pulpal cavity big, pulpal tissues morphologically and functionally underdeveloped and thus incapable to provide an acute reaction to inflammation.

PULPITIS OF TEMPORARY TEETH AT CHILDREN AGED BETWEEN 4 AND 6 YEARS

At the age of 4-6 years predominates the acute pulpitis followed by acute pulpitis with affections of periodontium and lymphatic ganglions; less often are the cases of chronic pulpitis and their exacerbation.

Morphological particularities of pulpitis at the age of 4-6

years:

- Typical localization of carious cavities;
- The pulpal tissue is morphologically and functionally mature;
- The roots are already formed;
- Between the roots of temporary molars and permanent dental buds is the maximum distance.

PULPITIS OF TEMPORARY TEETH AT CHILDREN AGED BETWEEN 7 AND 9 YEARS

The chronic forms and their exacerbation predominate.

Morphological particularities of pulpitis at the age of 7-9 years are the following:

- Typical localization of carious cavities;
- Pulpal tissues are on a stage of involution;
- Roots are subject to resorption;
- Permanent teeth buds are approached to the roots of temporary teeth to the maximum extent.

TREATMENT OF TEMPORARY TEETH PULPITIS

Presently the rationality of treating the pulpitis of temporary teeth is no more discussed, it being determined by the following factors:

- Complications of temporary teeth pulpitis affect the formation of temporary root and the processes of radicular resorption, as well as the normal development of the permanent teeth buds and their position on the dental arcade. Inflammatory complications can lead to the formation of follicular cysts and appearance of odontogenous infection nidus for the child's organism, etc.;
- Possibility of using the affected tooth in mastication, i.e. recovery of functioning;
- Maintenance of space on the dental arcade till the time of physiological substitution.

Treatment of pulpitis of temporary teeth is hard both from

technical and psychological points of view. The main difficulties are associated with:

- anesthesia, especially at small children, with use of vital treatment methods;
- use of endodontic tools;
- preservation of an isolated operation field during the intervention;
- absence of materials for the filling of channels to be perfectly adapted to the physiological formation processes and resorption of temporary dental root.

Methods of treating pulpitis of temporary teeth

A. Vital methods:
   - biological method (pulpal direct and indirect sealing);
   - amputation (pulpotomy);
   - extirpation (pulpectomy).

B. Non-vital methods:
   - amputation (pulpotomy);
   - extirpation (pulpectomy).

Indirect pulp cap (dentinogenetic treatment)

Principle: conservative (medicamentous) treatment of inflammatory process, stimulation of recovery processes and dentinogenesis in pulp with recovery of tooth function.

Indications: strictly limited to the following: serous pulpitis (reversible) and simple chronic pulpitis. Applicable only to healthy children (health groups I and II), children with caries of IInd degree, with roots on formation (stage I) or already developed roots (stage II) with localization in the center of carious cavity.

Technique. Visit I:

- isolation (application of rubberdam, etc.);
- removal of affected superficial tissues by excavation, without anesthesia until the signs of sensibility appear;
- in some cases anesthesia can be practiced with careful

preparation of cavity: removal of damaged dentine and preservation of dentine neighboring the pulp (without pressure, with cooling and frequent replacement of sterile drill);

- medicamentous treatment of cavity with non-irritating solutions (warm): proteolytic ferments, weak antiseptics, physiological serum;
- slight drying of cavity with air or sterile tampons;
- application of preliminarily prepared paste containing a combination of antibiotics, sulfanilamide, corticosteroids, anesthetics under bandage for 4-5 days;

Visit II:

- control of pulp vitality;
- isolation;
- removal of temporary bandage and curative paste;
- application of odontotropic medications on the floor of carious cavity;
- permanent filling of carious cavity; in some cases a temporary bandage can be applied and afterwards substituted by permanent filling in 15-20 days if no complications appear.

Clinical and radiological observation is necessary after 2 weeks, 3, 6 and 12 months.

Sealing materials include: pastes based on calcium hydroxide: Calmecine, Calcine (Russia); Dycal (De Trey); Life (Kerr); Reocap (Vivadent); Calcicur, Calcimol LC (Voco); Calxyl (Otto), etc.

Pastes based on zinc-oxide-eugenol: preliminarily prepared or commercially procured pastes: Cavitec (Kerr), Kalsogen Plus (De Trey), etc.

Direct pulp cap

Indications: Very low in cases of temporary teeth: accidental opening of pulpal chamber due to traumatic lesion (incisors) or during preparation (if well isolated from saliva) of carious cavities of teeth on the root formation stage.

Technique:

- isolation of tooth;
- preparation of carious cavity;
- preparation of cavity with non-irritating solutions (sol. NaCl 0.8%);
- after hemostasis calcium hydroxide-based preparations are applied and temporary filling for 3-6 weeks;
- permanent filling is applied after the expiration of period set out above if there are no accuses and the tooth is vital.

Clinical and radiological observation is necessary after 2 weeks, 3, 6 and 12 months.

Pulpotomy or vital amputation of pulp

Principle: removal of coronary pulp under local or general anesthesia followed with application of special material at the level of channel opening to attain two particular scopes:

- facilitation of cicatrice formation at the level of amputation with conservation of radicular pulp vitality;
- fixing the sub-adjacent tissues at the level of amputation in order to prevent the infection from penetration into the radicular pulp.

Indications: accidental opening of pulpal chamber during the first two days from trauma when the opening is too big and saliva penetrates in there, simple chronic pulpitis. This method is applies to temporary teeth at stages I or II of root formation.

Technique:

- local or general anesthesia;
- isolation of tooth (cotton tampons, dental dikes, etc.);
- preparation of dental cavity and medicamentous treatment;
- opening of pulpal chamber, amputation of coronary pulp with the help of excavator or sterile round bur, sectioning a radicular filets;
- hemostasis;
- treatment of radicular pieces with medicamentous

preparations;

- application of cement base and permanent filling.

Clinical and radiological observation is necessary after 2 weeks, 3, 6 and 12 months.

Materials used for the sealing of pulp amputation at temporary teeth are very different:

a) Materials based on calcium hydroxide. However, they require a precise diagnosis, strict observation of asepsis and can lead to the extension of inflammatory process into the radicular pulp and to internal resorption;

b) Materials based on zinc-eugenol (however, they can induce the inflammatory process into the radicular pulp);

c) Materials based on formocresol (the Buckley formula: 19 % formaldehyde, 35 % cresol and 15 % glycerin). Formocresol is applied on a bullet in the pulpal chamber for 5 minutes and then the zinc-eugenol paste is applied. If hemostasis is not assured, the bandage can be left for 24 hours: Formocresol in contact with vital pulpal tissue creates a fixation zone resistant to autolysis and microbial invasion. The radicular pulp remains vital, the formation or radicular resorption processes are facilitated.

Pulpectomy or vital extirpation

Principle: complete removal (extirpation) of pulp under local or general anesthesia.

This method is less often used for the treatment of temporary teeth due to the following reasons:

- use of this method requires longer time and therefore is less suitable for small children;
- the complications provoked by the children's behavior: sudden and uncontrolled movements, etc.
- limited visibility of operation field;
- filling of radicular channels straight to the apex that influences the process of radicular rhizalysis;
- danger of infection and lesion of periapical space

(including the buds of permanent teeth) with endodontic tools.

Indications: all forms of pulpitis of permanent teeth with developed roots.

Technique:

- local or general anesthesia;
- isolation of tooth (cotton tampons, rubberdams, etc.);
- preparation of cavity and medicamentous treatment;
- opening of pulpal cavity;
- ablation of coronary pulp;
- hemostasis;
- extirpation of radicular pulp;
- medicamentous and instrumental preparation of channels;
- drying of radicular channel;
- filling of channel;
- application of cement basis and permanent filling.

The materials used for the filling of radicular channels of temporary teeth are: zinc-eugenol-oxide paste, Walkoff paste (zinc oxide and Walkoff solution: chrolphenol crist. 2.0; Camphorae tritae 4.0; Thimoli 0.1; Mentholi 0.1; Iodoformii farinosi q.s.), resorcin-formalin paste.

For the filling of permanent teeth roots are contraindicated the cones of gutta-percha, plastics, silver, and metallic alloys.

Presently for the filling of channels is widely used the method called *Traitement SPAD*.

Indications: radicular filling at temporary and permanent teeth after amputation, vital or devital extirpation.

Technique:

a) Pulpotomy
   - hemostasis and medicamentous treatment of pulpal chamber;
   - treatment with sulfuric acid 45 %;
   - drying ;
   - application of paste on the bottom of pulpal chamber;

- permanent filling.

b) Radicular filling:
- medicamentous treatment of radicular channel;
- treatment with sulfuric acid 45 %;
- drying of channel;
- permanent filling.

Non-vital pulpotomy

Principle: removal of preliminary devitalized coronary pulp followed with mummification of radicular pulp. This method is most often used for the treatment of temporary teeth.

Indications: accidental opening; all forms of pulpitis of temporary teeth, without acute and serous pulpitis complicated with periapical tissues reaction and regional lymphadenitis.

Technique:

Visit I:
- preparation of carious cavity and medicamental treatment;
- opening of pulpal horn;
- application of the devitalizing paste on the opened pulp;
- temporary dressing for 24 hours t incisors, and for 48 hours at molars.

Visit II:
- tooth isolation;
- removal of temporary dressing and verification of effect;
- definitive preparation of carious cavity;
- removal of ceiling of the pulpal cavity and coronary pulp (with excavator or globular bur);
- medicamental treatment;
- application of cotton wool with mummification solution on the pulpal chamber: resorcin-formalin for 2-3 days and temporary dressing.

Visit III:
- tooth isolation;
- removal of temporary dressing and cotton wool;

- application of resorcin-formalin paste;
- application of cement basic and permanent filling.

For the pulp devitalizing are used arsenic and para-formaldehyde based pastes: Depulpin (Voco), Toxavit Paste (Lege Artis), Caustinerf.

Four groups of *curative pastes* with action of radicular pulp are: 1. mummifying action: Burkley (cresol 3, formalin 10%, watered glycerin q.s.); formaldehyde based pastes;

2. impregnating action: resorcin-formalin (impregnate radicular pulp, polymerizing and preserving);

3. meta-plastic action: tri-oxy-methyl based pastes; Gysy paste (contains para-formaldehyde);

4. odontotropic action: Kunzel (zinc-oxide-eugenol (5,0) and timol (1,0)); Ray (zinc oxide-- eugenol and iodoform).

Non-vital pulpectomy

Principle: removal of preliminary devitalized dental pulp with subsequent filling of all channels.

Indications: all forms of pulpitis of temporary teeth with developed roots.

Technique:

Visit I:

- preparation of carious cavity with removal of altered dentine;
- opening of pulpal chamber;
- application of devitalizing paste;
- temporary dressing for 24-36 hours.

Visit II:

- removal of temporary dressing and verification of effect;
- definitive preparation of carious cavity;
- removal of floor of the pulpal cavity and coronary pulp (with excavator or globular bur);
- removal of radicular pulp;
- medicamentous and instrumental treatment of channels;

- filling of channel with resorbable paste;
- filling of carious cavity.

It is recommended to introduce the extraction needle directly to the vicinity of the root top after that a turning movement is made (45^0) in order to fix and extirpate the radicular pulp. In large channels one can use simultaneously 2-3 pulp extractors. Medicamentous and instrumental treatment is alternated and limited to up to 2/3 of channel length, touching of apex shall be avoided.

For the filling of channels of temporary teeth are used the resorbable pastes: zinc-oxide-eugenol, the Walkoff paste, iodoform pastes (Rebel, Ray), Treatment SPAD, etc.

PARTICULARITIES OF TREATMENT OF TEMPORARY TEETH PULPITIS AT DIFFERENT DEVELOPMENT STAGES

1. Period of temporary tooth root formation

Practically to all forms of pulpitis the pulpal amputation method applies. Often gneral anesthesia is used, especially for the treatment of small children. Choice of curative pastes for this method is conditioned by the creation of adequate conditions for the formation of temporary teeth roots.

2. Period of functional maturity of pulp (stabilization of formed root)

The therapy of pulpitis at temporary teeth with already formed roots includes conservative and surgical methods.

The rational choice of the treatment method depends not only on the form of pulpitis, duration and localization of process but also on the child's age, his general state, psychic development, etc.

The conservative treatment method (biological) did not prove itself in the therapy of pulpitis at temporary teeth due to:

imperfection of diagnosing pulpitis at children of small ages, particularities of rapid evolution of pulpitis (with predomination of purulent and diffuse forms); particularities of pulpitis at temporary teeth characterized by the approximal localization of carious cavity; difficult observation of treatment technique conditions, etc.

The surgical method is widely used in therapy of pulpitis at temporary teeth, especially non-vital pulpotomy. One of the most widely used pastes in the country is the resorcin-formalin paste. Generally, the treatment of pulpitis of temporary teeth by pulpectomy is less efficient compared to the pulpotomy method.

Local anesthesia is usually applied beginning with the age of 5-6 years for the amputation and vital extirpation of pulp at molars. Until this age is attained general anesthesia must be used.

In case of acute diffuse pulpitis or chronic exacerbated pulpitis with affection of apical periodontium and lymphatic ganglions: during the first visit a drainage of exudation is made by opening the tooth cavity (under anesthesia, with the use of turbine, etc.) and subsequently in 4-5 days after liquidation of acute state pulpitis is treated by vital or non-vital pulpectomy in 2-3 visits.

Treatment of chronic gangrenous pulpitis is performed in 3-4 visits by the method of vital or non-vital pulpectomy, with application of medicamentous bandages in the channel (proteolytic ferments, antiseptics, silvering, etc.); careful and thorough instrumental processing is required. Finally it is necessary to impregnate and filling the channels with resorcin-formalin paste.

Sometimes due to the too big curve of the channels of temporary molars it is very difficult to extirpate the apical part of radicular pulp. In such cases one must not pursue complete extirpation but impregnate the channel with resorcin-formalin solution and obturate it with resorcin-formalin paste.

3. The period of pulp involution (radicular resorption)

During this period we must pay particular attention to the radicular resorption terms of temporary teeth, it may be determined more objectively by the radiographic method.

The tooth with pulpitis must be extracted indifferently of the form of pulpitis, if till the physiological substitution remain about 6-8 months, Pulpitis of temporary teeth with root resorpted up to 1/3 and if the physiological substitution is expected to occur relatively late (in 1.5 – 3 years) conservative treatment can be used. In all forms of pulpitis the amputation method is used.

PULPITIS OF PERMANENT YOUNG TEETH

The ethiopathogenesis of permanent young teeth involves three important causes:

1. Dental caries;
2. Dental traumatism;
3. Therapeutic procedures.

1. Inflammatory processes in pulp - result of evolution of dental caries

In the first phase in the dentine and in pulp act the defense mechanisms characterized by: intra- and peri-channel calcification of dentine; formation of secondary dentine proportional to the destroyed dentine, by odontoblastic irritation at the level of pulp. When the carious processes advance the defense mechanisms become insufficient and their approach to the pulpal chamber determines irritation of pulp in the odontoblastic and sub-odontoblastic zone. Signs of chronic inflammation appear: slight dilation of vessels and defense cells. This phase is reversible and a-symptomatic.

In the more advanced phases of dental caries continuous irritation leads to the capillary and fibroblastic proliferation, lymphocytes and histiocytes also get involved in this process. In consequence of this process granulated tissue is formed that

allows replacing the destroyed tissues. This phase is partially reversible.

When the pulpal chamber is opened a direct aggression of bacterial germs and toxins occurs. Thus the chronic inflammatory process gets accompanied by an acute inflammation characterized by cellular infiltrate with neutrophils preceding the formation of an abscess in the zone of opening. Evolution of this process is possible in two directions: a) in some cases the proliferation of granulated tissue becomes dominant, a polyp covered with a fibrous capsule is formed; b) when the chronic inflammatory processes repeat and get complicated by acute accidents, they get displaced into the depth of the channel, causing in fine the necrosis of pulp.

At young permanent teeth inflammatory processes in the coronary pulp are reversible and practically do not hamper the activity of apical zone. In case of acute diffuse processes, chronic and chronic exacerbated processes an essential role is played by the considerable vasculation of the growth zone and width of apex that assure the continuance of root growth- apexogenesis. Even in more advanced cases when the inflammatory process is extended onto the radicular pulp the apical zone remains intact while its autonomy gives a possibility to substitute the activity of odontoblasts with the mesenchymal reserve cells both from pulp and periapical tissues.

2. Degenerative and inflammatory processes in the pulp of permanent young teeth – as consequence of dental traumas

Post-traumatic degenerative changes comprise:

- numeric reduction of odontoblasts, fibroblasts and non-differentiated mesenchymal cells;
- growth of collagen followed by partial calcination of fibers;
- obliteration of capillary network leading to the reduction of blood supply;
- deposits of secondary dentine in considerable quantities;

- degeneration of nervous branches.

All these at last lead to the reduction of pulpal volume simultaneously with abundant deposits of mineral salts. Initially these processes are symptomatic, owing to compression and traction exerted onto the vascular-nervous packet during the trauma and subsequent installation of inflammatory or degenerative process in the pulp and in the tissues of periodontium. However, in most cases the symptoms are missing or are reversible, they do not lead to the lesion of root growth zone.

3. Inflammatory processes as consequences of therapeutic interventions

Therapeutic procedures in the young permanent teeth (preparation of carious cavity, treatment of dentinary lesion, drying, negative properties of filling materials, accidental opening o pulpal chamber, etc.) can provoke irritation and inflammatory processes at the level of pulp.

During the first phases take place the changes of odontoblasts with production of a considerable quantity of secondary dentine. This phase is asymptomatic, transient and reversible.

On the next stage occurs the severe lesion of odontoblasts with their aspiration into the dentinary channels. The odontoblatic stratum develops an acute inflammatory process with dilation of vessels and exudation. Affection of odontoblasts is irreversible. They are substituted by reserve mesenchymal cells that produce unstructured dentine in important quantities for the purpose of root formation. In parallel occurs the complete resorption of exudation meaning that this phase is also reversible, but symptomatic: acute short-lasting pains.

In more advanced phases the growing dilation of vessels leads to the appearance of edema. The resulting stasis leads to margination and leukocytal diapedesis, respectively, purulent

exudation appears and develops. This phase is partially reversible, the healing is possible owing to the appearance of proliferative phenomena at the periphery of purulent processes and formation of granulated tissue that absorbs the exudation and replaces the mortified tissues. This phase is symptomatic, characterized by persistent and diffuse pain, and exacerbation.

CLASSIFICATION OF PULPAL AFFECTIONS OF PERMANENT TEETH AT CHILDREN

Traditional classification – based on the clinical symptomatology criterion combined with other different clinical tests (by Euler and Meyer):

1. Acute pulpitis
   a) serous - partial
      - total.
   b) purulent - partial;
      - total.
2. Chronic pulpitis
   a) open - ulcerous;
      - granulomatous.
   b) close.
3. Pulpal necrosis.

Classification of pulpitis by Selter and Bender

1. Reversible pulpitis (treatable):
   - intact non- inflamed pulp;
   - partial acute or chronic pulpitis, but without necrosis.
2. Irreversible pulpitis (untreatable):
   - partial chronic pulpitis with partial necrosis;
   - total chronic pulpitis;
   - total pulpal necrosis.

This classification of reversible and irreversible pulpitis is based on the criterion of possibility of treatment.

Classification of pulpitis by Kuntzel

- Pulpal hyperemia (profound caries);
- Asymptomatic or reversible pulpitis;
- Symptomatic or irreversible pulpitis;

- Simultaneous symptomatic and asymptomatic pulpitis (partially recoverable).

Classification of pulpitis of permanent teeth at children by T. Виноградова

1. Acute pulpitis:
   a) serous partial;
   b) serous total;
   c) purulent partial;
   d) purulent total.
2. Chronic pulpitis:
   a) simple;
   b) proliferative;
   c) hypertrophic proliferative;
   d) gangrenous.
3. Exacerbation of chronic pulpitis.

Pulpitis of permanent young teeth has the same manifestations as at permanent mature teeth with certain specific particularities:

- Dental caries more often provokes chronic inflammatory processes to which later-on centers of acute inflammation.
- Therapeutic manipulations can often result in acute transient inflammations that sometimes turn into chronic affections.
- Traumas lead to degenerative changes with slow evolution but with prolonged maintenance of integrity in the apical zone.
- The pulp reacts to slightest external irritants without the compulsory presence of clinical signs.
- Pains are relatively weaker pronounced but the inflammation of pulp can remain asymptomatic for a longer time.
- The phase of partial pulpitis is very short.
- Inflammation from the coronary pulp rapidly extends into the radicular one.
- Purulent processes are more often.
- Irradiating pains are less often.
- Acute forms quickly get transformed into chronic ones, while the chronic ones can facilitate the appearance of acute

centers (exacerbation).

- Often the inflammatory process penetrates into the periodontium.
- Acute forms often are accompanied by general intoxication of organism.
- Favorable evolution of inflammatory processes in the pulp of young teeth is to a consider able extent determined by the specific properties of the apical zone (growth zone).
- Immune reactions of young teeth are significant, they block the evolution of inflammatory process, but simultaneously they can provoke extended destruction processes.

EVOLUTION PARTICULARITIES OF PULPITIS OF PERMANENT TEETH AT DIFFERENT DEVELOPMENT STAGES AT CHILDREN

1. Root formation period

Acute pulpitis The clinical evolution of serous inflammatory process in this period practically lacks the usual clinical image for this form.

In cases of acute serous pulpitis the children mainly suffer from spontaneous appearance of a slight pain. The accesses typical for pulpitis are not manifested. This pain usually doesn't repeat spontaneously but is provoked by mechanical or chemical irritants. Such an evolution can be explained by the possibility of exudation drainage via the wide dentinary channels into the carious cavity and via the wide apical orifice into the periapical tissues.

At some children this process is manifested severely: fever, headaches, tiredness, weakness, disorders of sleep, tachycardia, etc. In cases of acute diffuse pulpitis intense pains occur that by evening or by night repeat and get stronger. The children loose appetite, are irritated, and breathe frequently and have tachycardia. On the next day the duration of pain increases and so

on until the pain becomes permanent and irradiate into the temple, into the occipital, into the sub-orbital region or into the ear. Analgesics are of little help. The carious cavity displays damaged dentine and is slightly pigmented. Probing of cavity floor is painful. EOM data is not reliable.

Acute purulent pulpitis in a tooth with incompletely developed root is characterized by acute and pulsating, sometimes irradiating pains. Pulpitis is very often associated with collateral edema, hyperemia, and hyperesthesia of skin in the affected region.

Lymphatic ganglions are enlarged and slightly painful at palpation. Profound carious cavity or filling are detected in the affected teeth. Hot food intensifies the pain while cold food reduces it.

Chronic pulpitis Chronic pulpitis clinically is manifested weakly; however unpleasant sensations (heaviness) in the affected tooth may be present. Slight pains can appear when leaving a warm and entering a relatively colder environment. Also acute pains in the affected tooth can appear.

At examination loss of enamel gloss is observed, the tooth becomes darker, a profound opening cavity with or without opening of pulpal chamber may be detected. The pulp is ash-gray, lax, and painful at probing. EOM data is not reliable.

Chronic exacerbated pulpitis is characterized by slow evolution with repeated recurrent acute pains. From anamnesis one may conclude that the tooth reacts on thermal irritants, however the pain was not intense. Remission periods at some children can last for a long time.

The children have accuses on acute, sudden and pulsing, sometimes irradiating pains. Periodontal reaction is observed very often.

2. Period of developed roots

Clinically doesn't differ from the manifestations of pulpitis of

permanent teeth at adults

Differentiated diagnosing of pulpitis at children

1. Acute focal pulpitis is differentiated by:
   - acute forms (serous or purulent);
   - acute diffuse pulpitis;
   - chronic fibrous pulpitis;
   - profound caries;
   - papillitis.
2. Acute diffuse pulpitis is differentiated by:
   - between the forms (serous or purulent);
   - acute focal pulpitis;
   - chronic exacerbated pulpitis;
   - acute apical periodontitis;
   - chronic exacerbated apical periodontitis.
3. Chronic simple (fibrous) pulpitis is differentiated by:
   - profound caries;
   - acute focal pulpitis;
   - chronic gangrenous pulpitis;
   - chronic apical periodontitis.
4. Chronic gangrenous pulpitis:
   - chronic fibrous pulpitis;
   - chronic apical fibrous pulpitis, granulated and granulomatous.
5. Chronic hypertrophic pulpitis:
   - proliferation of gingival papilla;
   - granulated apical periodontitis.
6. Chronic exacerbated pulpitis:
   - acute pulpitis;
   - acute apical periodontitis;
   - chronic exacerbated apical periodontitis.

TREATMENT PARTICULARITIES OF PERMANENT TEETH PULPITIS AT CHILDREN

1. Period of root formation at permanent teeth

Pulpitis of permanent teeth with incompletely developed roots is treated by biological method or amputation methods (vital amputation).

The problem of the role of pulp in the formation of dental root is still under discussion. Continuation of root formation at majority of children after non-vital pulpotomy allowed H. Чупрынина to state that the main role in this process is played by the growth zone.

The remote investigation data confirm the high efficiency (90, 5%) of conservative treatment at children with teeth on the root formation stage and with already developed roots. After treatment growth and formation of permanent dental roots continue.

The indirect pulp cap (dentinogenous treatment) is prescribed to practically healthy children: with acute focal pulpitis (at teeth with incompletely developed or developed root); with simple chronic pulpitis (fibrous), if there are favorable conditions for the preservation of filling (cavities of class I) and there are no signs of reaction in the apical periodontium.

Direct pulp cap is indicated in cases of small accidental openings of pulpal chamber and during the processing of carious cavity, in cases of coronary fractures with opening of pulpal chamber not exceeding 2 mm and lasting for several hours (up to 6 hours).

Use of arsenic-based medications in the teeth with incompletely developed roots is dangerous due to the possibility of damaging the growth zone that has primordial importance for the root formation. This is why at this age conservative methods or vital amputation must be used.

In cases of allergic states one must use the method of non-

vital pulpotomy. In order to reduce the toxic action of arsenic, pastes based on para-formaldehyde (with antiseptic property, devitalizing the pulp without toxic action on apical periodontium) are indicated. Indications for non-vital pulpotomy: acute focal pulpitis (in case of failure of biological method), acute diffuse pulpitis, simple chronic pulpitis.

Vital amputation is indicated in the cases when it is not possible to use the biological method: in cases of simple chronic pulpitis, acute focal pulpitis, acute diffuse pulpitis, in cases of accidental opening of pulp during the first two days following the trauma. The technique is identical to the one described for temporary teeth. At the level of amputation one shall use pastes based on calcium hydroxide followed by zinc-oxide eugenol (ZOE) with accelerated hardening. The tooth is definitely obturated with silver amalgam, composite material or prefabricated crown.

High pulpotomy

High pulpotomy (partial pulpectomy) consists in the removal of coronary pulp and of some part of radicular pulp in order to preserve the apical part of the pulp and the growth zone of the root. Indicated only for teeth with incompletely developed roots, when not only the coronary, but also the radicular pulp is involved in a chronic pronounced inflammatory process for a long time. Also used in cases of chronic gangrenous pulpitis and chronic exacerbated pulpitis that involve the tissues of periodontium, coronary fractures with opening and profound infection of pulp, if more than 48 hours have passed from the moment of trauma.

Technique:

- Local anesthesia and isolation of tooth;
- Preparation of carious cavity;
- Opening of pulpal chamber;
- Removal of coronary pulp;

- Amputation of radicular pulp (1/3, ½, 2/3 of channel) with round burs or pulp extractors – very careful with minimum affection for pulpal edge;
- Hemostasis and washing of channels without pressure with physiological serum, calcium hydroxide solutions;
- Application of sterile tampons to the channel;
- Application of sealing paste (based on calcium hydroxide with rapid hardening);
- Application of cement base and definite filling.

Clinical and radiological verification must be performed after 2 weeks, 3, 6 and 12 months. The basic material of calcium hydroxide can be resorpted and thus require refilling. After the end of root formation total extirpation of pulp an radicular filling are performed (with non-resorbable materials).

The Frank procedure of closing the apex (bio-pulpectomy – apexification) The same indications as for high amputation apply. After removal of coronary pulp the radicular pulp is removed up to the level of radiological apex using endodontic tools with special care in order not to affect the thin and incompletely developed walls of the root and not cross the open apical foramen. Irrigation with sodium hypochlorite solution or non-irritant solutions must be performed. Calcium hydroxide is applied to the end of the channel, the channel is thereafter obturated with ZOE-based paste and then definite filling is applied. If the paste is resorpted, it has to be refilled periodically. After the formation of apex in terms of 6-12 months the apical barrier is established and the channel is obturated with an "inverse" gutta-percha cone. Complete apical closing is confirmed radiologically in the form of a cementoid osteo-dentinary bridge. The osteo-dentinary bridge is made of cement produced in the concentric rings from periphery towards the center and towards the apex.

The Fortier technique Filling of channel in bio-pulpectomy is made with ZOE, with or without apical application of calcium

hydroxide based paste, a gutta-percha central " inverse" cone , while the rest of the cones are applied normally, using the lateral material condensation technique for radicular filling.

In cases of pulpal necrosis the author recommends a "layered" filling: in the apical third a resorping paste is applied, then preceded as mentioned above (Fortier J.P., Demars-Fremault C., 1987).

Consequences of treating a tooth with open apex

The consequences can be the following:

a) apexogenesis – formation of roots with normal dimensions, shape and length, with subsequent "closing" of radicular apex. It is a positive result of vital treatment of pulpal inflammation, allowing the continuation of root growth and closing of open apex;
b) apexification – affection of the growth zone leads to the interruption of root formation, in result we have a root of smaller dimensions and of atypical shape;
c) acute or chronic apical processes without closing of apex, requiring surgical periapical interventions or extraction of the permanent tooth.

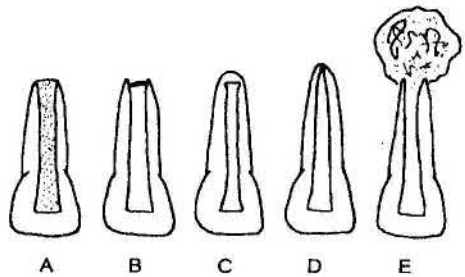


Figure 7.1 **Modalities of apical closing (by Mathewson R.J, 1995): A - apparently open apex (fibrous closing); B - calcified bridge at the apical level; C- wide apical closing apexification); D - normal apical closing (apexogenesis); E- open apex with periapical inflammatory reaction.**

2. Period of developed root – functional maturity of pulp

During this period one may recommend all the previously described methods, including the extirpation method.

At children can be applied the method of vital extirpation with local or general anesthesia (less often). The non-vital methods are not recommended for the treatment of permanent teeth. In particular cases, depending of the anatomic-physiological specifics of permanent teeth at children (wide radicular channels and apex, etc.) in order to avoid the toxic action of arsenic one may use pastes based on para-formaldehyde.

Control questions and topics:

1. Causes of pulpitis at children.
2. Form of pulpitis most often detected in temporary teeth at children.
3. Classification of pulpitis of temporary teeth.
4. Evolution particularities of pulpitis of temporary teeth.
5. General clinical particularities of chronic forms of pulpitis of temporary teeth.
6. Pulpitis of temporary teeth at children under 4 years.
7. Pulpitis of temporary teeth at children aged between 4 and 6 years.
8. Pulpitis of temporary teeth at children aged between 6 and 9 years.
9. Methods of treating pulpitis of temporary teeth.
10. Methods of treating pulpitis during the period of root formation of temporary teeth.
11. Methods of treating pulpitis during the period of developed roots of temporary teeth.
12. Methods of treating pulpitis during the period of root resorption of temporary teeth.
13. Classification of pulpitis of permanent teeth at children.
14. Evolution particularities of pulpitis of permanent teeth at

children.

15. Methods of treating pulpitis during the period of root formation of permanent teeth.
16. Methods of treating pulpitis of permanent teeth with already developed roots.
17. Consequences of treating a tooth with open apex.

CONTROL TESTS

Simple compartment

1. Appearance of pulpitis of temporary and permanent teeth at children is provoked by the following factors:

A. Chemical;
B. Toxic-bacterial;
C. Traumatic;
D. Thermal;
E. All answers are correct.

2. The most frequent factors that determine the appearance of temporary teeth pulpitis at children are:

A. Chemical;
B. Toxic-bacterial;
C. Traumatic;
D. Thermal;
E. All answers are correct.

3. Basic particularities of evolution of temporary teeth pulpitis are:

A. Most often are observed the chronic forms;
B. Rapid transition of one form of pulpitis into another, evolution with variable symptomatology;
C. Disturbance of general state of organism;
D. Often are observed inflammations of periodontal tissues and lymphatic ganglions;
E. All answers are correct.

4. The examination methods used in the diagnosing of pulpitis of temporary teeth are:

A. The same as for adults;
B. Only the basic examination methods;
C. The basic examination methods and one secondary method: electroodontodiagnosis;
D. The basic methods and radiography;
E. All the basic and secondary examination methods.

5. The big percentage of non-coincidence between clinical and patho-morphological diagnosis of pulpitis is determined by the following factors:

A. Imperfection of diagnostic methods for pulpitis;
B. Late visit to the dentist;
C. Very often - close dental cavity;
D. Hyperergic character of inflammation.

6. The duration of acute serous pulpitis of a temporary tooth is:

A. Less than 24 hours;
B. 1-2 days;
C. 2-3 days;
D. 3-4 days;
E. 4-5 days.

7. Duration of acute purulent pulpitis of temporary tooth is:

A. Less than 24 hours;
B. 1-2 days;
C. 2-3 days;
D. 3-4 days;
E. 4-5 days.

8. Acute focal pulpitis is characterized by the following symptoms:

A. Permanent pains intensifying with mastication;

B. Intensive, spontaneous, nighttime pains with short pauses between and irradiation;
C. Acute pains provoked by thermal and mechanic excitants disappearing immediately upon the elimination of excitant;
D. Acute spontaneous pains with long painless intervals;
E. Hyperesthesia.

9. Probing of carious cavity in case of acute focal pulpitis is:
A. Painful in a point of cavity floor;
B. Painful on the entire cavity floor or in several points of the cavity floor;
C. Not painful;
D. Painful at cavity walls;
E. Painful in the region of enamel-dentine junction.

10. Percussion of tooth in case of acute focal pulpitis is:
A. Not painful;
B. Slightly painful;
C. Painful;
D. Very painful;
E. Painful in some cases.

11. In case of acute diffuse pulpitis a child of 3 years can present the following symptoms:
A. Agitation, fever, refuse to eat, permanent pains intensifying during mastication, the child can point at the affected tooth;
B. Agitation, fever, intense pains, spontaneous or provoked by any excitants, most often appearing in the evenings with short painless intervals. The child can not indicate the affected tooth;
C. Acute pains provoked by thermal or mechanical excitants, disappearing immediately upon the removal of excitant,

the child can point at the affected tooth;

D. Agitation, fever, acute spontaneous pains with long painless intervals, the child can point at the affected tooth;

E. All answers are correct.

12. Probing of carious cavity in case of acute diffuse pulpitis is:

A. Painful in a point of cavity floor;

B. Painful on the entire cavity floor or in several points of the cavity floor;

C. Not painful;

D. Painful at cavity walls;

E. Painful in the region of enamel-dentine junction.

13. Percussion of tooth in case of acute diffuse pulpitis is:

A. Not painful;

B. Slightly painful;

C. Painful;

D. Very painful;

E. Painful in some cases.

14. Electro-excitability of pulp in case of acute focal pulpitis is equal to:

A. 0-6 mkA;

B. 6-12 mkA;

C. 12-20 mkA;

D. 20-30 mkA;

E. 30-60 mkA.

15. Electro-excitability of pulp in case of acute diffuse pulpitis is equal to:

A. 0-6 mkA;

B. 6-12 mkA;

C. 12-20 mkA;

D. 20-30 mkA;

E. 30-60 mkA.

16. Clinical manifestations of chronic fibrous pulpitis are the following:

A. There are no accuses, sometimes paroxysmal pains provoked by different excitants: thermal, mechanical and chemical ones;

B. Nighttime spontaneous pains without irradiation, provoked by any excitant;

C. Rarely, pains after change of temperature from cold to warm;

D. Piercing pains, often caused by mechanic excitants, often hemorrhages from dental cavity;

E. Permanent pains, gradually intensifying, especially during mastication or when touching the tooth.

17. In case of simple chronic pulpitis pains appear:

A. Due to diverse excitants, however, they can disappear with removal of excitan;

B. After meals, after some time from taking hot food;

C. Due to diverse excitants, remaining for a long time even after removal of excitants;

D. Spontaneous, nighttime.

18. Chronic gangrenous pulpitis is characterized by:

A. There are no accuses, sometimes paroxysmal pains provoked by different excitants: thermal, mechanical and chemical ones;

B. Nighttime spontaneous pains without irradiation, provoked by any excitant;

C. Rarely, pains after change of temperature from cold to warm;

D. Piercing pains, often caused by mechanic excitants, often hemorrhages from dental cavity;

E. Permanent pains, gradually intensifying, especially during mastication or when touching the tooth.

19. In case of chronic gangrenous pulpitis pains appear:
   A. Due to diverse excitants, however, they can disappear with removal of excitant;
   B. After meals, after some time from taking food;
   C. Due to diverse excitants, remaining for a long time even after removal of excitants;
   D. Spontaneous, nighttime.

20. Chronic hypertrophic pulpitis is characterized by the following:
   A. There are no accuses, sometimes paroxysmal pains provoked by different excitants: thermal, mechanical and chemical ones;
   B. Nighttime spontaneous pains without irradiation, provoked by any excitant;
   C. Rarely, pains after change of temperature from cold to warm;
   D. Piercing pains, often caused by mechanic excitants, often hemorrhages from dental cavity;
   E. Permanent pains, gradually intensifying, especially during mastication or when touching the tooth.

21. In case of chronic hypertrophic pulpitis pains are characterized by the following:
   A. Spontaneous appearance, usually nighttime;
   B. Permanent pains;
   C. Appearance after meals, after some time from taking hot food;
   D. Appearance after the penetration of nutrients and are associated with hemorrhage;
   E. Appearance due to any excitants and maintained for a long time after removal of excitants.

22. Among the various forms of pulpitis most often at children are

observed:

A. Acute focal form;
B. Acute diffuse form;
C. Chronic fibrous form;
D. Chronic gangrenous form;
E. Chronic hypertrophic form.

23. Among the various forms of pulpitis at children are observed rarely:

A. Chronic exacerbated form;
B. Acute diffuse form;
C. Chronic fibrous form;
D. Chronic gangrenous form;
E. Chronic hypertrophic form.

24. The carious cavity communicates with the dental cavity in case of pulpitis of:

A. Acute focal form;
B. Acute diffuse form;
C. Chronic fibrous form;
D. Chronic gangrenous form;
E. Chronic hypertrophic form.

25. Electro-excitability of pulp in case of chronic fibrous pulpitis is equal to:

A. 0-6 mkA;
B. 6-12 mkA;
C. 12-20 mkA;
D. 20-30 mkA;
E. 30-60 mkA.

26. Electro-excitability of pulp in case of chronic gangrenous pulpitis is equal to:

A. 0-6 mkA;
B. 6-12 mkA;

C. 12-20 mkA;
D. 20-30 mkA;
E. 30-60 mkA.

27. Probing in case of chronic gangrenous pulpitis is painful:
A. In the point of pulpal horn projection;
B. On the entire floor of the carious cavity;
C. On the entire floor of the carious cavity and in the point of pulpal chamber opening;
D. In the orifices of channels or in the depth of channels;
E. In the coronary part of pulp.

28. Chronic fibrous pulpitis is differentiated by:
A. Chronic gangrenous pulpitis, profound caries, chronic apical periodontitis;
B. Chronic gangrenous pulpitis, chronic hypertrophic pulpitis, profound caries, chronic apical periodontitis;
C. Medium caries, profound caries, acute focal pulpitis, chronic hypertrophic pulpitis, chronic apical periodontitis;
D. Profound caries, acute diffuse pulpitis, chronic gangrenous pulpitis, chronic hypertrophic pulpitis, chronic apical periodontitis;
E. Profound caries, proliferation of gingival papilla, chronic apical periodontitis, chronic gangrenous pulpitis.

29. Chronic gangrenous pulpitis is differentiated by:
A. Profound caries, acute focal pulpitis, chronic apical periodontitis;
B. Fibrous chronic pulpitis, chronic apical periodontitis;
C. Medium caries, profound caries, chronic apical periodontitis;
D. Profound caries, proliferation of gingival papilla, chronic apical periodontitis;
E. Chronic fibrous pulpitis, chronic hypertrophic pulpitis,

chronic apical periodontitis.

30. Affection of apical periodontium tissues is most often detected radiologically in the following form of temporary teeth pulpitis:

A. Acute diffuse pulpitis;
B. Acute pulpitis with affection of periodontium and lymphatic ganglions;
C. Chronic fibrous pulpitis;
D. Chronic gangrenous pulpitis;
E. Chronic hypertrophic pulpitis.

31. Chronic hypertrophic pulpitis is differentiated by:

A. Medium caries, profound caries of approximal surfaces, chronic fibrous pulpitis, chronic apical periodontitis;
B. Proliferation of gingival papilla, chronic apical granulated periodontitis;
C. Profound caries, chronic gangrenous pulpitis, proliferation of gingival papilla;
D. Proliferation of gingival papilla, chronic fibrous pulpitis;
E. Chronic apical periodontitis, chronic fibrous pulpitis, chronic gangrenous pulpitis.

32. The essence of conservative (biological) treatment of pulpitis consists of:

A. Removal of coronary pulp and preservation of radicular pulp vitality – under local or general anesthesia;
B. After devitalization of pulp - amputation of coronary pulp and mummification of radicular pulp;
C. Removal of coronary pulp and a part of radicular pulp under local or general anesthesia;
D. Treatment of inflammatory processes with medicamentous preparations;
E. Removal of coronary and radicular pulp under local or

general anesthesia.

33. The essence of vital amputation of pulp consists in:
A. Removal of coronary pulp and preservation of radicular pulp vitality – under local or general anesthesia;
B. After devitalization of pulp - amputation of coronary pulp and mummification of radicular pulp;
C. Removal of coronary pulp and a part of radicular pulp under local or general anesthesia;
D. Treatment of inflammatory processes with medicamentous preparations;
E. Removal of coronary and radicular pulp under local or general anesthesia.

34. The essence of vital pulp extirpation method consists in the following:
A. Treatment of inflammatory process in pulp with medicamentous preparations;
B. Removal of coronary pulp and a part of radicular pulp under local or general anesthesia;
C. After devitalization of pulp - amputation of coronary pulp and mummification of radicular pulp;
D. Removal of coronary pulp and preservation of radicular pulp vitality – under local or general anesthesia;
E. Removal of coronary and radicular pulp under local or general anesthesia.

35. The essence of profound pulp amputation method consists in the following:
A. Treatment of inflammatory process in pulp with medicamentous preparations;
B. Removal of coronary pulp and a part of radicular pulp under local or general anesthesia;
C. After devitalization of pulp - amputation of coronary pulp

and mummification of radicular pulp;

D. Removal of coronary pulp and preservation of radicular pulp vitality – under local or general anesthesia;
E. Removal of coronary and radicular pulp under local or general anesthesia.

36. The essence of non-vital pulp amputation method consists in the following:

A. Treatment of inflammatory process in pulp with medicamentous preparations;
B. Removal of coronary pulp and a part of radicular pulp under local or general anesthesia;
C. After devitalization of pulp - amputation of coronary pulp and mummification of radicular pulp;
D. Removal of coronary pulp and preservation of radicular pulp vitality – under local or general anesthesia;
E. Removal of coronary and radicular pulp under local or general anesthesia.

37. The essence of non-vital pulp extirpation method consists in the following:

A. Treatment of inflammatory process in pulp with medicamentous preparations;
B. Removal of coronary pulp and a part of radicular pulp under local or general anesthesia;
C. After devitalization of pulp - amputation of coronary pulp and mummification of radicular pulp;
D. Removal of coronary pulp and preservation of radicular pulp vitality – under local or general anesthesia;
E. Removal of coronary and radicular pulp after devitalization of pulp.

38. The most rational treatment method of pulpitis of temporary teeth at children aged above 5 years is:

A. Biological method;
B. Vital amputation;
C. Non-vital pulpotomy;
D. Non-vital pulpectomy;
E. Vital pulpectomy.

39. Devitalizing pastes (with arsenic) are contraindicated for application during the first visit in the treatment of:
A. Acute purulent pulpitis;
B. Acute pulpitis with involvement of periodontium tissues;
C. Chronic gangrenous pulpitis;
D. Chronic hypertrophic pulpitis;
E. Chronic fibrous pulpitis.

40. Application of biological method at children is limited by:
A. Absence of objective tests for diagnosing;
B. Insufficient efficiency of anesthesia;
C. Wide use of cements for fillings;
D. Big percentage of children with III rd activity degree of caries;
E. Late addressing by children.

41. Removal of altered dentine during biological treatment is performed:
A. Pneumatic machine (turbine);
B. Mechanic tool;
C. Excavator only;
D. Mechanic tool and excavator;
E. Mechanic tool only.

42. In case of acute focal pulpitis, when the cavity of a young permanent tooth is not opened, the following actions are necessary:
A. To open the tooth cavity and apply the method of direct sealing;

B. To apply the method of direct sealing;
C. To perform vital amputation;
D. To perform profound amputation;
E. To perform vital extirpation.

43. Antibiotics and sulfanilamide preparations are used in the biological treatment of pulpitis of permanent teeth for a period not exceeding:
A. 2 minutes;
B. 2 hours;
C. 24 hours;
D. 48 hours;
E. 3-7 days.

44. Glucocorticoids are used in the biological treatment of pulpitis of permanent teeth for a period not exceeding:
A. 2 minutes;
B. 2 hours;
C. 24 hours;
D. 48 hours;
E. 3-7 days.

45. Hormonal preparations are used for the biological treatment of pulpitis for a limited time, as they:
A. Possess antiphlogistic, antiseptic properties and facilitate the regeneration of pulp;
B. Possess antiphlogistic, dehydrating properties, hamper the regeneration processes in pulp;
C. Possess antiphlogistic, analgesic, anti-allergic properties, can disturb the hormonal status of organism;
D. Possess antiphlogistic, anti-allergic and plastics stimulation properties;
E. Possess antiphlogistic, plastics stimulation properties and disturb the immune state of organism.

46. Antibiotics are indicated for the biological treatment of pulpitis for a limited term, as they:
A. Can alter the color of dental crown;
B. Can provoke sensibility of organism;
C. Impede to the root formation;
D. Impede regeneration processes;
E. Can lead to the appearance of pulpal denticles.

47. For the medicamentous treatment of carious cavity in the biological method are used:
A. Sol. Furacillin 1: 5000, alcohol 70^0, ester;
B. Sol. Furacillin 1: 5000, Tripsin, Chlorhexidine 0.06 %;
C. Sol. Furacillin 1: 5000, Tripsin, Camphor-phenol;
D. Lysozyme, Dicaine 3%, Oxygenated water 3%;
E. Sol. Furacillin 1: 5000, Iodinol 1%, Dimexide 10 %, ester.

48. Complications of biological method by indication are caused by:
A. Incorrect choice of medicamentous preparations;
B. Insufficient sealing during filling of carious cavities;
C. Failure to observe the requirements of asepsis and antiseptics;
D. Incorrect technique.

49. Vital amputation of pulp is performed with:
A. A spherical bur with a pneumatic tool;
B. A spherical bur with an usual tool or with the excavator;
C. A cylindrical bur with an usual tool;
D. A pulp extractor;
E. Drill.

50. Hemostasis during vital amputation is done:
A. By diathermocoagulation;
B. With oxygenated water 3 %;
C. With 1 % sol. Adrenaline;

D. With orthophosphoric acid;
E. With temporary bandage.

51. For the vital amputation of pulp one shall use the pastes:
A. Endodont;
B. Camphor-phenol;
C. With antibiotics and sulfonamides;
D. With glucocorticoids;
E. Pastes based on calcium hydroxide.

52. The principal stages of the biological method of treating pulpitis are:
A. Preparation of carious cavity, application of arsenic paste, opening of tooth cavity, removal of coronary pulp, application of resorcin-formalin solution, application of resorcin-formalin paste, isolation material and permanent filling.
B. Anesthesia, preparation of carious cavity, medicamentous treatment, opening of tooth cavity, application of calcium hydroxide, application of isolation material and permanent filling.
C. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of pulp, hemostasis, application of calcium hydroxide paste, application of isolation material, permanent filling.
D. Preparation of carious cavity, medicamentous treatment, application of curative paste, application of isolation material, permanent filling.
E. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of coronary pulp, removal of a part of radicular pulp, hemostasis, medicamentous treatment, application of paste based on calcium hydroxide, filling of channel, application of isolation material, permanent filling.

53. The principal stages of vital pulpotomy are:
    A. Preparation of carious cavity, application of arsenic paste, opening of tooth cavity, removal of coronary pulp, application of resorcin-formalin solution, application of resorcin-formalin paste, isolation material and permanent filling.
    B. Anesthesia, preparation of carious cavity, medicamentous treatment, application of calcium hydroxide paste, application of isolation material and permanent filling.
    C. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of pulp, hemostasis, application of calcium hydroxide paste, application of isolation material, permanent filling.
    D. Preparation of carious cavity, medicamentous treatment, application of curative paste, application of isolation material, permanent filling.
    E. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of coronary pulp, removal of a part of radicular pulp, hemostasis, medicamentous treatment, application of paste based on calcium hydroxide, filling of channel, application of isolation material, permanent filling.

54. The principal stages of profound pulpotomy are:
    A. Preparation of carious cavity, application of arsenic paste, opening of tooth cavity, removal of coronary pulp, application of resorcin-formalin solution, application of resorcin-formalin paste, isolation material and permanent filling.
    B. Anesthesia, preparation of carious cavity, medicamentous treatment, application of calcium hydroxide paste, application of isolation material and permanent filling.
    C. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of

pulp, hemostasis, application of calcium hydroxide paste, application of isolation material, permanent filling.

D. Anesthesia, preparation of carious cavity, medicamentous treatment, application of curative paste, application of isolation material, permanent filling.

E. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of coronary pulp, removal of a part of radicular pulp, hemostasis, medicamentous treatment, application of paste based on calcium hydroxide, filling of channel, application of isolation material, permanent filling.

55. Principal stages of non-vital pulpotomy are:

A. Preparation of carious cavity, application of arsenic paste, opening of tooth cavity, removal of coronary pulp, application of resorcin-formalin solution, application of resorcin-formalin paste, isolation material and permanent filling.

B. Anesthesia, preparation of carious cavity, medicamentous treatment, application of calcium hydroxide paste, application of isolation material and permanent filling.

C. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of pulp, hemostasis, application of calcium hydroxide paste, application of isolation material, permanent filling.

D. Anesthesia, preparation of carious cavity, medicamentous treatment, application of curative paste, application of isolation material, permanent filling.

E. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of coronary pulp, removal of a part of radicular pulp, hemostasis, medicamentous treatment, application of paste based on calcium hydroxide, filling of channel, application of isolation material, permanent filling.

56. Principal stages of non-vital pulpectomy are:
    A. Preparation of carious cavity, application of arsenic paste, opening of tooth cavity, removal of coronary pulp, application of resorcin-formalin solution, application of resorcin-formalin paste, isolation material and permanent filling.
    B. Anesthesia, preparation of carious cavity, medicamentous treatment, application of calcium hydroxide paste, application of isolation material and permanent filling.
    C. Anesthesia, preparation and medicamentous treatment of carious cavity, opening of tooth cavity, amputation of pulp, hemostasis, application of calcium hydroxide paste, application of isolation material, permanent filling.
    D. Anesthesia, preparation of carious cavity, medicamentous treatment, application of curative paste, application of isolation material, permanent filling.
    E. Preparation of carious cavity, application of arsenic paste, opening of tooth cavity, removal of coronary pulp, removal of radicular pulp, hemostasis, medicamentous and instrumental treatment, filling of channel, application of isolation material, permanent filling.

Multiple compartment

57. Structural particularities of pulp at children are the following:
    A. Structure of coronary and radicular pulp is identical;
    B. Radicular pulp contains more fibers, less fundamental substance;
    C. Nervous myelinic fibers;
    D. Most of nervous fibers are non-mielynized;
    E. Contains many cellular elements;
    F. Contains fewer fibers;
    G. Is weakly vasculated.

58. Evolution particularities of pulpitis at children:
   A. Presence of residual pulpitis;
   B. Appearance of primary chronic pulpitis;
   C. Irradiating pains;
   D. Involvement of periodontium and regional lymphatic ganglions into the inflammatory process;
   E. Prevalence of chronic forms compared to the acute ones;
   F. Presence of acute pains.

59. Chronic forms of pulpitis of temporary and permanent teeth:
   A. Simple;
   B. Granulated;
   C. Hypertrophic;
   D. Proliferative;
   E. Gangrenous.

60. Preservation of growth zone vitality and subsequent root formation is possible if the following treatment methods are used:
   A. Non-vital pulpectomy;
   B. Biological method;
   C. Non-vital pulpotomy;
   D. Vital pulpotomy.

61. Indications for the use of biological method in the treatment of pulpitis of permanent teeth:
   A. Healthy child;
   B. Chronic evolution of carious process;
   C. Acute purulent pulpitis;
   D. Simple chronic pulpitis;
   E. Chronic hypertrophic pulpitis;
   F. Acute serous pulpitis;
   G. Electro-excitability of pulp 40-50 mkA.

62. Indications for the application of biological method in the treatment of pulpitis of temporary teeth:

A. Age – under 5 years;
B. Carious cavity - Class I Black;
C. Healthy child;
D. Chronic hypertrophic pulpitis;
E. Simple chronic pulpitis;
H. Acute serous pulpitis;
I. Acute evolution of carious process;
J. Chronic evolution of carious process.

63. Indications for the treatment of pulpitis of temporary teeth with use of vital amputation method:
A. Acute serous pulpitis;
B. Trauma;
C. Simple chronic pulpitis with activity degree III of dental caries;
D. Chronic gangrenous pulpitis.

64. Acute focal pulpitis is differentiated by:
A. Profound caries;
B. Chronic fibrous pulpitis;
C. Acute diffuse pulpitis;
D. Hyperesthesia of solid dental tissues;
E. Papillite.

65. Acute diffuse pulpitis is differentiated by:
A. Chronic exacerbated pulpitis;
B. Acute apical periodontitis and exacerbation of chronic forms of apical periodontitis;
C. Acute focal pulpitis;
D. Neuralgia of trigeminal nerve;
E. Profound caries.

66. Indications for the use of biological method in the treatment of pulpitis are:
A. Chronic fibrous pulpitis;

B. Accidental opening of pulpal chamber during the preparation of medium or profound carious cavities;
C. Acute focal (serous) pulpitis of permanent teeth;
D. Fracture of dental corona with affection of pulpal chamber, if no more than 6 hours have expired from the moment of trauma;
E. Chronic gangrenous pulpitis.

67. What relative contraindications exist for the application of biological method:
A. Age and behavior of the child;
B. Degrees II or III of carious activity;
C. General diseases of the child's organism;
D. Caries of activity degree I;
E. Localization of carious cavity.

68. In case of chronic fibrous pulpitis, when the cavity of a young permanent tooth is open, may be applied:
A. Direct pulp cap;
B. Indirect pulp cap;
C. Vital pulpotomy;
D. Profound pulpotomy;
E. Vital pulpectomy.

69. In the biological treatment of pulpitis are used the following preparations:
A. Antibiotics, sulfanilamide preparations;
B. Zinc-oxide-eugenol paste;
C. Preparations based on calcium hydroxide;
D. Analgesics;
E. Corticosteroids.

70. Particularities of preparation of carious cavities in the biological treatment of pulpitis:
A. Without pressure, with the simple tool;

B. At high rotations – with pneumatic tool;
C. Sterile burs must be changed frequently;
D. One must avoid penetration of buccal liquid into the cavity;
E. Periodic washing of carious cavity with weak antiseptics.

71. In case of accidental opening of pulp during the preparation of carious cavity shall be applied:
A. Paste with antibiotics;
B. Paste with hormonal preparations;
C. Calcium hydroxide-based paste;
D. Pastes with ferments and anesthetics;
E. Zinc-oxide-eugenol based pastes.

72. In cases of vital pulpotomy of pulp the following methods of anesthesia are used:
A. Applicative;
B. Infiltrative;
C. Trunk method;
D. General method;
E. All answers are correct.

73. The following preparations are used for the devitalization of pulp:
A. Resorcin-formalin;
B. Arsenic;
C. Paraformaldehyde;
D. Tricresol-formalin;
E. Vagotil.

74. Indications for the vital pulpotomy of pulp:
A. Chronic fibrous pulpitis of temporary and permanent teeth;
B. Accidental opening of pulpal chamber (during preparation);

C. Acute focal pulpitis of permanent teeth;
D. Chronic gangrenous pulpitis;
E. Fracture of dental corona with large opening of pulpal chamber.

Correct answers:

1.E.	26.E.	51.E.
2.B.	27.D.	52.D.
3.E.	28.A.	53.C.
4.D.	29.B.	54.E.
5.A.	30.D.	55.A.
6.B.	31.B.	56.E.
7.C.	32.D.	57. A, D, E, F.
8.D.	33.A.	58. B, D, E.
9.A.	34.E.	59.A, C, D, E.
10.A.	35.B.	60. B, D.
11.B.	36.C.	61.A, B, D, F.
12.B.	37.E.	62.A, B, C, E, H, J.
13.B.	38.C.	63. A, B, C.
14.C.	39.B.	64. A, B, C.
15.D.	40.D.	65.A, B, C, D.
16.A.	41.D.	66.A, B, C.
17.C.	42.B.	67.A, B, C, E.
18.C.	43.E.	68.A, C, D.
19.B.	44.E.	69. A, B, C, E.
20.D.	45.B.	70.A, C, D, E.
21.D.	46.D.	71. C, E .
22.C.	47.B.	72.B, C, D.
23.E.	48.D.	73.B, C.
24.E.	49.B.	74.A, B, C, E.
25.D.	50.C.	

8 APICAL PARODONTITIS (PERIODONTITIS)

Anatomic- morphological particularities of periapical radicular tissues at children

Periodontium is of mesoderm origin. Periodontium is observed for the first time already on the follicular development stage of the tooth, simultaneously with the beginning of root formation. It is developed simultaneously with the dental root and the compact lamina of the alveole. On the radiogram of a tooth with incompletely developed roots periodontium is found only in the developed region of root, but in the "palm" radicular area it is in fusion with the growth zone that is clearly delimited by the compact lamina of the alveolar bone. To the extent of development of the apical portion of root the dimensions of the growth zone gradually decrease to the very disappearance, simultaneously with the formation of radicular apex. The periodontium gets completely developed within one year from the end of formation of radicular apex. In this period the periodontal space is widened, especially in the apical region, constituting around 1-1.5 mm. Thickness of periodontium in average constitutes 0,2-0,25 mm; however, it can vary during the root development, depending on age, functional modifications and in result of pathologic processes.

The main anatomic-physiological particularities determining the dynamic evolution of inflammatory processes in the apical zone of periodontium are the following:

- Lack of stability in the apical region represented by a mixed tissue: between pulp and periodontium, established of weakly differentiated structures with high regeneration potential.

- The apical orifice is wide; the pulp is in contact with the alveolar bone.
- Taking into consideration the structural particularities of pulp and channel dimensions, any intra-pulpal inflammatory process can easily propagate into the alveolar bone, becoming thus an internal inflammatory process in bone.
- Roots can be at different development stages (in development, already developed and in resorption).
- The tissues of periodontium are immature, they contain many cellular elements, possibilities of delimiting the inflammatory process are very low, and this is why inflammation easily penetrates in all the tissues.
- The cortex of the alveolar bone is thin, the alveolar bone contains more medullar tissues, it is well vasculated and enervated, internal septa are thin, fine and elastic, the bone is less mineralized, contains comparatively more organic substances than inorganic, etc. All these lead to the rapid and massive destruction of periapical tissues
- The bones of maxillary-facial region at children are in permanent growth and reparative (regenerative) processes dominate over the resorption processes. Productive (proliferative) processes dominate at children.
- The periosteum is fragile, active, well vasculated, easily removable from bone and thus favors the continuous propagation of inflammatory process.
- Periapical tissues are abundantly vasculated, enervated, they contain a vast lymphatic network; however, they are immature.

Etiology of apical periodontitis

Apical periodontitis at children can be provoked by diverse factors:

1. Infectious ones;

2. Non-infectious ones:
   - mechanical (traumatic)
   - chemical;
   - allergic.

The etiological factors provoke the appearance and subsequently influence the evolution of pathological process. This is why the doctor must correctly identify the causes of the process and perform the treatment taking into consideration the etiology. Localization of process is also important. If the inflammatory process penetrates into the periapical tissues - apical periodontitis appears, if it enters the marginal periodontium – marginal periodontitis is determined. At children apical periodontitis can be provoked simultaneously by several etiologic factors: infectious, traumatic, chemical ones.

Infectious apical periodontitis

In the pedodontic practice most frequent form is the infectious periodontitis. The penetration ways for infection are different. Usually the germs, toxins and the pulp disintegration products penetrate into the periodontium via the radicular channel in cases of pulpitis and pulpal gangrene. The investigation showed that periodontitis is provoked by the polibacterial flora. Most frequently met microorganisms are the aerobic and anaerobic forms of streptococcus, staphylococcus, Veilonela, Lactobacteria, Leuriform fungi, etc.

In the process of tooth development the pulpal and periodontal tissues are in tight connection. To the extent of root formation histological are differentiated three types of tissues: coronary pulp (similar to the lax conjunctive tissue), radicular pulp (a denser conjunctive tissue) and the periodontium (even denser conjunctive tissue with greater content of fibrous structures).

The anatomic limits that separate these tissues are the orifices of radicular channels and apical orifices. To the extent of formation (narrowing) of these limits, the difference between

these tissues becomes more pronounced. Particularities of histological structures of these tissues determine the different characters of reaction to the inflammatory process. In the teeth with already developed roots the inflammatory processes are often stopped not only in the coronary pulp, less often in the radicular pulp, while the periodontium remains unaffected. In the teeth with incompletely developed roots or on the stage of radicular resorption these tissues are insufficiently developed (and thus are less differentiated) the involutive and resorptive processes begin and the anatomic points delimiting them are wide. This is why during these periods the inflammatory process in pulp is diffused and easily penetrates into the periodontium. Respectively, acute periodontitis at children appears in result of pulpitis not treated in due time. Also the infection can penetrate into periodontium via the periodontal saccules. Sometimes the inflammatory process can penetrate into the periodontium with blood in cases of some acute infectious diseases: flu, angina, scarlatine, etc. Simultaneously with periodontium is often affected the radicular pulp. The inflammatory process can continuously be propagated with sinusitis, osteomyelitis, etc. The inflammatory process in the apical periodontal tissues plays a role of a barrier, preventing the infection from penetrating into the depth.

Traumatic apical periodontitis

It appears in the result of some important traumatic mechanic action: punch, fall from height, etc. In such cases the frontal teeth are affected. Also traumatic apical periodontitis can be caused by repeated or permanent micro traumas: high fillings, orthodontic devices, pathology of occlusion, pernicious habits of nibbling solid objects, musical instruments, etc, as result of incorrect endodontic treatment: affection of periodontium with radicular needles, brutal and profound extirpation of pulp – these can cause a detachment of periodontal tissues and obturation of channel

after apex.

Chemical apical periodontitis

Pathology of this kind appears during and after the treatment of pulpitis, being caused by the action of arsenic-based preparations and antiseptics: formalin, phenol, tri-cresol-formalin, etc., materials for the filing of channels. Having established the diagnosis of acute arsenic pulpitis one must take into account the fact that the symptoms of acute inflammatory process in periodontium can be provoked by the direct toxic action of arsenic over the periodontium and in the sequence of spreading of the inflammatory process in the pulp. It is known that the devitalizing action of arsenic during the first 1-2 hours is manifested by paresis and followed by the paralysis of nervous terminations of vessels (vessel constricting and dilating action). Appear the conditions simulating the progress of inflammatory process in the pulp.

Allergic apical periodontitis

Appear at children with increased sensitivity of the medications used in the endodontic treatment. Medications with complex chemical structure or protein composition act as antigens, leading to the formation of antibodies. Substances with simple chemical structure (haptens) in the organism can associate with proteins of sanguine serum and under the complex antigenic form provokes the formation of antibodies. In a stage-by-stage contact with these medications appears the specific allergic reactions (local or general). Most often they are provoked by antibiotics, eugenol, iodine, formalin, etc.

Pathogenesis of apical periodontitis

Acute inflammatory process in the periodontium of temporary and permanent teeth evolves in correspondence with the general laws characteristic for the acute inflammatory process

in the conjunctive tissues. The character of evolution of the inflammatory process in periodontium depends on the intensity and duration of etiologic factors, on the particularities of the affected zone and on the reactivity state of the child's organism.

The initial period of acute serous periodontitis is manifested by an edema, pronounced hyperemia, and migration of leukocytes from the sanguine vessels into the surrounding tissues. In line with the development of process the periodontium becomes infiltrated with serous exudation with an insignificant number of neutrophyl leukocytes.

Continued evolution of the inflammatory process in periodontium leads to the transition of the affection from serous into purulent form that is characterized by pronounced hyperemia, focal or diffuse infiltration of leukocytes; disorientation of collagen fibers in some of the sectors, loss of fuxinophilia, destruction and decomposition of fibers.

In result of action of pathological factors on the periodontium in the affected zone appears an inflammatory process: are formed and concentrated the mediators of inflammatory process (histamine, serotonine, acetylcoline, etc.) and the proteolytic tissular ferments (leukotoxin, hialuronidase, collagenase, necrozine, exsudine,etc.) that lead to the alteration of the structures of periodontium and disorders of circulation and metabolism.

Morphologically, in the apical region during inflammatory processes (by A. Marmasse, 1974), 4 zones can be distinguished: infection zone, contamination zone, irritation zone and stimulation zone. In the infection zone microorganisms can be found and identified. In the contamination zone are found the cells and their fragments destroyed by bacterial toxins, a considerable number of lymphocytes, no microorganisms are detected there. In the irritation zone the levels of toxins that penetrate through the lymphocyte barrier are reduced, among

cellular elements dominate the histiocytes and the osteoclasts that destroy the fibers of collagen and lead to the bone resorption (progressive resorption leads subsequently to the delimitation of nidus in the bone tissue). In the stimulation zone the concentration of toxins is considerably smaller, the irritation effect disappears, intense activity of fibroblasts and osteoblasts is observed, and they produce fibers of collagen and peripheral bone.

For the temporary teeth can be characteristic the following particularities: existence of wide communication ways between the infected pulp and periodontium allows for the permanent aggression of microorganisms and toxins (especially in the period of underdeveloped root or resorption). Particularities of the apical zone facilitate the extension of exudation; radicular resorption associated with inflammatory process leads to the growth of the activity of osteoclasts, to the permanent destruction of bone that becomes replaced by granulated tissue; the limitation and recovery trends in the periapical tissues are weaker.

In parallel with the alteration and exudation processes appear the processes of proliferation characteristic for chronic and exacerbated inflammation. Depending on these, one or another form of chronic periodontitis can appear: granulated, granulomatous or fibrous. The proliferation process ends with the formation of granulated tissue that subsequently is transformed into fibrous tissue. To the extent of evolution of the inflammatory process take place the qualitative changes of the character of exudation (serous, purulent, and hemorrhagic) and of the diffusion ways. Most often exudation is spread via the radicular channel, via the periodontal channel, through the bone, through the sub-periosteum, and sub-mucous.

Inflammatory processes in the periodontium depending on pathological and clinical evolution can be:

1. Acute (serous or purulent) characterized by predomination

of alterative-exudation processes, with shorter but more intense evolution;

2. Chronic characterized by proliferative-regenerative processes, slow evolution and weakly pronounced clinical image.

CLASSIFICATION OF APICAL PERIODONTITIS

Classification of periodontal affections by WHO

K.04. Maladies of periapical tissues

K.04.4. Acute apical periodontitis of pulpal origin
FAJ acute apical periodontitis (as infection)

K.04.5. Chronic apical periodontitis
Apical granuloma

K.04.6. Periapical abscess with fistulae.
Including: - dental abscess with fistula;
- dental-alveolar abscess with fistulae;
- periodontal abscess of pulpal origin.

K.04.60. Open fistula in the maxillary sinus

K.04.61. Open fistula in the nasal fossa

K.04.62. Open fistula in the buccal cavity

K.04.63. Fistula on skin

K.04.69. Periapical abscess with unidentified fistula

K.04.7. Periapical abscess without fistula
Dental abscess
Dental-alveolar abscess
Periodontal abscess of pulpal origin without fistula

K.04.8. Radicular cyst
Including: Apical cyst
Periapical cyst

K.04.80. Apical and lateral cyst

K.04.81. Residual cyst

K.04.82. Paradental inflammatory cyst

K.04.9.Other imprecise affections

Classification of apical periodontitis of temporary and permanent teeth at children by T. Виноградова:

I. Localization of process:
- apical;
-marginal;

II. Etiology:
- infectious;
- traumatic;
- toxic;
- medicamentous.

III. Clinical evolution:
- acute: a)serous; b) purulent.
- chronic: a) fibrous; b) granulated; c) granulomatous.
- exacerbation of chronic forms of periodontitis.

IV. Pathological-morphological changes:
- acute inflammation: serous, purulent;
- chronic inflammation: fibrous, gangrenous, proliferative.

To the opinion of some researchers (W. Hess, 1955; H. Rebel, 1950, etc.) the notion "periodontitis" doesn't fully describe the inflammatory process that occurs not only in the periodontium but also in the adjacent tissues.

Classification of apical periodontitis by H. Taatz, 1975

1. Acute apical periodontitis.
2. Chronic apical periodontitis:
   a) edematous thickening of periodontium;
   b) diffuse resorptive ostitis;
   c) limited resorptive ostitis (granuloma);
   d) progressive granulated;
   e) radicular cyst;
   f) sclerotic ostitis.

Incidence of apical periodontitis at children

Periodontitis is most often identified at children in the cases of lack or insufficient organization of regular treatment care of buccal cavity at children. At children, the same as at adults, the chronic or exacerbated forms of apical periodontitis prevail. Among chronic ones most often are found the granulated ones, less often - granulomatous and fibrous ones (see color insert fig. 8.1).

At temporary teeth prevails the granulated form, while the granulomatous and the fibrous ones are found in very rare cases.

The incidence of apical periodontitis grows in accordance with age. If at the age of 2-3 years the apical periodontitis is diagnosed at 0.1% of patients, at the age of 7-8 years it is found at 20-30 % of children. Periodontitis of young permanent teeth at the age of 6-9 years is found at 24 % of children, while at the age of 10-14 years its incidence reaches 71-72 %.

ACUTE APICAL PERIODONTITIS

At children the acute apical infectious periodontitis evolves in parallel with acute diffuse pulpitis or chronic exacerbated pulpitis (gangrenous pulpitis).

Accuses: Permanent and localized pains, intensifying with evolution, pulsing pains, becoming more pronounced during mastication or intercuspidation. At children of relatively smaller ages is possible the rapid infection of periodontal tissues with generalized clinical manifestation: symptoms of general intoxication, fever of 38°C and greater. Children become capricious, whining, loose appetite, are afraid to open the month, have disorders of sleep, etc. The teeth become mobile, very painful at touch, progressive edema of soft facial tissues and growth of regional lymphatic ganglions are observed. If during the first several hours no medical assistance is provided, acute periostitis or acute osteomyelitis are developed.

Objective characteristics: Usually the children can point at the affected teeth that may have medium-sized or deep carious cavities with damaged and pigmented dentine. The carious cavity in most cases doesn't communicate with the pulpal cavity, its probing being not painful. In some cases the teeth can be unaffected or obturated. Percussion is painful. The teeth can be mobile. Alveolar gums are hyperemic and swollen in the region of the affected teeth that at palpation in the projection of radicular apex is painful. In some cases on the passage patch an infiltrate can be detected, while in more severe cases a collateral edema of soft facial tissues may be found. Lymphatic ganglions are bigger in size, slightly painful and mobile. In the periodontitis of inferior incisors sub-mental lymphatic ganglions are increased, periodontitis of superior incisors, canines and superior or inferior premolars causes the increase of the anterior sub-mandibular lymphatic ganglion respective to the side of the process. In case of periodontitis of superior or inferior molars are increased the medial and the posterior sub-mandibular ganglions.

Blood analysis: leukocytosis raised RSE.

EOD: above 100 mkA.

Radiological signs: usually the changes can not be detected, sometimes an insignificant increase of periodontal space is found.

Depending on the etiology the clinical image of the acute periodontitis at children can have individual specifics.

Acute traumatic periodontitis (mechanical)

To a considerable extent the clinical image is determined by the state of dental pulp after the action of acute trauma. In the case of vital pulp the evolution of the process becomes smoother, and the vindication perspectives are better. In case of pulpal necrosis after the trauma the periodontium is infected and clinical signs of acute apical infectious periodontitis appear.

Affection of periodontium at children can be caused by brute use of endodontic tools or by the obturation of channels beyond

the limits of apical orifice.

Acute medicamentous periodontitis

At children more often is found the acute periodontitis provoked by the action of arsenic-based medications that can occur due to overdosing or over-exposure (more than 48 hours) during the treatment of pulpitis. Children have accuses on the permanent pains that get stronger during intercuspidation. Usually the percussion of tooth is moderately painful.

Relatively rare are the cases of acute periodontitis caused by preparations provoking necrosis of periodontium (powerful antiseptics, tri-cresol-formalin, etc.) or endodontic obturation materials (eugenol, phenol, etc.). Medicamentous periodontitis has a persistent evolution and highly resistant to therapy.

CHRONIC APICAL PERIODONTITIS

Chronic apical periodontitis at children is a consequence of acute periodontitis or chronic gangrenous pulpitis. Chronic periodontitis is often associated with chronic forms of pulpitis (especially gangrenous ones) due to the anatomic-histological particularities of the roots of temporary teeth and tight connections between the radicular pulp and periodontium. The chronic inflammatory process in pulp through the apical orifice or supplementary channels in the region of bifurcation are propagated into the periodontium, involving the periodontal apical tissues, the alveolar bone and the follicles of permanent teeth. Due to this reason the chronic apical periodontitis can be diagnosed at teeth with pulpitis, teeth with obturated radicular channels, teeth with obturated carious cavities, etc. They are often primary-chronic processes not preceded by acute inflammatory processes. Very often this diagnosis is established during the prophylactic examination of children, on radiograms, in cases of appearance of fistulas on the gums, etc.

Clinical evolution particularities of chronic periodontitis at children

- found at teeth with superficial carious cavities (superficial to medium) , with close dentinary cavity, both at temporary and permanent teeth;
- chronic granulated forms prevail, at temporary teeth they affect the follicles of permanent teeth;
- fistulas often appear in cases of chronic granulated forms of periodontitis of temporary teeth;
- destructive process at temporary teeth is localized in the region of radicular bifurcation (trifurcation) or is propagated into the apical region of the two or three roots
- chronic periodontitis of teeth with incompletely developed roots can slow down or even stop the apexogenesis;
- the fibrous form is not found and the granulomatous form is very rarely found at temporary teeth.

Chronic apical periodontitis displays a very vague clinical image. Often the children do not have accuses at all, the affected teeth are used in mastication and do not react to thermal agents.

At examination the affected teeth can appear intact or can have carious cavities of different depth, sometimes obturated. Dental corona can be destroyed considerably or completely. The enamel color is modified (with gray nuances and opaque appearance). At examination an edema is detected, hyperemia of gum in the region of tooth, sometime fistulas with purulent elimination and granular tissue. Probing of carious cavity is painless. In cases of granulated periodontitis in the dental cavity some granulated tissue can be observed that at probing is slightly painful and possibly, bleeding. Percussion of tooth is painless, percussion sound is damped. The tooth can be mobile.

The main distinct signs of chronic apical periodontitis allowing determining not only the existence but also the character and the spread of the process, the state of surrounding tissues are

the radiological images. Only radiologically one can determine the form of chronic apical periodontitis:

1. Granulated - one nidus of alveolar bone destruction with affection of cortical plate, with regular borders, candlé fire – shaped.

2. Granulomatous - one nidus of alveolar bone destruction with affection of cortical plate, with regular borders, of spherical or oval shape;

3. Fibrous - widening of periodontal space, mainly in the region of radicular apex.

At children most often is found the granulated form. Granulomatous periodontitis is rare, while the fibrous periodontitis is not found at all at temporary teeth.

Granulated periodontitis of temporary teeth is typically localized in the region of radicular bifurcation (trifurcation). The character of the process, its localization and structural particularities of the bones at children determine the rapid propagation of inflammatory process into the surrounding tissues.

Massive nidus of destruction of the bone can occupy the entire space around the roots and get propagated over the buds of permanent teeth. For such states at children was proposed the notion of chronic granulated ostitis (by Виноградова Т., 1978).

Complications of apical periodontitis characteristic for temporary teeth

- Periostitis, osteomyelitis of maxillaries;
- Necrosis of permanent dental buds;
- Follicular cyst;
- Lymphadenitis;
- Formation of a chronic nidus of odontogenous infection;
- Modifications of radicular resorption;
- The Turner's dental syndrome;
- Displacement of the permanent dental bud with subsequent formation of position anomalies.

For the periapical inflammatory processes are characteristic the disorders of physiological radicular resorption, often-pathological resorption of roots of temporary teeth. Pathological resorption of temporary teeth is provoked by the giant poly-nucleic cells and cells of the inflammatory infiltrate. Processes of osteogenesis are minimaly and remain behind the radicular resorption. This is why in cases of pathologic resorption the main radiologically detectable signs are the destruction and the decomposition of bone in the region of bifurcation of molars or around the roots. Simultaneously with the progress of pathological process the roots of temporary teeth and the follicles of the permanent teeth withdraw from each other while in physiological resorption they get closer. Interference of pathological resorption with physiological resorption can lead to the loss of rhythm, to continuous and rapid resorption of roots. In cases of pathological resorption can start the resorption of temporary teeth with undeveloped roots, resorption of neighboring teeth, etc. The process of pathological resorption can spread onto the follicles of permanent teeth; can result in the premature resorption of the compact lamina of dental follicle and resorption of permanent tooth. Under the influence of chronic apical periodontitis can appear the follicular cysts, focal hypoplasia of permanent teeth, position anomalies of permanent teeth, etc.

Also under the action of pathologic process various disorders of structure and shape of permanent teeth can appear. One of the first authors to describe such teeth was Turner. Most often are affected the second premolars, such affections being characterized by insufficient development of the dental corona, the teeth are cask-shaped, lack of enamel (aplasia), change of color (brown), dental surface defects, etc. The Turner's dental syndrome is found in relatively rare cases regardless of quite high incidence of apical periodontitis, as the appearance if this syndrome is determined by

the extension of osteitis within the limits of permanent dental follicle and degree of its mineralization. If a half of the dental corona is mineralized the danger of affection of the permanent dental follicle is very small. The first permanent premolar can be affected till the age of 4 years while the second premolar can be affected till the age of 5 years.

TREATMENT OF APICAL PERIODONTITIS OF TEMPORARY TEETH

Treatment of apical periodontitis at children is associated with numerous difficulties conditioned by the anatomic-physiological particularities of temporary teeth and tissues of apical periodontium during the different development periods, being simultaneously determined by the processes of growth and radicular resorption, general state of health, behavior, etc. When treating apical periodontitis one must take into consideration the factors that may provoke physiological imbalance in the child's organism (cooling, tiredness, acute infectious diseases, allergic reactions, etc.). At children suffering from chronic diseases (rheumatism, chronic nephritis, cardiac vices, etc.) chronic periodontitis is often exacerbated or can intensify the general disease.

The success of treatment and perspectives of the apical periodontitis are to a considerable extent determined by the reactivity of the child's organism, by the efficiency of medicamentous methods and preparations used in the endodontic treatment, by the assurance of painless endodontic manipulations.

Treatment of such affections at children implies the use of surgical method (dental extraction), the conservative method and the mixed methods (apical resection, hemisection, etc.). For temporary teeth dental extraction or conservative treatment are used.

When determining indications for the conservative treatment of apical periodontitis at children the main criterion is not the

child's age or the terms of permanent teeth' eruption but the character and the spread of the inflammatory process, the degree and the mode of radicular resorption of the temporary teeth, the extension of the inflammatory process on the buds of permanent teeth and the child's general state of health. Choice of rational method must be done individually taking into consideration all these factors.

Conservative treatment is generally more rational as it is aimed at the rehabilitation of apical periodontal tissues and preservation of the tooth with its real functional value. The compulsory conditions for an effective conservative treatment of apical periodontitis are: accessible radicular channel for the mechanic and medicamentous treatment with subsequent obturation in the limits of apical orifice.

The main scopes followed in the treatment of apical periodontitis at children consist of:

1. Exclusion of the cause or reduction of its action (extirpation of inflamed pulp, treatment of pulpal gangrene, removal or inhibition of the toxic medications (arsenic preparations, etc.), etc.
2. Choice of the most rational way for the evacuation of exudation;
3. Action over the microflora of macro- and micro –dentinary channels;
4. Neutralization of action of biogenic amines;
5. Removal of inflammatory process from periodontium;
6. Obturation of macro- and micro –dentinary channels;
7. Stimulation of reparative processes in the tissues of apical periodontium and root formation.
8. Increase of the reactivity of child's organism.

Treatment of the acute forms of apical periodontitis of temporary teeth

The main scope of the treatment of acute apical periodontitis

consists in the creation of drainage for exudation from the periodontal space. Drainage can be assured in three ways: through radicular channel; through the alveolar bone, sub-periosteum, sub-mucosal or formation of fistula; through the periodontal space or through the dental alveoli (in case of dental extraction). The most preferred method is through radicular channel as it allows the vindication of the inflammatory process and the preservation of tooth. The most radical method is dental extraction.

Contraindications for the conservative treatment of apical periodontitis at children:

1. Lesion of the integrity of compact lamina of the permanent tooth bud.
2. Pathological resorption above 1/3 of the root length.
3. Affected tooth- cause of the periostitis or osteomyelitis.
4. Affected tooth – cause of septicemia.
5. Apical periodontitis of temporary incisors with incompletely developed roots.
6. Complete destruction of dental corona.
7. Perforation of the dental cavity floor.
8. Inefficiency of conservative endodontic treatment.
9. Loss of functional importance of the temporary tooth if there are 1.5-2 years to its physiological substitution.
10. Chronic infectious-allergic diseases of the child if the affected tooth is a source of odontogenous infection.

Principles of treatment of acute apical periodontitis

Treatment of acute apical periodontitis implies:

1. Removal of cause;
2. Provision of drainage for exudation.
3. Instrumental and medicamentous treatment in order to favor the transition of acute form into the chronic one.
4. Treatment of chronic inflammatory process. Tactics of treating the apical periodontitis depends on the etiologic factor, on

the extension and the gravidity of clinical evolution of inflammatory process, on the general state of the child's health.

Stages of treating acute apical periodontitis

Visit I. Using the turbine one must open wide the dental cavity, remove the infected or necrotizing pulp, treat the dental cavity with antiseptic preparation and use the pulp extractors to remove the necrotizing masses from the radicular channel. Antiseptic and instrumental preparation of the radicular channel is required. For a tooth with developed apex it is necessary to open the radicular apical orifice in order to assure the drainage from periodontal space. The tooth remains with open cavity. Rinsing with sol. 1 % sodium bicarbonate, 0.2 % chlorine-hexetidine, etc.

In case of sub-gingival abscess or sub-periosteum abscess an incision under local anesthesia is required. The incision is followed by endodontic drainage. Treatment with sulfanilamide, calcium gluconate, analgesics is indicated.

In more severe cases of purulent periodontitis or in the absence of improvements after endodontic drainage and incision the child must be hospitalized and treated in the oro-maxillary-facial section.

Visit II. Instrumental and medicamentous processing of dental cavity revision of radicular channels and their treatment with antiseptics. For the antiseptic treatment of radicular channels are used the solutions: ectericide, chloramine 2 %, chlorhexetidine 0.5 %, iodinol, decamine 0.2 %, etonium 1 %, etc. Also are recommended the solutions with proteolytic ferments: trypsine, chimotrypsine, lyzozyme. Instrumental treatment of channels is performed with special care and attention, with endodontic tools corresponding to the thickness of radicular channel, taking into consideration the length of channel, in order to prevent the traumas of apical zone and of the follicles of permanent teeth.

Hermetic medicamentous bandage or obturation of channels

can be performed only after liquidation of acute inflammatory process, i.e. when there are no pains at percussion and then the child's general state of health is normalized. The channels of temporary teeth are obturated within the limits of the apical radicular orifice. For the obturation of channels of temporary teeth resorbable pastes are used: zinc-eugenol, thymol-based pastes, iodinol-based pastes, Ghenis paste, etc.

Physiotherapeutic methods are rarely used in the treatment of apical periodontitis of temporary teeth.

Treatment of medicamentous apical periodontitis

Toxic periodontitis of temporary teeth is most often provoked by the arsenic preparations when treating pulpitis with use of devitalizing methods. Treatment includes preparation of carious cavity, wide opening of pulpal chamber, amputation and extirpation of radicular pulp, instrumental processing and antiseptic treatment of channels, application of arsenic antidote (5% unithyol, sol. Iodine 5 %, sol iodinol 1%, etc.) under hermetic bandage. After liquidation of pain at intercuspidation and percussion the channels are obturated.

Treatment of chronic forms of apical periodontitis of temporary teeth

The methods of treating chronic periodontitis of temporary teeth with developed roots and permanent ones do not have major differences. The main difference consists in the choice of endodontic obturation material. For the obturation of channels of temporary teeth one uses pastes with prolonged antiseptic action that are resorped in agreement with the radicular resorption. Treatment of chronic periodontitis of temporary teeth on the stage of formation or radicular resorption must begin only after the performance of radiography allowing determining the efficiency of conservative treatment and the length of radicular channel.

During the first visit the carious cavity is prepared; the pulpal

chamber is open wide in order to assure access to the radicular channels and to remove the necrotizing material from there. If the channels are large enough, one can use simultaneously 2-3 pulp extractors. These operations must be done with major caution in order to prevent the necrotizing materials from penetration into periodontium and avoid the lesions of periapical tissues that are associated with pains and hemorrhage. In order to prevent the exacerbation of chronic inflammatory process during the treatment the instrumental processing must be performed only after a good medicamentous treatment of the dental cavity. Medicamentous treatment of the dental cavity is performed with antiseptic non-irritating solutions: sol. 1 % iodinol, sol. 3 % oxygenated water, sol. 2.5 % sodium hypochlorite, sol.2 % chloramine, sol.0.5 % chlorhexidine, 1 % metronidasol suspension, etc. Also for these purposes may be used the ferments in combination with antimicrobial preparations: solutions of trypsin, chimotrypsine, chimopsine, terylitine, lyzozyme in combination with microcide, monomycine, streptomycine solutions, etc.

At temporary teeth can be used the treatment of radicular channels with solution of resorcin-formalin or silvering method. However, during the period of radicular resorption complications of toxic nature or exacerbation of inflammatory processes can occur. In case there is any granular tissue in the channel (often observed at temporary teeth) it is recommended to extirpate it under local anesthesia or intra-channel anesthesia with solution of phenol-anesthesine, with sol, camphor-phenol, etc. Anesthetic solutions are introduced with precautions into the channel with the help of slightly impregnated cotton tampons in order to avoid the chemical lesion of the buccal mucous tissues.

Instrumental processing is aimed at the removal of infected dentine of the walls of the radicular channel. Different endodontic tools are used for this purpose. The choice of tools is made

depending on the dimensions of the channels (thickness, length). The permeability of channels of the temporary teeth is usually not difficult as the channels are wide, however, at molars the curve angle of the channels can be significant. This is why the widening and passage of the channel can easily result in the perforation of root or apical orifice followed by trauma or ingress of necrotizing tissues into the periodontium. In order to avoid such complications certain concessions are made in the mechanical processing of channels. The treatment continues with obturation of radicular channels. Before obturation the channel must be dried with cotton tampons or paper cones. Endodontic materials used for the obturation include: zinc-eugenol- based pastes, Walkoff paste, iodoform paste, thymol, etc.

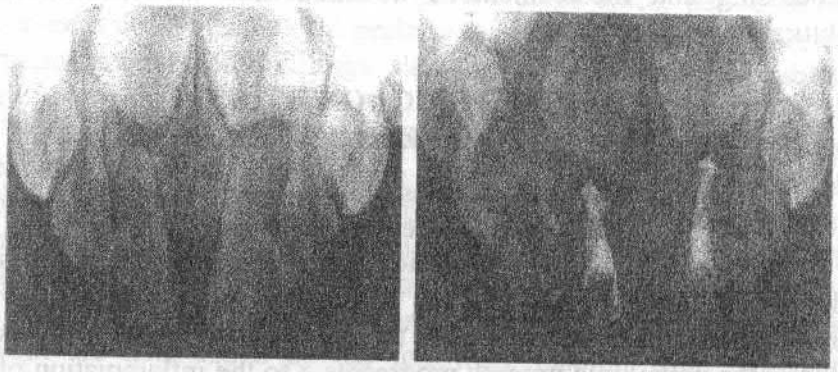


Figure 8.2. **Radiographic image. Treatment of chronic granulated periodontitis of 51 and 61.**

Chronic granulated periodontitis of temporary teeth with permeable channels or during the period of physiological resorption of roots, in the presence of fistula or of a granulated tissue in the channel must be treated in one visit. When the channels are not completely permeable and can not be processed instrumentally, in cases of intense pains or when the child is

agitated, as well as in the cases of low resistance of organism (acute infectious diseases, dyspepsia, etc.) treatment is performed in two visits. During the first visit after the opening of pulpal chamber the necrotizing masses are removed from the dental cavity and orifices of channels. A small tampon with solution of camphor-phenol or resorcin-formalin is inserted under bandage and left for 2-3 days. During the second visit, after the removal of infected residuals from channels, mechanic processing and medicamentous treatment the channels are obturated.

It is not rational to treat temporary teeth with pulpal gangrene in a single visit. In such cases the channel is treated with 1 % suspension of metronidasol or bactrim emulsion and left there in the channel for 1-2 days. During the second visit after mechanical processing and medicamentous treatment and the channel is obturated.

APICAL PERIODONTITIS OF PERMANENT TEETH AT CHILDREN

Etiology and pathogenesis of apical periodontitis of permanent teeth at children

The most often cause of periodontitis of permanent teeth is the dental caries. Germs and the products of their vital activity induce inflammatory processes in the pulp and lead to its necrosis and as the pathologic process progresses – to the inflammation of the periodontal tissues. According to Т. Виноградова the appearance of periodontitis is provoked by associations of bacterial germs of 2-5 species. The streptococcus germs (98.8%), Gram-positive bacillus (56%), staphylococcus germs (27.5 %), diplococcus germs, fungi, etc. predominate in the necrotizing pulp. Such associations of microorganisms aggressively affect the periapical tissues by producing ferments: hialuronidase, fibrinolyzine, plasmocoagulase, etc. An important role in the initiation of pathological process is played both by the bacterial

germs and their toxins n the radicular channels and by the biogenic amines – products of decomposition of pulpal tissues and dentine.

Periodontitis of permanent teeth can appear in teeth with obturated channels during or after the treatment of pulpitis (lesion of periodontium with endodontic tools, traumatic amputation or extirpation of pulp, obturation of radicular channel beyond the apical level, action of medicamentous preparations and endodontic materials on the periapical tissues, etc.).

Periodontitis of frontal superior and inferior teeth is often caused by traumas (contusion, coronary or radicular fractures, etc.). Acute dental traumas at children can occur in result of hitting against various objects, falls during sportive activity, recreation, road accidents, etc. In rare cases apical periodontitis can be a consequence of a prolonged action of permanent dental micro-traumas (high fillings, dental coronas, pernicious habits, etc.).

In relatively rare cases apical periodontitis is a result of exerting excessive force during orthodontic treatment, or of the premature dental eruption, of the propagation of inflammatory process from the nidus of the neighboring tooth and destruction of the periodontal tissues, of the radicular cyst pressure, etc.

Particularities of pathogenesis of periodontitis of permanent teeth are determined by the following factors: wide radicular channels, presence of a weakly mineralized dentine layer (predentine) that during the period of root formation exceeds the thickness of strongly mineralized dentine; direct connection of pulp with the periodontium on all development stages of permanent teeth, especially during the period of root formation; a significant number of capillaries and cellular elements in the periodontium; porous structure of the cortical alveolar bone; weak mineralization of the alveolar bone and its large medullary spaces, etc.

All these particularities in ensemble induce the break-out and the rapid propagation of the inflammatory process in periodontium by spreading and action of bacterial germs and toxins in the radicular channel.

SYMPTOMATOLOGY OF ACUTE APICAL PERIODONTITIS OF PERMANENT TEETH AT CHILDREN

From the clinics and pathological anatomy point of view there can be distinguished three successive phases of development of acute apical periodontitis (by M. Грошиков). Phase one is the periodontium intoxication phase (in case of acute diffuse pulpitis, acute purulent pulpitis, etc.). Infection has not yet penetrated into the periapical tissues, but reactive hyperemia of sanguine vessels is already present. Children feel the heaviness and tension in the region of the affected tooth that becomes "bigger and higher" than other teeth. Simultaneously with the beginning of treatment irritation of periodontium gradually reduces.

The phase two is characterized by the appearance of exudation serous process. It is manifested by progressive evolution, debuting with permanent moderated and localized pains. Pains usually become more intense during the nighttime and practically are not controlled by analgesics. At the incipient stage the children can point at the affected tooth. The affected tooth has a medium or profound carious cavity. Usually the carious cavity doesn't communicate with the dental cavity. Probing of carious cavity is painless. Sometimes the tooth may be intact but have a modified color, or filling. Percussion of tooth is very painful. The tooth can be mobile. The gum in the region of affected tooth is swollen and hyperemic, painful at palpation. In more advanced cases of the inflammatory process on the vestibular passage fold in the region of projection of the affected tooth inflammatory infiltrate and collateral edema of soft tissues

can be observed. Regional lymphatic ganglions are enlarged, mobile and slightly painful at palpation.

Acute medicamentous periodontitis (toxic periodontitis) at children appear most often during or after the treatment of pulpitis with use of arsenic (overdosing, late removal of pulp after application of arsenic, etc.). In cases of overdosing and over-exposure the children can mention that the acute pains have disappeared but on the second or third day the pains resumed and gradually intensified, especially during mastication or intercuspidation. Percussion of the affected tooth is weakly or moderately painful.

Acute medicamentous periodontitis at children can appear during or after the treatment of pulpitis with use of powerful disinfecting substances (tri-cresol-formalin, phenol, etc.) or endodontic pastes (based on tri-cresol-formalin, eugenol, etc.) provoking irritation of periapical tissues. Such affections are characterized by intensive, permanent pains resistant to therapy. Affections can transit from serous into purulent form.

The clinical image of mechanic (traumatic) periodontitis to a considerable degree is determined by the state of pulp after the traumatic action. If the pulp is vital, the process evolves in an easier way and the vindication perspective is more favorable. In case of pulpal necrosis infection always appears followed by intoxication of periodontal tissues and later takes one or another form of infectious periodontitis.

In cases of mechanical lesion when processing the radicular channel with endodontic tools bacterial germs penetrate into the periodontium and shortly provoke a purulent process in there. In case of obturation of the radicular channel beyond the apical level the serous inflammatory process appears.

The third phase of acute periodontitis is the purulent one. Compared to the serous one it is characterized by vertiginous evolution with symptoms of general intoxication. In the beginning

the pains are localized, permanent, however, gradually they get more and more intense, can become irradiating and in all cases the child can point at the affected tooth that is "higher" than others and is very painful at any touch. The child avoids any contact with the tooth and doesn't masticate food on the affected part. Sometimes the children are afraid to close the mouth, to swallow the saliva as it presumes dental occlusion, while the saliva flows out of buccal cavity (reflex ptialism). Pains become stronger at warm and become less intense at cold. Also they get intensified in the horizontal position.

At examination the tooth can be intact, but have somehow different color, most often the affected tooth is carious or filled. The pulpal chamber is open or close. Probing is painless. Percussion is very painful. The tooth may display accented mobility and can be "higher" compared to the neighboring ones. The mucous tissue in the region of root projection is hyperemic and swollen. Fetid smell from buccal cavity is felt. In the region of passage fold inflammatory infiltrate can be observed, such infiltrate being painful at palpation. Facial asymmetry, collateral edema of soft tissues is observed.

In cases of purulent periodontitis the regional lymphatic ganglions are always enlarged, mobile and painful at palpation. In contrast to serous form, the child's general state is difficult, with symptoms of general intoxication: headache, insomnia, absence of appetite, and fever up to 38-39^{0}C. As the pains are strong, the child can not asleep, can not eat and in result of intoxication appear the signs of tiredness: pale skin, dark rings around the eyes, etc.

If no urgent stomatological assistance is provided, the inflammatory process wills rapidly progress and turn into periostitis, osteomyelitis of maxillaries, phlegmon, etc.

Blood analysis: leukocytosis 15-25 x 10/l with deviation of leukocytary formula to the left; VSE after 2-3 days is increased

(20-30 mm/hour).

Radiologically the modifications can be detected after 24-48 hours: loss of contrast of the spongious bone, widening of periodontal space. The cortical bone lamina is intact.

CHRONIC PERIODONTITIS OF PERMANENT TEETH AT CHILDREN

Chronic periodontitis is a consequence of acute apical periodontitis, chronic pulpitis and micro traumas of periodontium. Chronic periodontitis at children can be a primary chronic process.

General evolution particularities of the chronic periodontitis of permanent teeth at children are the following:

- can appear at permanent teeth with superficial and medium carious cavities;
- granular form predominates;
- granular form is often associated with formation of fistulas;
- destructive processes can be often localized in the region of radicular bifurcation (trifurcation) rather than at apex;
- chronic periodontitis of permanent teeth with incompletely developed roots can stop or slow down the root formation (apexogenesis).

The clinical signs of chronic periodontitis are clear enough and there generally are no difficulties in setting the diagnosis. The basic symptoms of periodontitis are the following:

- hyperemia of mucous tissues in the vestibular part of the alveolar process in the region of radicular apex projection;
- the child has sensations of discomfort , heaviness, sometimes pain in the region of the affected tooth;
- the affected tooth can be felt as "higher than others" in occlusion;
- percussion is painless or sensitive;

- at palpation in the region of radicular apex projection unpleasant sensations or weak pain can be observed, sometimes hyperostosis or crepitation in case of radicular cyst.

At examination the dental crown can be considerably destroyed or a superficial carious cavity can be present. The dental cavity can be either open or closed. In most cases the color of the affected tooth is modified: the dental corona is more opaque, with nuances of gray or brown, especially in the region of collar. Probing is painless. Usually percussion is not painful, but unpleasant sensations can appear. The gingival mucous tissues are often swollen, hyperemic, fistulas with elimination or granularities can be detected. The clinical symptoms at the level of marginal and alveolar gum are often characteristic for the granulated form of chronic periodontitis.

The affected tooth doesn't react to thermal irritants. The electro-excitability of pulp in case of chronic periodontitis of permanent teeth is usually 100-120 mkA.

The data of clinical objective examination are not sufficient for setting the diagnosis and for choosing the tactics of treatment as the enumerated clinical signs do not allow evaluating the state of dental root (degree of formation, existence of pathological resorption, dimensions of apical orifice, etc.) and the character and the extension of pathological process into the periapical tissues. This is why in all cases radiological investigation is always necessary.

There are some particularities of radiological investigation of teeth with chronic apical periodontitis at children. In more than a half of all cases the child's age doesn't correspond to the dimensions and normal root formation degree for the tooth affected by chronic periodontitis.

When analyzing the radiograms one must determine the degree of root formation, the dimensions of apical orifice, the state of growth zone and only on the basis of these data the

character and extension of destructive processes can be determined.

Figure 8.3. **Chronic granulated periodontitis of 11.**

Most often the chronic inflammatory processes of the tissues of apical periodontium of mono-and poly-radicular teeth evolve in the granulated form that can vary by degree of extension.

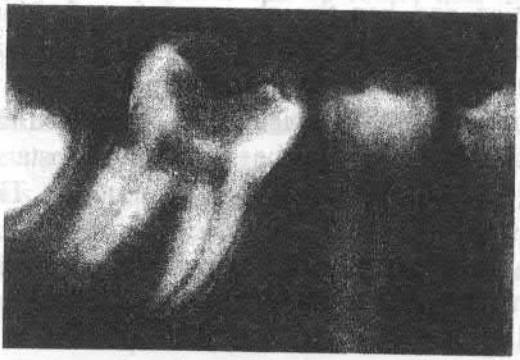


Figure 8.4. **Chronic granulated periodontitis of 46.**

Radiologically can be distinguished three degrees of destructive modifications characteristic for the granulated form of

chronic periodontitis:

1. Widening of periodontal space without essential destructive processes in the periapical tissues. The pathological process s based not on the deformation of cortical lamina as in the fibrous form of periodontitis but the destructive process. Widening of periodontal space occurs on the account of resorption of cortical bone lamina.
2. The granular tissue grows out of the limits of periodontal space with lesions of bone structure. Cement resorption is possible. Extended rarefaction of bone tissue can be seen on the radiograms.
3. The extended rarefaction nidus of bone tissue with missing bone in the middle.

Radiological image of granulated periodontitis of teeth with incompletely developed roots

The root is incompletely developed: walls are thin, getting thinner and thinner gradually to the growth zone, the radicular channel is wide, and the radicular channel ostium is narrower than the apical orifice and has the shape of funnel. Normally this funnel and the growth zone from periphery are limited by the cortical alveolar lamina of semi-arc shape. In case of chronic granulated periodontitis the cortical is resorpted and the radicular channel is not delimited from the spongious substance of bone that is involved into the inflammatory process. The state of growth zone is determined by the state of cortical lamina of the alveolar bone. If the resorption processes have affected the cortical lamina that delimits the growth zone, it is also partially or fully resorpted.

The growth zone is constituted by two layers of soft tissues: of pulpal and periodontal origin. The fibroblasts of oval shapes similar to the fibroblasts of the cambial bone tissue predominate in the pulpal layer that differs from the other fibroblasts of pulpal

stratum. The big fascicles of collagen fibers predominate in the periodontal layer. This complex of well vasculated tissues constitutes the growth zone.

Radiologically can be determined only the space in which this tissue is located but there is no way to determine its actual state. So, the radiogram can not give any information on the state of the growth zone and only in cases of extended destructive processes we can assume that the growth zone is also destroyed. Taking into consideration the structural particularities of the growth zone, its embryonic character and its regeneration potential, despite of the inflammatory processes it can still maintain its functions with the condition of correct treatment.

The other forms of periodontitis of permanent teeth at children are observed very rarely. Until the age of 9-10 years the chronic fibrous periodontitis is not detected due to the normally wide periodontal space. In this age the granulomatous periodontitis is also detected very rarely. This form of the affection is more often found at children aged 11-12 and above.

At the pluriradicular teeth at children different forms of periodontitis at different roots can be found and their state can also vary depending on the structural particularities and vascularity of pulp.

Particularities of treating periodontitis of permanent teeth at children

The tactics of treating apical periodontitis of permanent teeth depends on the etiology, clinical evolution and severity of pathological process, on the stage of root formation and periapical tissues, on the general state of the child's organism.

In order to make precise the indications for one or another treatment method one has to determine the character of the inflammatory process and its extension, the state and the root formation degree, the dimensions of apical orifice and of the radicular channel, permeability and the curve of the radicular

channel, etc. When pluriradicular teeth are affected these aspects must be made clear for each root in separate.

Taking into consideration the above described specifics, the treatment of apical periodontitis of permanent teeth at children can be performed with use of conservative, surgical and mixed methods.

The conservative method is the most rational one if implemented correctly in a suitable situation as it allows the root formation of the permanent tooth to continue allows the vindication of periapical tissues and maintains the functioning of the affected tooth. Conservative treatment of apical periodontitis is indicated for the teeth with accessible channels, i.e. when it is possible to influence the periapical inflammatory process via the radicular channel.

Contraindications for the conservative treatment of periodontitis of permanent teeth at children:

- Exacerbation of a general systemic disease with decompensation (of cardiac system, of kidneys, allergic states, etc.).
- Acute purulent or chronic exacerbated form of periodontitis when the inflammatory process progresses and there are no signs of improvement, with symptoms of septicemia, despite of nidus drainage and treatment with antibiotics.
- Extensive and destructive inflammatory processes of the alveolar bone (osteomyelitis, phlegmon, etc.).
- Acute purulent or chronic exacerbated periodontitis of teeth with impermeable channels;
- Massive destruction of dental crown under the gingival level;
- Profound perforation of radicular channel.

In such cases the pluriradicular teeth are extracted while for uniradicular teeth apical resection can be used.

Treatment of acute or chronic exacerbated periodontitis

The main principles of treating acute and chronic exacerbated forms of apical periodontitis:

1. Removal of cause.
2. Creation of most rational way for the drainage of exudation: if the exudation is in the periodontal space- via radicular channel, if it is under the periosteum or is sub gingival - by incision of infiltrate.
3. Treatment of the inflammatory process in the periapical region.
4. Choice of most rational physiotherapy method.
5. Raising the general resistance of the child's organism: antimicrobial therapy, anti-histamine treatment, stimulation of reparative processes, etc.

The factors that positively influence the evolution of treatment of periodontitis at children are: good or satisfactory state of health, high non-specific resistance of organism, accessible carious cavity, permeable radicular channels, etc.

The acute serous and purulent periodontitis is most often the consequence of acute diffuse pulpitis or chronic exacerbated pulpitis. Treatment includes pulpectomy or removal of necrotizing masses from the dental cavity and radicular channels with creation of drainage for exudation.

During the first visit with the pneumatic tool without pressure one must process the carious cavity, open the pulpal chamber and make access to the radicular channels. Necrotizing masses from the pulpal chamber are removed and gradually, with the help of pulp extractors on antiseptic protection, are removed the necrotizing masses from the radicular channels. One must use the endodontic tools with due care when treating the teeth with incompletely developed roots in order not to affect the periapical tissues.

At permanent teeth with developed roots often there is no

need to open the apical orifice to create drainage (unlike at adults) as it is wide and assures a satisfactory drainage from periapical region. The dental cavity remains open and washings with 1% sodium hydrocarbonate are performed.

In cases of severe evolution associated with serous periostitis, after pulpectomy and antiseptic treatment of channel can be indicated antibiotics, sulfanilamide preparations, anti-histaminic and analgesic preparations, etc.

When treating acute purulent periodontitis the child must be invited to the doctor on every day in order to permanently keep the situation under control and correct the tactics of treatment depending on the evolution of inflammatory process.

During the second visit instrumental processing and antiseptic treatment of dental cavity and radicular channels are performed.

Antiseptic treatment of radicular channels is performed with the following solutions: ectericide, microcide, sodium hypochlorite, chloramine, chlorhexetidine, proteolytic ferments, etc.

Hermetic closing of dental cavity with use of antiseptics or filling of channels can be performed only after liquidation of acute inflammatory process, disappearance of pain at percussion and normalization of general state of the child.

In order to raise the efficiency of endodontic treatment various physiotherapeutic methods can be used: intra-channel electrophoresis, ultrasound, microwaves, etc.

The response reactions and the results of treatment at children appear earlier than at adults regardless of smaller doses and shorter periods of treatment. The active biological substances are formed much earlier and in greater quantities compared to the organisms of adults in similar conditions.

Galvanic current is applied at intensity 2-3 times smaller than at adults, duration of electrophoresis is reduced to 15 minutes, and the total number of sessions is also reduced. Intra-channel

electrophoresis is indicated for acute purulent periodontitis and chronic exacerbated periodontitis of permanent teeth with narrow and impermeable channels, in cases of breakage of endodontic instruments in the channel, apical periodontitis exacerbated at capsulation, especially in the period of incompletely developed roots. For electrophoresis is used the solution of iodine, solution of potassium iodide 10%, isotonic solution of sodium chloride, etc. 3-4 sessions of electrophoresis are indicated daily or every other day. Duration of treatment depends on individual tolerability; nut can not exceed 30 mA/min (J. Mitrega et al., 1976).

Physiotherapy with ultrasound is indicated in acute and chronic exacerbated periodontitis associated with periostitis and regional lymphadenitis, performed daily, the total number of sessions being 5-6.

Also ultrasound can be used for the treatment of skin in the region of projection of the inflammatory process through a stratum of Vaseline during 5-6 minutes at 0,2V/cm.

Treatment of acute toxic arsenic periodontitis of permanent teeth consists in the pulpectomy and application of antidotes: sol. 5 % unithiol, sol. 5% iodine, sol 1% iodinol. Treatment is done in 3-4 visits, tampons with antidote solution are left in the channel until the pain at percussion and intercuspidation disappears.

If the acute periodontitis is provoked by the toxic action of phenol, after extirpation of pulp in the channel is inserted a wick drain or a paper cone with ricin oil or 10% emulsion of anestesine on ricin oil under provisory bandage.

If the acute periodontitis is provoked by the combined mechanic and chemical action in result of obturation of radicular channel beyond the level of radicular apex, especially with resorcin and formalin pastes during the treatment of pulpitis by pulpectomy or is caused by formation of haematoma after the traumatic pulpectomy different physiotherapeutic methods are

indicated, including ultrasound therapy and ultra-short waves -- 5-6 sessions. In cases of intense pains ionophoresis with 10% sol. calcium chloride (4-5 sessions daily) can be indicated in complex with analgesics.

In cases of infected haematoma and appearance of acute purulent periodontitis the radicular channel must be dis-obturated and drainage through it must be assured.

In cases of acute periodontitis provoked by obturation of channel beyond the radicular apex level (treatment of pulpitis or periodontitis by pulpectomy) control radiography is necessary. When the channels are obturated and endodontic material is found in the periodontal space, physical methods of treatment are used: buccal rinsing with warm sol. 0,5% sodium hydrocarbonate, analgesics - in cases of weak pains. If the pains are strong or symptoms of acute serous periostitis appear, electrophoresis with sol. 2 % lydocaine (2-4 procedures) are indicated. In cases of infiltrate on the passing fold 5-6 procedures of high frequency currents are performed. If one of the radicular channels is not obturated to the apex, the tooth and the channels are de-obturated, the tooth is left open and physiotherapeutic treatment is prescribed. After the liquidation of acute inflammatory process the channels are obturated to apex and permanent obturation is applied.

Treatment of chronic forms of apical periodontitis of permanent teeth at children

The main scopes of treating apical periodontitis at children are the following:

1. Action on the microflora of the radicular channel and macro dentinary channels.
2. Removal of action of biogenic amines.
3. Removal or reduction of apical inflammatory process.
4. Creation of favorable conditions for the radicular apexogenesis.

5. Stimulation of regeneration of all tissues of apical periodontium.

In cases of chronic apical periodontitis of uniradicular tooth with developed roots and close apical orifice, or in the presence of granular tissue in the apical region of radicular channel painful at probing and associated with hemorrhage treatment in one session is indicated. After the processing of carious cavity and clearance of access in the channels the granular tissue is removed under infiltration anesthesia (trunk) or under local anesthesia in the channel (sol. lydocaine, sol. Phenol with anesthesine), the channel is processed instrumentally and treated with medicines and at last obturated with endodontic material during the same visit. If the radicular channel is not obturated during the first visit, the granular tissue will continue to grow in the channel and complicate the situation with exacerbation of inflammatory process. In such cases instrumental processing of channels can cause hemorrhage, however, obturation of channel must be performed during the same visit. Repeated application of antiseptics, haemostatic and cauterization preparations used for complete drying can lead to the successive exacerbation of inflammatory process. Regardless of all above stated the channel must be obturated in a single visit.

One of the main stages of endodontic treatment is the instrumental processing of radicular channel.

The scopes of instrumental processing are the following:

- clearance of channel from residues of food, necrotizing pulpal tissues, damaged and infected dentine ;
- widening of narrow or obliterated portions of the channel;
- widening of channel and reduction of curve by creation of favorable conditions for obturation;

Successful instrumental processing of the channel implies:

- use of high quality endodontic tools;

- assurance of good access of endodontic tools to the radicular channel (straight line access);
- widening of channel orifices;
- use of endodontic tools in ascending order: from small to large without omitting dimensions (phases);
- examination of tool tips after removal from channel in order to detect deformations and prevent fractures of tools;
- working with endodontic tools only in the humid channel (treated with antiseptics);
- cleaning of endodontic tools from necrotizing tissues and dentine before successive introduction into the channel;
- use of tools only in the limits of radicular channel in order to prevent the lesions of periapical tissues and penetration of necrotic tissues beyond apex;
- widening of the channel is done at least to the diameter of 2-3 nos. bigger than initial diameter of the endodontic tool;
- strict observation of rotation angle of endodontic tools (Hedstroem: $90\text{-}360^0$ in wide channels, $45\text{-}90^0$ in narrow channels, drills (K-file reamer) – 360^0 in wide channels, $90\text{-}180^0$ in narrow channels).

Before proceeding with instrumental treatment, the tools with stops must be selected depending on thickness and length of channel. There are many methods of instrumental processing of channels (the standard technique, the step-back method, the crown-down method, etc.). Removal of necrotic from channels is done in sequences of 1/3 of channel length with antiseptic protection.

Mechanic processing must be effected with extreme care, covering each wall of the wide radicular channels and due protection of the apical zone. Endodontic tools of big numbers with sectioned heads must be used. They are introduced into the wide channels from the part opposed to the one to be processed. Usually, for the passage and widening of channels, are used the

Hedstroem needle and the drill. The endodontic tool is inserted gradually into the channel and rotated in the clockwise direction till 180^0. The tool is taken out of the channel by rotation movement in the inverse direction, leaning against the radicular channel wall.

After the instrumental processing of radicular channel it must be treated with medicines: oxygenated water 3%, chloramine 2%, sodium hypochlorite 3%, chlorhexidine 0.2%, iodinol 1%, proteolytic ferments (trypsin, terylitin, etc.).

After the drying of channel with paper cones or cotton wool the channel is filled with the help of Lentulo needle with endodontic pastes and gutta-percha cones (in normal or "inverse" direction), control radiography is performed and permanent filling is applied.

Treatment of periodontitis of permanent teeth with incompletely developed roots

Treatment of periodontitis of permanent teeth with incompletely developed roots is associated with certain difficulties. The vindication perspective to a considerable extent depends on the state of growth zone. Maintenance of vitality of cellular elements in the growth zone favors the growth of roots, closing of apical orifice and regeneration of apical zone tissues in the nidus of inflammation.

After the preparation of carious cavity, opening of dental cavity and assurance of access between the channels, step by step the necrotizing masses under antiseptic protection are removed with the help of 2-3 pulp extractors, avoiding the lesions of apical tissues and penetration of necrotizing masses beyond the root limits. With extreme care one has to remove the infected predentine from the channel walls. Endodontic tools of suitable diameters are used (big sizes) with stops in order to avoid excess of working length. Lesion of apical zone usually results in abundant hemorrhage. The wider is the apex the more difficult

becomes the treatment: danger of mechanical lesion of apical zone, ingress of necrotic masses beyond the limits of root, persistence of radicular secretion and bleeding, possibility of exacerbation of the chronic inflammatory process. For the medicamentous treatment are used the medications of wide antimicrobial action without cytotoxic effects. In the case of gangrenous smell or wet necrotic masses in the channel it is recommended after processing to leave in the channel a wick drain with bactrim, metronidasol, camphoro-phenol, etc. under bandage. 2-3 courses of intra-channel electrophoresis can be indicated. During the second visit, unless the patient has accuses and if there are no pains at percussion, after the instrumental processing and medicamentous treatment of channel it is obturated in the limits of developed root (with radiological control). It is recommended to obturate the incompletely developed roots in stages: paste based on calcium hydroxide, zinc-oxide-eugenol, and inverse gutta-percha cone. This favors the processes of apexogenesis. After the formation of apex the radicular channel is de-obturated, processed instrumentally, treated with medicines and obturated to the very apical orifice.

The situation is more difficult if the growth zone is completely affected by the periapical inflammatory process. Often this process has a prolonged and slow evolution associated with fistulas. In such cases the major danger comes from the stratum of infected pre-dentine, as it has a very large contact with periapical tissues containing the inflammatory process and inhibiting the regeneration. Treatment includes instrumental processing and thorough antiseptic treatment with use of proteolytic ferments. Obturation of channel is done in stages: Ghenis paste in the apical zone and zinc-oxide-eugenol. After the definite apex formation it is recommended to open the channel obturation and apply permanent obturation of radicular channel.

The children with apical chronic periodontitis must be

registered with the dispensary. The state of treated teeth must be controlled clinically and radiologically 2 times a year. The dispensary control is cancelled after the recovery of periapical tissues, formation of radicular apex or apexification in result of destruction of the growth zone and closing of the root apex of the incompletely developed root.

Control questions and topics:

1. What factors provoke the appearance of apical periodontitis of temporary and permanent teeth at children?
2. Clinical evolution particularities of acute apical periodontitis of temporary and permanent teeth at children.
3. Clinical evolution particularities of chronic apical periodontitis of temporary and permanent teeth at children.
4. Radiological image of the chronic forms of apical periodontitis of temporary and permanent teeth at children.
5. Radiological image of the apical periodontitis of teeth with incompletely developed roots.
6. Complications of apical periodontitis of temporary and permanent teeth at children.
7. Main scopes in the treatment of apical periodontitis at children.
8. Principles of treating acute apical periodontitis.
9. Treatment stages of acute apical periodontitis at children.
10. Treatment stages of chronic apical periodontitis at children.
11. Contraindications of conservative treatment of periodontitis of temporary teeth at children.
12. Contraindications of conservative treatment of periodontitis of permanent teeth at children.
13. Treatment of apical periodontitis of permanent teeth with incompletely developed roots.
14. Materials for the obturation of channels of temporary and permanent teeth at children.

15. Control of treatment efficiency in cases of apical periodontitis of temporary and permanent teeth at children.

CONTROL TESTS

Simple compartment

1. Apical periodontitis at children is most often provoked by the following factors:
   A. Toxic-bacterial;
   B. Traumatic;
   C. Chemical;
   D. Medicamentous;
   E. Allergic.

2. The germs that lead to the affection of periapical tissues are mostly found in:
   A. Periapical tissues;
   B. Radicular cement;
   C. System of radicular channels.

3. In the bacterial endodontic pathogenic flora predominate:
   A. Aerobic microorganisms;
   B. Microorganisms of many types;
   C. Anaerobic microorganisms;
   D. Viruses.

4. Most often at children is found the apical periodontitis:
   A. Chronic granulomatous;
   B. Chronic granulated;
   C. Exacerbation of chronic periodontitis;
   D. Acute serous;
   E. Acute purulent.

5. Fistula is detected most often in case of:
   A. Acute serous apical periodontitis;
   B. Acute purulent apical periodontitis;

C. Chronic granulated apical periodontitis;
D. Chronic fibrous apical periodontitis;
E. Chronic granulomatous apical periodontitis.

6. Granulomas and cysts are constituted of:
A. Flat multi-layered epithelium of buccal cavity vegetating through the dental-gingival saccule;
B. Flat multi-layered epithelium of buccal cavity vegetating through the fistula;
C. Epithelium of idiopathic provenience;
D. Malassez epithelial residues proliferating in the inflammatory process.

7. For the chronic apical periodontitis are characteristic:
A. Spontaneous pains, disappearing in short time;
B. Permanent and stabbing pains;
C. Permanent pains intensifying at intercuspidation;
D. No pains are characteristic;
E. Irradiating pains.

8. The diagnosis of chronic apical periodontitis is established on the basis of:
A. Anamnesis and examination;
B. EOD;
C. Radiogram;
D. Probing and percussion;
E. All answers are correct.

9. The radiological image of acute apical periodontitis is characterized by:
A. Absence of essential changes, widening of periodontal space, vague trabecular image in the region of inflammatory process;
B. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with irregular shape or candle fire –

shaped, etc.;

C. Widened periodontal spaces mostly in the region of radicular apex;

D. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with regular limits of spherical or oval shape.

E. Narrowing of periodontal space in the region of radicular apex.

10. The radiological image of chronic fibrous apical periodontitis is characterized by:

A. Absence of essential changes, widening of periodontal space, vague trabecular image in the region of inflammatory process;

B. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with irregular shape or candle fire – shaped, etc.;

C. Widened periodontal spaces mostly in the region of radicular apex;

D. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with regular limits of spherical or oval shape.

E. Narrowing of periodontal space in the region of radicular apex.

11. The radiological image of chronic apical granulated periodontitis is characterized by:

A. Absence of essential changes, widening of periodontal space, vague trabecular image in the region of inflammatory process;

B. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with irregular shape or candle fire – shaped, etc.;

C. Widened periodontal spaces mostly in the region of

radicular apex;

D. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with regular limits of spherical or oval shape.

E. Narrowing of periodontal space in the region of radicular apex.

12. The radiological image of chronic apical granulomatous periodontitis is characterized by:

A. Absence of essential changes, widening of periodontal space, vague trabecular image in the region of inflammatory process;

B. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with irregular shape or candle fire – shaped, etc.;

C. Widened periodontal spaces mostly in the region of radicular apex;

D. Presence of a destruction nidus in the alveolar bone with lesion of cortical plate with regular limits of spherical or oval shape.

E. Narrowing of periodontal space in the region of radicular apex.

13. Chronic apical granulated periodontitis differs from the granulomatous one by:

A. Color of the tooth;

B. Appearance of vase paresis;

C. Shortage of sound at percussion;

D. Presence of fistula;

E. Characteristic radiological image - destruction of periapical bone tissue with irregular edges.

14. Main principles of treating apical periodontitis at children are:

A. Removal of cause;

B. Choice of the most rational way for the drainage of exudation;
C. Treatment of inflammatory process in the periapical region;
D. Raising the general resistance of the child's organism;
E. All answers are correct.

15. The main scopes of treating apical periodontitis at children are:
A. Action over the microflora of radicular channel and micro –dentinary channels;
B. Neutralization of action of biogenic amines;
C. Removal or inhibition of apical inflammatory process;
D. Creation of conditions for apexogenesis, stimulation of reparative processes in the tissues of apical periodontium.
E. All answers are correct.

16. Instrumental processing of radicular channels is aimed at:
A. Clearance of channel from residues of food and necrotizing pulpal tissues;
B. Clearance of channel from damaged and infected dentine;
C. Widening of narrow or obliterated portions of the channel;
D. Widening of channel and reduction of curve with creation of favorable conditions for obturation;
E. All answers are correct.

17. For the treatment of toxic arsenic periodontitis at children are used the following preparations:
A. Camphor-phenol;
B. Unithiol;
C. Sodium hypochlorite;
D. Resorcin-formalin;
E. Eugenol.

18. The most important thing in the endodontic treatment is:

A. Endodontic treatment of radicular channel;
B. Mechanical processing and medicamentous treatment of radicular channel;
C. Hermetic obturation of radicular channel in the apical and coronary portion.

19. The optimum alternative of treating chronic apical periodontitis of a tooth with developed roots at a child suffering from rheumatism is:
A. Obturation of channels with zinc-eugenol paste;
B. Obturation of channels with resorcin-formalin paste;
C. Surgical treatment;
D. Obturation of channels + physiotherapeutic treatment;
E. Obturation of channels with zinc-eugenol paste + gutta-percha cone.

20. The main indication for surgical treatment of chronic apical periodontitis at temporary teeth is:
A. Child's age;
B. Eruption periods of the permanent tooth;
C. Character of destruction of periapical tissues and degree of permanent dental bud involvement in the pathologic process;
D. Degree of destruction of dental crown;
E. Presence of fistula.

21. In case of hemorrhage after removal of granular tissue from the radicular channels:
A. Channels are filled;
B. Channels are not filled;
C. Extraction of temporary tooth is recommended.

Multiple compartment

22. Apical periodontitis can be provoked by the following factors:
A. Toxic-bacterial;

B. Traumatic;
C. Chemical;
D. Physical;
E. Allergic.

23. Chronic apical periodontitis at children can be:
A. Simple;
B. Granulated;
C. Fibrous;
D. Gangrenous;
E. Granulomatous.

24. Acute apical periodontitis at children can be:
A. Partial;
B. Diffuse;
C. Serous;
D. Purulent;
E. Fibrous.

25. For the acute apical periodontitis are characteristic:
A. Nocturnal spontaneous pains;
B. Permanent stabbing pains;
C. Permanent pains intensifying at intercuspidation;
D. Irradiating pains;
E. Absence of pains.

26. Apical periodontitis at children affects the teeth:
A. With superficial carious cavities;
B. With medium carious cavities;
C. With profound carious cavities;
D. With incipient caries;
E. Intact teeth.

27. Complications of apical periodontitis at children include:
A. Adamantinoma;

B. Periostitis;
C. Osteomyelitis of maxillaries;
D. Alveolitis;
E. Follicular cyst.

28. Complications of apical periodontitis of temporary teeth are the following:
A. Necrosis of permanent dental bud;
B. Position anomalies of permanent teeth;
C. Formation of a chronic nidus of odontogenous infection;
D. The Stainton-Capdepont syndrome;
E. The Turner syndrome.

29. Complications of the apical periodontitis of temporary teeth are the following:
A. Regional lymphadenitis;
B. Changes in the periods of radicular resorption;
C. Apexification;
D. General intoxication of organism at small children;
E. Allergic reactions.

30. Complications of the apical periodontitis of temporary teeth are the following:
A. Amelogenesis imperfecta;
B. Systemic hypoplasia of permanent teeth;
C. Local hypoplasia of permanent teeth;
D. Follicular cyst;
E. Radicular cyst;
F. Dentinogenesis imperfecta;
G. Necrosis of permanent dental bud.

31. Differentiated diagnosis of acute apical periodontitis is made with:
A. Acute pulpitis with involvement of periapical tissues and regional lymphatic ganglions;

B. Acute diffuse purulent pulpitis;
C. Acute odontogenous sinusitis;
D. Radicular cyst;
E. Periostitis of maxillaries.

32. Contraindications for the treatment of apical periodontitis at children include:
A. Lesion of the integrity of compact lamina of permanent dental bud;
B. Pathological resorption of 1/5 of root length;
C. Pathological resorption of 1/3 of root length;
D. Pathological resorption of 1/2 of root length;
E. Child's age.

33. Treatment of apical periodontitis at children is contraindicated when the affected tooth is cause of:
A. Periostitis;
B. Osteomyelitis;
C. Septicemia;
D. Mediastenitis;
E. Gastritis.

34. Contraindications for the treatment of apical periodontitis at children include:
A. Complete destruction of corona of temporary tooth;
B. Perforation of dental cavity floor;
C. Absence of effect of conservative endodontic treatment;
D. Apical periodontitis of temporary teeth with incompletely developed roots;
E. Apical periodontitis of permanent teeth with incompletely developed roots.

35. Contraindications for the treatment of apical periodontitis at children include:
A. Loss of functional importance of the temporary tooth if

there remains one year till physiological replacement;
B. Loss of functional importance of the temporary tooth if there remain two years till physiological replacement;
C. Loss of functional importance of the temporary tooth if there remain three years till physiological replacement;
D. The tooth is a nidus of odontogenous infection;
E. Apical periodontitis of permanent teeth with incompletely developed roots.

36. Endodontic materials used for the obturation of channels in temporary teeth include:
A. Zinc-oxide-eugenol paste;
B. Ghenis paste;
C. Phosphate cement;
D. Endodont;
E. Calmecin.

37. Endodontic materials used for the obturation of channels in the temporary teeth include:
A. Intradont;
B. Paste + gutta-percha cone;
C. Paste + silver pivot;
D. Zinc-oxide-eugenol paste;
E. Iodoform paste.

38. The negative action of eugenol pastes in the endodontic practice is possible in cases of:
A. Allergic reaction;
B. Obturation beyond the limits of radicular channel apex;
C. Incomplete obturation of radicular channel;
D. Eugenol is completely inoffensive;
E. Eugenol is completely neutralized with zinc oxide.

39. For the antiseptic treatment of radicular channels in the treatment of apical periodontitis at children are used:

A. Trypsin;
B. Iodinol;
C. Esther;
D. Sodium hypochlorite;
E. Capropher;
F. Schiler-Pisarev solution.

40. Clinical and radiological supervision (dispensary control) after the treatment of apical periodontitis of temporary and permanent teeth is performed after:
A. 2 days;
B. 2 weeks;
C. 3 months;
D. 6 months;
E. 12 months.

Correct answers:

1. A.
2. C.
3. B.
4. B.
5. C.
6. D.
7. D.
8. E.
9. A.
10. C.
11. B.
12. D.
13. E.
14. E.
15. E.
16. E.
17. B.
18. C.
19. C.
20. C.
21. A.
22. A, B, C, D, E.
23. B, C, E.
24. C, D.
25. B, C.
26. A, B, C.
27. B, C, D, E.
28. A, B, C, E.
29. A, B, C, D.
30. C, D, G.
31. A, B.
32. A, C, D.
33. A, B, C, D.
34. A, B, C, D.
35. A, B, D.
36. A, B.
37. D, E.
38. A, B.
39. B, D.
40. D, E.

9 AFFECTIONS OF PERIODONTIUM AT CHILDREN

Periodontium is a complex of tissues that surround the tooth: the gums, the circular ligament, the periodontium, and the alveolar bone - cement - joined functionally and morphologically.

At children the tissues of periodontium for a long time are in development, i.e. in processes associated with appearance, eruption and formation of temporary and permanent dental roots.

STRUCTURAL PARTICULARITIES OF PERIODONTIUM AT CHILDREN

The gums: a) are comparatively better vasculated, epithelium has a thinner layer of keratinated cells and thus their color is more vivid, b) they have a less granulated surface, as the epithelial papillae are less pronounced; c) the conjunctive tissues are of comparatively smaller density; d) the gingival sulcus is deeper; e) during dental eruption the marginal gum has rounded contours, it is slightly edematous and hyperemic.

Radicular cement: a) it is thinner, b) it is of smaller density; c) it has a tendency to hyperplasia in the region of epithelial insertion.

Periodontium: a) it is widened, b) it has thin and fine fibers; c) it differs by hydration due to vasculation and developed lymphatic system.

Alveolar bone: a) it is flatter; b) cortical lamina is thinner; c) contains more medullar space; d) is less mineralized; e) contains a smaller number of trabeculae; g) has a rich network of sanguine and lymphatic vessels.

Dynamics of histological and histo-chemical formation of gums depending on age

In temporary dentition: the gingival epithelium is thin, slightly differentiated, with weakly pronounced epithelial papillae, without keratinization. At children aged less than 3 years the mucous tunic of the gums contains much glycogen. After this age the gingival epithelium (in contrast to other locations in the buccal cavity) no glycogen is detected. Appearance of glycogen at children aged above 3 years is a sign of pathological changes of inflammatory character.

Mixed dentition suffers evident changes: the stratum of gingival epithelium grows bigger, the epithelial papillae become more pronounces, the basal membrane becomes thicker, collagen fibers become denser and generally more oriented. At this age takes place the maturing of collagen structures and decreases the predisposition to diffuse reactions. Permeability of vessels becomes lower. All these contribute to a chronic and slow evolution of pathogenic processes in gums.

During the period of permanent dentition the gums have a differentiated structure. The superficial layer is composed of epithelium with keratinization capacity. An exception is the mucous tunic of gum around the dental collar (in the gingival sulcus) that doesn't possess this property and due to this it is affected first of all. In this zone the epithelium contacts with microorganisms and the contents of the gingival sulcus. Under the action of ferments produced by microorganisms the permeability of epithelium grows higher.

Formation particularities of periodontium

Periodontium at children is characterized by lack of a stable form and structure. Its development begins from dental bud. During the dental eruption at the edge of alveolar process the fibrous structures of the dental bud interact with the collagen fibrous structures of the gums. This interconnection of fibers

takes place before the eruption of tooth into the buccal cavity and after eruption until the functional occlusion is attained.

Structural particularities of the maxillary bones depending on age

In the temporary dentition the radiological image of the bone tissue is not clearly determined. One can see well only the compact lamina of permanent dental buds and of the periodontium of temporary teeth, the solid dental tissues of mineralized teeth. The structure of bone is weakly differentiated, the septums are weakly pronounced, one can observe the wide medullar spaces. The periodontal space is almost twice as wide as at adults.

In mixed dentition at erupted teeth the tops of interdental septums are sectioned in the direction of tooth in process of eruption and are at the level of enamel margin and cement. One may get the impression that periodontal space at the collar of erupted teeth is enlarging. At the end of eruption the edges of interdental septums can have different shapes (normal variants): sharp, oval, flat, duplicated, etc. Formation and mineralization of septum tops and compact lamina take place after the "closing" of radicular apexes. Absence of compact lamina of interdental septums at children with incompletely developed roots is a sign of immaturity of the periodontal bone tissue.

In permanent dentition the bone tissue is already developed. The spongious bone has big cells, fine trabeculae and wide red tissue zones. The tops of interdental septums are at the level of enamel-cement and only in spacing cases can be under this level. The septums can have various shapes. However, the healthy periodontal bone is characterized by clear cortical laminae.

The principal particularity of the structure of periodontal tissues at children consists in the permanent development and restructuring, i.e. in the dynamic formation of periodontium of a tooth, of a group of teeth and of the entire dental-maxillary

system. The components of developed periodontium have a special structure and specific morphological and functional characteristics. They have the same sanguine and lymphatic systems and unique enervation and do to these reasons the pathological processes are uniform - indifferently on etiology and localization of excitant the entire complex of tissues reacts on it. All pathological processes in periodontium evolve in the morphological and functionally immature tissues, thus leading to inadequate reaction on excitants able to produce periodontal affections at adults.

Disproportion in the development of structures and functions at children predetermine the formation of chronic juvenile periodontitis and gingivitis that are a consequence of juvenile hypertension, disturbances in the metabolism of carbohydrates (sugar diabetes, etc.). Pathological processes at children can disappear without consequences, with or without the influence of certain factors or can keep on progressing regardless of applied treatment.

Pathological processes in periodontium can have the following particularities:

- practically in all cases they start from the gingival margin and progress towards the apical region;
- they are not limited and may affect several teeth;
- most often they are of chronic character;
- they have a tendency to permanent extension.

Classification of periodontal affections

WHO classification

K.05. Gingivitis and periodontal disease, including the affections of edentate alveolar edge;

K.05.00. Acute streptococcal gingivo-stomatitis;

K.05.08. Other precise acute gingivitis

K.05.1. Chronic gingivitis

K.05.10. Simple marginal

K.05.11. Hyperplasic
K.05.12. Ulcerative
K.05.13. Desquamative
K.05.18. Other chronic precise gingivitis
K.05.2. Acute periodontitis
K.05.20. Periodontal abscess of gingival origin without fistula
K.05.21. Periodontal abscess of gingival origin with fistula
K.05.22. Acute pericoronitis
K.05.28. Other acute precise periodontitis.
K.05.3 .Chronic periodontitis
K.05.30. Simple
K.05.31. Complex (complicated)
K.05.32. Chronic pericoronitis
K.05.33. Chronic hyperplasic folliculitis
K.05.38. Other precise chronic periodontitis
K.05.4. Chronic periodontitis
Juvenile periodontosis
K.06.00. Gingival retraction (including post-infective, post-operative)
K.06.00. Localized
K.06.01.Generalized
K.06.09.Gingival retraction without precision
K.06.1 Gingival hyperplasia
K.06.10 Gingival fibromatosis
K.06.18 Other precise gingival hyperplasia
K.06.2 Gingival and edentate alveolar margin lesions associated with traumatism
K.06.20 Provoked by traumatic occlusion
K.06.21 Provoked by brushing
K.06.22 Functional keratosis
K.06.23 Hyperplasia by irritation (with prosthesis)

Classification of affections periodontium by ASUS (1983)

1. Gingivitis - inflammation of gums generated by the pathologic action of local and general factors evolving without affecting the gingival-dental ligaments.

- Clinical forms: catarrhal, ulcerous and hypertrophic;
- Manifestation forms: light, medium and severe;
- Form of evolution: acute, chronic and exacerbated;
- Extension degree: localized and generalized.

2. Periodontitis – inflammation of periodontal tissues characterized by the progressive destruction of periodontium and alveolar bones.

- Manifestation forms: light, medium and severe;
- Evolution: acute, chronic, exacerbated , abscess and remission;
- Extension degree: localized and generalized.

3. Periodontosis - dystrophic affection of periodontium

- Manifestation forms: light, medium and severe;
- Evolution: chronic and remission;
- Extension degree: generalized.

4. Idiopathic diseases with progressive destruction of periodontal tissues *(periodontolysis)*: Papillion-Lefevre syndrome, neutropenia, agama-globulinemia, decompensated sugar diabetes, etc.

5. Periodontoma - tumefaction and tumefacient affections (epulis, fibromatosis, etc.).

According to the data of WHO (1980) affections of periodontium are found at 80 % of children and in some cases at 100 % (Finland). The researches performed by Poulsen and Moller in 1972 in Denmark revealed that 25 % of children aged 3 years and 80 % of children aged 5 to 14 years are affected by this disease. At 12 years most often are affected the girls, while at the age of 14 years – the boys, probably due to earlier attaining of

puberty by the girls.

Out of all periodontal affection at children predominates the catarrhal gingivitis (80-85 % of cases), followed by hypertrophic and atrophic gingivitis (12-15% of cases). Marginal periodontitis is formed at 3-5 % of adolescents aged 15. Idiopathic affections and parodontoma are very rarely found at children.

The incidence of periodontal affections at children in the Republic of Moldova is presented in Table 9.1 below, being higher at children from urban localities and attaining around 96,8% at the age of 15 years.

Table 9.1

Incidence of periodontal affections at children in the Republic of Moldova

Age	Years	Villages, %	Cities , %
3-4 years	1992	-	-
	1996	-	-
6 years	1989	27.2	28.1
	1992	31.3	33.33
	1996	33.4	35.2
12 years	1989	42.3	61.2
	1992	46.5	68.4
	1996	49.3	69.0
15 years	1989	83.0	92.4
	1992	85.5	96.0
	1996	86.7	96.8

Spreading of periodontal affections at children depends on many factors, among these factors can be specially mentioned: social-economic level of the country, cultural level of the population, level of buccal cavity hygiene, particularities of nutrition.

Local inflammation forms of periodontal tissues predominate

at children of smaller ages: between 2.5 and 6 years, while the generalized forms predominate at children aged 6 and higher, especially after the age of 12 years. Periodontitis and dystrophic forms are most often detected at ages above 12 years. Indifferently on ages, gingivitis predominates.

GINGIVITIS

Gingivitis can appear in result of bacterial or viral infection, failure to observe the hygiene of buccal cavity, local allergic reaction, mechanic trauma (dental –alveolar anomalies, short phrenum of lips and tongue, small vestibule, caries of approximal surfaces, incorrect filling, etc.), chemical lesions, etc. As separate morphological unit, it can be acute or chronic; catarrhal, ulcerous or hypertrophic; localized or generalized. Generalized gingivitis (as a symptom) can appear in cases of acute herpetic stomatitis, infectious stomatitis, periodontitis, beriberi, endocrine disturbances at children during puberty age, systemic maladies of organism (CNS diseases, tuberculosis, affections of gastrointestinal tract, gastritis, gastroduodenitis, colitis, hepatitis, etc.).

CATARRHAL GINGIVITIS

Catarrhal gingivitis at children is detected most often. The acute process is characterized by the appearance of hyperemia (gums have a bright red color), edema and hemorrhage of papillae and marginal gums. The process can be localized (1-5 teeth) and generalized. The acute form is most often provoked by local causes (mechanic trauma, chemical lesion, etc.) or general diseases (catarrhal symptomatic gingivitis).

Chronic catarrhal gingivitis most often evolves without accuses, however, hemorrhage can be present (during brushing and biting solid food), unpleasant sensations (burning pain, pressure).

At *examination:* hyperemia and edema at the level of papillae

and marginal gums, soft or solid deposits can be identified. Probing can result in hemorrhage, periodontal pockets are absent.

Diagnostic methods: Schiuller-Pisarev test positive; hygienic index above 1.0, PMA above 0, haematoma formation time :< 60-70s, CPITN – code 1-2.

Differentiated diagnosis: with other forms of gingivitis and periodontitis.

Treatment: 1. Removal of cause. 2. Hygiene of buccal cavity. 3. Anti-inflammatory therapy: antiseptics, ferments, vegetal solutions - chamomile tea, sol. Salviae, Romazulan, etc. anti-edematous action preparations – polimínerol, maraslavin, keratoplastics – keratolin, oil of Fructis Rosae, etc. Also are recommended flushing and hydro-massage with mineral waters, electrophoresis (with heparin, calcium gluconate, sol. Romazulan 2-5%, etc.). In cases of symptomatic gingivitis most important is the treatment of general malady.

HYPERTROPHIC GINGIVITIS

Hypertrophic gingivitis is manifested by the proliferation of fibrous or granulated tissues of gums and formation of false pockets. Dental—maxillary anomalies, incorrect obturation, disturbances and endocrine diseases are contributing factors, as well as some medications (hidantoine, diphenine), etc. Hypertrophic gingivitis is preceded by the catarrhal inflammation of gums; however, it can be primary as well. Most often it is found at children of pubertal and pre-pubertal ages. There are two clinical forms: edematous and fibrous. Depending on the degree of coverage of dental corona one can distinguish: the light form – when 1/3 of the dental corona is covered, medium - more than 1/3 is covered and severe - when 2/3 of corona is covered.

The edematous form is characterized by the following accuses: evident hemorrhage during dental brushing, when biting solid food, unpleasant smell from the buccal cavity. The gums are hypertrophic, edematous and cyanotic, and have a smooth

surface. Probing causes hemorrhage, soft and solid deposits and false periodontal pockets can be detected.

Supplementary diagnostic methods: positive Schiuller-Pisarev test, hygienic index > 1.0, PMA> 0, reduction of haematoma appearance time (<60s).

Clinical image of the fibrous form is characterized by hypertrophy of gums, the color of gums doesn't change, and the gums do not bleed, and have an opaque and irregular surface can be false periodontal pockets.

Complementary methods: negative Schiuller-Pisarev test, variable hygienic index, PMA-0.

Differentiated diagnostics: with gingival fibromatosis.

Treatment of edematous form:

1. Removal of cause (dental deposits, dental-maxillary anomalies, etc.);
2. Oral hygiene;
3. Anti-inflammatory treatment and anti-edematous treatment in the form of washings, flushing, use of solutions, creams and films, inhalations with aerosols, gingival injections, etc.
4. Only after the removal of inflammatory process one may use the therapy of superficial sclerotization (Maraslavin, Phytodont, Sangvinarin, potassium iodide, etc.) and profound treatment (injections into gingival papillae of glucose solution 40 %, oxygenated water 10%).

Treatment of fibrous form:

1. Oral hygiene;
2. Sclerotization therapy: injections into the gingival papillae or electrophoresis with Lydase 0.1-0.2 ml, Ronidase, electrophoresis with Heparin 5000 Un per 1 ml, 15 min, 15-20 sessions.
3. Cryodestruction, laser therapy and gingivotomy.

ULCEROUS GINGIVITIS

Often appears after chronic catarrhal inflammation, however, can also be primary. Ulcerous gingivitis is favored by the worsening of general state of organism, reduction of immune levels of any etiology. On the background of inflammatory process (hyperemia, cyanosis, edema) appears the ulceration of gingival margin. Ulcerated surfaces are covered with fibrous or necrotic deposits and unpleasant smell from buccal cavity. Teeth can be slightly mobile. Regional lymphatic ganglions are enlarged, mobile and painful at palpation. The children with such affections are pale, atonic, have fever, refuse to eat due to the pains. Radiologically no evident changes are detected.

Differentiated diagnostics: with periodontal syndromes of sugar diabetes, X-histiocytosis, leucosis, etc.

Treatment: Treatment is complex: therapy of the general disease and improvement of the general resistance of the child's organism.

General treatment: anti-histaminic preparations are administered (Dimedrol 0.003, promethazine 0.0025, pipolphen, tavegil 0.001), vitamins C and P. Foodstuff with high content of proteins and vitamins, easily assimilated and liquids in big quantities.

Local treatment: after application anesthesia proteolytic fermens are applied (trypsin, chemotrypsin, terylytine). Necrotizing deposits must be removed. Afterwards it is recommended to perform irrigation and treatment with antiseptics (furacillin, ectericyde, metronidazole, etc.), applications with anti-inflammatory and keratoplastic preparations. Systematic observation of oral hygiene.

PERIODONTITIS AT CHILDREN

Unless the local and general factors are removed the inflammatory process will progress and turn into periodontitis – inflammatory process characterized by the progressive destruction

of the gingival-dental ligament and bone tissues. Besides inflammation of gums, periodontal pocket is formed.

Depending on localization periodontitis can be: local or generalized, and depending on evolution - acute or chronic. Acute periodontitis is rarely found at children, being in most cases caused by considerable traumas of periodontal tissues (inflicted by tools during tartar removal, in result of installation of orthodontic devices, caused by medicaments (arsenic), etc.).

Chronic periodontitis is found most often, being provoked by local factors.

In the pathogenesis of periodontitis the disturbances of sanguine circulation were found to be a triggering factor of the pathologic process. On the incipient stages appears the inflammation of gums, periodontium, pulp and alveolar bone. Progress of this process results: in the desquamation of gum epithelium and gingival sulcuss; widening of periodontal space; osteoplastic resorption of bone, etc. Clinical symptoms are more pronounced during the pubertal ages, when appear the development disproportion of periodontium and hormonal disharmony. Hormonal arrhythmia leads to disharmony in the evolution of epithelial gingival cells, reduces their defense properties and aggravates the inflammatory and dystrophic processes in periodontium.

Depending on the severity of the process three forms can be distinguished: light, medium and severe.

Light form: debuts with gum inflammation, insignificant hemorrhage, sub – and supra-gingival deposits can be present, periodontal pockets can be determined with depth up to 3.5 mm, with possible elimination. Teeth can display insignificant mobility.

Radiological image: widening of periodontal space in the collar region, destruction of cortical plate and tops of interradicular and interdental septums, hotbeds of osteoporosis.

Medium form: characterized by progress of the pathological process. Inflammatory process becomes more pronounced, hemorrhage becomes more significant, periodontal pockets can have a depth up to 5 mm, simultaneously they can contain granulation and purulent exudation, teeth can display mobility of degree II.

Radiological image: bone resorption between 1/3 and ½ of the intra-dental septum.

Severe form: continuous progress of inflammatory process. Periodontal pockets with depth exceeding 5-6 mm, mobility of degrees II-III, often with traumatic occlusion caused by teeth displacement. Abundant sub- and supra-gingival deposits are observed. The severe form of the disease is also characterized by retraction of gums and baring of roots followed by hyperesthesia. Sometimes retrograde pulpitis or periodontal abscesses can appear.

Radiological image: osteoporosis and destruction of alveolar bone for more than ½ of dental root; the vertical resorption variant with formation of bone pockets predominates.

Complementary methods: positive Schiller-Pisarev test, hygienic index > 1.0, PMA > 0, CPITN may vary 1+2+3+4, reduction of haematoma appearance time down to 10-40 s.

Differentiated diagnosis: with gingivitis, periodontal syndrome in sugar diabetes, X-histiocytosis, etc.

Treatment: A complex of curative measures is implemented: local conservative treatment, physiotherapeutic treatment, surgical, orthodontic and general treatment.

Local conservative treatment: removal of cause, removal of tartar, hygiene of buccal cavity; symptomatic treatment of gingivitis: with antiseptics (dioxydine 1% chlorhexidine bi-gluconate 0.3 %, etc.), wide spectrum antibiotics, anti-protozoan (metronidazole), antimycotic preparations (nistatine, levorine,

etc.), ferments (trypsin, chemotrypsin, lysozyme 1%, etc.), corticoids (unguents "Phtorocort", "Deperzolon") - only in severe cases; phytotrophic preparations (Chamomile extraction, Aloe extraction, etc.); prostaglandins inhibitors (ung. Butadioni 5%); antioxidants (galascorbine 1%, dibunole emulsion 1%, etc.).

Physiotherapeutic treatment is indicated after the removal of acute inflammatory process: electrophoresis (with calcium gluconate, with sodium fluoride, vitamins B1, C, etc.), ultrasound with medicaments (phonophoresis), gingival massage, hydrotherapy, etc.

Surgical treatment: phrenuloplastic and vestibuloplastic operations. Curettage of pockets, gingivotomy and gingivoectomy are not indicated for children.

Orthodontic treatment: amelioration of state of periodontium at children with dental-maxillary anomalies depends on the success of orthodontic treatment of such anomalies. Without good orthodontic treatment no sustainable results can be expected and indifferently on the curative measures the inflammatory process in periodontium will reoccur. In cases of traumatic occlusion it is recommended to polish selectively the points of excessive contact. Also it is recommended to immobilize the mobile teeth with the help of mobile orthodontic devices, buses and fixed devices.

AFFECTIONS OF PERIODONTIUM IN THE PUBERTAL PERIOD

Pubertal period comprises two stages: pre-pubertal stage (8-9 years to 13-14 years) and pubertal period (13-14 to 18-20 years). Namely during this period most cases of gingivitis occur due to the influence of sexual hormones on the gingival epithelium. Often during the pre-pubertal occurs the chaotic and irregular elimination of sexual hormones.

At girls of pubertal age increased excretion of sexual hormones and insufficiency of progesterone are noticed, thus

creating conditions for the predomination of proliferating processes in the epithelium of endometrium, other mucous tissues of the organism, including the gums. Under the influence of progesterone occurs the desquamation of epithelium that can result in the desquamative (ulcerous) gingivitis. In case of predomination of estrogen hyperplastic processes appear, i.e. hypertrophic gingivitis. Pubertal period is characterized by arrhythmia of hormonal status. Development of syndromes of hypertrophic gingivitis or desquamative gingivitis depends on the predomination of one or another hormone. Most frequent form of gingivitis during this period is the hypertrophic one that at children follows after the catarrhal form.

If gingivitis evolves during a long period of time and has a chronic character, it can gradually transform into marginal periodontitis with lesions of gingival-dental ligament and alveolar bone, especially when the pathological process is of hormonal origin and complicated by the action of other factors: non-observation of oral hygiene, dental-maxillary anomalies, excessive contact in occlusion, etc. In such cases juvenile gingivitis as a development factor of periodontitis persists after the stabilization of hormonal status after the pubertal age.

At adolescents with hypertrophic gingivitis may appear false pockets (without the lesion of enamel-epithelium insertion integrity). Hemorrhage in cases of hypertrophic gingivitis is provoked by the influence of hormones but not of the granular tissues as in cases of periodontitis at adults. This is why for the treatment of false pockets one must use anti-inflammatory and sclerotizing preparations. At adults with success can be used the method of curettage of the granular tissues from periodontal pockets with cicatrizing of the pocket surface. Curettage of false pockets at adolescents leads to the lesion of circular ligament and progress of the process, i.e. favors the evolution of gingivitis into periodontitis. Cicatrizing (in result of periodontal surgical

treatment) in conditions of hormonal arrhythmia often leads to the intensification of hyperplasic processes in gums. This is why surgical methods (curettage, gingivotomy) are contraindicated for children.

Treatment of chronic hypertrophic gingivitis consists in the removal of dental deposits, application of anti-inflammatory remedies, etc. and at last – of sclerotizing therapy. Physiotherapy is recommended: electrophoresis (KI 5%, 0,4-0,6mA, 20 min, 15-20 sessions, Lydase, Ronidase, heparin 5000Un, 15 min, 10-15 sessions, vacuum massage (300-400 mm col. Hg.).

Treatment of erosive (desquamative) gingivitis begins with adequate oral hygiene, removal of dental deposits, anti-inflammatory treatment, proteolytic ferments, keratoplastics.

Due to hormonal misbalance at adolescents it is not possible to fully remove the symptoms of juvenile gingivitis, however it is necessary to reduce its intensity by conservative methods, by an adequate level of oral hygiene, treatment of dental-maxillary anomalies, polishing of excessive contact points, phrenuloplasty, vestibuloplasty, etc.

PERIODONTOSIS AT CHILDREN

Periodontosis is rarely met at children but is characterized by destruction of periodontal tissues. Clinically it is characterized by recession of gums; their color is pale, without bleeding. Dental deposits usually are not found on teeth, the affection evolves without formation of pockets, while the teeth remain immobile even at advanced stages of the disease. Due to the baring of roots hyperesthesia from thermal and chemical excitants can appear.

Radiological examination: osteoporosis and osteosclerosis of certain sectors with reduction of bone volume, periodontal space becomes narrower.

Treatment: symptomatic.

IDIOPATHIC AFFECTIONS AT CHILDREN

Generalized periodontitis in sugar diabetes

Sugar diabetes s a well-known and widely spread disease, most often found at children of 3, 6 and 12 years. It is a severe disease with disturbances of metabolism, affection of sanguine system, especially capillaries and arterioles. The influence of all these negative processes on the periodontal tissues depends on the severity of the disease, timely diagnosis, treatment and its efficiency. For these reasons the symptoms of periodontitis and at incipient stages of generalized gingivitis at children (especially in cases of temporary dentition) shall be carefully considered by the doctor.

Symptomatology: clinical signs are manifested only at the advanced stages of the disease, this is why it is very important to determine the level of carbohydrates in blood and determine the glucose tolerance degree. Gingivitis is a disease of proliferate character. Cyanosis of marginal gums, granulation in the periodontal pockets, hemorrhagic and purulent exudations are characteristic. The teeth become mobile even in cases of superficial pockets - mobility of teeth doesn't correspond to the destructive process. Teeth can change their positions, thus leading to secondary anomalies and distortion of occlusion and complicating the evolution of pathological process. The teeth may have supra and subgingival deposits (see color insert fig. 9.1).

Radiological examination: one may notice a specific funnel- or cup-shaped alveolar bone destruction process that doesn't include the mandibular bone.

Treatment: complex, in collaboration with the endocrinologist.

Local treatment - symptomatic, with anti-inflammatory and sclerotizing remedies, ferments, etc.

Orthodontic treatment – as indicated. Regular observation of oral hygiene.

Generalized periodontitis in cases of hereditary neutropenia

The disease may have a permanent or cyclic evolution, being manifested by the considerable reduction of neutrophils with simultaneous increase of monocytes and in many cases of eosinophiles under normal or slightly reduced total number of leukocytes. A similar image is observed in the bone marrow. In cases of cyclic neutropenia changes are periodical, appearing after certain intervals of time.

Symptomatology: The disease is manifested from the first months of the child's life by the appearance of dermatitis, furuncles, abscesses, stomatitis, bronchitis, pneumonia, etc. Generalized periodontitis begins its manifestation simultaneously with dental eruption. First appears the ulcerous gingivitis, then after the age of 3 years appear the periodontal pockets, begins the destruction of alveolar bone and appears the mobility of temporary teeth. Similar phenomena are observed at permanent teeth after eruption, so that by the age of 12-14 almost all teeth may be extracted.

Radiological examination: the process is limited to the alveolar bone and has clear contours.

Treatment: Treatment is complex and requires the involvement of hematologists, pediatricians, etc. Treatment is local and symptomatic.

Generalized periodontitis in cases of hypoimmuneglobulinemia

This disease is provoked by the insufficiency of certain immune humoral factors and is characterized by the deficit of one or more groups of immune globulins and reduced immune reaction on the administration of antigen and sensitivity to bacterial infections.

Symptomatology: frequent development of purulent processes in different organs (abscess, phlegmon, piodermia, pneumonia, pyelonephritis, arthritis, etc.). Hypertrophic chronic gingivitis of

2-3 degree with moderate hemorrhage and jelly-like gingival papillae are characteristics. In cases of periodontitis the gingival pockets are profound, without dental deposits. There are no radiological signs allowing distinguishing between this syndrome and the usual periodontitis. However, absence of effect from the treatment of periodontitis, polymorph image of the disease and the investigation of immune globulins of sanguine serum allow to establish the diagnosis.

Treatment is complex, requires involvement of pediatrician, etc.

Local treatment of periodontitis is symptomatic.

THE PAPILLION-LEFEVRE SYNDROME

It is a primary dystrophic process based on the lesion of dental-gingival ligament and of the tissues of periodontium. Can appear in the localized form (monoalveolysis Orban) in the region of frontal teeth and primary permanent molars and in the generalized form (the Papillion-Lefevre syndrome) affecting both temporary and permanent teeth.

Symptomatology: the process begins with rotation, migration and mobility of teeth. Initially the gums have an usual appearance, but periodontal pockets appear. Afterwards inflammation appears, usually without pains, dental roots are bared and covered with whitish deposits, dental deposits are present in small quantities. The first symptoms are clear in the age of 2-3 years.

Children with this symptom suffer from transpiration, hyperkeratosis and deep cracks on the surfaces of feet and palms. Temporary teeth become very mobile and soon require extraction. Similar processes begin after the eruption of permanent teeth. Internal organs are not affected. The affection is suspected to be caused by the disturbances in the metabolism of amine acid named tryptophan.

Radiological examination: the process of alveolar bone

destruction has a vertical –oriented direction, other sectors of the maxillaries are unchanged.

Treatment is complex and must be performed in the specialized sections in collaboration with other specialists.

Local treatment: symptomatic.

X- HISTIOCYTOSIS

Actually, based on the character of evolution and involvement of bone lymphatic tissues and the visceral affections four clinical forms of the same disease are distinguished:

1. Affection of one skeletal bone;
2. Generalized affection of the bone system;
3. Generalized affection of both bone and lymphatic system;
4. Generalized affection of both bone and lymphatic system associated with visceral manifestations.

The form 4, also known as the ***Letterer-Zive disease***, evolves severely and is characterized by sleepiness, loss of body weight and appetite. In parallel with the progress of disease on the child's skin appear the sharpened papules of red or dark red color with tendency to conglomerate. Afterwards they are covered with crust. The affected children often suffer from stomatitis, otitis, mastoiditis and lymphadenitis. During the acute period of disease may appear such symptoms as polypepsia (5-10 liters of water per day), exophtalmia, and splenohepatomegaly, and diarrhea, disturbances of respiratory and cardiovascular system.

Laboratory analyses of blood show progressive anemia, thrombocytosis, eosinophilia, increased reaction on sedimentation of erythrocytes. Marrow bone puncture reveals reticulohistiocytic proliferation processes.

Ulcerous-necrotic gingivitis is characteristic for this affection: gums are inflamed, periodontal pockets contain granulation and exudation, gingival recession and bearing of dental roots are observed, teeth become mobile.

Radiological examination: the alveolar bone or the

maxillaries can have sectors of oval- and round-shaped destruction. Similar changes can be found in all the flat bones (skull, pelvis, scapulae).

Forms 2 and 3 (the ***Hend-Schiuler-Cristcen disease***) are manifested by gradual generalization of the process and slow evolution with remission periods. The maximum manifestation periods are characterized by exophtalmia, polypepsia, splenohepatomegaly, etc. Gingivitis appears after the progressing of general symptoms and can be useful in timely diagnostics. Often ulcerous gingivitis can be detected with unpleasant smell, dental mobility, gingival recession, profound periodontal pockets, etc.

Radiological examination: unlike in the other forms, space-type destruction of alveolar bone in combination with affections of other sectors of maxillaries can be observed.

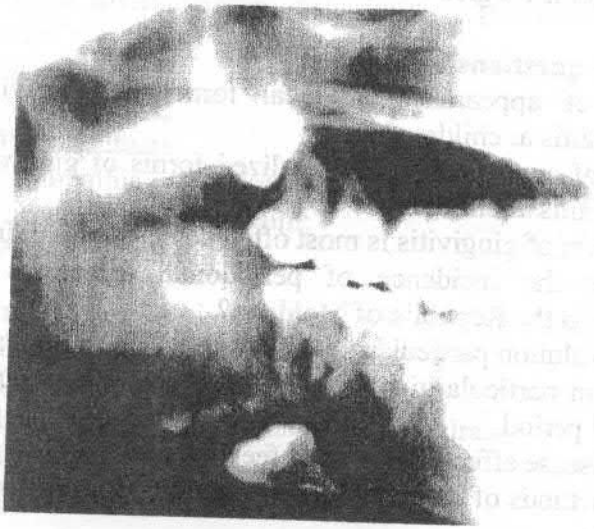


Figure 9.2. **Radiological image of X-histiocytosis: diffuse affection of spongious bone, interdental and interradicular septums, "floating" teeth.**

The form 1, or the ***Taratynov disease*** (also known as eosinophilic granuloma) is the lightest variant. The process has a chronic evolution and has favorable forecasts. Affection of a single flat bone is attested. In case the process is localized in the buccal cavity - symptomatic signs of pronounced marginal periodontitis appear.

Treatment: Local symptomatic treatment of periodontitis doesn't assure stable results and only the treatment of the general disease can lead to improvements in periodontal tissues.

PERIODONTOMA

It is a generic name of several periodontal affections of neoplastic origin, manifested by malign or benign tumor processes. In clinical practice often is met the gingival fibromatosis, papillomatosis and epulis.

Treatment is surgical.

Control questions and topics:

1. Causes of appearance of local forms of gingivitis and periodontitis at children
2. Causes of appearance of generalized forms of gingivitis and periodontitis at children
3. What form of gingivitis is most often detected at children?
4. What is the incidence of periodontal affections among children in the Republic of Moldova?
5. Basic evolution particularities of gingivitis at children.
6. Evolution particularities of periodontium diseases during the pubertal period.
7. What disease affects the skin of feet and palms?
8. Main methods of diagnosing the affections of periodontium at children.
9. Role of oral hygiene in the treatment of periodontal affections at children.
10. Radiological image characteristic for the affections of

periodontal tissues at children in cases of sugar diabetes.
11. Radiological image characteristic for the affections of periodontal tissues at children in cases of X-histiocytosis.
12. Particularities of treating catarrhal gingivitis at children.
13. Treatment of hypertrophic gingivitis at children.
14. Role of pediatrician in the diagnosis and treatment of periodontal affections at children.
15. Symptomatic treatment of periodontal affections at children.
16. What systemic diseases frequently lead to the affections of marginal periodontium at children?
17. Treatment of affections of marginal periodontium during the pubertal period.

CONTROL TESTS

Simple compartment

1. The most often detected pathology of periodontal tissues at children is:
   A. Gingivitis;
   B. Periodontitis;
   C. Periodontosis;
   D. Idiopathic affections with progressive destruction of tissues of periodontium;
   E. Periodontomas.

2. The main cause of gingivitis at children of preschool age is:
   A. Disturbance of hormonal status;
   B. Failure to observe the oral hygiene;
   C. Excessive consumption of carbohydrates;
   D. General diseases;
   E. Hereditary predisposition.

3. At children under 3 years can be positive the test:
   A. With bensidine;
   B. With formalin;

C. Schiller-Pisarev;
D. Kavetki;
E. Kulajenko.

4. Characteristic signs for edematous form of hypertrophic gingivitis include:
A. Hyperemia, gingival edema, periodontal pocket of 3.5 mm. dental mobility of degree I;
B. Hyperemia, gingival edema, gingival hemorrhage, dental-gingival sulcus of 3 mm;
C. Hyperemia, gingival edema, gingival hemorrhage, dental-gingival sulcus of 2-2.5 mm;
D. Hyperemia, gingival edema, false gingival pocket of 4 mm;
E. Hyperemia, gingival edema, periodontal pocket of 3 mm.

5. The characteristic signs of the light form of marginal periodontitis are the following:
A. Periodontal pocket of 4 mm, resorption of the top of intra-alveolar septum of 1/3, dental mobility of degree I.
B. Periodontal pocket of 3.5 mm, osteoporosis, reduction of the intra-alveolar septum height less than by 1/3, absent dental mobility.
C. False periodontal pocket of 3.5 mm, osteoporosis, absent dental mobility.
D. Hyperemia, edema, gingival hemorrhage, dental-gingival sulcus of 2 mm.
E. All answers are correct.

6. The quantity of hialuronidase in the periodontal pockets is:
A. Relatively higher;
B. Significantly higher;
C. Absent;
D. Insignificant;

E. Lowered.

7. Radiological examination in periodontology may provide data about:
   A. Bone density;
   B. Bone relief;
   C. Depth of periodontal pockets;
   D. Value of dental implantation;
   E. All answers are correct.

8. Physiotherapeutic treatment in cases of X-histiocytosis are:
   A. Indicated;
   B. Contraindicated;
   C. Must be combined with general treatment.

Multiple compartment

9. Periodontium consists of:
   A. Alveolar bone;
   B. Gums;
   C. Periodontium;
   D. Interradicular and interdental septums;
   E. Dentine;
   F. Cement;
   G. Circular ligament.

10. The destructive action of bacteria on periodontium is a consequence of:
   A. Interruption of sanguine circulation;
   B. Destruction of nervous terminations;
   C. Complex histolytic mechanism;
   D. Participation of exo- and endotoxins;
   E. Participation of enzymatic factors and cytokinines.

11. The destructive action of bacteria on marginal periodontium is a consequence of:

A. Complex histolytic mechanisms;
B. Participation of exo- and endotoxins;
C. Perturbations of local trophycity;
D. Reduction of sanguine circulation;
E. All answers are correct.

12. The structure of bacterial plaque comprises:
A. Serous inflammatory exudation;
B. Bacterial aggregates;
C. Desquamated epithelial cells;
D. Products of cellular metabolism;
E. All answers are correct.

13. Adherence of bacteria on the film of supragingival plaque is explained by the following mechanisms:
A. Precipitation;
B. Agglutination;
C. Hydrophobia;
D. Electrostatic phenomena;
E. Specialized bacterial structures.

14. The mineralized bacterial plaque covering the surface of tartar n the subgingival zone is characterized by the presence of:
A. Coccus;
B. Bacillus;
C. Filaments without characteristic orientation;
D. Bacteria oriented at straight angle to the surface of the tartar;
E. Specific inter-bacterial connections.

15. The components of saliva resisting to the formation and maturing of bacterial plaque are:
A. Mucin;
B. Lipase;
C. Lactoferrin;

D. Salivary tampon systems;
E. The L Po system.
F.

16. The factors determining the particularities of clinical evolution of periodontal affections at children are:
A. Morphological and functional immaturity of the tissues of periodontium;
B. Changes in the reactivity of organism;
C. Disproportion in growth and maturity of the tissues of periodontium, structures and systems assuring the adaptation of organism to the external environment;
D. Infectious diseases at children;
E. Acute herpetic stomatitis.

17. Particularities of clinical evolution of periodontal affections at children:
A. The inflammatory-destructive process is the major characteristic sign for all forms of periodontal affections.
B. Idiopathic affections of periodontium with progressive destruction of periodontal tissues and neoplasic processes are rare;
C. Affections of periodontium are associated with abrupt reduction of the immune state;
D. Most often the catarrhal and hypertrophic forms of gingivitis are met;
E. Pathological processes can disappear without traces under the action of minimum interventions or without them, or even after removal of cause, can become progressive.

18. The characteristic signs for catarrhal gingivitis are:
A. Hyperemia, gingival edema, periodontal pocket of 3.5 mm, dental mobility of degree I;
B. Hyperemia, gingival edema, gingival hemorrhage, dental-gingival sulcus of 3 mm;

C. Hyperemia, gingival edema, gingival hemorrhage, dental-gingival sulcus of 2-2.5 mm;
D. Hyperemia, gingival edema, false gingival pocket of 4 mm;
E. Hyperemia, gingival edema, periodontal pocket of 3 mm.

19. During the orthodontic manipulations are possible:
A. Increase of dental mobility of a tooth or of a group of teeth;
B. Destruction of supporting periodontium;
C. Appearance of true pockets;
D. Widening of periodontal space;
E. All answers are correct.

20. General diseases:
A. Are contributing factors for periodontal affections;
B. Give rise to periodontal diseases;
C. Accelerate the evolution of periodontal diseases;
D. Contribute to the appearance of complications;
E. Contribute to the appearance of recurrence.

21. General diseases:
A. Contribute to the appearance of periodontal disease;
B. Determine the appearance of periodontal disease;
C. Accelerate the evolution of periodontal affections;
D. Contribute to the appearance of complications and recurrence of periodontal affections;
E. Reduce the effect of local treatment.

22. The diseases that evidently influence the evolution of periodontal affections are:
A. Chronic rheumatism;
B. Respiratory infections;
C. Cardiovascular diseases;

D. Hypovitaminosis;
E. Diabetes.

23. Primary gingival lesion in gingivitis is represented by:
A. Ulceration;
B. Edema;
C. Congestion;
D. Irritation of tartar nature;
E. Septic inflammation.

24. Traumatic occlusion:
A. Produces an inflammation of periodontium;
B. Leads to the appearance of periodontal pockets;
C. Influences the pathological process of beginning and evolution of periodontal pockets;
D. Is a secondary factor in the pathogenesis of periodontal pathologies;
E. Produces non-inflammatory pathological modifications.

25. Hypertrophic and hyperplasic forms of gingivitis are most often met:
A. During puberty, at girls ;
B. At boys;
C. During pregnancy;
D. During certain physiological periods;
E. All answers are correct.

26. The test of dental percussion in periodontology is performed:
A. By moderate strike;
B. Longitudinally;
C. Transversally;
D. Only in the direction of dental axle;
E. At the level of vestibular surfaces.

27. Examination of patients with chronic marginal periodontitis is

performed:

A. By the explanation of the reasons for applying for assistance;
B. By anamnesis;
C. By subjective paraclinical examination;
D. By objective clinical examination of marginal periodontium;
E. By complementary examinations.

28. The clinical examination of marginal periodontium is performed by:

A. Exerting pressure;
B. Inspection;
C. Percussion;
D. Auscultation;
E. Palpation.

29. In periodontology radiography provides information on:

A. Degree of bone atrophy;
B. Shape of bone atrophy;
C. Morphology of periodontal space;
D. Evolution of bone resorption;
E. All answers are correct.

30. Radiological examination provides information on:

A. Localization, size and character of bone affections;
B. State of internal cortical structure of the alveolar bone;
C. State of spongious bone at the level of interdental septums;
D. Depth of false periodontal pockets;
E. Degree of pathologic mobility.

31. The radiological signs characteristic for periodontitis are:

A. Displacement of teeth;
B. Osteoporosis with preservation of anatomic shape of

interalveolar septums and cortical lamina;
C. Destruction of cortical lamina, osteoporosis of the interalveolar septum bone;
D. Resorption of the interalveolar septum bone from 1/3 to 2/3 and more;
E. Resorption of dental root.

32. Affection of the skeleton bones and maxillaries are characteristic for:
A. Papillion-Lefevre syndrome;
B. Sugar diabetes;
C. Letterer-Zive disease;
D. Hydroimmunoglobulinemia;
E. The Hend-Schiuller-Cristcen disease.

33. The characteristic signs for marginal periodontitis of moderate degree include:
A. Depth of pocket up to 4 mm;
B. Depth of pocket up to 6 mm;
C. Resorption of interalveolar septum of ½;
D. Dental mobility of degree 1;
E. Dental mobility of degrees 2-3;
F. Resorption of interalveolar septum of less than 1/3;
G. False pocket of 6 mm;
H. Resorption of interalveolar septum of 2/3.

34. The characteristic signs for severe marginal periodontitis include:
A. Depth of pocket up to 4 mm;
B. Depth of pocket up to 6 mm;
C. Resorption of interalveolar septum of ½;
D. Dental mobility of degree 1;
E. Dental mobility of degrees 2-3;
F. Resorption of interalveolar septum of less than 1/3;

G. False pocket of 6 mm;
H. Resorption of interalveolar septum of 2/3.

35. In cases of chronic hyperplasic gingivitis are indicated:
A. Laser therapy;
B. Ionophoresis with KI 5%;
C. Gingival self-massage;
D. Pointed diathermocoagulation;
E. Electrophoresis with lidase, ronidase.

Correct answers:

1. A.
2. B.
3.C.
4. D.
5. B.
6. B.
7. E.
8. B.
9. A, B, C, F, G.
10. C, D, E.
11. A, B.
12. B, C, D.
13. C, D, E.
14. A, B, C.
15. C, D, E.
16. A, C.
17. B, D, E.
18. B, C.
19. A, D.
20. A, C, D, E.
21. A, C, D, E.
22. A, C, D, E.
23. B, C.
24. C, D, E.
25. A, C, D.
26. A, B, C, E.
27. B, D, E.
28. A, B, C, E.
29. A, B, C, D.
30. A, B, C.
31. C, D.
32. C, E.
33. A, C, D.
34. B, E, H.
35. B, E.

10 ORAL MEDICINE AT CHILDREN

Structure of mucous tunic of the buccal cavity during the different development stages

The buccal cavity on its entire surface is covered with mucous tunic that in its turn is composed of three layers: epithelial, mucous itself and submucous.

The epithelium of buccal cavity consists of a basal layer and a spinous one. The epithelium of gingival mucous tissue and of the palate is pronouncedly keratinized.

The tunic of the mucous tissue itself is constituted of connective tissues rich in cellular elements and sanguine vessels.

Depending on the morpho-pathological particularities there are 3 types of mucous tunics: covering, masticatory and specialized. The covering mucous tissue is located on the lips, in the jugal region, on the palatal fold of the oral cavity, etc. Masticatory mucous tissues are located on the gums and on the solid palate, while the specialized ones – on the tongue.

The tunic of covering mucous tissue is characterized by lack of keratinization and by the existence of a pronounced submucous stratum.

The masticatory mucous tunic has a keratinization capacity and adheres closely to the periosteum as it doesn't have a submucous layer. The special mucous tunic on tongue has specific receptors (taste receptors). The mucous tunic on tongue doesn't have a submucous layer and thus is fixed straight to the muscle.

The mucous tunic of buccal cavity performs a series of specific functions; protective, sensitive, plastic and absorption.

At children the structure of mucous tunics in the buccal cavity (histologically and histo-chemically) changes considerably depending on age.

Three age periods with significant differences may be distinguished; these periods characterize the dynamics of development processes in the mucous tunics of buccal cavity:

I – newborn's period (from birth to 10 days) and sucker's period (till the age of 1 year);

II – from one year to three years;

III – from 4 years to 7 years and from 8 years to 12 years.

At newborns one can distinguish one similar structure (analogical) of mucous tunics in all the areas of buccal cavity (i.e., regional uniformity) that is determined by an inferior degree of epithelial and connective tissue differentiation. The epithelium is thin and contains much glycogen, RNA and a considerable quantity of acid mucopolysaccharides.

In the proper lamina one can observe some kind of incompletely developed connective tissue. The fibrous structures (collagen fibers and argyrophile) are insufficiently differentiated.

The submucous stratum contains many cellular elements: fibroblasts, histiocytes and lymphocytes. The number of macrophages is small and present in young forms only.

At newborns these particularities determine the structural fragility of the mucous tunic and higher regeneration capacities.

At suckers in the region of the mucous tunic of masticatory type gradually appear the centers of parakeratosis, glycogen gradually disappears, fibrous structures condensate in the proper lamina, the number of sanguine vessels and cellular elements decreases.

The connective tissues are weakly differentiated. With all these, simultaneously with the existence of higher quantities of glycogen and ARN, in al the sectors of the mucous tunic at newborns pronounced fucsinophilia may be observed, being

considered as a sign of existence of mature protein structures (that compose the collagen and elastic fibers). This phenomenon may be explained by the placental transmission of certain mature protein structures and mucopolysaccharides from mother to the fetus. At suckers fucsinophilia decreases, thus denoting the beginning of losses in the immune properties of tissues inherited from mother. By this fact one can explain the resistance of the child's organism to the viral and bacterial infections during the first year of life and the predisposition of buccal mucous tissues to the fungous affections.

During the period between 1 and 3 years of life clear regional differences in the mucous tunics become to appear. The quantity of glycogen in epithelium decreases. Fibrous structures are thin. The proper lamina displays an increase in the quantity of cellular elements and sanguine vessels, thus favoring the pronounced permeability of vascular walls. A greater number of mastocytes is determined in the connective tissues.

The particularities of mucous tissue at this age determine the reduction of immune reaction and the increase of vascular permeability and or these reasons it suffers more often from acute viral affections.

At the age of 4-7 years children display quantitative and qualitative modifications of the mucous tissues; increase of glycogen and ARN levels, reduction of the number of sanguine vessels and cellular elements - all these being evidences of substantial reduction in metabolic processes.

During the period between 8 and 12 years takes place the reduction of glycogen levels and the increase of protein structures. In the proper lamina considerably increases the number of lymphoidal-histiocytic elements. During this period occurs the reduction of predisposition to diffuse inflammatory reactions of the buccal mucous tunics and increases the incidence of allergic reactions.

After the age of 12 the histological and histochemical properties are characterized by the changes influenced by humoral factors, predomination of juvenile gingivitis and soft leukoplakia.

Generally, the acute evolution of inflammatory process is characterized by the appearance of alteration and exudation processes on the oral mucous tissues.

Classification of buccal mucous tissue affections by WHO

K.12. Stomatitis and related affections:

With exception of:

- Solid chancre;
- Streptococcal gingivo-stomatitis;
- Herpangina;
- Pyostomatitis;
- Allergy;
- Candidosis;
- Flu affections;
- Mycosis;
- Red lichen;
- Epizootic aphthe;
- Medicamentous stomatitis;
- Fusospirillary stomatitis;
- Mycotic stomatitis;
- Ulcerous-necrotic stomatitis;
- Vesicular stomatitis in acute fevers;
- Viral stomatitis;

K.12.0. Recurrent buccal aphthe:

- Mucilitch Aphthe;
- Recurrent ulcerous-aphthous affections;
- Aphthous stomatitis.

K.12.01. Recurrent necrotic periadenitis of mucous tunic:

- Sutton Aphthe;

- Major aphthe;
- Cicatrizing aphthous stomatitis;

K.12.02. Herpetiform stomatitis;
K.12.03. Bednar Aphthosis;
K.12.04. Traumatic ulcerations (tongue ulcerations excluded);
K.12.08. Other recurrent buccal aphthae – precise and imprecise.
K.12.1. Other forms of stomatitis:
K.12.10. Unusual and uncommon stomatitis,
K.12.11. Geographic stomatitis;
K.12.12. Proteic stomatitis;
K.12.13. Polypous palatal hyperplasia;
K.12.14. Contact stomatitis;
K.13.20. Idiopathic leukoplasia;
K.13.21. Tabbagic leukoplasia;
K.13.22. Erythroplasia;
K.13.23. Leukoedema;
K.13.24. Nicotinic leukokeratosis of palate;
K.13.3. Tricholeukoplasia;
K.13.4. Granulomas and pseudogranulomatous lesions of mucous tunic;
K.13.40. Pyogenic granulomas;
K.13.41. Eosinophilic granulomas of buccal cavity;
K.13.42. Verucous xantomas (histiocytosis);
K.13.6. Inflammatory hyperplasia of buccal mucous tissues;
K.13.7. Lesions of buccal mucous tissues of other imprecise origin;
K.13.70. Melanic exaggerated pigmentation (melanoplasia, smoker's melanosis);
K.13.71. Buccal fistulas;
K.13.72. Pigmentation of oral mucous tissues;
K.13.73. Oral mucinosis;

K.13.78. Other stomatitis;

K.14. Affections of tongue:

K.14.0. Lingual abscess;

K.14.01. Traumatic ulcer

K.14.08. Other precise glossitis;

K.14.1. Geographic tongue

- Marginal exfoliative glossitis;
- Benign migrating glossitis;

K.14.2. Losanic median glossitis

K.14.3. Hypertrophy of lingual papillae;

K.14.30. Charged tongue

K.14.31. Black tongue except for black hairy tongue in antibiotics and tricholeukoplasia.

K.14.32. Hypertrophy of foliated papillae;

K.14.38. Other hypertrophy of papillae;
Black tongue in antibiotics;

K.14.4. Atrophy of lingual papillae;

K.14.42 Atrophic glossitis;

K.14.5. Plicate tongue:

- fissured;
- scrotal;
- stripped;

K.14.6.Glossodynia:

K.14.60. Glossospirosis (hot tongue);

K.14.61. Glossodynia (painful tongue).

K.14.80. Crenellated tongue;

K.14.81. Tongue hypertrophy;

K.14.82. Tongue atrophy, hemi-atrophy of tongue.

Classification of affections, modification and diseases of mucous tunic at children (Виноградова Т. Ф., 1987)

I. By etiology:

1. Viral affections:

- acute herpetiform stomatitis;
- recurrent herpetiform stomatitis;
- vesicular stomatitis;
- viral verrucosis

2. Mycotic affections:
   - Acute and chronic candidosis;
   - Candidomycosis, etc.
3. Bacterial affections:
   - Vincent ulcerous-necrotic stomatitis;
   - Stomatitis in case of tuberculosis;
   - Gonococcic stomatitis;
   - Syphilitic stomatitis;
4. Allergic affections:
   - Exudative polymorphic erythema;
   - Stomatitis, gingivitis, glossitis, allergic contact cheilitis (medicamental, etc.);
5. Affections of the buccal mucous – as symptoms of pathology of internal organs and systems:
   - At the level of digestive tract: recurrent aphthae of the buccal mucous tissues, etc.
   - Infectious diseases: scarlatina, diphtheria, varicella, flu, etc.
   - Sanguine diseases: desquamative Hunter glossitis in anemia; ulcerous stomatitis in leucosis, etc.
   - Dermatosis: Duhring dermatitis, Lupus erythematosis, planus red lichen;
   - Endocrine diseases;
   - Cardiovascular diseases;
   - Neural-psychic diseases;
6. Lesions of mucous tissues of buccal cavity caused by the action of mechanical, thermal and chemical factors

(Bednar aphthae, decubital aphthae, erosion, etc.);

II. By clinical evolution: acute and chronic (recurrent and permanent ones);

III. By localization: stomatitis, papillitis, gingivitis, glossitis, cheilitis, palatinitis, pareitis, etc.

IV. By morphological manifestations:

- Primary elements: vesicles, bubbles, papules.
- Secondary elements: erosions, aphthae, ulcers, cicatrices, maculae.
- Inflammation: catarrhal, fibrinous, alterative and proliferative.

VIRAL AFFECTIONS

Herpes Simplex

Acute herpetiform stomatitis (AHS)

It is one of the most widespread infections at people and met in 80% of all cases of stomatitis at children (Виноградова Т.Ф., 1973). It is the primary clinical form of herpetic infection.

Etiology: AHS is provoked by the virus named Herpes Simplex. Sources of infection are the ill peoples and the carriers of the virus. Most receptive are the children aged between 6 months and 3 years, as at this age the antibodies procured intra-placentarely from mother gradually begin to disappear while the own immune mechanisms are not yet in place. The virus penetrates into the organism through the place of direct contact with an ill person or carrier of virus through the common household use objects, toys, nipples, etc. and via respiratory ways. In the collectives of pre-school institution epidemics of herpetic stomatitis can occur.

Pathogenesis: AHS evolves as an acute infectious disease in 5 periods: incubation, prodromal period, period of clinical manifestation (catarrhal, eruptive), curing period (epithelization) and clinical recovery.

Appearance of AHS is favored by the lesion of mucous tunic, by dental eruption and morphological and immune particularities of the buccal mucous tissues, organism's reactivity, etc. The viruses develop and reproduce in epithelial cells of oral mucosa, spreading through the lymphatic ways. This is why one of first signs of infection is regional lymphadenitis.

The organism engages the following defense mechanisms: phagocytosis of cells affected by virus, formation of interferon, hyperthermic reaction, and formation of antibodies. However, at children these reactions are still imperfect.

The incubation period lasts from 2 to 17 days. If the defense forces of the child's organism (local and general tissue immunity) can not extinguish the virus, primary virusemia appears and thereafter the virus affects the cells of liver, spleen and other organs, reproducing at high rates. In result of such process appear the centers of necrosis in these tissues and secondary virusemia evolves - the prodromal and catarrhal periods. After this, generalized affection of epithelial cells of the mucous tissues occurs. The centers of mucous tissue necrosis appear in result of the direct action of the virus on the cells and due to the formation of capillary thrombi. Systemic blood coagulation disorders occur and the organism becomes more predisposed to hemorrhages.

Also may be affected the skin, the mucous tissues of eyes, of gastrointestinal tract, of genital organs. Often clinical forms of herpetic infection are associated.

Evolution of the following morphologic elements may be observed: maculae, vesicles, aphthae (erosions), maculae.

The Herpes Simplex virus leads to immune-depressive states in the organism, being manifested by the changes in the immune status at different stages of the disease (content of immune globulins, lysozyme, interferon, phagocytosis of neutrophils - B-lysine, etc.).

Symptomatology: General intoxication syndromes and local

manifestations on the mucous tissues of the buccal cavity predominate and determine the severity of the disease.

Light form of the disease is characterized by absence of visible intoxication in the organism. The prodromal clinical stage is not distinguished. The patients can have fevers with temperature up to $37,5^0$ C, the general state is usually satisfactory. The mucous tissues of buccal cavity and especially the gums are hyperemic and edematous (catarrhal gingivitis). In most cases such a state is preserved for no more than one day, after which the eruptions appear. Usually, rare maculae or bubbles are detected on the mucous tissues. Most often are met the aphthae. Aphthae have rounded, oval or figured shapes with clear edges, with smooth bottoms, covered by gray- white films and surrounded by hyperemic rings, painful. In case of light form the number of aphthae doesn't exceed 3-5. This stage usually lasts for 2-3 days.

During the curing period, after the removal of necrotic mass elements begins the epithelization. Usually gingivitis is preserved for 2-3 days after epithelization; lymphadenitis is preserved for 7-10 days (see the color insertion, figure 10.1).

In saliva, especially at the beginning of the disease are increased the fractions IgA and IgG. No considerable deviations from the standard sanguine formula are observed: insignificant lymphocytosis by the end of the disease.

Medium form: During the prodromal stage the general state is affected: weakness, absence of appetite, the child becomes capricious. Often catarrhal angina or the signs of an acute respiratory disease are observed. Fever with temperature up to $37,5^0$ C. Lymphatic ganglions are enlarged and painful at palpation.

The period of clinical manifestation is characterized by headaches, retching. The skin is pale; body temperature can reach $38 - 39^0$ C. In the buccal cavity and in the perioral region, on the background of hyperemia and edema, can appear 5 to 25 herpetic

elements (aphthae). This period is also characterized by catarrhal gingivitis of medium severity, saliva excretion is intensified (hyper salivation occurs). The child refuses to eat, doesn't sleep well, elements of secondary toxicosis become more and more pronounced. Often recurrent eruptions appear (see the color insertion, figure 10.2).

The curing period very much depends on the reactivity of organism. Epithelization of elements lasts up to 4-5 days. Gingivitis, hemorrhage and lymphadenitis are preserved for a longer time.

Blood analysis: most often - leukopenia, sometimes leukocytosis, lymphocytosis, RSE up to 20 mm/hour. Essential disturbances in the immune status are present. The activity of neutrophils during the development period of the disease is 3 times smaller and begins to grow only during the period of curing. The cellular immune and humoral factors are not fully reinstated during the recovery period.

Severe form: The severe form of this affection is observed much more rarely compared to the other forms. During the prodromal period it is characterized by adynamia, headaches, arthralgia, etc. Sometimes nasal hemorrhages are observed, retching, vomiting, etc. Disturbances of various systems of organism are possible. Pronounced lymphadenitis of submandibular and cervical ganglions is present. During the development period of the disease the child's body temperature can reach $39\text{-}40^0$ C. Sore throat, cough, hyperemia and edema of eye conjunctiva are observed. Lips are dry, ruby red. Endobuccal examination one can determine that the mucous tunic is edematous, hyperemic, catarrhal gingivitis is present. After 1-2 days eruptions appear not only in the buccal cavity but also on the skin in perioral region, on eyelids, on fingers of hands appear the bubbles characteristic for herpetic affection. Eruptions in the buccal cavity can appear for many times and there may be

observed more than 100 elements at different development stages. Often the aphthae get joined together, forming large necrotic surfaces in the buccal cavity. Sometimes the catarrhal gingivitis turns into ulcerous necrotic form. Saliva and nasal elimination can contain traces of blood, sometimes abundant nasal hemorrhages are observed (see color insertion, figure 10.30.

Blood analysis: leucopenia, eosinophilia, young forms of neutrophiles.

Urine analysis: traces of proteins.

Immune investigations: during the period of eruptions IgA, IgG, IgM and S-IgA are increased. During the curing period the IgG continues to grow, IgM is normalized; IgA and S-IgA are reduced.

The indicators of phagocytosis are low. At 80 % of children gastric juice looses acidity. The curing period depends on the quality of treatment, on the suffered complications and parallel evolution of other diseases. Clinical recovery is despite, after the severe form of herpetic affection substantial negative changes of homeostasis follow: reduction of bactericidal and lysozyme activity.

Diagnosing: Laboratory methods include the virusological ones, serological, cytological and immune fluorescent ones (see the color insertion, figure 10.4).

Differentiated diagnosing: with herpetic angina, polymorphic exudative erythema, aphthous fever, allergic affections.

Treatment: volume and character of treatment measures depend on the disease evolution stage, on the severity of the disease, character of evolution and associated infections.

Treatment includes a complex of curative measures, both local and general. The light and medium forms are usually treated in ambulatory conditions, while the severe forms require hospitalization.

General treatment: Antiviral preparation Bonafton 0,025 g 1-4 times a day during 3-5-7 days in doses corresponding to age; Virolex tablets 200 mg 5 times a day; Acyclovir during the first 24 hours 0,2 g 5 times a day – during 5 days.

In severe cases, taking into consideration the intoxication of organism, disintoxication therapy may be prescribed (intravenously: haemodesum, reopoliglucinum, glucose 5%, isotonic solution, etc.). Depending on age, antihistaminic preparations may be indicated:

- Dimedrol – to children aged under 2 years 0,002 -0,05 g, at the age of 2-6 years – 0,005-0,015 g, to the ones aged between 6 and 12 years – 0,015 -0,05 g;
- Suprastine - to children aged under 1 year 0,002 -0,05 g, at the age of 1-5 years – 0,005-0,015 g, to the ones aged between 5 and 12 years – 0,015 -0,05 g 2-3 times a day;
- Diprasine – to the children aged less than 6 years – 0,008 - 0,01 g 2-3 times a day, after the age of 6 years – 0,012 -0,015 g 2-3 times a day;
- Diasoline – 0,02-0,05 g 1-2 times a day.

Antihistaminic preparations are administered in combination with vitamin C (Ac. ascorbinici).

In cases of severe forms complicated with fusobacteriosis metronidazole or wide spectrum antibiotics shall be prescribed. In conditions of hospital local and general immune stimulation preparations may be used: prodigiosan 15-15 mlg (2-3 injections) once per 3-4 days, lysozyme 75-100 mlg per day during 5-7 days, gamma-globulin 1,5 – 2 ml once per 3-4 days (1-3 injections), methyluracyl 0,15-0,25 g, pentoxyl 0,05- 0,1 g, sodium nucleinate 0,001-0,002 2-3 times a day during 3-5 days.

If necessary, symptomatic therapy is prescribed: antipyretics (for the prophylaxis of hyperthermic syndrome – at hyperthermia above 390C), cardiovascular action preparations in doses adequate for the age, etc. Vitamins A, C and group B are also

recommended.

Local treatment: During the periods of incubation, prodromal and clinical manifestation periods local antiviral preparations are applied (unguents: tebrofen 0.5%, Bonafton 0.5%, Acyclovir 5% cnume, Florenal 1%, Ryodoxol 0,25%, Gossipol liniment 3%, Oxolin 0,25 %, etc. A similar action is produced by solutions and unguents of leukocytal interferon and 0,2 % solution of deoxyribonucleic acid (DNA). These medications are applied onto the mucous tissues of the buccal cavity for 5-6 times a day.

As the eruptions in the buccal cavity are very painful, 5-10% anesthesine in vegetal oils must be applied, solutions or unguents 1-2% of pyromecaine, Anaestho Gel (Voco), 1% solution trimecaine, plastocaine, etc.

Antiseptic treatment of teeth involves the removal of soft dental deposits with weak antiseptics (sol. Furacillin 1:5000, etonium 0,5 %, etc.). Proteolytic enzymes are used for the removal of necrotizing tissues (trypsin, chemotrypsin, lysozyme, terylitin, etc.).

In order to stimulate the epithelization processes during the curing period the antiviral preparations are gradually substituted by keratoplastic preparations: oily solution 3,44% of vitamin A, carotoline, Fructis Rosae oil, aloe liniment, aekol, vinylin, Solcoseryl unguent and gel, "Livian"and "Vinizol" preparations, etc.

Physiotherapeutic methods include: ultraviolet rays, helium and neon laser (only at the beginning of epithelization).

Patient care: Special regime is required to include the curative, anti-epidemic measures, bed regime and special nutrition. It is recommended to have the child consume as much liquids as possible. Fresh vegetable soups are recommended, milk products (milk, yogurt, cheese), boiled eggs (if the child tolerates them), fruit and vegetable juices (carrot, cabbage, etc.). The food must be warm and not irritate the buccal mucous tissues. Before

eating the mucous tissues of the buccal cavity must be treated with anesthetics and after meals - washed with antiseptics.

Prophylaxis: AHS is a contagious infectious viral affection and represents a special threat for the children of risky groups (aged between 6 months and 3 years).

As mass prophylaxis of AHS is not possible, certain anti-epidemic measures are rational to be undertaken in the nidus of infection. First of all, the affected children must be isolated from the other children (they shall not attend the preschool institutions). For the purpose of prophylaxis, all children that were in contact with the affected child shall be administrated antiviral preparations. The affected child must stay in his individual bed; have individual bed linen, clothing, toys and individual plates. Persons taking care for children in the institutions of preschool education shall regularly pass medical examination.

In cases of AHS the pediatrician dentist must undertake the following actions: announce respectively the kindergarten or the school, visit the respective group of children and organize the performance of anti-epidemic measures: disinfection of objects, toys, etc. with 1-2 % chloramines, etc., irradiate the rooms with ultraviolet rays, administer antiviral medications to the children, isolate the sick child and report the case of infection to the center for preventive medicine.

Chronic recurrent herpetic stomatitis (CRHS)

The herpetic infection has a chronic character, being characterized by periodic exacerbation. It appears at children of different ages primarily infected with the Herpes Simplex virus and already having antibodies to neutralize the virus.

According to the data of T. Виноградова, every tenth child that ever suffered from AHS thereafter suffers from recurrent affection with gradual transition into chronic recurrent form, at 69.3% of children the first recurrent affection occurs in terms of

up to one year after AHS.

The mechanisms of chronic herpetic infection are complicated. It is considered that transition into a latent form is the result of the influence produced by the virus onto the immune mechanisms and their accommodation. During remission periods the virus is localized in the ganglions of nerves enervating the mucous tissues and the portions of skin where the herpetic elements appear in cases of recurrence. When the immunity levels of organism decrease (acute respiratory diseases, bronchitis, pneumonia, tonsillitis, antritis, intoxications, stress, administration of immune depressants and steroids, traumas of mucous tissues, etc.) the virus is activated. One of the most important signs of CHRS is the typical and permanent localization of elements (on lips, on the mucous tissues of buccal cavity, on eyelids, on genital organs). Localization of recurrent eruptions depends on the primary localization of infection. Most often recurrent eruptions appear during the spring, summer and autumn. Frequency of eruptions varies considerably.

Symptomatology: Patients have accuses on permanent pains in the regions of herpetic elements on the mucous tissues of buccal cavity, pains become stronger when eating and speaking. General state of health may be affected: weakness, children get tired quickly and are irritated.

Appearance of herpetic elements on the mucous tissues of buccal cavity is preceded by sensations of burning and pains. The herpetic elements usually have a typical localization: on the red margin of lips, skin of perioral region, solid palate, tongue, cheeks. On the mucous tissues appear some easily bursting vesicles, thus forming small red erosions (3-5 mm) of round or oval shape, localized in groups, sometimes unified and covered with white-grayish patch

The affection may have three forms of severity:

The severe form: at children this form is detected rarely (at 7

% of children with CHRS). This form of affection is characterized by recurrence exceeding 4 times a year. The permanent recurrent form is determined in very rare cases.

Disorders of general state of health in such cases are observed mainly at children aged less than 3 years. Older children support lightly the recurrent affection, however, they also display disturbances of general health, suffer from pains in articulations, etc.

The medium form is determined at 12 % of affected children. Recurrences are manifested 1-2 times per year. Disturbances of general state of health are characteristic for the children aged less than 3 years.

The light form at children is met most often – in 81 % of cases. Recurrences are rare – 1-2 times per 3 years. Unitary herpetic elements appear on the mucous tissues of buccal cavity (1-2 pieces), usually in the same places. General disturbances of health are rare.

Diagnosing: Sometimes correct diagnosing is associated with difficulties, in such cases use of virusological, immunological and cytological methods are necessary.

Differentiated diagnosing: differentiated from recurrent aphthae on the mucous tissues of buccal cavity and allergic eruptions.

Treatment is mostly oriented to the avoidance of recurrences: general prescriptions aimed at raising the immune status, removal of infection centers (assanation of buccal cavity, etc.), liquidation of vicious habits (lesions of mucous tissues, biting, lip sucking, etc.).

Local and general treatment in cases of CHRS recurrences doesn't substantially differ from the AHS therapy.

During remission periods immune stimulation preparations are indicated (gamma-globulin, prodigiosan, pentoxyl, decaris (laevamizol), anti-herpetic vaccine, etc.), vitamins (C, A, vitamins

of group B).

Dispensary control: Children with CHRS must be registered and observed dispensary. The volumes and frequency of measures depend on the severity of the disease. However, first of all one must implement the complex of primary and secondary prophylactic measures against herpetic infection at children.

AFFECTION OF MUCOUS TISSUES OF BUCCAL CAVITY IN CASES OF ACUTE VIRAL RESPIRATORY INFECTIONS

The group of acute viral respiratory infections (AVRI) is characterized by polyetiology and analogous clinical manifestations on the wide background of clinical evolution and localization on respiratory organs.

Influenza - viral infectious disease characterized by symptoms of intoxication and catarrhal inflammation of upper respiratory ways. The pathogenic agent of flue is a RNA virus.

The flu virus is epitheliotropic and has a general toxic action on the organism. For flu are characteristic the desquamation of epithelium of respiratory ways, degeneration, superficial necrosis, especially in the region of pharynges. In some cases these affections are extended onto the mucous tissues of cheeks, tongue, soft palate, etc.

The para-influenza infection is characterized by moderate intoxication and affection of the internal nasal and laryngeal mucous tissues. The virus of human para-influenza belongs to the family of Paramyxoviridae, such viruses contain RNA. Most often are affected the children aged under two years.

Adenoviral infection is acute respiratory affection characterized by fever, moderate intoxication, affection of respiratory ways' mucous tissues, sometimes are affected the eye conjunctiva and the lymphatic system. By the age of 5 years practically all the children have already had the adenoviral

infection. This infection is transmitted by air (less often by contact).

Symptomatology: Appearance of eruption elements in the buccal cavity is preceded by fever with temperature 37-38^0 C. Regional lymphadenitis is also observed. The mucous tissues of buccal cavity become evidently hyperemic and edematous. Sometimes catarrhal gingivitis appears. Salivation is intensified. Mucous tissues can develop hemorrhages. After 1-2 days the temperature falls and the mucous tissue develops small vesicles with yellow or transparent contents, gradually these vesicles increase in diameter, turning into bubbles or papules. One or two days later erosions (aphthae) appear. Epithelization processes begin after 3-4 days.

Viral respiratory infections may be complicated by acute herpetic stomatitis, Vincent's ulcerous-necrotic stomatitis, etc.

Treatment is complex, involves consultation with pediatrician and other specialists. Usually, special treatment of affections of buccal cavity is not required, symptomatic treatment is indicated most often.

ENTEROVIRAL INFECTION (*KOXAKI AND ECHO*) HERPANGINA OR KOXAVIRAL STOMATITIS

The transmission mechanism of enteroviruses is both respiratory and fecal-oral. Children are highly susceptible to enteroviruses. Most often are infected the children aged between 3 and 10 years. This infection is characterized by seasonal manifestation (summer and autumn). The enteroviral infection is contagious and can cause epidemics in the children's communities. The Koxaki viruses contain RNA. Replication of viruses takes place in the epithelial cells and in the lymphatic system of upper respiratory organs and in the intestine. Thereafter through the blood system the virus penetrates into various organs and systems. In most cases the nasal-pharyngeal mucous tissues are affected.

The diseases provoked by the ECHO and Koxaki A viruses may have various manifestations. By the main clinical syndrome the disease may take the following forms: serous meningitis, epidemic myalgia, enterovirus fever, enterovirus eczema, herpetic angina, etc.

Herpangina was for the first time described in 1927 by I. Zagorsky who proposed this term not anyway corresponding neither to the etiology no to the clinical signs of the disease. Herpangina is met at children of different ages, most often in organized children's collectives. Affections of adults are rare. The incubation period lasts for 2-12 days. The pathology debuts suddenly, body temperature rises up to 39-40^{0}C. Children have accuses on headaches, weakness, disturbances of sleep, absence of appetite, myalgia (most often in the abdomen and pains when moving the ocular globes). Recurrent vomiting can also be noted. Hyperemia of cutaneous tissues of the upper part of the body (especially of face and neck) is characteristic for this affection (see the color insert, figure 10.5).

With beginning from the very first days of the disease, on the plications of palate, on tongue, on the solid and soft palate appear small red papules with diameter 1-2 mm that shortly turn into fine vesicles surrounded by inflammatory areas of red color. Eruptions may be multiple, sometimes unitary elements can appear. After 1-2 days the vesicles burst and turn into superficial erosions covered with grayish-white detritus. Often the mucous cavity is of normal color; in some cases it may be moderately hyperemic. New elements may appear during the evolution of disease, leading to increase of body temperature and worsening of general state of health. Elements are painful at touch; however, pain is felt only when swallowing. Regional lymphatic ganglions are enlarged but not painful. Fever persists during 1-3 days. The period of element regeneration is long-lasting. In the peripheral blood, during the first 10-15 days the number of leucocytes may be slightly

increased, VSE is normal.

Treatment: During the period of acute manifestations confinement to bed is strongly recommended, followed by ambulatory regime and diet. In cases of hyperthermia and headaches antipyretics and analgesics are indicated respectively. Hypo-sensitization preparations are prescribed. Due to the particularities of element location the local therapy is provided in the form of irrigations with solutions or aerosols containing antiseptic and analgesic substances, proteolytic ferments, antiviral remedies and keratoplastic agents. Helium-neon laser can also be used.

Prophylaxis: For the purpose of prophylaxis in the nidus of infection human interferon is administered in doses of 5 drops for 3-4 times a day during 10-15 days or other antiviral preparations.

ZOSTER ZONE AND VARICELLA

The Zoster Zone (Zoster Herpes) is provoked by the virus Varicella Zoster. It was established that during the non-immune period the virus provokes an acute infection- varicella, while during the immune period (at children that have already had the varicella) the virus provokes the Zoster Herpes. Recurrence in the form of Zoster Zone appears in result of reactivation of the latent form of the virus persisting in the Glasser ganglion of the trigeminal nerve under the action of numerous factors, such as acute diseases, mechanic traumas, treatment with glucocorticoids and immune depressants, etc. The infection is spread by air and by direct contact. There are several described cases when children got infected in result of contact with adults affected by Zoster Zone. Most often the infection occurs in autumn and winter.

Symptomatology of varicella: The incubation period lasts for 7-14 days. Most often are infected the children aged between 1 and 3 years. The prodromal period is characterized by absence of appetite, the infected children become capricious, have headaches, etc. Later appear the fevers with temperature of 38-

39^0C, general intoxication syndromes, and vesicular erythematosus elements on skin and mucous tissues of buccal cavity. Sometimes similar elements may be found on the eyes conjunctive and on genital organs (see the color insertion, figure 10.6).

Elements erupt on the edematous and hyperemic mucous tissues. As a rule, vesicles in the buccal cavity appear simultaneously and rapidly turn into erosions of oval shape with clear contours, similar to aphthae. In some cases recurrent eruptions are possible. Simultaneously with the appearance of eruptions in the buccal cavity the children begin to complaint on pains intensifying during mastication and speaking. Submandibular lymphatic ganglions are enlarged and slightly painful.

Symptomatology of the Zoster Zone: Most often this disease is found at adults, however, children aged above 7 years can also be affected. The disease begins acute with a rise of body temperature up to 38-390C, followed by headaches, absence of appetite, intense pains in the places where eruptions are to appear. One to three days later on the skin and on the mucous tissues of buccal cavity alongside the neuro-vascular fascicles appears a multitude of vesicles (vesicular form). Vesicles erupt and turn into erosions. Sometimes vesicles may have hemorrhagic content (hemorrhagic form) or necrotic content (gangrenous form).

Differentiated diagnosing: with acute herpetic stomatitis, allergic affections and pemphigus.

Treatment comprises a series of complex measures and is performed in collaboration with pediatrician, infectiologist, etc.

General therapy: antiviral preparations (Bonafton, DNA-se, etc.); hyposensitizing, analgesics, vitamins of group B, vitamin C, etc.

Local therapy: antiviral unguents, anesthetics, antiseptics, keratoplastic preparations.

Anti-epidemic activities are required. In prophylactic

purposes to healthy children antiviral preparations are administered. Sick children must be isolated at home for 5 more days after the appearance of last eruptions.

MEASLES or RUBEOLA

Measles is an acute infectious disease, characterized by fever, intoxication, catarrhal inflammation of upper respiratory ways, mucous tissues of eyes, eruptions in the form of maculae and papules.

The pathogenic agent of this disease is a virus from the Family of paramixoviruses. The virus contains RNA. The disease can occur during any season of the year. However, most often it occurs during the autumn-winter period and in the springtime. The source of infection is a sick person. The patients are most contagious during the catarrhal period and during the first day of eruptions. The infection is transmitted by respiratory way. The virus penetrates into the organism through the mucous tissues of upper respiratory organs. Further the virus penetrates into the blood. The virus displays tropism to CNS, respiratory and gastrointestinal system. In pathogenesis the most important feature of this virus is its general and local immune reduction capacity. After the influence of this virus, the assimilation of vitamins C and A decreases.

Evolution: the incubation period lasts for 8-12 days. The disease begins with fever (38-390C), dry cough, rhinitis, conjunctivitis. Hyperemia of conjunctiva, photophobia, and palpebral edema are observed and further associated with purulent secretion. General state of health is worsened.

One to three days before the appearance of eruptions on skin and on the mucous tissue of solid and soft palate appears an enanthema in the form of small maculae of pink-reddish color. Thereafter, on the mucous tunic of cheeks in the region of molars, sometimes on the mucous tissues of lips appear numerous grayish-white points sized as sand particles, surrounded by red

inflammation background. The buccal mucous tissues become lax, rough, hyperemic and dull. These signs are known under the name of Koplick-Filatov-Belski maculae. They develop in result of partial necrosis of superficial epithelial layers of the inflamed buccal mucous tissues. These maculae represent some white-yellowish sectors projecting out of the surface of epithelium, by size not exceeding a needle eye. These elements can not be removed from the mucous tissues and can get melted together. И. Новик (1971) describes them as "drops of lime". In line with evolution they obtain a reddish color.

As the resistance of organism to other infections is reduced, especially during the prodromal period of rubeola, it can get complicated by AHS or by exacerbation of CHRS at children aged above 10 years.

Treatment: Of major importance in the complex therapy in cases of rubeola are the adequate sanitary-hygienic conditions. Local treatment is symptomatic: anesthetics, antiseptics, antiviral unguents for the prophylaxis of herpetic infection.

RUBELLA

Rubella is an acute infectious disease characterized by eruptions similar with ones of varicella, insignificant general disturbances and increase of lymphatic ganglions in the occipital regions, parotid and posterior cervical region. The disease has a seasonal character (usually occurs during the wintertime). Epidemics are possible.

The disease affects children aged between 1 and 7 years. The source of infection is the sick person, contagious not only during the period of clinical manifestations but also during the incubation and recovery periods. Contamination occurs via the respiratory ways. The virus penetrates into the organism through the mucous tissues of upper respiratory organs, initially replicates in the lymphatic ganglions and thereafter penetrates into the blood. Eruptions appear in two weeks. One week later after the

appearance of eruptions the virus disappears from blood due to the formation of neutralizing antibodies.

Symptomatology: The incubation period lasts for 15-24 days. Eruptions are the first sign of the disease. The patient's general state of health is slightly disturbed, sub-fever temperatures, headaches, weakness may be attested. Catarrhal manifestations at the level of upper respiratory ways take the form of cough, conjunctivitis appearing simultaneously with eruptions. In buccal cavity: hyperemia and laxity of amygdales, enanthema on the buccal cavity. These are some pink small maculae with size of needle eye, appearing on the mucous tissues of soft palate and cheeks. The Koplick-Filatov-Belski sign is not detected.

The typical symptom is the growth of lymphatic ganglions, especially of the occipital and posterior cervical ones. The lymphatic ganglions grow in size before eruptions and may remain so for some time after their disappearance.

Treatment: Symptomatic treatment with observation of hygiene of buccal cavity is required.

INFECTIOUS MONONUCLEOSIS (THE FILATOV-PFEIFFER DISEASE)

It is an acute infectious disease characterized by fever, increase of all groups of lymphatic ganglions, affection of oro-pharynges, hepatolienal syndrome and presence of typical mononuclears in blood.

The pathogenic agent is the Epstein-Barr virus that leads no to cytolysis but to the division of affected cells – B-lymphocytes. The virus can persist in them for a long time. The disease is most often exacerbated in autumn and spring. People of any ages may be affected; however most often children and adolescents are affected. Sources of infection may be ill persons and carriers of the virus. Contamination takes place via respiratory ways and by direct contact. The virus penetrates into the mucous tissues of oro-pharynges and upper respiratory ways.

Symptomatology: The incubation period last for 5-21 days, sometimes even 1-2 months. The disease breaks out suddenly, body temperature may rise up to 39-40^{0}C. The characteristic signs of infection appear by the end of first week of the disease. Patients have complaints on pains in throat, weakness, retching. The most characteristic symptom is the increase of cervical lymphatic ganglions. At palpation they are elastic, slightly painful, not accreted between. Sometimes around the increased lymphatic ganglions fat tissues may be edematous. Lymphatic ganglions are not suppurated. Another important symptom is polyadenitis.

In the buccal cavity: the mucous tissues are hyperemic, increase and edema of palatal amygdales and uvula is noticed. Nasal respiration is complicated due to the hypertrophy of nasal-pharyngeal amygdale. Yellow-grayish patch appears on the amygdales, this patch may be detected in the very first days of the diseases, or on the third or fourth day, simultaneously is registered the increase of body temperature and worsening of general state. Liver and spleen grow in size as well. Blood analysis reveals appearance of leukocytosis, increase of VSE. A determining sign is the presence of typical mononuclears.

Local treatment includes antiseptic treatment of buccal cavity, antiviral treatment, keratoplastic.

VIRAL VERRUCOSIS ERUPTIONS (PAPILLOMAE) OF BUCCAL MUCOUS CAVITY

The disease is provoked by the virus of human papilloma. Usually, sources of infection are people and animals. Traumas, fissures of the buccal mucous tissues, vicious habit to suck fingers, bite the lips, etc. are contributing factors for the breakup of the disease. The incubation period may last from several days to two months.

Symptomatology: On the mucous tissues of lips and cheeks

appear the papillomatous concrescences, small and multiple, "cauliflower" –shaped. Small concrescences may have small stems. The color of verruca is pale pink, in some cases they may be of dark color. As a rule, children do not have accuses on pain in buccal cavity.

Treatment: Local treatment includes application of antiviral unguents. Depending on the localization of verrucosis, their number, deepness of the process and duration of evolution antiviral and surgical treatment are recommended (cryocautery). Antiviral therapy must be followed by adequate surgical intervention. Assanation of buccal cavity with observation of hygiene is compulsory.

AIDS

AIDS is an infectious contagious disease with grave evolution and lethal result. The disease is provoked by the human immune deficiency T-lymphotropic virus (HIV). The virus affects the T4 lymphocytes, destroys the cellular immune system and in result of this the organism looses resistance to the conventionally pathogenic flora, becomes vulnerable to the non-specific infection and tumor processes. Interaction of T-helpers (T4 lymphocytes) with specific antigens leads to the stimulation of division and destruction of cells. Upon penetration into the cells the viruses irreversibly alter the as they use the cellular genetic materials for their own replication. The normal ratio of T-helpers to T-killers is 2:1, but in case of AIDS the situation becomes exactly the opposite. The mechanism of cellular immune inhibition includes the cytospastic action of the virus, the toxic action of its components, the cytotoxic action of the macroorganism on the own T-lymphocytes with antigenic properties. The virus leads to the reduction of cellular immune mediators (interleukins), macrophages. In this way appears leukocytosis that afterwards develops into leucopenia, lymphopenia, thrombocytopenia or anemia, the quantity of IgG rises considerably, etc. In some cases

the virus affects monocytes, macrophages, histiocytes, (of derma, mucous tissues, lymphatic ganglions, spleen, liver, etc.).

The virus is transmitted from ill persons or from carriers. Presently the number of infected persons is growing in geometrical progression. The risky groups include: homosexuals, drug-addicted persons, persons ill of hemophilia, prostitutes. Transmission ways include: sexual contact, parenteral way (with blood during injections, transfusions, etc., in donor-provided organs) and intrauterine way (from mother to child). Also has been demonstrated the possibility of infecting a child via the mother's milk. The possibility of transmission via saliva, air, through common use objects (including toys), stings of insects has not been proved. Evolution of the disease depends to a considerable extent on the initial health status of the macroorganism.

When treating diverse dental and periodontal affections, performing surgical interventions, treating the affections of mucous tissues the dentists to a certain degree are exposed to the risk of infection. The probability of getting infected for a stomatologist is 5-10 times higher compared to the other groups of population, as they are in contact with the patient's infected material (blood, saliva). Practical experience shows that in fact the risk of infection for stomatologists is very low (0.05%), however, it doesn't reduce the danger. In their turn stomatologists may be sources of HIV infection for their patients if they are carriers of HIV (through the epithelial lesions of hands, through the saliva and blood of patients). Use of non-sterilized tools after having treated an infected patient can result in the infection of a healthy child when performing stomatological treatment (from infected patient or carrier to another patient). Several cases were attested when children got infected in medical institutions due to the breach of aseptic requirements therein (via intravenous infusions, etc.).

AIDS at children evolves much faster compared to the adults. Most children with signs of the disease are aged less than two years, 79% of ill children are aged less than 5 years. The boys-to-girls ratio is 1,2:1.

According to the data of WHO, manifestations in buccal cavity are polymorphic and include: mycotic, bacterial, viral affections, tumors, affections of unknown etiology (recurrent ulcerations decubital and trophic ulcers, xerostomia, lymphadenopathies, hyper pigmentation), etc.

In order to diagnose the affections of oral mucous tissues at patients with AIDS the doctors of London in September 1992 have proposed a working classification, according to which the affections are divided into 3 groups:

Group 1 - affections strongly related to the HIV infection:

- Candidosis (erythematosis, pseudo membranous, hyper plastic);
- Hairy leukoplakia;
- Marginal gingivitis;
- Ulcerous-necrotic gingivitis;
- Destructive periodontitis;
- Kaposi sarcoma;
- Hodgkin lymphoma.

Group 2 – affections poorly related to the HIV infection:

- Bacterial infections;
- Affections of salivary glands;
- Viral affections;
- Thrombocytopenic purple;

Group 3 – affections that may be registered in the course of HIV infection but are not anyway related to it.

The buccal mucous tissues are usually affected after the appearance of the main signs of disease: loss of body weight, loss of appetite, adynamia, increase of lymphatic ganglions, etc. At infected children on the buccal mucous tissues acute medium and

severe candidomycosis may be found. Especially at adults, besides acute candidomycosis, atrophic or hyperplasic form of chronic candidomycosis, mycotic cheilitis, and hystoplasmosis of buccal mucous tissues may be identified. According to the specialized literature, incidence of candidosis of buccal mucous tissues varies from 31% to 94%. Usually, the disease progresses but the antimycotic therapy is of little effect.

There exist descriptions of cases when the buccal mucous tissue is affected by viral infection and such affections are manifested as first symptoms of AIDS: acute herpetic stomatitis, herpes Zoster, varicella, viral verrucosis, etc.

Bacterial infection can take the form of gingivitis and ulcerated –necrotic stomatitis with characteristic symptoms: necrosis of gingival papillae, etc., with progressive and long-lasting evolution. Often recurrent apical periodontitis is observed and marginal periodontitis provoked by endogenous microflora.

The idiopathic thrombocytopenic purple is characterized by the appearance of stains and hemorrhages on skin and on the buccal mucous tissues. The buccal mucous tissues and the genital organs may have limited centers of leucoplasia as consequences of keratinization processes.

Manifestations in the buccal cavity may be very various and may also include symptoms characteristic for dermatosis, somatic, allergic and other diseases.

Signs of alarm for a dentist to raise suspicions on AIDS at children include: different forms of candidomycosis, bacterial infections and viral affections of oral cavity, aggressive forms of periodontitis (HIV-periodontitis), chronic recurrent aphthous stomatitis, and idiopathic thrombocytopenic purple, affections of salivary glands associated with xerostomia, exfoliative cheilitis, desquamative glossitis, hairy leukoplakia, Kaposi sarcoma, and epidermoid carcinoma.

The clinical image of AIDS at children differs essentially

from the clinical image observed at adults: primary affections of central nervous system with clear neural-psychic signs are frequent, simultaneously infection with hepatitis B, sarcoma Kaposi, lymphomas are observed less often. Frequent áre the cases with hypergammaglobulinemia, lymphopenia is rarely diagnosed; normally the Th/Ts ratio is unchanged; interstitial lymphoid pneumonia, bacterial sepsis - as one of the causes of death, etc.

Plasmocytary pneumonia is one of the most frequent pathologies found at patients, especially children, being diagnosed in 61 % of cases.

AIDS AT CHILDREN DURING THE PERIOD OF FIRST CHILDHOOD

The first signs of AIDS are candidosis or the herpes of buccal mucous tissues and skin, pneumonia, enteritis, loss of body weight, fever, increased of lymphatic ganglions. Maculated and papular eruptions and thrombocytopenic hemorrhages may be identified. Plasmocytary pneumonia, as a rule, is detected during the first months of the child's life. Lethal results occur in short periods of time.

The clinical signs characteristic for the congenital HIV infection are: loss of body weight, hepatosplenomegaly, thrombocytopenia, anemia, stops in growth and development, icterus, flattening of nose, strabismus, blue sclera, often – signs of fever, enanthema, diarrhea, lymphadenopathy, etc.

Children are much more vulnerable to HIV, as their immune system is on the stage of formation. Duration of latent period is shorter compared to adults, especially at children aged under 1 year. Diagnosing during the first year of life is difficult, as the provenience of antibodies is unclear - either they provided from mother or produced by the child's organism. The clinical and immunological signs of the disease may be clarified only after the age of one year.

Infected children first of all suffer from affections of the central nervous system. One of the major consequences of infection is the delays in development of organism. Immunological particularities of AIDS at children include high levels of immune globulins and incapacity to produce antibodies at the introduction of antigens.

Diagnosing:

- determination of virus-specific antibodies;
- immune-ferment analysis;
- immune blotting or immune fluorescence;
- determination of virions in the lymphatic ganglions.

Differentiated diagnosing: At this age AIDS at children must be differentiated from primary immune deficits, or immune deficiency states caused by the prolonged administration of corticosteroids or chemical therapy. At children of greater age differentiated diagnosing is performed with infectious mononucleosis and blood diseases.

Treatment: For the moment specific treatment methods are not elaborated. As antiviral preparations one may use azidothymidine, interferon, virazol, etc. In order to recover the immunity are administered thymisine, tymalin, levomyzol, etc. Secondary infection may be neutralized with antibacterial preparations. Locally – symptomatic treatment. In cases of mycotic stomatitis amphoterycin B, etc. is administered. Also of considerable importance are the removal of dental deposits and observation of dental hygiene.

Prophylaxis: Prophylaxis comprises a series of complex measures. Dentists must strictly observe the following requirements:

- thorough consideration of the patient's anamnesis;
- use of gloves (before proceeding with the next patient the dentist must wash the hands and change the gloves);

- use of protective mask and glasses;
- the working cloths must be changed daily or immediately if stained with blood, washed in hot water with detergents and disinfecting agents;
- all tools, mirrors, etc. must be thoroughly disinfected;
- when working with sharp tools special care must be taken in order to prevent skin lesions. In cases of skin lesions the dentist may not continue the reception of patients;
- use of single-use syringes and needles;
- compulsory sterilization of surgical cutting tools after each patient;
- periodic disinfection of stomatological equipment (sodium hypochlorite 1:10, lysoformine 1%, etc.);
- the prints, print-spoons, etc. must be cleaned from blood, saliva and disinfected before transmission to laboratory;
- reduction of contact with buccal liquid to minimum by use of cofferdams, aspirators, etc.

SCARLATINA
(Scarlet fever)

Scarlatine is an acute infectious disease transmitted through the air. It is characterized by fever, general intoxication, and angina and dotted eruptions. Most often are affected the children aged 2 to 7 years during the autumn and wintertime.

Etio-pathogenesis: The disease is caused by the toxygenic Beta-hemolytic streptococcus of group 4 that affects the nasal-pharyngeal mucous tunic; in rare cases it affects the skin, provoking local inflammatory processes (angina, regional lymphadenitis). The exotoxin produced by streptococcus provokes general intoxication. Further in the pathological process a major role is played by allergic mechanisms that participate in the pathogenesis of hypertoxic scarlatine and appearance of complications.

Simptomatology: The incubation period lasts for 2-7 days. The disease begins suddenly with fever, headaches, general disturbances, pains at deglutition, vomiting, etc. A typical and permanent sign is the angina characterized by pronounced hyperemia of soft palate, increase of amygdales. Amygdales and their lacunae may have patch. Angina may be catarrhal, lacunar or necrotic. Simultaneously with increase of body temperature and development of intoxication, on the hyperemic sectors appears the dotted enanthema that confers an irregular aspect to the buccal mucous tissues. Eruptions spread on the jugal and gingival mucous tissues. Then, during the first 2-4 days on the body skin small dotted eruptions of pink or bright red color begin to appear. Regional lymphatic ganglions are increased and painful during the first several days of the disease. Eruptions stay for 2-5 days, then they become dimmer and dimmer and the general state of organism begins to improve gradually.

At the debut of the disease the tongue is covered with grayish patch difficult to detach (in severe cases the patch may be brown). During 2-3 days the tongue gradually gets cleaned, beginning with the top and ending with dorsal surfaces, such cleaning being conditioned by the desquamation of epithelium. On the surfaces free of patch the mucous tissue of tongue becomes bright red (raspberry-colored), fungi form papillae are edematous and bigger in size than usually. In 4-5 days the tongue gets completely cleaned from patch and obtains a very characteristic aspect: its surface is smooth, "polished" and painful when the patient takes meals (see the color insertion, figure 10.7).

At the beginning of disease the lips are edematous, become raspberry- or cherry- colored. In some cases on the 4th or 5th day lip erosion can occur.

Differentiated diagnosing: with diphtheria, measles, angina, blood diseases.

Treatment is organized at domicile; the child must be isolated

to prevent contacts with healthy children. In severe cases the child must be hospitalized. Confinement to bed for 5-6 days is strongly recommended. Antibiotics are prescribed in doses adequate to age. In severe cases: in toxic form - disintoxication treatment, in septic form - intensive treatment with antibiotics. In the absence of complications the child is released from hospital after 10 days from the appearance of disease. The child may be admitted to organized collectives only after 23 days of quarantine. Children that have not previously had scarlatina but were in contact with a sick child, may be admitted to organized collectives after 7-days isolation at domicile. Regular disinfection at domicile must be performed.

DIPHTHERIA

Diphtheria is an acute infectious disease transmitted by air and characterized by toxic affection of cardiovascular and nervous system, specific inflammatory process with formation of fibrinogenous patch.

Etio-pathogenesis: The pathogenic agent of the disease is the diphtheria bacillus Leffler (Corynebacterium diptherie) with high resistance in the external environment. The pathogenic action is provoked by specific exotoxins. Corynebacteriae vegetate on the mucous tissues of buccal cavity and other organs where further appear a diphtheric inflammatory process with formation of films. Exotoxins generated by the pathogenic agent are absorbed into the blood and cause the general intoxication of organism, affection of myocardium, peripheral and vegetative nervous system, kidneys and suprarenal glands.

Clinical evolution: The incubation period can last for 2-10 days. Most often are infected the children aged between 1 and 5 years.

Often affected are the mucous tissues of pharynges, larynges, while the mucous tissues of nasal and buccal cavities are affected rarely.

The diseases occur rapidly and have a progressive character. The body temperature rises up to 38-39^{0}C, general state disturbances begin to occur. The affected child has accuses on pain in throat. During examination hyperemia and edemas of mucous tissues of amygdales, palatal tissues and uvula are determined. At the beginning of disease the patch is present in the form of a thin net, in several days it develops into a smooth or undulated surface with clear contours, looking like covered with adjacent mucous tissue. The films are of white or gray color and can not be detached from the mucous tissues. They appear in result of fibrinogenous inflammatory process, as consequence of local reaction of tissues on the action of germs and toxins. The films are composed of fibrin, desquamated epithelium, leukocytes, in some cases the provoking germ can also be identified in the film. With the progress of disease the patch advances onto new sectors, becoming thicker and getting a yellowish-gray or grayish color. Bleeding turns the color of patch into rusty. After removal the films reappear again soon. They are localized on the gingival margins, on tongue and in other areas. Buccal cavity emanates a specific sweetish smell of putrefaction.

Another characteristic sign of this disease is regional lymphadenitis with considerable edema of soft tissues that can afterwards spread onto the throat down to the region of clavicle. The clinical image of the disease at previously vaccinated children is different: diphtheria evolves in the form of catarrhal or lacunar angina, films are laxer, without tendency to spread, are easily detachable from the mucous tissues, etc.

Differentiated diagnosing is done with angina (follicular, lacunar, phlegmonous), with scarlatina (necrotic angina), with infectious mononucleosis, with sanguine diseases (with necrotic processes in buccal cavity), etc.

Treatment: The child must be isolated immediately and placed in the section for infectious diseases in hospital. Regime to

bed is obligatory. The basic method of treatment consists in the intramuscular administration of anti-diphtheria serum in doses adequate to the degree of intoxication and time of addressing for medical assistance. To prevent complications, intra-dermal test with serum 1:100 is recommended. In severe cases disintoxication therapy is indicated. In cases of necessity symptomatic treatment is prescribed. Antibiotics are administered in order to prevent pneumonia. Should the risk of stenosis of larynges appear - glucocorticoids are administered, in severe cases intubation or tracheotomy must be performed.

Prophylaxis: Active immunization of children with diphtheric serum and anatoxin. To children were in contact with ill antibiotics are recommended during 7 days (tetracycline, erythromycin, etc.).

MYCOTIC AFFECTIONS

In most cases mycosis at children is provoked by the fungi of Candida family (monilia, oidium, and endomyces). The fungi of Candida family are widely spread in the surrounding environment and permanently vegetate as saprophytes in the buccal cavity and on skin. According to

E. Мороз (1971), 97% of children less than 3 years are carriers of Candida. In the oral cavity at children are usually found: Candida albicans (68,5%), Candida pseudotropicalis (15,12%), Candida Krusei (10,46%), Candida tropicalis (6,98 %) (Курякина Н. В., 2001).

Affection ways include: contact with the person ill of candidosis or transformation of conditionally pathogenic fungi into pathogenic ones. In certain circumstances the fungi from latent states may become pathogenic: different general diseases (diseases of digestive tract, hypovitaminosis, endocrine diseases, etc.), i.e. when the general resistance of organism decreases, administration of antibiotics, corticosteroids, cytostatics, failure to observe the hygiene of buccal cavity, inflammatory processes of

mucous tissues and gums, carious teeth, orthodontic devices, etc.

The major pathogenic factor is the reduction of organism's resistance (both general and local). At newborns (especially at prematurely born children) and suckers (especially ones fed artificially) due to the imperfect development of local immunity this pathology is met often enough. Infection usually occurs during the delivery from mothers suffering from candidomycosis of genital organs, during contact with the infected hands of the medical personnel and relatives, via the common use objects, etc. With age the resistance of children to candidae increases: if at suckers and newborns predominate the acute forms, then at children aged above 3 years the chronic forms are more often detected.

We distinguish three forms of candidosis: 1) superficial cutaneous, of mucous tissues and nails form of candidosis; 2) chronic generalized (granulomatous) forms at children; 3) visceral (systemic ones). All these forms may cause affections of the buccal mucous tissues.

By evolution at children the acute and chronic forms are distinguished. By localization: cheilitis, glossitis, stomatitis, etc. Depending on clinical symptoms: superficial or profound, localized or diffused.

Acute candidosis

(thrush, acute pseudomembraneous stomatitis, etc.)

Most often found at suckers and rarely at children of greater age – at children weakened by chronic general diseases, sugar diabetes, hypovitaminosis, etc., children treated with antibiotics, etc.

Simptomatology: Characterized by hyperemia of buccal mucous, appearance of white or yellowish patches. The affection may evolve into the light, medium or severe form.

Light form: The buccal mucous tissues display limited hyperemic sectors (on tongue, on lips, on cheeks), white dotted

patch. Further the dots may grow in size and join, forming films looking like condensed milk. This kind of patch is easily detachable, after detachment a hyperemic surface is opened.

Medium form: The medium form is characterized by the appearance of patch in the form of films on some areas of tongue, lips, cheeks, etc. The patch is not always easily detachable, after detachment an erosive surface is opened, sometimes with hemorrhage (see the color insertion, figure 10.8).

Severe form: The severe form is characterized by diffuse affection of buccal mucous cavity. The most pronounced patch of grayish color and infiltration of tissues is localized on all the surfaces of tongue, lips, cheeks, et. Often angular cheilitis and sub-mandibular lymphadenitis are observed. The patients have accuses on dry mouth, burning in buccal cavity, foamy saliva. In certain cases affections of respiratory ways, skin, urinary bladder, and genital organs are determined. Unless treated adequately, this form can turn into chronic and provoke candidomycotic sepsis.

Chronic candidosis

There exist two forms of chronic candidosis: hyperplasic and atrophic. Chronic candidosis often appears at children suffering from other chronic diseases without any accuses on buccal cavity. Sometimes pains at mastication appear, children prefer semi-liquid food, avoid speaking. Sometimes they consume greater quantities of liquids due to drying in buccal cavity.

Simptomatology: Most often found on tongue or in the angular region of lips. On the mucous tissues this affection is manifested by dense patch of yellow or light-brown color, well seated on the tissues of tongue, cheeks, etc. The patch projects out of hyperemic and edematous mucous tissues. The patch may be painful at palpation. Detachment of patch results in pain and hemorrhage.

If the fungi penetrate deep into the tissues, consistent

granuloma appears. Sub-mandibular lymphatic ganglions are increased, mobile and consistent.

Diagnostics: Laboratory methods: microscopy, cultivation of fungi in dynamics.

Differential diagnosing: with allergic catarrhal stomatitis; with leucoplasia, with physical and chemical lesions of buccal mucous tissues.

Treatment: A spectrum of complex measures with anti-fungal orientation, removal of pathogenic factors, treatment of general diseases, mobilization of general reactivity of organism, improvement of buccal health and amygdales, rational nutrition, etc.

In cases of *light forms* treatment is local. Treatment implies use of solutions changing the acid environment in the buccal cavity into alkaline: 1-3 % sol. sodium bicarbonate. Iodine preparations are administered: 1 % sol. Iodinol, sol. Lugoli in glycerin, Kastellani sol., 1% clotrymasol (canesten), etc.

In cases of *medium forms* additionally antimycotic preparations are prescribed: nystatin 100 000 U/g, 5 % levorine, 5% decamine, amphoterycin 30 000 U/g, 15 clotrymasol, 1-5% mycoheptin, myconasol gel 2%, etc.

In cases of *chronic forms* to the children of greater age, especially to the ones that for long time use orthodontic devices, Decamine drops (0,15mg) are prescribed – 1 drop per each 3-4 hours. Simultaneously, alternated use of several preparations is recommended.

In *severe cases*, besides local treatment, peroral antimycotic preparations are prescribed in doses adequate to the age and state of the patient: Nystatini 500 000 U 5-6 times a day, Amphotericini 200 000U two times a day, Mycoheptini – 250 000 U two times a day, Ketoconasol, Nizoral in tablets 200 mg for 10 days two times a day, further - 1 tablet a day, Fluconasol – 50 mg two times a day. In order to amplify the antimycotic action one

must avoid the administration of the same medication locally and per oral. Tablets are better to be sucked or crushed with the teeth (to suckers they are administered in the form of suspension with the mother's milk), i.e. they must remain in the oral cavity for as long time as possible. The treatment with antimycotic preparation shall last for 7-10 more days after the disappearance of clinical symptoms.

In cases of chronic candidosis general sensibilization of organism occurs, so hyposensitization preparations are indicated. Polyvitamins are administrated (vitamins of groups B, C).

Generalized and visceral forms of candidomycosis are treated in conditions of hospital.

The patient's ration must be rich in vitamins and microelements (dairy products, fruits, vegetables). Consumption of products with high content of carbohydrates (sweets, pastry, potatoes, etc.) must be limited.

Prophylaxis: Must begin already in the prenatal period, i.e. pregnant women must timely treat mycosis of genital organs, etc. In order to prevent candidosis, to all newborns on the second-fifth day of life suspension of Nystatin is administered per oral. Observation of oral hygiene is necessary. In case of treatment with antibiotics and glucocorticoids antimycotic preparations must be administered. Observation of sanitary-hygienic regime in maternity sections, hospitals, kindergartens is obligatory.

ORAL MANIFESTATION OF SPECIFIC INFECTIONS

Tuberculosis

It is a chronic infectious disease provoked by *Mycobacterium tuberculosis* (bacillus *Koch*). The main source of infection is ill human or bovine. Contamination is on respiratory way, with sputa; in majority events is produce in childhood, frequently less of disease provocation. Therefore in organism is formed allergic tuberculin state, production concomitantly a certain level of immunity. This state of primary tuberculin infection is tracing

with specific reactions of organism to tuberculin introduce intradermal (*Mantoux*) and epidermal (*Pirquet*).

At level of primary localization of mycobacterium is developing primary tuberculoses nidus, then primary complex, which is composed of: primary nidus, lymphangeitis and lymphadenitis. Encapsulation of primary nidus and its petrifaction are appreciated for a favorable evolution of a tuberculoses process. In these nidus bacilli can to persist a long time less manifestation, and when the immunity of organism decrease (infections, intoxications etc.) the process can to reevaluate. Next development of tuberculosis is determinate of immune and biological factors. Clinic-morphological manifestations of tuberculosis have three variants: primary tuberculosis (at the first contact with bacilli), disseminate tuberculosis (develop over one considerable period after primary infection, supported with residual modifications of nidus) and secondary tuberculosis (affection in organism that supported primary infection and to developed habitually in lungs).

At children affection of oral mucosa is secondary to pulmonary, lymphatic and bones tuberculosis, and contamination is upon lymphatic and sanguine ways from primary nidus. On the oral mucosa the secondary tuberculosis infection is manifested for lupus tuberculosis (vulgar) and miliary ulcerous tuberculosis.

Lupus tuberculosis is a form of oral cavity tuberculosis that appear at persons with high reactivity to pathogen agent. At 75% of ill children are affected the skin of face, the red margin of lips (especially the superior lip).

In the first 2-3 years of life at children is determined the primary ulcer, localized frequent on the tongue or gum. It presents an infiltrate with smaller dimensions, less signs of inflammation, on its surface is formed an ulcer with granular tissue. Regional lymphadenitis is present.

Primary element is lipoma – a red-yellowish nodule of 1-3 mm in diameter, at palpation – soft and painless. At pressing of glass lamellas on lipoma the red color despair and are observed a yellow nodule ("honey-jelly" symptom). Buttoned explorer break down in lipoma (Pospelov phenomenon), this is explicated of elastic and collagen fibers in nodule.

Four forms of lupus tuberculosis of oral mucosa at children are distinguished:

1. Oval ulcer with cu lucid margins and high quantities of fresh hemorrhagic granulations. For gingival affection is characteristic the alveolar bone resorption with mobility and loss of teeth. This form presents a risk of malignity.
2. Recurrences of ulcerations in region of cicatrices where appear fresh nodules. To labial affection are formed hemorrhagic and purulent crusts, after their removing is determined ulcerous surfaces with irregular margins covered by yellow deposits. Lips are enlarged in volume and can to have profound pain fissures.
3. Papillomatsis proliferation with diffuse tuberculosis infiltration. Process is localized to limit of hard and soft palate. Nidus has a hard, nodular consistence with fissures. At destruction of nodules are produced profound ulcers.
4. Smooth cicatrices surface with solitary nodules. Cicatrisation of ulceration on red margin of lips can to provoke a stenosis of oral cavity. Can to appear recurrences of lipoma on cicatrices.

For all forms is characteristic regional lymphadenitis. Lymphatic ganglions are enlarged, adherent and pain to palpation.

Miliary ulcerous tuberculosis is characterized of appearance, in special, on dorsal surface of tongue, inferior lip and retro-molar region of smaller ulcer that subsequently extended to periphery. Ulcer is painful, round or oval form with irregular contour, at palpation its surface is smooth, bleeding, covered of granular tissue and grey-yellowish deposits.

Diagnostic: *Mantoux* reaction is positive.

Treatment: general therapy is realized in specialized hospital.

Local treatment includes assanation of oral cavity, elimination of dental plaque; exclusion of oral mucosa traumatisms, regarding to oral hygiene, appliance of anesthetics, antiseptics, keratoplastics on the affected mucosa.

Syphilis

It is a chronic infectious disease provoked by *Treponema pallidum*. At children frequently is attested the congenital syphilis –transmitted of fetus through placenta from ill mother. Obtained syphilis of children is rarely. Congenital syphilis of children less 6 years is delimited: precocious congenital syphilis of suckers and of preschool period, tardy syphilis and latent syphilis.

At congenital syphilis on the skin and oral mucosa are determined typical papules, analogue with secondary obtained syphilis. Papules are localized on mucosa of soft palate, palatine pillars, tonsils, cheeks, lips, tongue and gum. Papules are round or oval form, red-cyanotic color; size varied 5mm - 2 cm. In case of localization on the tongue is present atrophy of papillae in region of papules. At children rarely is observed the erosion or ulceration of papules surfaces.

For congenital syphilis are characteristic diffuse infiltrates of lips red margin, skin and labial mucosa. In region of infiltrate, in special, in angular region, is traced profound fissures, after their epithelization remain cicatrices.

Tardy congenital syphilis is traced of children after 5 years of life. In this period appear grave pathological processes, which make profound lesions of organs tissues of organism.

Diagnostic: necessity laboratory confirmation. At children with tardy congenital syphilis classic serologic reactions are positive.

Treatment is made in specialized dispensary.

Local treatment is symptomatic assanation of oral cavity,

elimination of dental plaque; exclusion of oral mucosa traumatisms, regarding to oral hygiene, appliance of anesthetics, antiseptics, keratoplastics on the affected mucosa.

Gonorrhea

At children is traced rarely. New-born is infected in time of birth from mother or other ill persons. Usually are affected concomitantly oral and nasal mucosa, and conjunctiva. Clinical evolution of a gonococcic stomatitis is asymptomatic. The first signs of gonococcic stomatitis are hyperemia, edema of mucosa, superficial erosions and ulcerations. In grave cases, in absence of treatment the process progress with appearance of multiple erosions and ulcerations on jugal, lingual and gingival mucosa. Ulcerations are superficially, with smaller size, painful, of yellow-grayish secretion in which are traced gonococci. Regional lymphadenitis is present.

Diagnostic: in base of bacteriologic examination of eliminations are established more quantities of gonococci.

Treatment is made in specialized dispensary with administration of antibiotics and sulfanilamide.

Local treatment: application with anesthetics, antiseptics and keratoplastics, elimination of dental plaque; exclusion of oral mucosa traumatisms, regarding to oral hygiene.

Prophylaxis: oral cavity of new-born from ill mother is painted with 2% silver nitrate solution.

Vincent ulcero-necrotic gingivostomatitis

This disease is provoked by saprophyte of oral cavity that in certain conditions become pathogenic. Frequently are affected adolescents and younger, but the small children are affected rarely. Affection appear in autumn and winter seasons, organism resistance reduction caused by general diseases, insufficient alimentation, imperfect oral hygiene, as to complication of teeth eruption, after teeth extraction, etc. This pathology is associated

with hipovitaminosis, hematological deceases, etc. In pathogenesis of Vincent ulcero-necrotic gingivostomatitis is importance locale immune reactions of Arthus phenomena type to microbial allergen action that provokes of oral mucosa necrosis associated with fusospirillosis.

Vincent ulcero-necrotic gingivostomatitis is: acute and chronic forms, and light, medium and severe degree of process gravity.

Disease develops typically for one infectious disease. The general state of children is severe and it is characterized by marked intoxication, fever, asthenia, enlarged lymphatic ganglions, and hyper-salivation.

Ulcero-necrotic gingivostomatitis starts at level of gingival papilla and expand to gingival margin. The process progresses quickly and affects other sectors of oral mucosa. Mucosa is edematous and lax, with hyperemia. On lips, cheek, soft palate and tongue mucosa appear ulcerations with yellow-grayish deposits. Children accuse to insupportable pains in oral cavity that are intensified in time of alimentation or speaking. Out of oral cavity is eliminated hemorrhagic saliva and fetid smell. Sometime gingivostomatitis is complicated with angina (Plaut-Vincent angina). In grave cases ulcerations on the mucosa are profound, and on the gum can provoke necrosis and alveolar bone sequestration. The disease evaluates 9-14 days and ends with epithelization of ulcerations and sometime with cicatrisation.

Differential diagnostic: with oral mucosa pathologies in course of hematological diseases, with allergic ulcero-necrotic stomatitis.

Treatment of Vincent ulcero-necrotic gingivostomatitis at children must to be complexly in function of disease severity and children age. Child is rationally to hospitalize in oro-maxillo - facial section.

General treatment:

1. antimicrobic and antitrichomonadic (metronidazol of 0,25 g 2 time/day, 5-10 days; oxacilină of 0,25-0,5 g 4-5 time/day; rondomicină of 0,15-0,3 g 2-4 time/day);
2. antihistamine (dimedrol, clemastin, cloropiramină etc.);
3. vitamins (C, A, B, P);
4. analgesics and antipyretics;
5. in severe cases – disintoxication therapy;
6. caloric diet, liquids, juices etc.

Local treatment – paint oral cavity with:

1. anesthetics (5-10% anesthesine in vegetal oils; solution or unguent 1-2% piromecain; solution 1% trimecain; plastocain, Anaestho Gel (Voco) etc.)
2. antiseptics (solution 0,06% clorhexidin, 0,5% etoniu, 1% dioxidin, 0,5% chloramines, oxygenate water 3%, 2% sodium bicarbonate etc.);
3. proteolytic enzymes (tripsin, chemotripsin, lizozim, terillitin etc.) – for remove of necrotic tissues;
4. anti-inflammatory and keratoplastics (vinilin, liniment aloe; aekol; unguent and gel Solcoseril; aerosol "Livian", "Vinizol", „Levovinizol", „Legrazol", solution 1% galascorbine, alcoholic solution 0,2-0,5% sanguiritrine, alcoholic solution 2% gramicidine la 200 ml distillate water, solution 0,02 nitrofural, solution stomatidin etc.

Oral rinsing with anesthetics and antiseptics with oral hygiene assessment is indicated. After removing of acute inflammatory process – assanation of oral cavity, treatment of periodontal diseases and removing of dental deposits.

Prophylaxis: regular assanation of oral cavity and oral hygiene observation in case of general diseases of organism, in special infectious diseases etc. If at acute ulcero-necrotic gingivostomatitis don't realized intense therapy, then process is transformed in chronic faze, recurs and progresses. Prophylaxis is important in reason of: frequent recurrences contribute to severe

evolution of destructive process. Children are taken at III group of dispensary control, are investigated and implemented preventive measures for 3-4 time on the year.

ORAL MANIFESTATION OF ALERGIC DISEASES OF CHILDREN

Allergic diseases of children are frequently in present and are characterized by permanent ascension of frequency and evolution severity. This is in connection with: pollution of extern environment with gazes, with production residues, with using of synthetic materials, colorants and alimentary supplements etc. Appearance of allergy at children is conditioned by: hereditary predisposition, system pathology, character of alimentation, and climacteric factories etc. Early large utilization less discernment of medicaments to children provokes increase of frequency allergic diseases at children.

Allergy is exaggerate and denatured pathologic reaction of organism to action of some factors called allergens. Nature of allergy consists in antigen-antibody reaction.

Exo-allergens and endo-allergens exist. Exo-allergens penetrate in organism through digestive and respirator tract, derma and mucosa. They may be of: 1. non-infectious origin – alimentary products, medicaments, dust of room, pollen of plants etc. 2. infectious origin - microorganisms, viruses, fungi and their metabolites. Endo-allergens are changed proteins proper of organism (auto-allergens), that may be primarily (crystalline, thyroid-globulin) and secondary – are formed in organism in case of metabolism disorders by action of various factors (ionized radiation, hypothermia, combustions, bacteria, viruses, fungi etc.). Allergens may be complete and incomplete. It provokes allergic reaction in connection with macromolecules of organism that induce antibodies production, and to formation of antigenic complex with molecules of organism, while antibodies are produced only for complexes, but not for its component.

Antibodies are molecules of globulin, specific modified in result of antigenic stimulation. Exist the following varieties of antibodies: cellular; fixed on cellules; anaphylactic (aggressive); of inhibition (blockage of allergens, don't provoke allergy); humoral and free (in blood); witness (don't participate in reaction).

Allergic reactions may be immediately, slowly and mixed. In pathogenesis of immediate allergic reaction are praised three stages (А.Д.Адо, 1978): immunological, pathochemical (biochemical) and patho-physiological (functional and structural disorders).

Immunological stages starts through contact of allergen with organism, in result organism is sensitized. Only at repeated action of allergen on the sensitized organism is produced allergen-antibody complex.

Pathochemical stage is characterized through elimination of biologic active substances, allergic mediators: histamine, serotonin, bradychinine, acetylcholine, heparin etc. This is in result of allergic modification of tissues that are more mastocytes (serous membrane, lax connective tissue etc.).

Pathophysiological stage is result of allergic mediators' action on the tissues. Spasm of bronchial and intestinal smooth muscles, sanguine serum modification, blood coagulation (hematopexis) disorder, cytolysis etc. is present.

Four ms of allergic reactions exist in dependence of evolution mechanism:

1. *Type 1 allergic response (immediate, anaphylactic, reagent, and atopic type of reaction).* This allergic reaction develops with formation of antibody-reagents, which are distributed to group of IgE and IgG_4. They settle on mastocytes and basophile leukocytes, and in result are eliminated following mediators: histamine, serotonin, and prostaglandin etc. After

contact with specific allergen the clinical symptoms of allergic reaction appear above 15-20 min.

2. *Type II allergic response (citotoxic).* Antibodies (capably to active the complement) are produced by tissues cells and are presented of IgG and IgM. Antibodies settle with modified cells of organism and induce complement activity, which provoke cells lesions and phagocytosis. Allergic drug reaction has citotoxic evolution.

3. *Type III allergic response (tissues lesion by immune complexes - Artius reaction, immune-complex)* is provoked by immune circular complexes formation (IgG and IgM). Antibodies settle with antigen and precipitates. This reaction is decisive in serum disease, allergic alveolites, alimentary and drug induced allergies, some auto-allergic diseases (rheumatoid arthritis, lupus erythematosis systemic etc.).

4. *Type IV allergic response or slow allergic reaction (hyper-sensitivity of slow type, cellular hyper-sensitivity)* has by antigens sensitized T lymphocytes, which have on their membranes receptors for antigens. At interaction of lymphocyte with allergen the cellular mediators (lymphochines) are eliminate. They provoke agglomeration of macrophages and other lymphocytes and in result appear inflammation. Slow allergic reaction develops in sensitized organism above 24-48 hours after contact with allergen. Clinically symptoms are hyperergic inflammation, mononuclear infiltrates around small sanguine vessels, allergic inflammation.

Anaphylactic shock

It is the most severe allergic state. Anaphylactic shock appears more frequent after parenteral administration of medicaments, in special intravenous of antibiotics (penicillin, streptomycin etc.), vitamins (thiamine, ciano-cobalamina), anesthetics (Novocain, Procaine, lidocain etc.); preparations of iodide, sulfanilamides, acetylsalicylic acid, etc. at per oral

administration, and sometimes the filling materials (in special endodontic).

Symptoms of anaphylactic shock are followings:

1. Hemodynamic shock is characterized by acute cardiac insufficiency: thin thready pulse, hyperemia of skin alternant with pale color, intense transpiration, decrease of arterial tension, etc.

2. Cerebral shock- agitations, convulsions, are present signs of cerebral edema (headache, epileptic crises, aphasia etc.).

3. Asphyxia – bronchospasm, laryngeal and pulmonary edema, etc.

4. Abdominal shock – abdominal pain, diarrhea, nausea, etc.

Distinguish tree degree of anaphylactic shock: light, medium and severe; and by evolution: flashing, recurrent, abortive.

Treatment must start immediately after appearance of first symptoms:

1. Stop administration of allergen in organism or reducer its absorption. Up to 0,3-0,5 ml 0,1% adrenaline solution in place of injection.

2. Place patient supine with legs raised, if possible.

3. For increase the arterial tension: up to 0,5 ml 0,1% adrenaline solution or 0,3-1,0 ml 0,1% mezaton solution intra-cutaneous or intramuscular.

4. Antihistamines are administrated after arterial tension normalization: intramuscular 1% dimedrol solution, 2,5% diprazin solution, suprastin etc.

5. In severe cases – intravenous hydrocortisone 50-150 ml with 5% glucose solution; prednizolon 1-2 ml to 1 kg body weight; 4-20 ml dexametazon.

6. For abolition of bronchospasm – intravenous 5-10 ml 2,4% euphilline solution in 10 ml isotonic solution of NaCl or in 10 ml 10% or 40% glucose solution.

7. In case of convulsions or supra-excitation are indicated tranquilizers: seduxen, elenium, droperidol etc.

8. Of shock provoked by penicillin: intramuscular penicillinaza 1000000 U in 2 ml isotonic solution of NaCl.

9. Symptomatic treatment depends of affected systems and functions.

Prophylaxis: Detailed anamnesis is necessary. In case of children with allergic reactions in anamnesis is recommended using of antihistaminic preparations.

Quincke edema

Angio-neurotic edema is an allergic reaction by immediate type, which appears later than alimentary, medicamental, and chemical allergens action, hypothermia etc. An important role is heredities, chronic infection, diseases of digestive system etc.

Edema appears spontaneous and develops rapid. After administration of allergen in few minutes develops on diverse sectors of face (inferior lip, eyes lids, cheek), on oral mucosa, tongue is formed an edematous sector. Derma and mucosa color isn't modified. Edematous region has elastic consistence, painless of palpation. Pharynges-laryngeal and tongue edema can provoke asphyxia – obstruction and sensation of respiratory discomfort, cyanosis of tongue and face, aphonia (see color insert Fig. 10.9.)

Quincke edema during few minutes, but in severe cases some hours or days, after that it involves or recurs periodic.

Treatmen: 1. Stop contact with allergen. 2. Antihistamine. 3. In case of pharynges-laryngeal and tongue edema - intravenous hydrocortisone 50-150 ml with 5% glucose solution; prednisolon 1-2 ml/kg/body weight; 4-20 ml dexametazon. 4. Vitamin-therapy. 5. In case of hypotension – subcutaneous 0,3-0,5 ml 0,1% adrenaline solution.

Prophylaxis of recurrences consists in: exclude contact with allergen.

Rash

It is a temporary edema of derma or connective tissue of oral

mucosa and is characterized by rapid appearance of rash and edema on the skin and mucosa, which develops later than increased capillaries permeability. The rash develops fast on diverse regions of skin and oral mucosa and can persist few hours. More frequent they are localized on lips and cheeks (see insert color Fig. 10.10.).

Treatment: specific, immunologic, pathogenic, and symptomatic therapy. Local treatment includes use of antiseptics and keratoplastics.

Drug induced allergy

Complications of pharmacotherapy in pediatric dentistry are provoked by increase of medicaments number and sensitivity of children organism. Drug induced allergy may be provoked by anything medicaments but more frequent by antibiotics (penicillin and tetracycline preparations, streptomycin), sulfanilamide, analgesics, anesthetics, iodide etc.

Pathogenesis of medicamental allergy can include all variants of allergic reactions, which are conditioned of individual reactivity of child's organism, systemic pathology, character of medicamental allergen, mode of administration, dose of medicament, etc.

Symptoms of medicamental allergy in oral cavity are diverse. In function of localization on the oral mucosa may be observed: stomatitis, cheilitis, gingivitis, and glossitis. In accordance to: 1) degree of propagation may be localized and extend; 2) inflammatory reaction: catarrhal, catarrhal -hemorrhagic, erosive, ulcero- necrotic; 3) severity: light, medium, severe and more severe.

Catarrhal and catarrhal-hemorrhagic stomatitis (cheilitis, glossitis)

They are light forms of medicamental allergy. Children

accuse pruritus; trouble of taste, painful and dryness in time of alimentation. At examination is: diffuse hyperemia, edema of mucosa, teeth-print on the tongue and cheeks, pointed hemorrhages. On the tongue is present profound desquamation of filiform papillae– „shining tongue".

Differential diagnostic: hypovitaminosis C and B, diseases of digestive system, infectious and mycotic diseases.

Treatment: general treatment: exclude administration of medicament or substitute to other one, recommend antihistaminic preparations, calcium, more quantities of liquid and non-irritant food. Local treatment: anesthetics, antiseptics, keratoplastics. Respect the oral hygiene.

Erosive stomatitis (cheilitis, glossitis)

Children accuse pain in oral cavity that is intensify during alimentation or speaking; hyperemia and edema of oral mucosa. On palate, lips, cheeks, and tongue mucosa are bullas, erosions with fibrin deposits. Erosions can confluent making large erosive surfaces. Hyperemia edema and light hemorrhage of gingival papillae are attesting. General state of child aggravates: weakness, absence of appetite, fever to 38°C, etc. Lymphatic ganglions are enlarged and painful at palpation. Evolution of disease is in dependence of pathologic process extension on oral mucosa and presence of chronic infection nidus.

Differential diagnostic: with acute herpetic stomatitis, polymorph exudative erythema, and pemphigus.

Treatment: general treatment: exclude administration of medicament or substitute to other one, recommend antihistaminic preparations, calcium, in severe cases – glyco-corticoids. Recommend more quantities of liquid and non-irritant food. Local treatment: anesthetics, antiseptics, keratoplastics, proteolytic enzymes. Respect the oral hygiene.

Ulcero-necrotic stomatitis (cheilitis, glossitis)

This form usually develops on the base of severe general allergic reaction with derma, mucosa and organs lesions. It develops acute then allergic reaction to fusospirillosis.

General state of child is severe. Children accuse headache, weakness, absence of appetite, fever, pains in oral cavity that is intensify during alimentation or speaking; hyper-salivation, fetid smell in mouth.

At examination - adynamia, teguments are pale, fever. Intra-oral – hyperemia and edema of oral mucosa, hyper-salivation, isolated nidus of necrosis yellow-grayish colored. Interdental papillae are necrotized, with gray fibrin deposits, after their removing results an erosive and hemorrhagic surface. Sub-mandibular lymphatic ganglions are enlarged and painful at palpation.

Differential diagnostic: with Vincent ulcero-necrotic stomatitis, ulcero-necrotic stomatitis of blood pathology, and trophic ulcer of cardiovascular pathology.

Treatment: general treatment: exclude administration of medicament or substitute to other one, recommend antihistaminic preparations, calcium, in severe cases – glyco-corticoids; more quantities of liquid and non-irritant food; intramuscular - 5% unitiol solution at 5 ml of 2 time per day; intravenous 30% tiosulfat of sodium solution at 5-10 ml, etc. Local treatment: anesthetics, antiseptics, keratoplastics and proteolytic enzymes. Respect the oral hygiene.

POLYMORPH EXUDATIVE ERYTHEMA

Etiology and pathogenesis: it is a poly-etiologic disease. In pathogenesis an important role plays bacterial allergy more frequent provoked by staphylococci and streptococci. In 1/3 of cases is viral etiology (*Herpes Simplex, Coxachi,* etc. virus). Exist two varieties of disease: *allergic-infectious and toxic-allergic.* It is associated with exposure to certain drugs (antibiotics,

sulfanilamide, etc.) or infecting organism (acute respirator diseases, etc.) in a susceptible individual. Until 5 years old it doesn't develop. More frequent appears or recurs in springtime.

Simptomatology: begins acute with general asthenia, myalgia, rheumatoid pains, headache, and fever to 38°-39°C. In severe forms to 1-2 days appear eruptions (maculae, vesicles and bulla with serous and hemorrhagic contain, which form ulterior erosions) on oral mucosa and skin, hyperemia and edema of mucosa. *Nikolsky* symptom is negative. Erosions may be confluent with gray-yellowish deposits. More frequent are affected anterior regions of oral mucosa: lips, cheeks, and tongue. Usually gum doesn't affect. In severe cases is affected red bordure of lips, appear large hemorrhagic crusts. Eruptions are painful; children refuse open the mouth and to eat; determines hyper-salivation. Regional lymphatic ganglions are enlarged and painful at palpation (see color insert Fig. 10.11.)

Parallel with oral mucosa lesions appear polymorph eruptions on the skin, which more frequent are localized on hands, face, and legs. In debut appears rare maculae of 1 cm to 3- 5 cm, which ulterior are transformed in bulla and breaks.

Disease evolution may be accomplished whit bacterial disease association (fuzospirochetosis).

The light form is medium 7-8 days, and severe form is 3- 4 weeks. Recurrences are usually in autumn and springtime.

Fiessinger-Rendu (1917) and Stevens-Johnson (1922) syndrome

These are severe and specific forms of polymorph exudative erythema with acute evolution and grave disorders of general state: fever, bronchopneumonia, myocarditis etc.). Except of oral mucosa and skin pathology appear conjunctivitis, rhino-rhea, and genital pathology. Children are passive, have tachycardia (more of 100), frequent and superficial respiration (see color insert Fig.10.12.)

Allergic probes are positive to antibiotics and sulfanilamide. Without an opportune treatment may be lethal cases (until 20%) in result of central nervous system affection and coma.

F. Lyell (1956) syndrome

It is more severe form characterized by rapid, progressive and acute evolution. The general state is severe- symptoms of high intoxication. On the skin and mucosa appear big maculae and bulla. After that, develop erosions on the large surfaces of mucosa. *Nikolsky* symptom may be positive (see color insert Fig. 10.13.). Also mucosa of digestive system, genital organs, respirator system and interne organs are affected. Without an intensive treatment are frequently lethal cases.

Differential diagnostic: with acute herpetic stomatitis, medicamental stomatitis, and pemphigus.

Treatment: Children are hospitalized in specialized sections where there is administered a complex treatment. In acute period in first is general treatment with: antihistaminic preparations (dimedrol, suprastin, tavegil etc.); therapy of disintoxication (intravenous: hemodesis, reopoligliuchine, isotonic solution, etc.); 30% tiosulfat of sodium solution; corticosteroids (prednisolon, triamcinolon, dexametazon); vitamins of B group and C, etc.

Local treatment is symptomatic: anesthetics (anesthesine, piromecain, etc.); antiseptics; proteolytic enzymes; keratoplastics; in grave cases - unguents with corticosteroids (0,5% hydrocortisone, ftorocort, flucinar, etc.). At toxic-allergic form exclude administration of medicament (allergen).

After epithelization of elements on oral mucosa, is recommended assanation of oral cavity and infections nidus in organism. In periods between recurrences are recommended immune-stimulators: methyluracil, pentoxil, nucleinat of sodium, etc.

Recurrences are rare and develops slowly after nonspecific hyposensitivity with histo- globulin (0,5-2 ml with interval of 3

days subcutaneous); anti-staphylococci gamma-globulin 10-25 U/day time of 3-7 days.

Dispensary control: Children with polymorph exudative erythema are permanently at dispensary control.

Behçet syndrome

It is autoimmune disease characterized by chronic recurrent evolution. Any of the above types of oral ulceration may appear in the Behçet syndrome, which affects the mouth, skin, genital mucosa, eyes, heart, blood vessels, chest, joints, and nervous system. It is a severe and dangerous multisystem condition, commoner in males, which cannot be diagnosed on the basis of the oral presentations. There are no diagnostic tests for this condition. More frequent adolescent are affected.

Simptomatology: the aphthae appear concomitant on the intact oral mucosa in number of 3-5 elements, localized on dorsal region of oral cavity. Children accuse myalgia and arthralgia, fever, intoxication, central nervous system pathology, etc.

Treatment: in conditions of hospital with collaboration of dermatologist, pediatrics, neuropathologist, etc. the severity of the condition and the nature of multisystem disease usually necessitates systemic immunosuppression with corticosteroids, azathioprine, cyclosporine A, or dapsone. Local treatment in acute period: anesthetics; dezoxiribonucleasis, interferon of 4-5 time per day, 3-4 days; 0,02% clorhexidin solution, sol. 1% etacridină lactat.

Dispensary control: In period of recurrence children is of dispensary control.

RECURRENT APHTHAE OF ORAL MUCOSA

One of more frequent pathology of oral mucosa at children is recurrent aphthae syndrome. Etiologic factors are following: viruses, misbalance of vitamins (B_1, B_{12}, and C), neuro-dystrophic

disorders, hereditary and constitutional predisposition, pathology of immune system etc.

Following symptoms are characteristic: 1) disorders of appetite (frequent children refuse some food: milk, meat, etc.); 2) dolor syndrome (periodic pain in abdominal region, sometimes simulates appendicitis); 3) motor disorders of digestive system (constipation, etc.); 4) disorders of foodstuff fermentation (doesn't increase the body weight, at coprology: incomplete fermented substances (lipids, proteins, hydro-carbonates); 5) secondary pathology on basis of dermato-mucous syndrome (colitis, enteritis, etc.).

Three clinical forms of recurrent aphthae are distinguished: light, medium and severe.

Light form: Recurrences appear one time in few years. Children have not important accuses more frequent in time of mastication. Aphthae are solitary, little painful. Observe insignificant symptom of digestive system disorders. In anamnesis: constipation, periodical pain in abdominal region; exudative diathesis in first year of life. Coprology: not important quantities of fermented muscular fibers.

Medium form: Recurrences appear yearly (1-2-3 time on year). Aphthae are little painful, usually appear in anterior region of oral cavity, which may last 7-9 days. In anamnesis: absence of appetite, constipation, pain in abdominal region, etc. Coprology – disorders of proteins, lipids and hydro-carbonates fermentation. At investigation: pathology of digestive system: hyperacid gastritis, cholecystitis, etc.

Severe form is characterized through appearance of solitary or multiple aphthae, of various sizes and localized in anterior and posterior regions of oral cavity, which may last 9-20 days. Recurrences appear more frequent to 4 times per year, sometimes in each month. In first days may be fever. Parallel with epithelization may be appearing other ones. Sometimes develops

the ulcers after their epithelization may be appearing cicatrices. Children with severe form in oral cavity have diseases of digestive system: chronic gastritis, chronic gastro-duodenitis, nonspecific colitis, etc. Children accuse constipation, diarrhea, spontaneous pain in abdominal region, etc.

Endoscopies: intestinal pathology, frequently appearance of aphthae coincides with appearance of intestinal erosions. Investigation must be effectuating concomitant with paediatrist, gastrologer ant other specialists.

Treatment depends of general disease. Individually are indicated necessary diet and medicaments. Of children with severe form in recurrent period are indicated immune-stimulators: lizozim, decaris (levamizol), prodigiozan etc.

Local treatment has symptomatic character: anesthetics, ferments; keratoplastics; antiseptics; anti- inflammatory preparations, etc.

Dispensary control: children are investigated one time per year of light form and two times per year of severe form of recurrent aphthae of oral mucosa.

TRAUMATIC LESIONS OF ORAL MUCOSA AT CHILDREN

They may be later than action of mechanical, thermal, electrical and radioactive agents.

The most frequent of children is meet ***acute mechanical traumas*** of oral mucosa. In anamnesis: bite of oral mucosa, traumatism with toys, pencils, sharpened things, etc. Symptoms: haematoma, laceration (erosions or ulcerations). More frequent are affected lips, soft and hard palate, rare – alveolar bone. Diagnostic, usually, isn't difficult.

At little children diagnostic may be sometime difficult: in case of poor anamnesis and if from moment of trauma passed few days, then in oral cavity appears edema of cause of nonspecific inflammation association. Child refuses to eat; disorders of the

general state; fever; regional lymphadenitis; appears pint in time of mastication and speaking.

Treatment: medical care is indicated in function of sizes and depth of lesions. In first, must to start with remove traumatic agent action. Profound laceration, after local anesthesia and processing, is sutured.

In case haematoma, erosions or small ulcerations is sufficient painting with antiseptics (furacilin, etoniu, lactate etacridin ş.a.) and with keratoplastics. Acute mechanical traumas of the oral mucosa of children have fast epithelization.

Chronic mechanical traumas of children are meeting as to erosions, ulcers and leukoplakia. These traumas are caused by sharpened margins of teeth, roots, and fillings; vicious habits, etc. In function of: intensity, time of action, localization of lesion, ages of child, reactivity of organism, microflora of oral cavity, etc. are clinical symptoms.

Bednar aphthae

It is localized erosion in region of junction of hard and soft palate in consequence of trauma with nipple, feeding bottle, etc. Of children to artificial alimentation, hypo-trophic, premature born, illness, etc. is meeting. Usually appear of children in first days of life. Aphthae is oval erosion with gray-yellowish deposits and inflammatory halo. In some cases may be bilateral erosions localized on the hard and soft palate.

Treatment: exclude the cause; change the nipple on one shorter; antiseptics; keratoplastics and anti-inflammatory; anesthetics etc. Aphthae ends with epithelization in time of few weeks.

Habits to bite or to suck the lip, cheek and tongue, etc. are enough frequently of all aged children, because it is produced unconscious or voluntary. Chronic lesion of mucosa may be localized on the jugal mucosa, occlusal line or other region of mucosa which may be bite.

Soft leukoplakia

It is develop after a chronic inflammatory process. At the basis of affection are neuro-trophic changes of oral mucosa caused by chronic trauma, acute inflammatory processes, smoking, etc. Children don't have accuses. It has following characteristics: on the edematous lips and cheeks mucosa in - angular and occlusal line region appears an opacity, which obtains of surface a white nuance and has cornification (elements of hyperkeratosis). Affected epithelium is detached easily.

Exist typical (focal or diffuse) and atypical forms. Atypical form is characterized through absence of cornification and presence of mucosa opacity. Exacerbation at children is in period of control tests, exams, competitions, etc. - in situations of the neuro-psychic straining. Diagnosis is based on the presence of chronic trauma.

Differentiated diagnosis: with typical form of lichen planus with manifestations in oral cavity; Cannon spongy nevus; candidosis.

Treatment: exclude the cause, vicious habits and make assanation of oral cavity. For children with neuro-psychic disorders are indicated sedatives; poly-vitamins; vitamin A in oil to 5-10 drops of 2-3 time per day, 1,5-2 months. Recommend sport, psycho- and hypnotherapy. Local apply keratoplastics: oil solution of retinol acetate, caratolin, aecol etc. to 3-4 time per day.

Thermal lesions

Of children is meeting rarely and is provoked frequently by hot food (milk, soup etc.). Usually is affected: labial mucosa, apex and anterior surface of tongue, anterior region of hard palate. Appear hyperemia, edema, intraepithelial vesicles, superficial ulcerations and erosions, living pain. Severity depends of agent temperature, time of action, depth and surface of lesion, age of children etc.

Treatment is analog with treatment of acute lesions:

symptomatic. In severe cases children are hospitalized in section of combustions and is indicated a complex treatment (general and local).

Chemical lesions

Frequent is meeting of 1-3 aged children when they unconscious can consume diverse acid or alkaline solution, etc. Appear pain in region of the contact of mucosa with chemical agent. Symptoms are in correlation with chemical substance nature, its quantity, time of action, age of children, etc. Acids provoke coagulation necrosis: mucosa is covered to the membrane, which adheres to subjacent tissues; hyperemia and edema. Alkaline provoke colliquative (humid) necrosis which sometimes harms all stratums of mucosa. After few days appear erosions or ulcers.

Chemical lesion of children during the stomatological treatment may be provoked by diverse medicaments: formalin, alcohol, ester, phenol, arsenical paste etc.

Treatment: remove chemical substance of the mucosa. If contact with chemical agent takes place in presence of doctor immediate oral cavity is removing with neutralizing solutions. For acid - 1-2% solution of hydro-carbonate of sodium, water of soap, etc. For alkaline - 0,5-1% solution of citric or acetic acid. For arsenical paste is used magnesium oxide or iodide solution. For solution of resorcin-formalin - 3% solution of ammonium carbonate. For solution of argent nitrate is used solution Lughole or 2-3% solution of sodium chloride. For ammoniac - 0,5% solution of citric, acetic, hydrochloride acid. For alcoholic solution of iodine is using magnesium oxide, solution of hydro-carbonate of sodium, and for sodium fluoride - 10% solution of calcium chloride.

Local is applied: anesthetics; antiseptics; keratoplastics; proteolytic ferments etc. and general: poly-vitamins, nonirritant aliments, etc.

ABNORMALITIES OF TONGUE AND INDEPENDENT GLOSSITIS

Mucosa of tongue is affected concomitant with other region of oral mucosa to stomatitis of diverse etiology. But are some diseases only of tongue.

Fissured tongue

This anomaly of development is meeting rarely of small children (Down disease), more frequent may be of children at pre-pubertal and pubertal period, when concomitant with rise of organism the size of tongue grows caused by development of muscular stratums. On the lateral and dorsal surfaces of tongue detect some sulcus (one is central, deep and some lateral). Deepness and surfaces of sulcus are covered with filiform papillae. Tongue is enlarged, soft at palpation. Usually children don't have accuses. In case of microorganisms and fungi association appears an inflammatory process, in these cases appear accuses to pain and burn on the tongue surface (see color insert Fig.10.14.).

***Differential diagnostic*:** with diverse inflammatory process; lymphangioma of tongue.

Treatment is symptomatic, necessity the assanation of oral cavity, and remove of tongue deposits.

Geographic tongue
(benign migratory glossitis, erythema migrants)

It is considerate of congenital state, with hereditary character and may be the entire life. Also it may be in case of lingual neuro-trophic disorders, exudative diathesis, diseases of digestive system, etc. Sometimes is meeting of healthy children and can appear of first year of life.

Symptomatology: on lateral and dorsal surfaces of the tongue appears an opaque sector, white-yellowish or grayish, provoked by tumefaction and maceration of epithelium. After that starts

desquamation of corneous epithelium of filiform papillae and formation of red-bright smooth sector, which is evident on the environing epithelium. The red-bright color is because of thin stratum of the epithelium, but not inflammatory hyperemia. On the borders of the desquamation is evident high up epithelium. Concomitant, may be some nidus of oval, round or semicircular desquamation, which may be confluent similar to geographic map. For children is characteristic fast desquamation and regeneration of filiform papillae epithelium (2-3 days), that explain daily modification of region's form. Children don't have accuses, general state isn't modified. Sometimes may be little pain and burn from irritant aliments (see color insert Fig.10.15).

Differential diagnostic: with tongue desquamation in case of avitaminosis (A, B_1, B_6, B_{12}); rhomboid tongue.

Treatment is symptomatic: anesthetics; antiseptics; keratoplastics, calcium pantotenat 0,03-0,1 g to 3 time per day during one month, for 2-3 time per year; assanation of oral cavity, and treatment of general chronic diseases.

Rhomboid glossitis

It is chronic inflammatory disease rarely meeting of children. Symptomatology: appearance of one rhomboid or oval sector (0,5-2 cm on 1,5-3 cm), localized on the median line of tongue, anterior of circumvallated papillae. This sector doesn't have filiform papillae, but is very good outlined and has pink or red color. Usually develops asymptomatic.

Treatment is symptomatic; assanation and hygiene of oral cavity.

Hairy tongue

This pathology is rarely meeting of children, sometimes appears after various diseases and treatment with antibiotics. Children accuse absence of appetite; have sensation of foreign thing on the tongue, etc. On the dorsal surface of tongue in front

of circumvallated papillae, papillae filiform are elongated and thickened (hyperplasia) to 1 cm and more. Hyperplasia is caused by absence of physiologic desquamation of corneous epithelium of filiform papillae. Their color varied between brown and black. Hyperplasic sector has usually an oval form on the median line of tongue (see color insert Fig.10.16).

Differential diagnostic: with false hairy tongue (in result of oral irrigation with potassium permanganate, chloramines, some antibiotics and other medicaments, and some foodstuff).

Treatment: paint tongue with keratolytics: 1-2% solution of resorcin – for small children; for children more than 5 years - 1-2% solution of salicylic acid in alcohol of 70°. Removes tongue two times per day; assanation of oral cavity.

CHEILITIS

Pathology of lips is the most frequently of children.

Classification of cheilitis by A. Машкиллейсон and C. Кутин

1) independent cheilitis: exfoliative, glandular, of contact (simple and allergic), meteorological and actinic;

2) cheilitis symptomatic (atopic, eczematous, macro-cheilitis in Melkersson-Rozenthal syndrome, hypovitaminosis).

Exfoliative cheilitis is chronic affection with lesion of red margin of lips. In etiology and pathogenesis has an important role hereditary factor, immune-allergic disorders, dysfunction of nervous system, etc.

Dry form Children accuse appearance of grayish or gray-yellowish squame on the one or both lips, dryness of lips, and sensation of burn. Squame is fixated to epithelium of red lips margin only to one extremity other extremity is free. After removing of squame don't appear erosions, but is hyperemia. Evolution of disease is elongated (more years) with recurrences.

This form may be transformed in exudative form.

Exudative form Children accuse burn, pain, appearance of crusts which can hinder the speaking and eating. Crusts have gray-yellowish color, are localized more frequently in Klein zone, appear hyperemia and edema. One band of red lips margin to limit of skin and commissural labial isn't affected. Sometimes exudative processes are pronounced and crusts hung down. After removing of squame appear a moist surface, edema and hyperemia, isn't erosions.

Treatment: 1) Treatment of general disease. 2) Sedative, antidepressants, poly-vitamins, antihistamine, etc. 3) *Bucky* rays. 4) Laser with helium-neon. 5) Before each cure of treatment with *Bucky* rays recommend crusts removing, after application of antiseptics, anti-inflammatory preparation. 6) Local, in special of dry form, applies indifferent unguents, creams (hygienic lipstick, etc.).

Meteorological cheilitis Appears in result of diverse meteorological factors action (humidity, wind, cold, etc.) in certain conditions (constitutional particularities, predisposing factors: seborrhea, neuro-dermitis, etc.). Symptomatology: burn of lips, unpleasant sensations of constriction, exfoliated lips. Red margin of lips has hyperemia and infiltrate, frequent is covered by small squame. Distinguish slowly and chronic evolution.

Treatment: is symptomatic (unguents, cream, hygienic lipstick, vitamins B2, B_6, B_{12}; in severe cases – unguents with corticosteroids, etc.

Actinic cheilitis Chronic disorders of lips provoked by high sensitivity of red lips margin to action of the solar rays.

Dry form is characterized by burn, dryness, sometimes pain, erythema, small silvery-white squame, can appear erosions (see color insert Fig. 10.17).

Exudative form on the erythema and edema of mucosa and

red margin of lips appear vesicles, erosions, crusts. Children accuse pruritus, burn and pains.

Treatment is symptomatic: unguents with corticosteroids; vitamins B_2, B_6, PP: cream for prophylaxis of sunstroke.

Allergic cheilitis is a form of slow allergic reaction to action of diverse allergens (chemical compounds of lipstick, dentifrices, acrylic resin, etc.) on the red margin of lips.

Children accuse pruritus and burn. Usually is affected red margin of lips, but sometimes may be affected skin and mucosa of lips. Observe a limited erythema and desquamation, in severe cases may be vesicles, erosions, and exudation.

Treatment: Cause removing; local - unguents with corticosteroids (prednizolon, flucinar, ftorocort, etc.); antihistamine therapy; etc.

Glandular cheilitis appears more frequent on the inferior lip in result of hyperplasia, hyper-function of salivary glands of labial mucosa. Provocative agents may be traumatic lesions of lips with teeth, diverse things, vicious habits (suckling of lips), viruses, etc. At start children accuse the dryness of lips. Examination: observe on the labial mucosa the orifices of salivary glands canals are open in form of red points through that are eliminated saliva -- sign of "dew". Because of permanent humidity on lips appear macerations and erosions. Lips lose elasticity and appear fissures.

Treatment: 1. Local - anti-inflammatory unguents, in severe cases - glucocorticoids. 2. Electro-coagulation of the salivary gland. 3. Sialoadenectomy.

Atopic cheilitis is a symptom of atopic dermatitis or diffuse neurodermitis determined by genetic factors, which create predisposition to atopic allergy. Allergens may be: food, medicaments, microorganisms, physic factors, etc. more frequent is meeting of children after 7 years of life.

Symptomatology: is affected red margin of lips and always the skin preponderant in angular region. Never is affected labial mucosa. Disease starts with pruritus, appearance of erythema. Subsequently red margin of lips is infiltrated, appear little crusts which desquamate, appear fissures. The skin is dry and desquamated. Evolution is slowly, recurrences appear in autumn and springtime. Frequent after age of 20 may be auto-heals.

Treatment: antihistamine medication; vitamins of group B; in severe cases are indicated glucocorticoids: prednisolon 10-15 mg/day of children aged 8-14 or dexametazon; intravenous injections with tiosulfat of sodium 30%; local - unguents with glucocorticoids of 4-5 time per day; *Bucky* rays; exclude piquant, salty, citric, sweet, etc. foodstuff.

Eczematous cheilitis is a symptom of eczema – inflammation of skin superficial stratums with neuro-allergic character and acute or chronic development. For allergens may be diverse factors: food, medicaments, microorganisms, dentifrices, etc.

Acute form - appear pruritus and burn. Polymorphism is characteristic: step by step appear erythema, edema, vesicles, squame, and crusts. Ever is affected the skin in perioral region.

Chronic form – appears inflammatory infiltrate and compression of red margins and skin in perioral region, squame, crusts, fissures, cracks.

Sometimes microbial flora is associated and provokes microbial eczema: edema, erythema, appear vesicles covered by gray-yellowish crusts, and desquamation.

Treatment: antihistamine medication; sedative; local – unguents with corticosteroids (lorinden C, dermozolon, oxicort, etc.).

Melkerson – Rosethal – Mischer - Rossolimo syndrome is characterized by three symptoms: macro-cheilitis, neuritis and

fissured tongue. It is an infectious-allergic process, hereditary determined, with angio-nervous component.

Children accuse enlarged lip, appearance of pruritus, edema of lip, oral mucosa and facial skin. Paralysis of facial nerve may be present. The tongue is fissured. Disease has a chronic and recurrent development.

Treatment: corticosteroids; anti-malarias medicaments; antihistamine therapy; vitamins of group B, vitamin C; hyposensitivity therapy with bacterial allergens; local – electrophoresis with unguent of heparin, etc.

The main factor in pathogenesis of all labial pathology of children is labial architectonic trouble. For to normalize correct interposition of lips is necessary following:

1. Remove oral respiration (education, respiratory gymnastic, remove vicious habits; correct position in time of sleep in the firs year of life; remove of adenoids and deformation of nasal septum; etc.).
2. Treatment of dental-maxillary anomalies.
3. Labial myotherapy, massage of lips for reinforcement of muscular tonus, etc.

ORAL MANIFESTATIONS OF SYSTEMIC PATHOLOGY OF CHILDREN

Oral mucosa is in relationship with diverse organs and systems of organism. Receptors of oral mucosa have links with digestive system, central nervous system, cardiovascular system, endocrine system, etc. A lot of oral diseases are caused by disturb of diverse organs and systems function and the first symptoms of systemic pathology frequent appear on the oral mucosa and don't have specific manifestations. This creates difficulties in diagnostic.

Oral manifestations of gastrointestinal diseases

Chronic gastritis More frequent is present desquamative

atrophic glossitis and atrophy of lingual papillae. Hypertrophy of lingual papillae is established of hyperacid gastritis. Frequent accuse to perturb of taste and decrease of lingual mobility; recurrent aphthae of oral mucosa; acute or chronic herpetic stomatitis; cheilitis; etc.

Chronic enterocolitis of children has following oral manifestations: glossitis, angular cheilitis, recurrent aphthae (hypovitaminosis PP and B_2), desquamative glossitis (51% of children) and fissured glossitis (38% of children). Glossitis is characterized by edema, cyanosis and evident hyperemia.

Dysentery Appear catarrhal stomatitis and aphthosis after 2-3 days from the debut of disease, and desquamative glossitis late - after 7–14 days. Of severe form may be complicated with oral candidosis.

Hepato-cholecystitis Periodic may be desquamative glossitis with atrophy of lingual papillae.

Disbacteriosis is manifested frequently by acute candidosis, which is explained through modification in components and activity of intestinal flora.

Hepatic diseases The most frequent of children is **acute hepatitis** of viral etiology. In period of clinical manifestations establish hyperemia and edema of oral mucosa, desquamation of epithelium, xerostomia. The characteristic symptom is icterus oral mucosa. Tongue is edematous, cyanotic, with atrophy of filiform papillae and sectors of desquamation, gradually appears a red-bright and shining surface. Children accuse pains and burn of tongue. Frequent on the tongue appear many quantities of deposits. Also may be inflammatory and hyperplasic modifications of salivary glands ducts, telangiectasias on lips and soft palate.

Treatment: of general disease. Local treatment include: regarding of oral hygiene; oral cavity rinsing with antiseptics and anti-inflammatory, etc.

Oral manifestations in hematological diseases

Acute leucosis (acute leukemia) is characterized of hemorrhagic syndrome, leucemic infiltrates and ulcero-necrotic lesions on the gingival, palatine, lingual, and jugal mucosa. On the sectors with hemorrhages appear niduses of necrosis, which extend gradually. Tongue is edematous, covered by brown-grayish deposits, and on the apex and contact surfaces with teeth are ulcerations. Sometimes on the mucosa can appear vesicles that rapid crack and form erosions.

In oral cavity determine nidus of necrosis on mucosa and gum. Gingival papillae in severe cases can have necrosis of submucous stratum. Submucous gingival stratum is infiltrated by lymphatic, reticular, plasmatic and blastic cellules. Cellular infiltrate substitute connective tissue and circular dental ligament. The most frequent appear hypertrophic gingivitis. Progress of process induces of alveolar bone lesion and appearance of marginal periodontitis characterized by teeth mobility, hemorrhages, periodontal pockets, etc. In maxillaries predominates resorption of vertical type. Children accuse pains in maxillaries of intercuspidation of intact teeth. In acute leucosis of children are established blastic modifications in pulp. These disorders are more evident in teeth with undeveloped roots and last part of pulp. At 50% of children with leucosis on the oral mucosa are ulcerations.

Differential diagnostic: with Vincent ulcero-necrotic gingivo-stomatitis, hypovitaminosis C, and hypertrophic gingivitis.

Treatment is complex, in conditions of hospital - in hematological section for children. General treatment is indicated of hematologist. Regarding of oral hygiene is one of the main conditions. Assanation of oral cavity, removing of dental plague and calculus is making in conditions of hospital.

In case of: a) ulcero-necrotic affections of oral mucosa apply

local anesthetics, antiseptics, proteolytic ferments and keratoplastics; b) associated viral or mycotic infection - indicate local and general antivirotic or antimycotic therapy.

Children are of dispensary control in III group.

Myelomatosis Manifestations on the oral mucosa frequent are the first symptoms of disease. Precocious diagnostic and treatment can lead of disease remission.

Typical symptoms of disease are: hyperemia and cyanosis of gingival margin, gingival hyperplasia and hemorrhage, dental plaque and calculus. In submucous stratum establish reticular, lymphatic and many plasmatic cellules. Clinical and radiological: generalized progressive periodontitis. Resorption of interdental septums and of alveolar bone develops more intensively than in leucosis.

Agranulocytosis is a syndrome characterized by increase ore absence of granulocytes and neutrophils in peripheral blood.

At inspection of oral cavity determine on the tongue, soft and hart palate, and cheeks little sectors of necrosis. Affected surfaces are delimitated from environing tissues, have gray color and smell of putrefaction. Lymphatic ganglions are enlarged.

Differential diagnostic: with Vincent ulcero-necrotic gingivo-stomatitis and other hematological diseases.

Treatment is complex, in conditions of hospital - in hematological section for children. Local treatment: anesthetics, antiseptics, proteolytic ferments and keratoplastics; regarding of oral hygiene, etc. Children are of dispensary control in III group.

Hemorrhagic diathesis Pathology is manifested by predisposition of periodical hemorrhages which can appear simultaneous or in result of some unimportant lesions. Hemorrhagic diathesis may be provoked of: disorders of blood coagulation system; disorders of thrombocytopoiesis; lesions of

vascular walls, etc.

Hemophilia is a hereditary disease which is manifested only of boys. Exists three forms: 1. hemophilia A (classic) – insufficiency of VII factor; 2. hemophilia B – insufficiency of IX factor; 3. hemophilia C – increase of X factor activity.

Disease appears more frequent of 2-3 years aged children. Children are hypotrophic, have thin and pallid skin, and undeveloped adipose subcutaneous tissue. In case of traumas and in some cases without traumas appear hemorrhages. Hemorrhages are localized in region of joints and gastro-intestinal mucosa. Hemorrhage of marginal gum in period of teeth eruption or trauma by dental margins. Intense hemorrhage can appear after dental extraction. Disease can evaluate long time asymptomatic and is established only after dental extraction.

Children are of dispensary control of hematologist.

During the assanation of oral cavity must be avoided intervention on the pulp, apical and marginal periodontium, and dental extraction and chirurgical manipulations. In case of necessity of these manipulations children are hospitalized in specialized hematological centers. Dental extraction and chirurgical intervention are effectuated with minimum of traumatism. After assanation of oral cavity and chirurgical manipulations child is supervised lees one week in hospital.

Thrombocytopenic purpura (Werlhof diseased) is due to thrombocytes deficiency.

Characteristic symptom is appearance of hemorrhages without definite causes, usually after septic states, infections, influenza, pneumonia, etc. These hemorrhages are localized of the whole body, are asymmetrical and of diverse degree.

In oral cavity are established gingival hemorrhage and gingivitis, which are aggravated in period of teeth eruption and in pubertal period.

Anemia The nutritional deficiencies associated with anemia: iron, B_{12} and folate are all associated with recurrent oral ulceration and specific deficiencies may be present, even in the absence of a frank anemia. Atrophic glossitis was formerly the commonest oral symptom of anemia but less often seen now. Red lines or patches on a sore but normal looking tongue may indicate B_{12} deficiency. Candidosis may be precipitated or exacerbated by anemia, particularly iron deficiency, and angular cheilitis is a well-recognized association. The sore, clinically normal tongue (burning tongue) is sometimes a manifestation or even precursor of anemia.

Oral manifestations of cardiovascular disease

Affection of oral mucosa is remarked of decompensate stage of cardiovascular insufficiency, but of compensate stage of cardiovascular insufficiency it doesn't establish. Cardiovascular diseases of children with hypoxia symptom of decompensation are associated by edema and cyanosis of oral and gingival mucosa.

In case of vascular insufficiency are possible dystrophic processes, and in some cases - necrosis of oral mucosa in regions liable to mechanic action. Trophic ulcers are painful, have rugged margins, the depth is covered with white-grayish necrotic deposits and has slowly epithelization.

Oral manifestations of endocrine disease

Addisons' disease (adrenocortical hypofunction) classically, causes melanotic hyperpigmentation of the oral mucosa, commonly of the cheek. May also be part of endocrine-candidosis syndrome.

Cushing syndrome The appearance of a "moon face" and oral candidosis are the common head and neck manifestations of this syndrome.

Hypothyroidism Congenital hypothyroidism is associated with enlargement of the tongue, with puffy enlarged lips and delayed tooth eruption. In adult hypothyroidism, puffiness of the face and lips also occurs, but there are no particular oral changes.

Hyperthyroidism Not associated with any particular oral changes.

Hypoparathyroidism may be a component in the endocrine-candidosis syndrome, but there are no other specific changes.

Hyperparathyroidism is rare. It is caused by hyperplasia or adenoma of the parathyroids. Increase of parathormone cause increase of plasma calcium liberated from bone. Appears in the jaws as loss of lamina dura, a "ground glass" appearance of bone and cystic lesions (often looking multilocular), which contains dark-colored tissue, "brown tumor" histologically indistinguishable from a giant cell granuloma.

Diabetes No specific oral changes, although manifestations of decrease resistance to infection can be seen if poorly controlled (e.g. severe periodontal disease). Xerostomia and thirst are prominent features of ketoacidosis. Sialosis is sometimes seen as a late feature of diabetes. Burning mouth may be a presenting feature of diabetes, and oral or facial dysaesthesia may reflect the peripheral neuropathies seen in diabetics. There is a tendency to slower healing following surgery.

Sex hormones There is a well-recognized increase in the severity and frequency of gingivitis at puberty and in pregnancy. Some females have recurrent aphthae clearly associated with their menstrual cycle.

Oral manifestations of renal disease

More frequent oral mucosa is affected in glomerulonephritis

and chronic renal insufficiency. Children accuse: unpleasant taste (bitter) and dryness in oral cavity. Oral mucosa is edematous. In result of trophic disorders may appear nidus of necrosis on the alveolar mucosa, in jugal and retro-molar region etc. Chronic catarrhal gingivitis is characteristic with formation of periodontal pockets, dental mobility etc.

Treatment: of renal disease. Local is indicated antiseptics, proteolytic ferments, and keratoplastics; regarding of oral hygiene.

Oral manifestations of hypo- and avitaminosis

Hypovitaminosis A. Vitamin A normalizes metabolism, is necessary for development of the organism, maintains the health of skin, increase resistance of mucosa to pathogenic agents, etc. Symptomatology: dry and desquamated skin; dry conjunctiva and oral mucosa; hyperemia of oral mucosa; catarrhal persistent gingivitis and stomatitis; dry lips with superficial fissures and angular cheilitis; xerostomia. Associated secondary infection provokes erosions and ulcerations.

Treatment: indicate vitamin A (dragee of 3300 U or concentrate of vitamin A by 5-10 drops time of 1-2 months). Recommend food reach in vitamin A. Local treatment is symptomatic (antiseptics, keratoplastics etc.).

Hypovitaminosis B_1. Insufficiency of vitamin B_1 *(thiamine)* is manifested by pains and atrophy of lingual papillae, edema of oral mucosa and catarrhal gingivitis. Symptomatology: functional disorders of nervous system (neuroses, paraesthesia, asthenia, etc.); cardiovascular system and gastrointestinal system (nausea, vomit, decrease of appetite, etc.).

Treatment: tablets of Thiamine bromide or Thiamine 0,002-0,01 g and solution 2,5% and 5%; foodstuff reach in vitamin B_1 : beans, cereals, bread of rye, meat, etc..

Hypovitaminosis B_2 is manifested by following symptoms: dermatitis, cheilitis, and glossitis. Insufficiency of vitamin B_2 *(riboflavin)* provokes affection of oral, lips, eyes and ears skin and mucosa, etc. Eczema in perioral region; hyperemia and edema of red margin of lips with fissures, squame and crusts are characteristic. Frequent is determined angular cheilitis, associated with hemorrhages and maceration of epithelium, pains in time of speaking and eating.

The precocious characteristic sign is "raspberry" tongue (Hunter): lingual surface has hyperemia, atrophies of lingual papillae, and dorsum of tongue is red-bright, smoothly and radiant.

Treatment: is indicated sol.1% riboflavin mononucleotide 1 ml per day for 10-15 days or 0,01 g of 3 time per day for 1-1,5 months; foodstuff reach in vitamin B_2 – milk, eggs, meat, liver etc.

Hypovitaminosis PP. Vitamin PP *(nicotinic acid)* participates in processes of oxidoreduction, metabolism of hydrocarbonates, lipid, etc. In case of vitamin PP insufficiency appear pellagra, characterized by dermatitis, madness and diarrhea. Children accuse nausea, vomit, and decrease of appetite, persistent diarrhea, apathy, and asthenia. Dermatitis is characterized to pigmentation, hyperemia, dryness and desquamation of skin. Tongue is enlarged, edematous and with abundant deposits. Subsequently tongue is removed of deposits, appears atrophy of lingual papillae ("raspberry" tongue). Progressing of process provokes appearance of fissures, bulla, erosions and ulcerations on the labial, lingual („mosaic tongue"), gingival mucosa; hypo-salivation, dryness and intensive oral pains („fire in the mouth").

Treatment: In pellagra state is indicated 0,02-0,05 g vitamin PP of 2-3 time per day, injections intramuscular with solution 1% nicotinic acid of 1 ml (10-15 injections); dragee

0,015 g of 3 time per day and in solution 1%-5% for injections. Recommend foodstuff reach in vitamin PP: fruit, honey, liver etc.

Hypovitaminosis C *(scorbutus, Moeller-Barlow disease).* Insufficiency of vitamin C provokes decrease of child's organism reactivity, is manifested by decrease of phagocytosis capacity and forming of antibodies. Also, provokes disorders of capillaries permeability and of connective tissue, syntheses of collagen, blood coagulation etc. Hypovitaminosis C may appear in first year of life of children with artificial alimentation (sterilized milk).

Hypovitaminosis C is associated with asthenia, weakness, absence of appetite, decrease of body weight, myalgia, etc. Symptomatology: dry skin, appear haematoma, desquamation. In oral cavity: edema, cyanosis and hemorrhage of gingival mucosa; hemorrhagic diathesis and ulcero-necrotic processes, necrosis of gingival papillae, periodontal pockets, dental mobility. Gingivitis has a hyperplasic character, with intense hemorrhages. Sometimes, hypertrophic gum may cover surfaces of dental crowns. In case of secondary infection association: ulcero-necrotic gingivitis, fetid smells in oral cavity; severe intoxication of organism, fever, etc.

Treatment is complex. General treatment – remove principal cause, administrate vitamin C (intravenous, intramuscular or per oral): of 100 mg by 5 time per day (1 g / day); solution 5% 3-5 ml intramuscular; vitamins C and P of 50-100 mg by 2-5 time per day, galascorbine 0,5 g of 3-4 time per day; rutine 30-40 mg of; vitamin K and solution 10% calcium chloride; foodstuff reach in vitamin C.

Local treatment: galascorbine, vitamin C, caratoline etc., antiseptics; recording of oral hygiene; removing of dental plaque.

Control questions and topics:

1. What pathogenic agents provoke the most frequently disorders of oral mucosa of children in first year of life?
2. Clinical development and treatment of acute herpetic stomatitis.
3. Prophylaxis of acute herpetic stomatitis.
4. What factors support appearance of oral candidosis of children?
5. Symptoms and treatment of oral candidosis of children.
6. What pathogenic agent provokes Zoster Zone and varicella?
7. Symptoms and treatment of Zoster Zone and varicella of children.
8. Symptoms and treatment of diphtheria of children.
9. Symptoms and treatment of polymorph exudative erythema.
10. Symptoms of desquamate glossitis.
11. Symptoms of scarlatina on the oral mucosa of children.
12. Etiology, symptoms on the oral mucosa, treatment and prophylaxis of Bednar aphthae.
13. What substances can provoke frequently chemical lesions of the oral mucosa of children?
14. What diseases of digestive system have symptoms on the oral mucosa?
15. What cardiovascular diseases have symptoms on the oral mucosa of children?
16. What symptoms are characteristic for hematological diseases?
17. Oral symptoms of drug induced allergy of children.
18. Symptoms and treatment of atopic cheilitis (eczematous) of children.

CONTROL TESTS

Acute inflammatory diseases of oral mucosa of children, manifestations in cases of acute infectious diseases

Simple compartment

1. Acute inflammatory disorders of oral mucosa are characterized by following processes:
   A. Alterative;
   B. Alterative and exudative;
   C. Exudative and proliferative;
   D. Proliferative;
   E. Alterative and proliferative.

2. Manifestation of some particular pathologic signs is:
   A. Modification;
   B. Lesion;
   C. Disease.

3. Lesion of tissues and organs integrity with disorders of function is following:
   A. Modification;
   B. Lesion;
   C. Disease.

4. Bednar aphthae are:
   A. Disease;
   B. Modification;
   C. Lesion.

5. Polymorph exudative erythema is:
   A. Disease;
   B. Modification;
   C. Lesion.

6. Raspberry tongue is:

A. Disease;
B. Modification;
C. Lesion.

Multiple compartment

7. Primary elements of oral mucosa disorders are:
   A. Squame;
   B. Macula;
   C. Crust;
   D. Pustule;
   E. Erosion;
   F. Bulla;
   G. Papule;
   H. Cicatrix.

8. Secondary elements of oral mucosa disorders are:
   A. Afta;
   B. Tubercle;
   C. Fissure;
   D. Rush;
   E. Ulcer;
   F. Cyst.

9. Scarlatina is differentiated to:
   A. Diphtheria;
   B. Angina;
   C. Varicella;
   D. Acute herpetic stomatitis;
   E. Acute candidosis.

10. Antiviral unguents application in case of rubeola and varicella are necessary for prevention of:
   A. Infection with other viruses;
   B. Intoxication;
   C. Multiplication of viruses in cellules of oral mucosa;

D. Association of secondary infection.

Traumatic affections of oral cavity mucosa
Simple compartment

1. Mucosa of oral cavity has following functions:
   A. Sensitive;
   B. Absorptive;
   C. Plastic;
   D. Protective;
   E. All answers are correct.

2. Acute inflammation provoked by trauma of oral mucosa is maintained:
   A. 10-14 days;
   B. 1 month;
   C. 5-7 days;
   D. More than 1 month;
   E. 2 days.

3. Bednar aphthae more frequent appear of children with:
   A. Artificial alimentation and weakness;
   B. Artificial alimentation from 3 months of life;
   C. Cardiac vices;
   D. Vicious habits;
   E. Body weight of birth less than 3 kg.

4. Chronic trauma of oral mucosa is treated by following algorithm:
   A. Anesthesia, removing of cause, antiseptic treatment, keratoplastics.
   B. Removing of cause, antiseptic treatment, and keratoplastics.
   C. Removing of cause, anesthesia, antiseptic treatment, and keratoplastics.
   D. Antiseptic treatment, anesthesia, keratoplastics.

E. Antiseptic treatment, keratoplastics.

Multiple compartment

5. Classification of oral mucosa affections of children by Т.Виноградова contains following compartments:
   A. Clinical evolution;
   B. Localization of pathological processes;
   C. Etiological factors;
   D. Age of child;
   E. Morphological manifestations.

6. In function of etiology, exist following groups of oral mucosa affections (classification by Т. Виноградова):
   A. Mycotic, viral, bacterial;
   B. Modifications of oral mucosa than symptoms of organs and systems of organism pathologies;
   C. Mechanical, thermal and chemical lesions;
   D. Congenital;
   E. Allergic.

7. In function of localization of oral mucosa affections (classification by Т. Виноградова) are:
   A. Cheilitis;
   B. Glossitis;
   C. Pareitis;
   D. Adenitis;
   E. Papillitis.

8. In function of localization of oral mucosa affections (classification by Т. Виноградова) are:
   A. Stomatitis;
   B. Gingivitis;
   C. Lymphadenitis;
   D. Tonsillitis;
   E. Palatinitis.

9. In function of morphological manifestation of oral mucosa affections (classification by T. Виноградова) are:
   A. Primary elements;
   B. Secondary elements;
   C. Tertiary elements;
   D. Inflammation;
   E. Tumor.

10. Tunica of oral mucosa has following tissues:
   A. Connective;
   B. Epithelial;
   C. Adipose;
   D. Itself mucosa;
   E. Submucous.

11. In function of morpho-functional particularities, oral mucosa may be of following types:
   A. Masticatory;
   B. Covering;
   C. Specialized;
   D. Mixed;
   E. Secretory.

12. Primary elements on the oral mucosa are following:
   A. Bulla;
   B. Vesicle;
   C. Macula;
   D. Aphthe;
   E. Papule.

13. Secondary elements on the oral mucosa are following:
   A. Bulla;
   B. Erosion;
   C. Aphthe;
   D. Cicatrices;

E. Ulcer.

14. For chronic trauma of oral mucosa are characteristic following elements:

A. Bulla;
B. Bednar aphthe;
C. Erosion;
D. Cicatrices;
E. Fissure;
F. Macula.

15. Erosions and decubital ulcers are localized more frequently in following regions:

A. Hard palate;
B. On the apex or lateral surface of tongue;
C. On the velum;
D. On the transient plica;
E. In central region of dorsal surface of tongue;
F. In jugal region.

16. Differential diagnostic of oral mucosa traumas is realized with:

A. Candidosis;
B. Acute herpetic stomatitis;
C. Necroses of oral mucosa in leucosis, disorders of decompensate sanguine circulation;
D. Chronic recurrent stomatitis;
E. Syphilis.

17. For treatment of chronic trauma of oral mucosa are indicated:

A. Keratolytics.
B. Anesthetics.
C. Keratoplastics.
D. Antiseptics.

E. Corticosteroids.
F. Antibiotics.

Acute herpetic stomatitis
Simple compartment

1. Pathogen factor of acute herpetic stomatitis is:
   A. *Herpes Simplex* virus;
   B. Association of bacteria and viruses in oral cavity;
   C. Oral microflora with pathogenic capacities, in case of decrease of organism reactivity;
   D. *Candida tropicalis;*
   E. *Herpes Labialis* virus.

2. Spreading of *Herpes Simplex* virus in population is:
   A. Increased;
   B. Decreased;
   C. Depend of season.

3. Period of incubation of acute herpetic stomatitis lasts:
   A. 2 – 7 days;
   B. 7 – 14 days;
   C. 14 – 21 days;
   D. 21 – 30 days;
   E. Until 2 years.

4. A minim risk of infection by acute herpetic stomatitis has:
   A. Children later than 3 years;
   B. Children of natural alimentation in first age of life;
   C. Dark-haired;
   D. All answers are correct;
   E. Children of artificial alimentation in first age of life.

5. A maximum risk of infection by acute herpetic stomatitis has:
   A. Children later than 3 years;
   B. Children until 1 year;

C. Children between 1 and 3 years;
D. Children of natural alimentation in first age of life;
E. Children of artificial alimentation in first age of life.

6. Factors which provoke appearance of acute herpetic stomatitis are:
A. Super-cooling;
B. Administration of immune-depressants;
C. Hormonal and emotional fluctuations;
D. Diverse diseases of organism;
E. All answers are correct.

7. Polymorphism of acute herpetic stomatitis is:
A. True;
B. False.

8. The characteristic element of period of eruption on the oral mucosa of acute herpetic stomatitis is:
A. Macula;
B. Bulla;
C. Vesicle;
D. Pustule;
E. Aphthe.

9. Determination of clinical evolution period of acute herpetic stomatitis based on:
A. State of lymphatic ganglions;
B. Character of elements on the oral mucosa;
C. State of gum;
D. Signs of general intoxication;
E. Temperature of body.

10. The main symptom of acute herpetic stomatitis is:
A. Fever;
B. Lymphadenitis;

C. Erosions on the oral mucosa;
D. Gingivitis;
E. Eruptions on the skin of face.

11 Characteristic symptoms for light form of acute herpetic stomatitis:
A. Lymphadenitis;
B. Fever;
C. Erosions on the oral mucosa;
D. Gingivitis;
E. All answers are correct.

12. Characteristic symptoms for severe form of acute herpetic stomatitis:
A. Lymphadenitis;
B. Fever;
C. Erosions on the oral mucosa;
D. Gingivitis;
E. All answers are correct.

13. The main symptom of severe acute herpetic stomatitis is:
A. Gingivitis;
B. Fever (39-40°);
C. Lymphadenitis;
D. Eruptions on the oral mucosa;
E. All answers are correct.

14. The first symptoms of acute herpetic stomatitis of medium degree are:
A. Increase of body temperature;
B. Changes in child's comportment: slumber, adynamia, agitation etc.;
C. Dyspepsia, refuse to eat, vomit, etc.;
D. Regional lymphadenitis;

E. Vesicles on the skin, solitary or multiple aphthae on the oral mucosa.

15. The length of period of eruptions to acute herpetic stomatitis of light degree is:

A. Until 24 hours;
B. Until 72 hours;
C. 3 – 4 days;
D. 5 – 7 days;
E. 14 – 21 days.

16. The length of period of eruptions to acute herpetic stomatitis of medium degree is:

A. Until 24 hours;
B. Until 72 hours;
C. 3 – 4 days;
D. 5 – 7 days;
E. 14 – 21 days.

17. The length of period of eruptions to acute herpetic stomatitis of severe degree is:

A. Until 24 hours;
B. Until 72 hours;
C. 3 – 4 days;
D. 5 – 7 days;
E. 14 – 21 days.

18. Precocious diagnostic of acute herpetic stomatitis is based on the following symptoms:

A. Fever, adynamia, indisposition, sialorrhea.
B. Herpetiform eruptions on the skin of face and hands.
C. Catarrhal gingivitis, regional lymphadenitis.
D. Catarrhal gingivitis, regional lymphadenitis and herpetiform eruptions on the skin.
E. Regional lymphadenitis.

19. The most informative method of diagnostic of acute herpetic stomatitis is:

A. Cytological;
B. Serological;
C. Immune-fluorescence;
D. Biochemical;
E. Radiological.

20. Appearance of immature epithelial cells in smear corresponds of following period:

A. Incubation;
B. Clinical manifestations;
C. Catarrhal;
D. Convalescence;
E. Remission.

21. Principal criteria for diagnostic of acute herpetic stomatitis and herpangina are following:

A. Of herpangina isn't catarrhal gingivitis;
B. Number of pathological elements;
C. The length of disease;
D. Localization of pathological elements;
E. Character of pathological elements.

22. In treatment of chronic recurrent herpetic stomatitis of children more important are:

A. Antivirotic medicaments.
B. Anti-inflammatory, anesthetic and keratoplastic medicaments.
C. Establish and remove the nidus of organism's allergization.
D. Immune-stimulatory medicaments.
E. Antiseptics, ferments.

23. Local treatment of acute herpetic stomatitis in convalescence period includes:

A. Anesthetics;
B. Antivirotic;
C. Antiseptics;
D. Keratoplastics;
E. Proteolytic ferments.

24. What is more important in treatment of chronic recurrent herpetic stomatitis of children:

A. Longtime local treatment with antivirotic medicaments;
B. Immune-stimulatory medicaments;
C. Symptomatic treatment;
D. Keratoplastics;
E. Antibiotics.

25. Local treatment of acute herpetic stomatitis in first 3 days from falling includes in the first:

A. Antivirotic;
B. Keratoplastics;
C. Antiseptics;
D. Antivirotic and anesthetic unguents;
E. All answers are correct.

26. Local treatment of acute herpetic stomatitis in catarrhal period includes in the first:

A. Antivirotic;
B. Keratoplastics;
C. Antiseptics;
D. Antivirotic and anesthetic unguents;
E. All answers are correct.

27. Antivirotic therapy for child with acute herpetic stomatitis is prescribed:

A. In period of eruptions;

B. In moment of lymphadenitis appearance;
C. In prodromal period;
D. In moment of gingivitis appearance;
E. All answers are correct.

28. Unguents with antivirotic action are:
A. Bonafton 0,5%;
B. Flucinar;
C. Neomycin;
D. Polymixin;
E. Nistatine.

29. Unguent with the least antivirotic action is:
A. Florenal;
B. Tebrophen;
C. Oxolin;
D. Zovirax;
E. Interferon.

30. Local treatment of acute herpetic stomatitis includes:
A. Irrigation of oral cavity with antiseptics;
B. Hygienic antiseptic rinsing of teeth;
C. Therapy of oxygenation;
D. All answers are correct;
E. All answers are incorrect.

31. Children with acute herpetic stomatitis is treated:
A. In an isolate cabinet;
B. In therapeutic cabinet, but with special instruments;
C. In therapeutic cabinet;
D. All answers are incorrect;
E. All answers are correct.

32. The most important antiepidemic measures in case of acute herpetic stomatitis in kindergarten are:

A. Disinfection of rooms;
B. Isolation and treatment of all children;
C. Determination of infection's source;
D. Disinfection of common things;
E. Determination of sources and ways of infection's transmission.

33. Child with acute herpetic stomatitis isn't contagious in following period:
A. After epithelization of all eruptions in oral cavity;
B. Over 5 days after epithelization of all eruptions in oral cavity;
C. After disappearance of gingivitis;
D. After disappearance of lymphadenitis;
E. All answers are correct.

34. Antivirotic therapy in period of eruptions has purpose:
A. To help epithelization of elements;
B. To avert appearance of some new eruptions;
C. To stimulate nonspecific reactivity of child's organism;
D. To remove gingivitis;
E. To decrease child's body temperature.

Multiple compartment

35. The ways of transmission of *Herpes Simplex* virus are following:
A. Respiratory;
B. Through contact;
C. Alimentary;
D. Trans-placental;
E. All answers are correct.

36. Multiplication of *Herpes Simplex* virus in prodromal period is:
A. In epithelial cells of oral mucosa;
B. In liver;

C. In muscular tissue;
D. In lymphatic ganglions;
E. In lien.

37. High receptivity of children for viral infections is explaining through:
A. Large spreading of *Herpes Simplex* virus in nature;
B. Immaturity of interferon formation function;
C. Physiologic immunodeficiency;
D. High virulence;
E. Allergization of organism.

38. Urgent antiepidemic measures of acute herpetic stomatitis and chronic recurrent herpetic stomatitis:
A. Individual hygiene of oral cavity;
B. Daily investigation of children in scope of precocious diagnostic and treatment;
C. Disinfection of rooms and common things;
D. Using of antivirotic unguents (3-4 time / day);
E. Isolation and treatment of ill children.

39. For acute herpetic stomatitis are characteristic following elements on the oral mucosa:
A. Macula;
B. Bulla;
C. Vesicle;
D. Papule;
E. Aphthe.

40. Eruptive elements of acute herpetic stomatitis are localized:
A. On the oral mucosa;
B. On the skin of face;
C. On the course of neuro-vascular fascicules;
D. On the fingers of hands;
E. On the mucosa of nose, eyes, genital organs.

41. Severity of acute herpetic stomatitis is established by following criteria:

A. Number of elements on the oral mucosa;
B. Number of recurrences;
C. Body temperature;
D. Localization of eruptions;
E. General state.

42. In case of fever (above 39°C) of 2 years aged child with acute herpetic stomatitis is prescribed:

A. Antibiotics;
B. Sulfanilamide;
C. Sol. 2% aspirin;
D. Interferon.

43. Antivirotic unguents for treatment of acute herpetic stomatitis or recurrent chronic herpetic stomatitis are:

A. Florenal 0,5%;
B. Tebrofen 0,25%;
C. Liniment gossypol 3%;
D. Liniment sintomycine 1%;
E. Riodoxol 0,25%.

Mycotic affections of oral mucosa of children
Simple compartment

1. *Candida* fungi are:

A. Conditional-pathogenic;
B. Pathogenic;
C. Saprophyte;
D. Indifferent.

2. Candidosis than result of disbacteriosis is conditioned by:

A. Longtime treatment with antibiotics;
B. Longtime treatment with keratoplastics;
C. Chronic traumas of oral mucosa;

D. Non-observation of oral hygiene;
E. Nutritional deficiencies.

3. Fungi are fixed on the oral mucosa I the first year of life owing to:
A. Existence of specific receptors on the oral mucosa;
B. Sticky polysaccharides produced by fungi;
C. Immaturity of children oral mucosa;
D. Metabolic acidosis in oral cavity;
E. Epithelial papillae of mucosa

4. Candidosis of oral cavity is:
A. Contagious disease;
B. Light contagious disease;
C. Non-contagious disease.

5. In 1 ml of buccal liquid in candidosis are determined fungi colonies in number of:
A. 150-180;
B. 800-1000;
C. 1000-1500;
D. 3500-13500;
E. 15- 25.

6. In anamnesis of severe candidosis may be established:
A. Allergy from antibiotics;
B. Exudative diathesis;
C. Allergy from antibiotics and artificial alimentation;
D. Longtime treatment with antibiotics and premature birth of child;
E. Allergy from some foodstuffs.

7. Of light form candidosis removing of deposits provokes:
A. Erosion;
B. Hyperemia without erosion;

C. Erosion associated with hemorrhage;
D. Ulceration;
E. Ulceration associated with hemorrhage.

8. Treatment of acute candidosis of medium degree of oral mucosa with antimycotic medicaments lasts:
A. 7-10 days;
B. 10-14 days;
C. 14-21 days;
D. 21-30 days;
E. Not as much of 1 month.

9. For discontinue multiplication of fungi is important to create in oral cavity an environment:
A. Acid;
B. Alkaline;
C. Neutral.

10. For creation of one environment which stops multiplication of fungi in oral cavity are indicate:
A. Solution 0,5-1,0 % oxygenate water;
B. Solution 0,5% chloramines;
C. Solution 2% sodium bicarbonate;
D. Solution 0,5% citric acid;
E. Solution 1% Iodinol.

11. Daily dose of Nistatine for child over 1 year with candidosis constitute (for 1 kg body weight):
A. 10 000 UA;
B. 25 000 UA;
C. 50 000 UA;
D. 75 000 UA;
E. 100 000 UA.

12. Daily dose of Nistatine for child less 1 year with candidosis

constitute (for 1 kg body weight):

A. 25 000 – 50 000 UA;
B. 50 000 – 75 000 UA;
C. 100 000 – 150 000 UA;
D. 250 000 – 300 000 UA;
E. 300 000 – 350 000 UA.

13. After disappearance of symptoms of acute candidosis treatment must continue:

A. 2 – 3 days;
B. 5 – 7 days;
C. 7 – 10 days;
D. 14 – 21 days;
E. 21 – 30 days.

Multiple compartment

14. Penetration of *Candida* fungi in oral cavity of children is on the following ways:

A. Respiratory;
B. Through contact;
C. Of habitual things;
D. Intravenous infusion;
E. Through genital ways.

15. Development of candidosis of suckling is favorable in case of:

A. Decreased level of local immunity;
B. Traumas of oral mucosa;
C. Non-observation of child's hygiene;
D. Metabolism disorders of hydrocarbonates;
E. Sickly organism of newborn.

16. For diagnostic of candidosis are used following methods:

A. Cytological;
B. Immunological;
C. Bacterioscopy;

D. Immune-fluorescence;
E. Microscopically.

17. For acute candidosis of children are characteristics:
A. Fever;
B. Appearance of rounded erosions;
C. Appearance of some pellicles and white deposits;
D. Appearance of some white dotted deposits on the intact oral mucosa or hyperemia;
E. Gingival hyperemia and edema.

18. For treatment of acute candidosis apply following unguents:
A. Bonafton;
B. Alpizarin;
C. Decamin;
D. Canesten;
E. Levosin.

19. For general treatment of oral candidosis are indicated:
A. Iodide preparations;
B. Corticosteroids;
C. Antimycotics;
D. Antipyretics;
E. Antivirotics.

Simple joining

20. Diagnosis and laboratory results:
1) Acute candidosis.
2) Chronic candidosis.

A. Unitary cellular forms of fungi-form cells.
B. Fungi-form cells and pseudo-mycelium, with predomination of young forms.
C. Fungi-form cells and pseudo-mycelium, with predomination of pseudo-mycelium.

Polymorph exudative erythema

Simple compartment

1. Polymorph exudative erythema recurs:
   A. Sure;
   B. No.

2. Polymorph exudative erythema has a season character:
   A. Sure;
   B. No.

3. After polymorph exudative erythema appears stable immunity:
   A. Sure;
   B. No.

4. Nikolsky sign of polymorph exudative erythema is:
   A. Positive;
   B. Negative.

5. Recurrent chronic stomatitis of majority patients is caused by:
   A. Decreased protection of oral mucosa;
   B. Longtime treatment with antibiotics and corticosteroids;
   C. Microbial allergy;
   D. Chronic herpetic infection.

6. For Setton form are characteristic:
   A. Vesicles;
   B. Erosions;
   C. Superficial cicatrices;
   D. Maculae;
   E. Pustule.

Multiple compartment

7. Polymorph exudative erythema is of following nature:
   A. Viral;
   B. Manifestations of infectious allergy;

C. Manifestations of non-infectious allergy;
D. Bacterial;
E. Mycotic.

8. The polymorph exudative erythema appears of:
A. Preschool children;
B. School children;
C. Adolescents;
D. Anti-preschool children;
E. Less than1 year.

9. For polymorph exudative erythema are characteristic following symptoms:
A. Red-violaceous papule on the skin;
B. Vesicles on the red margin of lips;
C. Hemorrhagic crusts on the lips;
D. Erosions and hyperemia on the mucosa;
E. Bulla.

10. Typical localization of papule on the polymorph exudative erythema:
A. Sub-axillaries and inguinal regions;
B. Forearms and palms;
C. In region of face;
D. In region of neck;
E. In region of shanks.

11. Severe form of polymorph exudative erythema has following symptoms:
A. Eruptions on the skin of breast, palms, insteps and anogenital region;
B. Eruptions on the skin with typical localization – forearms, palms, shanks;
C. Bronchopneumonia, arthralgia;

D. Eyes affections;
E. Ears affections

12. Local treatment of polymorph exudative erythema is made with:
A. Antibiotics;
B. Keratoplastics;
C. Antiseptics;
D. Antivirotics;
E. Anesthetics;
F. Antihistamines;
G. Unguent with corticosteroids and antibiotics.

13. For treatment of oral mucosa in case of polymorph exudative erythema in habitual conditions are recommended:
A. Solution 2% sodium bicarbonate;
B. Tea of chamomile, eucalypt;
C. Iodine;
D. Emulsion of anesthesine;
E. Unguent of nistatine.

14. For Behçet syndrome is characteristic:
A. Periodic appearance of aphthae;
B. Typical localization eruptions;
C. Aphthae with profound cicatrisation, which provoke deformation of mucosa;
D. Painful aphthae;
E. Eyes affections;
F. Appearance of nodular recurrent erythema and elements of pyodermitis;
G. Appearance in place of injection of inflammatory infiltrate with central necrosis;
H. Aggravation of all symptoms in time of recurrence.

Simple joining

15. Allergens and groups of allergens:
    1) Habitual.
    2) Of insects.
    3) Epidermal.
    4) Medicamental.
    5) Of pollen.
    6) Alimentary.
    7) Industrial.
    8) Infections.

    A. Penicillin, Novocain, iodine.
    B. Dandruff, wool, pens, scale.
    C. Pollen de wormwood, herbs.
    D. Fish, wheat, beans, tomatoes.
    E. Oils, chromium, formalin.
    F. Pathogen agent of tuberculosis, leprosy, syphilis, etc.
    G. Powder of clothes, mushrooms, ticks, etc.
    H. Saliva, secretions, powder of insects.

16. Forms of polymorph exudative erythema measures of recurrence prevention:
    1) Infectious-allergic.
    2) Toxic-allergic.

    A. Discontinue introduction of medicaments which provoke allergy.
    B. Avoidance of super-cooling of organism and contact with infectious patients.
    C. Assanation of oral cavity.
    D. Removing of infectious nidus in organism.
    E. Specific hypo-sensibilities with bacterial allergen.
    F. Active immunization with staphylococcal anatoxin.

17. Severity and frequency of polymorph exudative erythema

recurrences:

1) Light.
2) Medium.
3) Severe.

A. 1-2-3- time / year.
B. More frequent of 4 time / year.
C. One time in some years.
D. In each month or permanent.

Cheilitis and glossitis of children
Simple compartment

1. The main etiologic factor of actinic cheilitis is:
   A. Infectious-allergic;
   B. Action of ultraviolet rays in cases of high sensitivity to sunstroke;
   C. Action of non-favorable meteorological factors;
   D. Labial architectonic disorders;
   E. Fungi.

2. Exudative diathesis of eczematous and atopic cheilitis is present in anamnesis:
   A. Sure;
   B. No.

3. Neurogenic mechanisms of cheilitis:
   A. Glandular;
   B. Exfoliative;
   C. Meteorological;
   D. Actinic;
   E. Angular.

4. In treatment of actinic cheilitis the main role has:
   A. Corticosteroid unguents;
   B. Unguents with antibiotics;

C. Restoration of labial architectonic;
D. Specific and nonspecific desensitization;
E. Creams for photo-protection.

5. In case of glandular cheilitis is prescribed:
A. Unguents with antibiotics;
B. Restoration of labial architectonic;
C. Specific and nonspecific desensitization;
D. Surgical removing of glands with hyperplasia or their coagulation;
E. Unguents with corticosteroids.

6. If angular cheilitis is accomplished with strepto-staphylodermitis appears:
A. Yellow-dark crusts;
B. Moist white deposits;
C. Erosions with purulent eliminations;
D. Vesicles with hemorrhagic content;
E. Massive crusts of red-brown color.

Multiple compartment

7. In appearance of meteorological cheilitis the main importances have:
A. Constitutional particularities;
B. Vicious habits;
C. Action of non-favorable meteorological factors;
D. Labial architectonic disorders;
E. Infectious-allergic factor.

8. Allergic reaction has a main role in pathogenesis of:
A. Glandular cheilitis;
B. Exfoliative cheilitis;
C. Meteorological cheilitis;
D. Atopic cheilitis;
E. Eczematous cheilitis.

9. Affection of red margin of lips, angular region and of skin in perioral region is characteristic for:
   A. Glandular cheilitis;
   B. Atopic cheilitis;
   C. Eczematous cheilitis;
   D. Exfoliative cheilitis;
   E. Actinic cheilitis.

10. In treatment of meteorological cheilitis the main role has:
   A. Unguents with antibiotics;
   B. Unguents with corticosteroids;
   C. Restoration of labial architectonic;
   D. Abolition of vicious habits;
   E. Specific and nonspecific desensitization.

11. Congenital anomalies of tongue are following:
   A. Catarrhal glossitis;
   B. Desquamative glossitis;
   C. Plicate (fissured) glossitis;
   D. Black hairy tongue;
   E. Rhombic glossitis.

12. Acute catarrhal glossitis develops after action of following factors:
   A. Physical;
   B. Infectious;
   C. Neuro-dystrophic;
   D. Exudative diathesis;
   E. Hormonal disorders.

13. Treatment of catarrhal glossitis includes:
   A. Assanation of oral cavity;
   B. Mouth-rinsing with antiseptics;
   C. Cryotherapy;
   D. Professional hygiene of oral cavity;

E. Unguents with corticosteroids.

14. Treatment of black hairy tongue includes:
    A. Hygiene of oral cavity;
    B. Assanation of oral cavity;
    C. Unguents with corticosteroids;
    D. Keratolytics;
    E. Keratoplastics.

15. Treatment of papillose rhombic glossitis includes:
    A. Consultation of surgeon;
    B. Dispensary control;
    C. Vitamin-therapy;
    D. Cryotherapy.

Correct answers:

Acute inflammatory affections of oral mucosa of children
Manifestations in acute infectious diseases

1. B.
2. A.
3. B.
4. C.
5. A.
6. B.
7. B, D, F, G.
8. A, C, E.
9. A, B.
10. A, C.

Traumatic affections of oral cavity mucosa

1. E.
2. C.
3. A.
4. C.
5. A, B, C, E.
6. A, B, C, E.
7. A, B, C, E.
9. A, B, D.
10. B, D, E.
11. A, B, C.
12. A, B, C, E.
13. B, C, D, E.
14. B, C, D, F.
15. B, F.

8. A, B, E.

16. C, D.
17. B, C, D.

Acute herpetic stomatitis

1. A.
2. A.
3. A.
4. A.
5. C.
6. E.
7. B.
8. E.
9. B.
10. C.
11. D.
12. E.
13. D.
14. B.
15. B.
16. D.
17. C.
18. C.
19. C.
20. D.
21. D.
22. A.
23. D.
24. A.
25. A.
26. A.
27. C.
28. A.
29. C.
30. B.
31. A.
32. B.
33. E.
34. B.
35. A, B, D.
36. B, D, E.
37. B, C.
38. B, C, D, E.
39. A, C, E.
40. A, B, D, E.
41. A, C, D, E.
42. C, D.
43. A, B, C, E.

Mycotic affections of oral mucosa of children

1. A.
2. A.
3. C.
11. C.
12. C.
13. C.

4. A.
5. D.
6. D.
7. B.
8. C.
9. B.
10. C.
14. B, C, E.
15. A, C, E.
16. A, C, E.
17. C, D.
18. C, D.
19. A, C.
20. 1B, 2C.

Polymorph exudative erythema

1. A.
2. A.
3. B.
4. B.
5. C.
6. C.
7. B, C.
8. B, C.
9. A, C, D.
10. B, E.
11. A, C, D.
12. B, C, E, G.
13. B, D.
14. C, E, F, G, H.
15. 1G, 2H, 3B, 4A, 5C, 6D, 7E, 8F.
16. 1BCDEF, 2A.
17. 1C, 2A, 3BD.

Cheilitis and glossitis of children

1. B.
2. A.
3. B.
4. E.
5. D.
6. A.
7. A, C, D.
8. D, E.
9. B, C.
10. C, D.
11. C, E.
12. A, B.
13. A, B, D.
14. A, B, D.
15. A, B, D.

11 DISPENSARY CONTROL OF CHILDREN

Dispensary control is an activity complex system of curative-prophylactic organizations that ensure prophylaxis, precocious traced and treatment of diseases what suppose an active and systematic evidence of children.

The individual assessment of children's health foresee theirs distribution in following groups:

1. Healthy child.
2. Healthy child with functional and morphological deviations or child with acute diseases (more of 4 time in the year) with high risk to develop of chronic diseases; deviation in child's development without endocrine pathologies.
3. Unwell child with chronic diseases and malformations in the stage of compensation.
4. Unwell child with chronic diseases and malformations in the stage of sub compensation.
5. Unwell child with chronic diseases and malformations in the stage of decompensation.

Dispensary control of children must to effect on stages, in function of different factors: epidemiological situation, level of stomatological assistance (means of insurance with medical personal, units, instrumentations, medications, etc.).

Application in practice of dispensary control relies, in the first, on the circumscription principle and is organized on following stages.

First stage: ***of registering***

Stomatological assistance is organized in one concrete sector (in children's collectivities: crèches, kindergarten, schools, gymnasiums, lyceums, etc.); establish relations with leadership of children's collectivity and with sanitary-epidemiological assistance for organization of stomatological assistance of children; gather the data of fluoride concentration in the drinking water; appreciate the epidemiological situation – in base of precedent data of children's oral health examination; conclude necessary medical documentation: children's register in dependence of age, medical history of each child, etc.

Second stage: ***examination and primary assanation***

Stomatological examination of children and primary assanation of buccal cavity are organized. Obtained data after the examination and assanation of buccal cavity lie at the basis of formation of children dispensary groups and make the graphic plan of doctor's activity for later years.

Dispensary groups of 0-3 aged children

1. Healthy children without stomatological pathology (are examined one time on year);
2. Healthy children, but with high risk to develop of stomatological diseases (are examined two time on year);
3. Children with stomatological diseases: caries and its complications, developmental anomalies of teeth, maxillaries, face; tumors and traumas of maxillo-facial region, that has suffered of osteomyelitis of facial cranium etc. (are examined three time on year).

Dispensary groups of 4-6 aged children

1. a) Healthy children (groups of health 1, 2, 3) with intact buccal cavity;

b) Children with risk to develop of stomatological diseases;

c) Healthy children that have I degree of caries activity (compensate stage), gingivitis bring about local factors.

Children at I group are examined one time on year.

2. a)Healthy children (groups of health 1, 2, 3) with II degree of caries activity (subcompensate stage); with localized gingivitis bring about number, position and occlusion anomalies, etc.;

b) Children (groups of health 4 and 5) with healthy buccal cavity or risk to develop of stomatological diseases, or with I degree of caries activity, with gingivitis bring about local factors;

c) Children (all groups of health) with chronic inflammatory diseases of maxillo-facial region, of salivary glands, with benign tumors.

Children at II group are examined two times on year.

3. a) Children with III degree of caries activity (forma decompensate);

b) Children (groups of health 4 and 5) with II degree of caries activity and gingivitis bring about dental-maxillary anomalies;

c) Children with developmental anomalies of teeth's structure;

d) Children with generalized gingivitis and periodontitis, with periodontal syndromes of somatic diseases etc;

e) Children with recidivated aphthe of oral mucosa;

f) Children with dental-maxillary anomalies, that require treatment with orthodontic systems;

g) Children that had tumors removed operation.

Children at III group are examined three times on year.

Dispensary groups of 7-18 aged children

1. a) Healthy children (groups of health 1, 2) with intact buccal cavity;

b) Healthy children with I degree of caries activity;

c) Healthy children with gingivitis bring about insufficient oral hygiene, absence of some teeth functioning, non qualitative

fillings and other local factors;

d) Healthy children with developmental anomalies of: labial, lingual frenum, small vestibule of buccal cavity etc.;

e) Healthy children with posttraumatic state of maxillo-facial region, without the cases of teeth with undeveloped root traumatism.

Children at I group are examined one time on year.

2. a) Children with chronic diseases of interne organs (groups of health 3-5), that don't have dental diseases, periodontal diseases, dental-maxillary anomalies, etc.

b) Healthy children with II degree of caries activity;

c) Healthy children with gingivitis bring about dental-maxillary anomalies that require treatment with orthodontic systems;

d) Children that have treated teeth with caries complication (in rehabilitation period);

e) Children that endured of inflammatory processes of maxillo-facial region (osteomyelitis, odontogenous lymphadenitis etc.); supernumerary teeth extraction; benign tumors removed operation;

f) Children that are of orthodontic treatment.

Children at II group are examined two times on year.

3. a) Children with chronic diseases of interne organs (groups of health 2-5), and with II and III degrees of caries activity;

b) Healthy children with III degree of caries activity;

c) Healthy children with demineralization and incipient forms of dental caries;

d) Healthy children with generalized or localized periodontitis;

e) Healthy children with periodontal diseases for syndrome of interne organs diseases (periodontal syndrome);

f) Healthy children that endured teeth with undeveloped roots trauma;

g) Children with factors for develop of dental-maxillary anomalies, that influence actively (disturb of deglutition, respiration, mastication, diction, vicious habits etc.);

h) Children in retention period after orthodontic treatment;

i) Children that are found at the complex treatment of grave stomatological diseases: the II and III degrees of caries activity, diseases of marginal periodontium, anomalies of occlusion, those who require the surgical correction of dental-maxillary anomalies, etc.;

j) Children that are found at evidence in Institute of Oncology.

Children at III group are examined three times on year.

Thirty stage: *implementation of the dispensary plan*

Implementation of the dispensary plan: organization of the examination and assanation of buccal cavity and application of the prophylactic methods at the children's groups level conformable with the dispensary terms (the dispensary group).

After the primary examination and assanation of children is recommended to begin the secondary assanation and application of prophylactic methods to children of III dispensary group, then those to II group, preschools, but then children to I dispensary group. This succession allow realizing more efficient the dispensary plan and to include maximum children in the dispensary control. At the end of year each doctor makes the graphic plan of work on the next year. The doctor, in accordance to special formulas, on the basis of current year dates can determine volume of work for the next year.

Graphic plan of activity on _____ year
Of stomatolog _____
City / Village_____ Sector _____
School / Lyceum / Kindergarten nr._____

Month	Day 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
September	
October	
November	in Kindergarten
December	
January	in Kindergarten
February	
March	in Kindergarten
April	
Mai	
June	in Kindergarten
July	in Kindergarten
August	holiday

Implementation of the dispensary plan can to last more years in function of incidence and prevalence of stomatological diseases, of de financial resources etc. If on a sector the prevalence and incidence of caries and other diseases are high then the good results are to be expected after 3 or 4 years of assanation and prophylaxis.

On this stage, if the child's health is improved then child is transferred in other dispensary group. The main is to respect the intervals of secondary assanation and prophylaxis in function of dispensary group.

Work's efficiency on this stage is appreciating of stabilization and afterwards decreases of incidence and prevalence of main stomatological diseases and its complications, etc.

Fourth stage: ***prophylaxis in dispensary system***

Prophylaxis in dispensary system is the main point of work. On the base of positive results of dispensary plan realization decrease the number of children with complicated caries, with periodontal diseases, etc. Thus the time for treatment decrease and time for prophylaxis increase in each dispensary group.

Estimate of caries prophylaxis program efficiency is performing in accordance to following indices:

1. Number of children with healthy teeth (estimate efficiency of primary caries prophylaxis).

2. Caries stabilization – with decrease of:

a) Caries ratio;

b) Frequency of recidivate caries (secondary caries);

c) Frequency of complicated caries;

d) Number of missing permanent teeth;

e) Frequency of children that require the treatment;

f) Frequency of children with III degree of caries activity.

3. Number of children with complicated caries, with chronic odontogenous nidus and organism allergization.

Implementation of prophylaxis program of main stomatological diseases is the most efficient then is making the dispensary control of children. At the start is adopting the community prophylaxis and next the individual prophylaxis.

Fifth stage: ***individual prophylaxis, rehabilitation in dispensary system***

Individual Prophylaxis, rehabilitation in dispensary system have the aim to perfect, to increase at the superior qualitative level the dispensary system and efficiency of prophylaxis program of the main stomatological diseases of children.In base of individual factors of risk for development of stomatological diseases for each child is planning and applying individual prophylactic methods.

Criterions of dispensary control efficiency:

1. Number of healthy children.
2. Number (percentage) of children that require the treatment.
3. Ratio of number of treated children at children that require the treatment (in percent).
4. Number of complicated caries at 1000 children.
5. Number of missing teeth at 1000 children.
6. Rate of caries.

7. Number of fillings at one child.
8. Number (percentage) of children with insufficient oral hygiene.
9. Number (percentage) of children to dispensary control.

One of the dispensary control efficiency criterion is the rate of children comprised of dispensary control and it is appreciate of following formula:

$$\frac{\text{Number of treated children + Number of children that not required treatment (healthy; treated previously)}}{\text{Number of children on sector}} \times 100\%$$

This index must be nearly to 100%.

Organization of children oral cavity assanation

The organization forms of oral cavity assanation are following:

1. Individual – child appeal to doctor without assistance or with assistance (parents, relatives, etc.) for treatment of dental and periodontal diseases, etc.

2. Periodical or one time organized assanation – revelation and thoroughly treatment of stomatological diseases in temporary children collectivities (hospital, holiday camp, etc.)

3. Pland curative-prophylactic assanation – one of principal component of stomatological dispensary system of children – is making over certain intervals of time in concordance with dispensary groups.

Organization methods of oral cavity assanation

Two organization methods of oral cavity assanation of children are: centralized and decentralized.

Centralized method foresees stomatological examination and assanation in medical department (policlinics of quarter, municipal policlinics etc. for children). Children in the group

accompanied of pedagogues go at the stomatological department. In policlinic is possibly multilateral examination and perfect and qualitative assistance of children from stomatologist and other specialists: orthodontist, surgeon, and radiologist. This method has some negative aspects: 1) stoppage of education process; 2) children avoid visits to stomatologist; 3) because of high number of children the quality of medical assistance decreases; etc. Through these reasons the centralized method can be efficiently then the policlinic has 5-6 doctors for qualitative stomatological assistance.

Decentralized method consists in assanation of children in stomatological consulting room organized in kindergartens, schools, lyceums, etc. Stomatological consulting room in children's collectivities can be placed temporary or permanent. If the number of pupils is more 1000 the stomatological consulting room functions every day throughout the year. If the number of pupils is smaller the doctor works parallel by twos schools by 2-3 times on the week in every school.

One variant of decentralized method is the method of group used in case of stationary stomatological consulting room absence in children collectivities. More profitable is the group from 2-3 doctors that work 1-3 months in one school. Treatment of children is doing in the mobile stomatological consulting room. With this method the rate of treated children increase, but quality of treatment decreases. Stomatological assanation must be realized planed, successively, systematically and perfect.

Plan of doctor's activity for next year is formed depending of stomatological diseases prevalence and incidence, efficiency of curative-prophylactic activity in precedent years, etc. This plan is approved of territorial stomatological and educational department.

Systematically assanation foresee regular realization of children's examination and assanation over certain intervals of time: for I degree of activity (compensate stage) – 13 months; for

II degree of activity (subcompensate stage) – 7 months; and for III degree of activity (decompensate stage) – 3,3 months. In period between examinations incipient forms of caries don't must to progress taking to complication. Periodicity of examination and curative-prophylactic method depend on the state of buccal cavity and organism individually of each child.

Assanation of buccal cavity must be perfect: all affected teeth are treated; put out the teeth after indication; are treated periodontal and oral mucosa diseases; start the orthodontic treatment of dental and occlusal anomalies.

Stomatological policlinic for children

Stomatological policlinic for children is a curative-prophylactic department what assure the diagnostic, treatment and prophylaxis of stomatological diseases of children.

The main obligations of stomatological policlinic for children are following:

a) prophylaxis of oro – maxillo - facial area diseases of children;
b) to trace the patients with oro – maxillo - facial pathologies;
c) qualified stomatological assistance.

Stomatological policlinic for children:

- makes the planed assanation of buccal cavity of children at collectivities from its region;
- makes stomatological assistance for appealing children;
- takes evidence and makes analyses of stomatological morbidity of children;
- appreciate the efficiency of prophylactic and curative methods applied to children;
- organize the sanitary education children and their parents in problems of primary and secondary prophylaxis of main stomatological diseases.

Evidence and report documentation of stomatological assistance for children

The stomatologist in policlinic is obligated to fulfill following evidence documentation:

1. Medical history of stomatological patient (f. 43).
2. Journal of evidence of stomatologist activity (f. 037/u).
3. Register of examination and assanation of patient at the dispensary control (f. 048/u).
4. Journal of evidence of stomatologist activity (f. 039/u).
5. Patient history of dispensary control (f. 030).

In the patient history of dispensary control (f. 030) is indicated effectuated treatment and time of next visit.

In children collectivities is reasonable to mark the medical history (f. 043) in function of dispensary groups and terms foresee of dispensary groups.

Recording of curative-prophylactic activity makes in passport of kindergarten or school. In passport is recording the number of examined and treated children; number of children that required the treatment; number of children involved prophylaxis.

In base of annual statistic analyze of obtained results is making the activity plane of stomatologist for next year.

Control questions and topics:

1. Definition of dispensary control.
2. What are the groups of health at children?
3. Describe the first stage of dispensary control of stomatologist.
4. Describe the second stage of dispensary control of stomatologist.
5. Stomatological dispensary groups of children aged 0-3 years.
6. Stomatological dispensary groups of children aged 4 - 6 years.
7. Stomatological dispensary groups of children aged 7-18 years.
8. How is realized the dispensary control plane?

9. What is prophylaxis in dispensary system?
10. Describe the individual prophylaxis and rehabilitation in dispensary system.
11. What are the efficiency criterions of dispensary control?
12. Name the forms of organization of buccal cavity assanation.
13. Methods of organization of buccal cavity assanation.
14. Describe the main obligation of the stomatological policlinic for children.
15. What is the evidence and report documentation in the stomatological policlinic for children?

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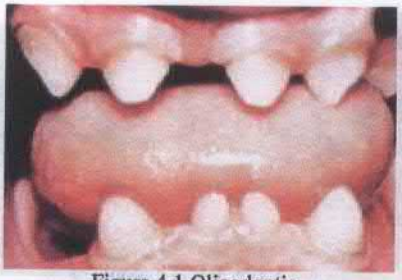

Figure 4.1 Oligodontia

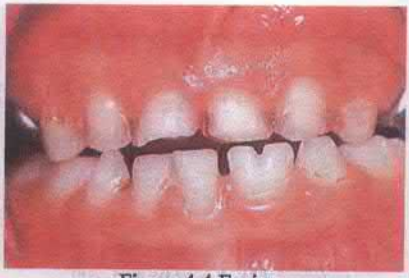

Figure 4.4 Fusion

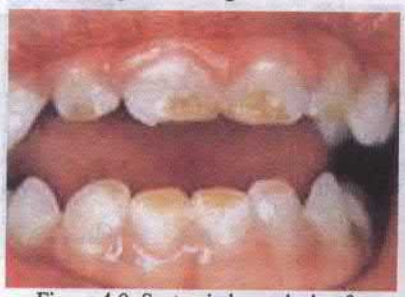

Figure 4.9. Systemic hypoplasia of temporary teeth complicated by caries.

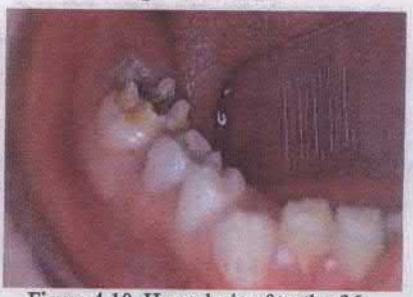

Figure 4.10. Hypoplasia of teeth of 6 years and incisors.

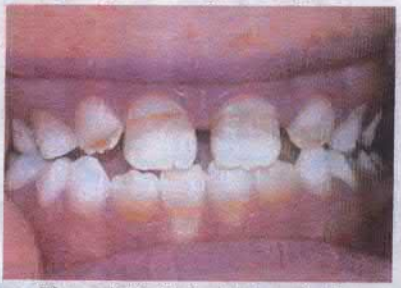

Figure 4.10a Systemic hypoplasia.

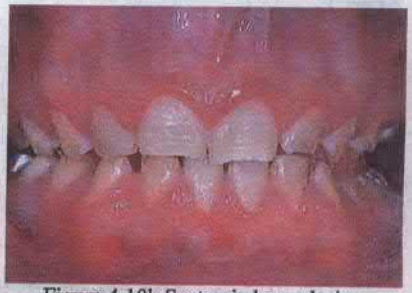

Figure 4.10b Systemic hypoplasia.

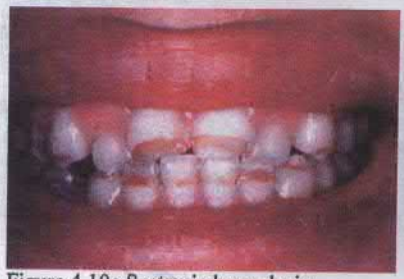

Figure 4.10c Systemic hypoplasia.

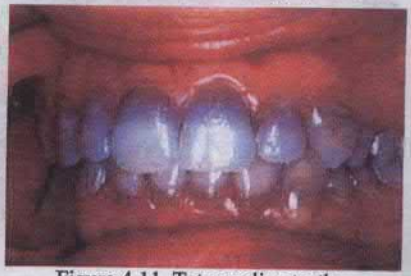

Figure 4.11. Tetracycline teeth.

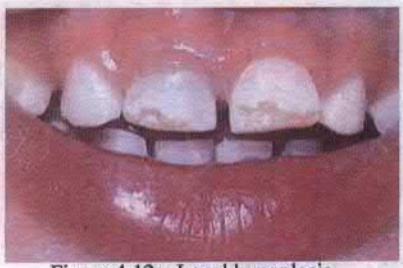

Figure 4.12a. Local hypoplasia.

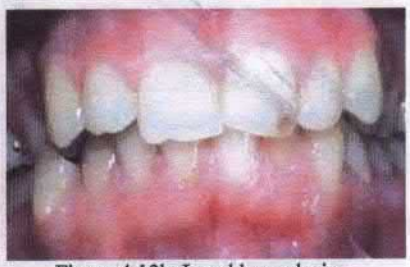

Figure 4.12b. Local hypoplasia.

Figure 4.13. Turner tooth.

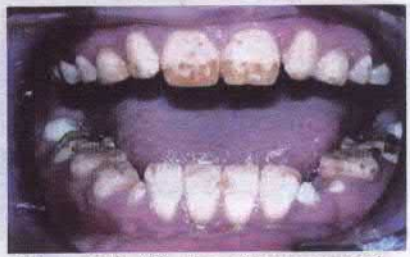

Figure 4.16. Affection of temporary and permanent teeth by fluorosis, degree V.

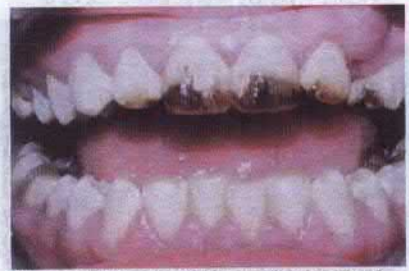

Figure 4.17a. Dental fluorosis, degree V.

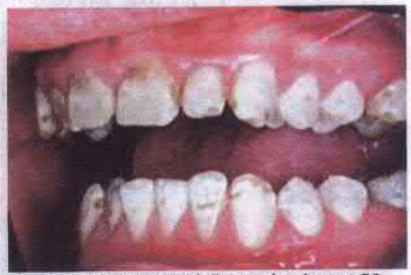

Figure 4.17b. Dental fluorosis, degree V.

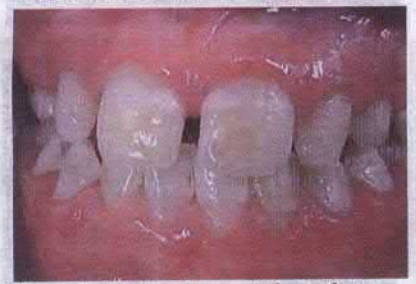

Figure 4.18a. Amelogenesis imperfecta.

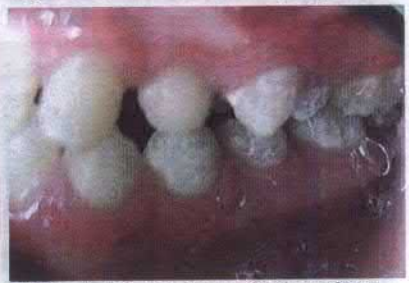

Figure 4.18b. Amelogenesis imperfecta.

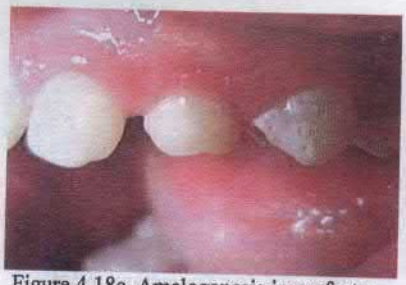

Figure 4.18c. Amelogenesis imperfecta.

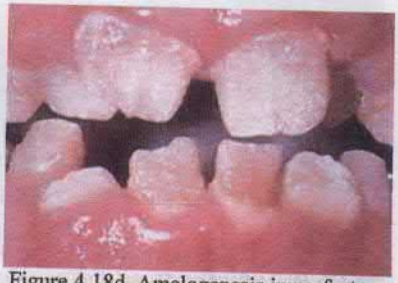

Figure 4.18d. Amelogenesis imperfecta.

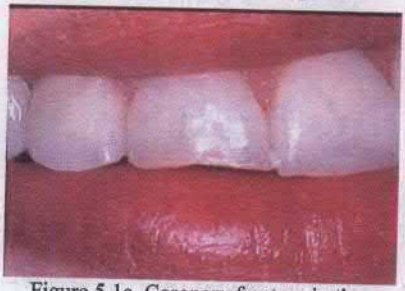

Figure 5.1a. Coronary fracture in the limits of dentine. Before restoration.

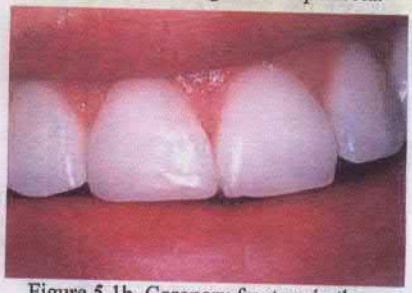

Figure 5.1b. Coronary fracture in the limits of dentine. After restoration.

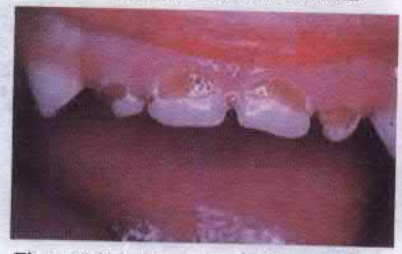

Figure 6.3. Bottle caries in the cervical region of temporary incisors.

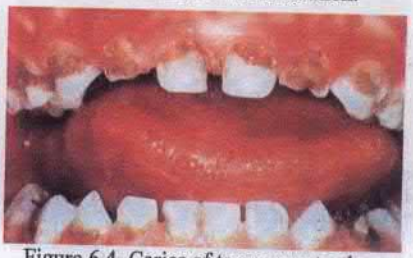

Figure 6.4. Caries of temporary teeth, activity degree III.

Figure 6.10a. Caries in the fissure of first permanent molar, appearance after processing of fissures.

Figure 6.10b. Acid engraving.

Figure 6.10c. Appearance after drying of cavity.

Figure 6.10d. Obturation of first permanent molar with glass ionomer.

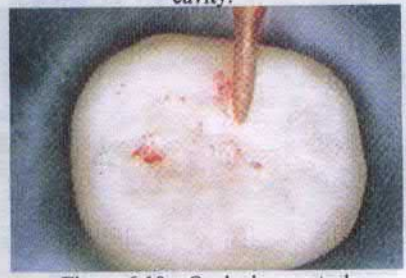

Figure 6.10e. Occlusion control.

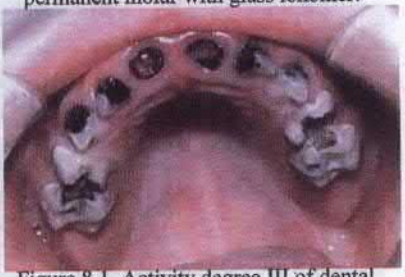

Figure 8.1. Activity degree III of dental caries.

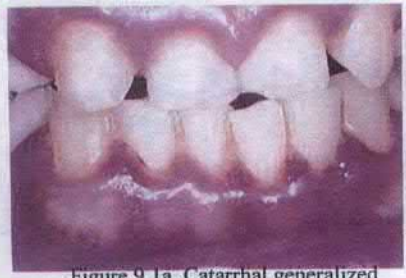

Figure 9.1a. Catarrhal generalized gingivitis.

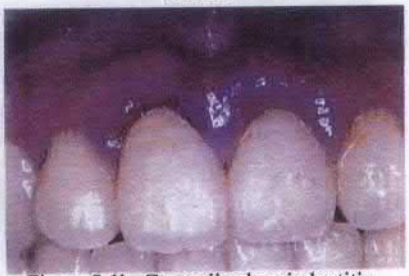

Figure 9.1b. Generalized periodontitis, medium degree.

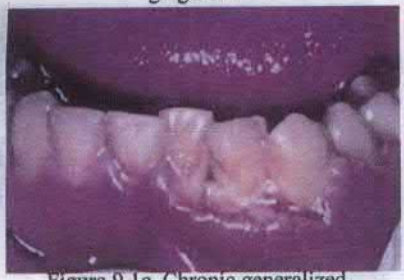

Figure 9.1c. Chronic generalized periodontitis in sugar diabetes.

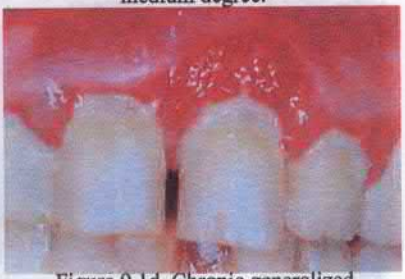

Figure 9.1d. Chronic generalized periodontitis in sugar diabetes.

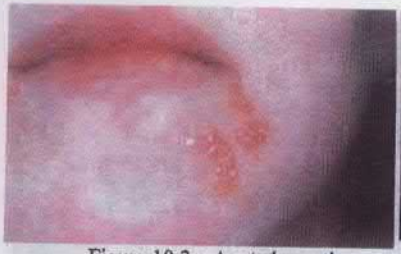

Figure 10.3a. Acute herpetic stomatitis, medium degree.

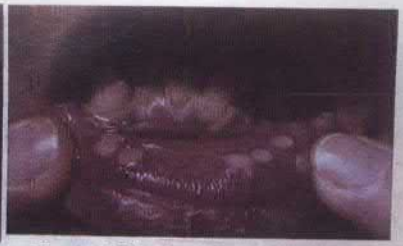

Figure 10.3b. Acute herpetic stomatitis, medium degree.

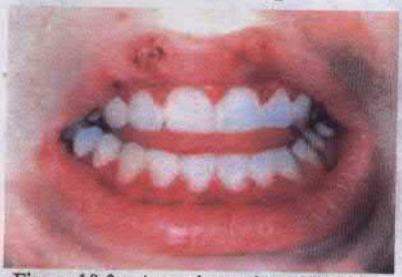

Figure 10.3c. Acute herpetic stomatitis.

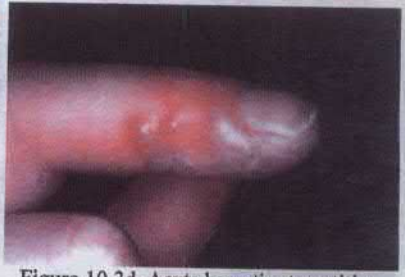

Figure 10.3d. Acute herpetic stomatitis

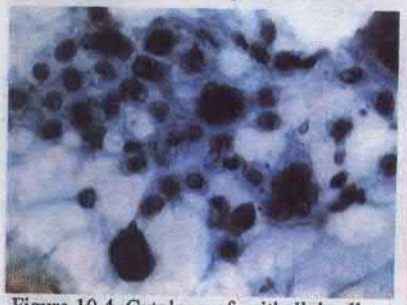

Figure 10.4. Cytology of epithelial cells in acute herpetic stomatitis.

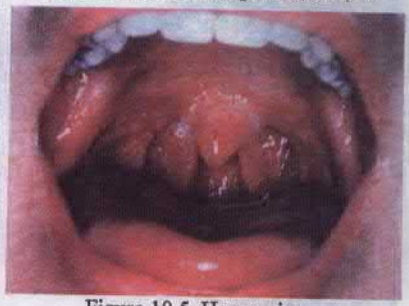

Figure 10.5. Herpangine.

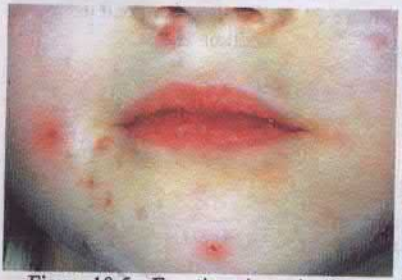

Figure 10.6a. Eruptions in varicella.

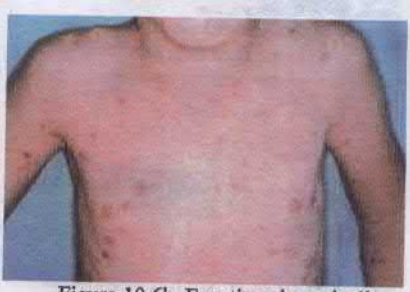

Figure 10.6b. Eruptions in varicella.

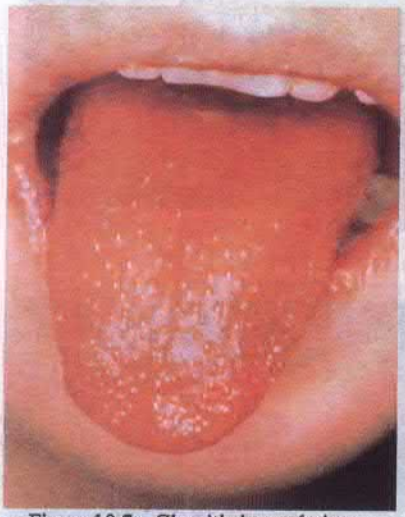

Figure 10.7a. Glossitis in scarlatina.

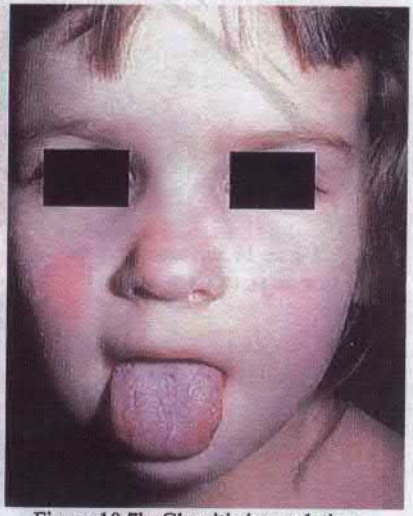

Figure 10.7b. Glossitis in scarlatina.

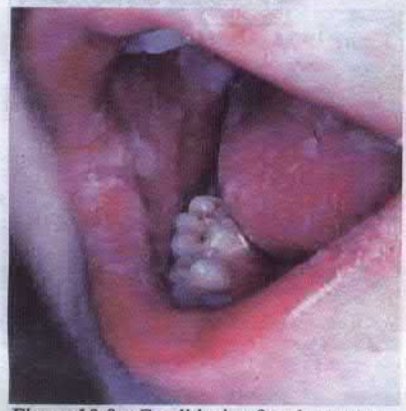

Figure 10.8a. Candidosis of oral mucous, medium degree.

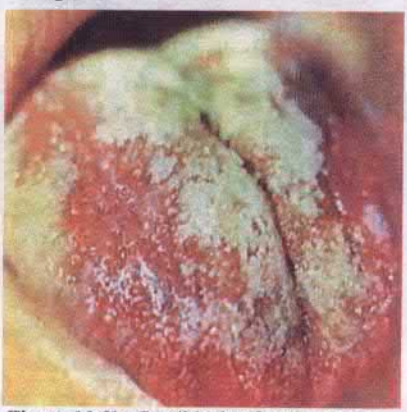

Figure 10.8b. Candidosis of oral mucous, medium degree.

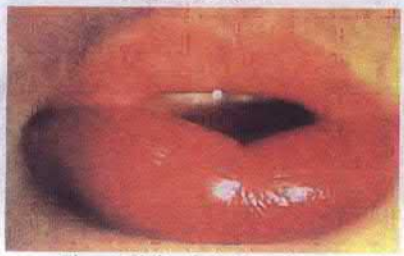

Figure 10.9a. Quincke edema.

Figure 10.9b. Quincke edema.

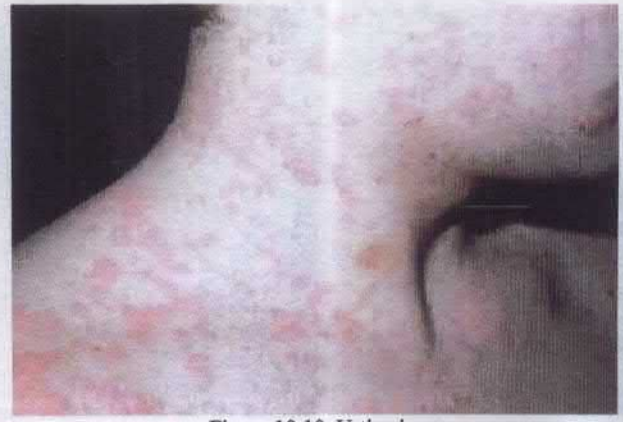

Figure 10.10. Urticaria.

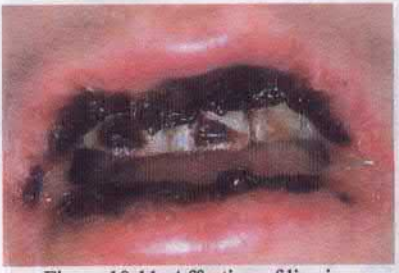

Figure 10.11. Affection of lips in exudative erythema.

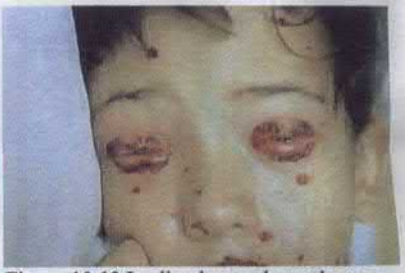

Figure 10.13 Lyell polymorph syndromes.

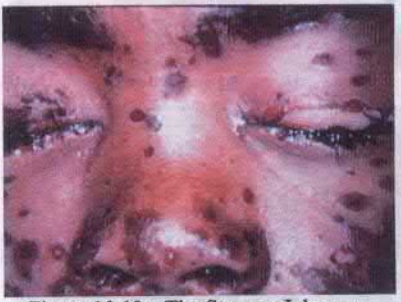

Figure 10.12a. The Stevens-Johnson syndrome.

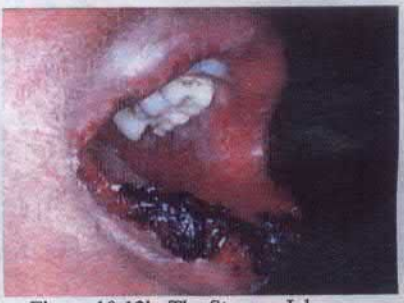

Figure 10.12b. The Stevens-Johnson syndrome.

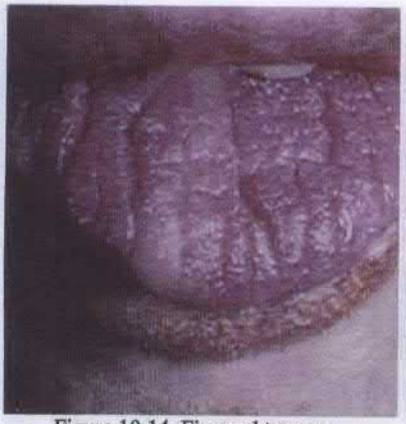

Figure 10.14. Fissured tongue.

Figure 10.15a. Desquamative tongue.

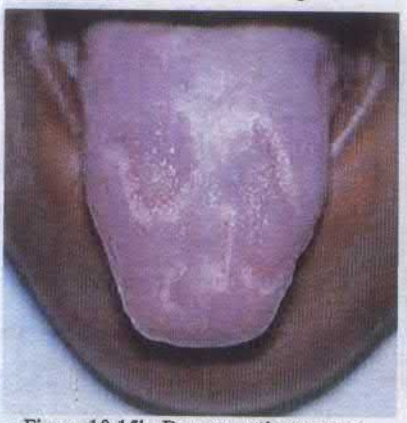

Figure 10.15b. Desquamative tongue

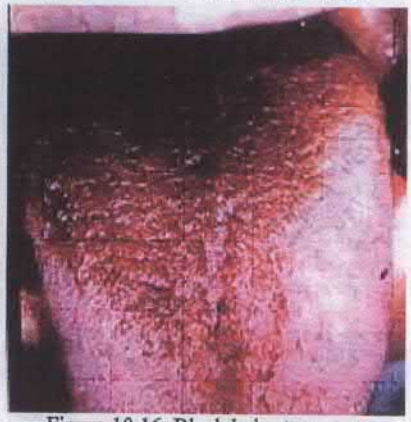

Figure 10.16. Black hairy tongue.

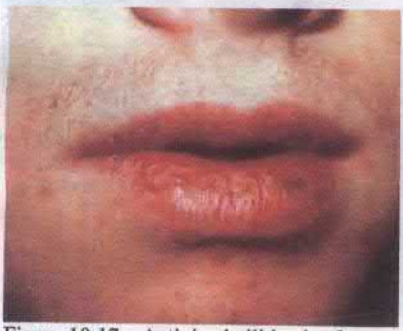

Figure 10.17a. Actinic cheilitis, dry form.

Figure 10.17b. Exfoliative cheilitis, dry form.