MINISTRY OF HEALTH OF THE REPUBLIC OF MOLDOVA
„NICOLAE TESTEMITANU”
STATE UNIVERSITY OF MEDICINE AND PHARMACY

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PRACTICAL COURSE OF PEDIATRIC ORAL AND MAXILLOFACIAL SURGERY

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MINISTRY OF HEALTH OF THE REPUBLIC OF MOLDOVA
„NICOLAE TESTEMITANU"
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FACULTY OF STOMATOLOGY
„ION LUPAN" DEPARTMENT OF PEDIATRIC ORAL&MAXILLOFACIAL SURGERY AND PEDIATRIC DENTISTRY

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Recommended for the students, residents, and medical practitioners of the Department of Pediatric Dentistry

The work was carried out at the „Ion Lupan" department of pediatric oral&maxillofacial surgery and pedodontics. The work is written in English and corresponds to the curriculum for dentistry students on the department of pediatric oral&maxillofacial surgery. The compartment includes emergency conditions - odontogenic and non-odontogenic infections, maxillofacial bone and soft tissue trauma in the head and neck region.

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CONTENTS

PREFACE

THE IMPORTANCE OF THE COURSE FOR STUDENTS AND RESIDENTS

Pediatric dentists may be involved in the primary assessment of pediatric trauma, infections, congenital malformations, andtumor patients. Due to continued training and practice, dentists acquire a keen eye for details, and due to their knowledge of facial anatomy, they are ideally suitedfor the diagnosis and treatment of facial lesions. Although dentists may not be involved in all aspects of craniofacial soft tissue treatment, they form an important part of the management team. Dental professionals may be one of the key health care providers who assess the patient postoperatively and provideorofacial reconstruction services. To be an effective member of the management team, dentists require thorough knowledge of the diagnosis and treatment of craniofacial injuries. There has been a gradual rise in the incidence of trauma in children, probably due to increased risk-taking and aggressive behavior in children, who are more commonly left on their own without any close supervision.

ABBREVIATIONS

ASA	–	American Society of Anesthesiologists
MRI	–	magnetic resonance image
CT	–	computer tomography
PET	–	Positron emission tomography
MEE	–	medial edge epithelium
ORL	–	oto-rhino-laryngologist
TMJ	–	temporo-mandibular joint
ECG	–	Electrocardiogram
ANS-PNS	–	the distance between the anterior nasal spine and the posterior nasal spine, maxillary dimension
S-N	–	Anterior cranial base, the line that connect point N= nasion, the most anterior point on the frontonasal suture and point S= sella, the center of the sellaturcica
ANS-PNS	–	the distance between the anterior nasal spine and the posterior nasal spine, maxillary dimension
SNA	–	position of maxilla to the cranial base

1. PECULIARITIES OF GROWTH AND DEVELOPMENT OF CHILDREN

1.1. Peculiarities of child development

The American Academy of Pediatrics defines pediatrics as „the specialty of medical science concerned with the physical, mental, and social health of children from birth to young adulthood." There are discrepancies in the pediatric population given the large age range and developmental stages encompassed by this field. Pediatric age categories (subpopulations) follow the US Food and Drug Agency and World Health Organization guidelines for clinical pharmacology, reflecting the underlying principle of a linear relationship between pediatric weight and growth, and are defined as:

a) neonates, 0-1 month,
b) infants, 1 month to 2 years,
c) children, 2-12 years,
d) adolescents 12-16 years.

In adolescence, further development stages based on pubertal matu rational stages should be considered. Because of differences in morphological, endocrine, cardiovascular, metabolic, and thermosregulatory responses, physiological responses to different internal and external factors differ between infants, children, adolescents, and adults. Infants and children have traditionally been considered an 'at-risk' group with greater susceptibility to adverse physiological responses and health events during environmental extremes.

The facial features of the human embryo develop rapidly during pregnancy, beginning around the fourth week after conception. From birth until the age of 18 years, the child develops intensively and passes through several stages. The younger the child, the more development peculiarities are found. Each period has some specific morpho-functional features. The development of the human body includes several periods:

1) intrauterine - 280 days (10 months);
2) premature - 2,5-3,5 weeks;
3) infant (suckling) period – up to 1 year;

4) early age – from 1 year up to 3 years;

5) preschool age – from 3 up to 7 years;

6) school age – from 7 up to18 years (small, medium and school puberty).

Intrauterine development. There are three prenatal periods: the pre-embryonic period - the first two weeks, the embryonic period - three to eight weeks, and the fetal period – nine weeks. Prenatal periods are critical for each developmental stage because of a higher risk of environmental teratogen exposure, resulting in an increased risk of infant morbidity and mortality. Most birth defects occur during the period when the organs are formed. During this period, the embryo is extremely vulnerable to the effects of drugs, radiation, and viruses.

The head and neck areas are most vulnerable to birth defects in the first 3 months of intrauterine development. The frontonasal prominenceis primarily responsible for the development of the forehead and nose. Following the developmental abnormalities of the frontonasal prominence, anomalies of the eyes and nose – hypotelorism or cyclopia, ethmocephaly, and cebocephaly - occur. In the first two months of development, the mergingdisorders of embryonic prominences may cause cysts, cervical and facial fistulas, and anomalies of the muscular, nervous, and vascular tissues. The anomalies occurring after two months result from development and growth abnormalities and have less importancefor the function of the organs (form anomalies of the skull and facial bones, nasal and auricular cartilage, eyeballs, etc.).

Premature development. The neonatal period (from birth to one month) is a time of extensive and ongoing system transition from the uterine environment to the external world. It includes the initial period after birth, which is referred to as the perinatal period. As a result of the pressure on the baby during delivery, postnatal subcutaneous bleeding manifests as swelling and extended edema of the soft tissue. It is mainly located in the occipital and parietal, frontal, and facial regions, depending on the child's position during pregnancy. They regress after a day and disappear in 2-3 days.

Neonatal jaundice refers to the yellow color of the skin, including the face and the sclera, resulting from the accumulationof bilirubin in the skin and mucous membranes. It is associated with a raised level of bilirubin in the blood (hyperbilirubinemia). About 60% of term and 80% of preterm babies develop jaundice in the first week of their lives.

Birth weight is relevant for preterm, term, and prolonged pregnancy. In the first 3-4 days of life, the birth weight decreases considerably, which is a physiological process. The neonatal period may be associatedwith respiratory, gastrointestinal, neural, and cardiovascular abnormalities because they have not been functionally or extensively remodelled. Birth defects with obvious clinical manifestations are observed immediately after birth. The defects with less obvious clinical signs may remain unnoticed for a long time; they may be observed in their functional disorders. For example, difficult breastfeeding or regurgitation and clicks of the tongue are caused by a short frenulum of the tongue and lips. Frenectomy shouldbe performedwhen there are clicks of the tongue during breastfeeding and the birth weight does not increase. Because children's hemostasis system is very unstable, post-surgery follow-up care is required for 2-3 days. The regurgitation of milk and the inability to suckle are signs of the soft palate defect.

The development of the jaws is due to the physical effort of the chewing muscles during breastfeeding. During the first days of life, the children suck about 300 g of milk; at the end of the second week, they suck about 700-800 g of milk due to the development and growth of the chewing muscles. Artificial feeding does not provide this function. For this purpose, the hole of the nipple has to be of such a diameter as to allow the milk to pass at the speed of one drop in two seconds, thus providing the feeding time in 12-15 minutes.

In neonates, the immune system is under-developed and relies on maternal antibodies transmitted *in utero* to modify and control the severity of neonatal diseases. The lack of antibodies and immunity means that neonates cannot fight off bacteria, viruses, or fungi. This makes infants more susceptible to infections, and they can catch a cold at any age.

Skin infections are often present in infants. Luckily, they respond very quickly to treatment if the infection is treated in time.

Infancy (suckling period) is the period of lifefrom 1 month to 1 year. This period is characterizedby the hypofunction of internal organs, especially the digestive tract. Significant changes occur in the endocrine system. At the age of 4-5 months, the function of the endocrine system is activated. In the first year of life, thyroid secretion increases, the pituitary gland and thymus begin to function. In some children, constitutional abnormalitiesare observed, as well as exudative diathesis, nervous and lymphatic abnormalities. The intensive growth of the skeleton generates a high risk of rickets.

The passiveprimary immunity gradually decreases and the acquired immunity is not developed yet. Under these conditions, the sensitivity of the organism is increased and the child is particularly exposed to various infections. Typically, the oral mucosa, respiratory organs, and teguments serve as gateways for germ penetration. The diffuse reactions and the inability to limit pathological processes within an organ or tissue are peculiar to this age.

During infancy, the action of external factors (sucking of fingers, lips, tongue, nipple) may cause dental-maxillary system deformities and abnormalities. For prophylactic purposes, the bottle should be used only during superficial sleep and taken away during deep sleep. The movements of the hands should be limited by the use of special devices to prevent finger sucking. During sleep, the pillow should be flat and small, and the chin should be extended and not left down, as it contributes to the insufficient development of the mandible. The neck should be free to avoid tension of the chewing muscles, which can keep the mandible in the posterior position. During this period, nasal breathing disturbances are observed, which also affect the development of the jaws.

In the first year of life, the processes of development and mineralization of the primary teeth continue, and the processes of development and mineralization of the permanent teeth begin. Metabolism changes, caused by general disorders of the body during this period, will influence the structure of dental tissues, causing diffuse hypoplasia or an increased predisposition to dental caries.

Early childhood, or the period of primary teeth. During this period of development, all the functional capacities of the body are gradually completed. Children have increased plasticity. Often, various disrupt-tions inendocrine function are observed.

The preschool period is different from early childhood qualitatively but not quantitatively in terms of the body's development. Milk teeth appear during early childhood and the preschool years. In the absence of complex prophylactic measures in children, there is a high incidence of dental diseases, such as dental caries, odontogenic infections, and dental-alveolar deformities.

The school period includes children from 7 until 12-13 years. In these children, the organs develop and differentiate. The central and peripheral nervous systems are formed. The child's muscular system and intellect develop quickly. At this age, neurological and endocrine disorders often occur.

Puberty is the last stage of a child's development. The period differs from one child to another and depends on the sex, functional, and anatomical capacity of the body. In boys, puberty begins at 14-15 years; in some, it begins at 18 years. It starts two years earlier in girls (from 12-13 to 16 years). The child's body becomes similar to the adult`s one. Besides dental diseases that are characteristic of the preschool period, gingivitis often occurs, and periodontal disease when oral hygiene is neglected.

1.2. Peculiarities of topographic anatomy of the cranial and maxillofacial regions in children

Skull. The base of the skull is proportionally developed, but a strong calvaria rises above it, while the short face is underneath it, being wide to the skull and narrow to the chin. The circumference of the newborn skull is, on average, 34 cm. An obvious contour of the front portion and a small chin are characteristic of the newborn's facial appearance. The spherical and relatively big head represents ¼ of the body length; ⅕ at 2 years, ⅙ at 6 years, and ⅐ at 12 years. In adults, the size of the head is 7½-8 of the body length.

The round face with chubby cheeks (due to abundant adipose tissue) is much smaller than the skull. The face has two regions, namely an upper and a lower one. The upper region contains the sensory organs and is located close to the skull, beingalmost the same size. The lower region is relatively smaller and is the beginning of the respiratory and digestive systems. The face develops until the age of 30, having an irregular shape with intensive periods of growth up to 6 months, from 3 to 4 years, and from 7 to 11 years. During this time, traumas and infections can affect the growth areas of the facial skeleton, resulting in a variety of deformities.

The small and wide nasal pyriform aperture with the anterior and lower nostrils is limited to the frontal region by a transverse cutaneous fold that disappears a week after birth. The external ears, apparently located more posteriorly than in adults, are larger in relation to the face (about 25mm in length and 16mm in width) and reniform (in adults they are triangular). They are placed just behind a vertical line, dividing the head into two equal halves. The cartilages of the ears and nose are hard, resilient, and elastic.

The neck is short with abundant adipose tissue. The position of the sternum, clavicle, and shoulders is higher in children than in adults. In newborns, the cervical region is proportionally larger than in adults - 28 cm, which constitutes 25,5% of the spine length, compared with 50 cm in adults, accounting for 22,1%. Due to the fact that the face is undeveloped, the cervical organs have a higher anatomical position than in adults. In the lateral region of the neck, the lymph nodes of the deep cervical chainare compact and located near the inner jugular vein, some of which are even located on the vein, which may be easily palpated. The largest amount of fat is located in the occipital region, in front of the protruding vertebrae. The thick fat layer under the chin can reach a thickness of 3,5 mm. The neck and chest are short; the abdomen is very long. The newborn seems to have only the head and abdomen.

The skin of young children has some characteristics that change over their life. It has a well-defined capillary network located in the papillary layer and a rich blood supply. The dermis is three times thinner; connective tissue and muscles are lacking. The baby's corneal layer is fragile. The corneocytes are not joined together so well, meaning that they are more permeable to outside factors and infections. In babies, the hydrolipidic film is thinner than in adults. It is less effective in protecting the epidermis against drying out and dehydration, making it much more vulnerable to attacks from the world around it. On palpation, the skin is soft, elastic, and slightly sensitive, which is why no concentrated ointments are applied. The adipose layer, localized proportionally, gives the child a specific, rounded appearance. The high proliferation capacity accounts for the rapid regeneration of soft tissue wounds. Wounds of 5-6 cm in length regenerate spontaneously in comparison to adults, who regenerate normally with wounds of up to 3-4 cm in length.

The epidermis is the uppermost layer of the skin. It is made up of several layers of cells referred to as keratinocytes. These cells start their life at the very bottom of the epidermis and then gradually move up to the uppermost layer of the skin. Once they reach the surface, they lose their nuclei, get filled with keratin and form what is known as the „corneal layer" (stratum corneum). This is a semi-permeable shield made up of degraded keratinocytes (corneocytes) linked together by lipid and protein complexes. The corneal layer serves as a barrier, protecting the body against outside attacks.

The surface of the epidermis is covered with a mixture of perspiration and sebum referred to as the „hydrolipidic film". This fatty substance keeps the skin moisturized and forms an antibacterial and antifungal barrier.

Located beneath the epidermis, the dermis serves as a support structure, being very important for skin firmness and elasticity. It contains elastin and collagen fibers, among other things.

The hypodermis is the deepest layer of the skin. It is mainly made up of fatty cells – adipocytes – which protect the body against variations in temperature and form a protective lining against the pressure to which the skin is subjected.

The oral cavity in newborns and infants is small until 3 months due to a short mandibular ramus, a short and broad palate, as well as the lack of teeth. The oral cavity is separated from the vestibule of the mouth through the gums, where teeth erupt. The hard palate is wide and flat, unlike in adults, where it is narrow and tall. On the posterior side of the hard palate, on both sides of the raphe, there is a depression where glandular holes open. In newborns and infants, the mucosa of the palate in the posterior region is symmetrical to the median line; there are epithelial cords and pearls made of epithelial tissue surrounded by a conjunctival capsule. They disappear after 2-3 years of life.

The mouth is small. The relatively small cavity lodges the relatively large tongue. The chewing muscles are well developed. In the deep spaces of the face, the Bichat's fat pad is located, which is well-defined, protruding into the oral cavity and being located on either side of the face between the buccinator muscle and several more superficial muscles, including the masseter muscles, the zygomaticus major, and the zygomaticus minor muscles. At the age of four years, it has the appearance of a slightly curved sphere, a portion of which forms a groove in which the anterior edge of the masseter muscle starts. The Bichat's fat pad is separated from the neighboring tissue by a thin membrane that gently fixes it on the buccinator muscle andeasily enucleates. With age, the fat pad grows and develops, resulting in temporal, buccal, and pterygoid extension. The primary function of the buccal fat pad is in relation to chewing and suckling. On the other hand, the fat pad facilitates the action of the muscles of mastication and, as a cushion protects the sensitive facial muscles from injury due to muscle action or exterior force. Because it is separated by a thin membrane and is very gently attached to the surrounding tissue, there is a favorable condition for infection to spread in the deep spaces through the buccal fat pad.

The mucous membrane of the oral cavity is thin, slightly dry, and well vascularized, giving it a special gloss. The gingival mucosa covering the alveolar arch and the alveolar edge is immobile and even thicker than in adults. The alveolar portion of the gingival cups is bounded by two grooves, namely a lateral larger one and a medial smaller one. Every half of the maxilla and the mandible has five protrusions, or dental cups, which form a covering for each deciduous and permanent tooth. Due to the abundance of vessels at the site of the projection of the primary teeth, the gum is white and red around them. On the inferior margin of the upper and lower gums there is a valuable folded envelope, themucosa namedRobin-Magitat envelope, more pronounced after breastfeeding. It is more evident at the level of the canine teeth and disappears when the primary teeth erupt.

In newborns, *the upper jaw* is short andwide. The transverse, anterior, and posterior bone diameters are greater than the vertical ones. The frontal process is well-marked, and the body of the bone consists of little more than the alveolar process, in which dental buds are located. The tuberosity of the upper jaw is undeveloped. The maxillary sinus has the appearance of a furrow on the lateral wall of the nose. The height of the alveolar process is small, and the vestibular cortical plate is thick, but the medial one is thin. The sockets of the superior deciduous teeth are large and markedon the anterior surface, giving the appearance of a maxilla projected forward. The jaw body is small in size, and therefore the dental buds are located immediately below the orbital floor.

Five processes underlie the growth of the upper jaw: sutural growth, alveolar process development, subperiosteal bone formation, the enlargement of the maxillary sinus, bone resorption and deposition. The sutural growth continues until 10 years of age and then becomes less significant. All the sutures (frontomaxillary suture, zygomaticomaxillary suture, zygomaticotemporal suture, pterygopalatine suture) that make the maxilla articulate with the other bones of the skull are parallel to each other and directed from upward anteriorly to downward posteriorly. The growth of these sutures will shift the maxilla forward and downward. The alveolar process development is associated with the eruption of the primary and permanent teeth and adds up to the maxilla height. Subperiosteal bone is formed throughout life and serves as a main factor in the growth of the maxilla. The enlargement of the maxillary sinus plays an important role in the growth of the body of the maxilla.

The sinus, which occupies most of the body of the maxilla, expands by bone resorption on the sinus side and bone apposition on the facial surface of the maxillary process. The enlargement of the nasal cavity is caused by bone resorption at the floor of the nasal cavity, which is compensated for by bone apposition on the oral surface of the palate.

The maxillary sinus in newborns appears as a bulging in the inferior meatus with three surfaces, namely the upper, the lateral, and the medial one. The fourth surface is a lateral oblique edge, which is the apex of the sinus. On the anterior side, it touches or exceedsthe nasolacrimal canal. The communication hole with the middle meatus is a mere cleft that is sagittally oriented. It becomes an elongated oval in the second month of life, a rounded oval in the seventh month, and rounded at the age of seven. At birth, the sinus has the following dimensions: vertically – 5 mm, longitudinally – 8 mm, transversally – 3.5 mm. Subsequently, the sinus developsthrough bone resorption. The sinus cavity increases anteriorly and laterally, where the bone is mainly spongious.

The sinus grows in height by leveling the inferior wall of the orbit. In the first year of life, the inferior sinus wall projects at a distance of 4.2 mm from the bottom of the nose. Every year, it descends by 0.5 mm. At the age of 7 years, the sinus is tetrahedral. Its growth is completed between the ages of 12 and 14 years, with the eruption of the molars and posterior growth. Because dental buds are separated from the sinus cavity by a thick wall, the height increase is not related to deciduous teeth.

Intensive sinus growth occurs at the age of 5 years. Its growth is slower between the ages of 5 and 15 years. During tooth eruption, the inferior margin of the sinus appears wiped out on the X-ray image. After the eruption of permanent teeth at 13-15 years, the maxillary sinuses mature and adopt the same anatomical form as the teeth. The paranasal sinuses reach their maximum width horizontally between the ages of 15 and 20 years. Vertically, they reach their maximum height. The left paranasal sinus is larger than the right one, and in boys it is more voluminous than in girls.

The overall growth changes during the first 5 postnatal years are generally greater than those between 5 and 16 years. Maxillary and anterior cranial base growth rates peak in the first year and then decline over the next four years.

Between the ages of 1 and 3, there is the most postnatal growth in facial depth. The growth of the cranial base during the first 5 years is greater than during the remaining postnatal years. The maxillary size (e.g., N-ANS, S-N, ANS-PNS) increases during the first 5 years, while SNAdecreases overall between the ages of 1 and 5 years. It does not change between the ages of 2 and 3 years, and it increases slightly between 4 and 5 years.

In newborns, the lower jaw is formed by a left and right piece of cartilage called Meckel's cartilage. At birth, the body of the bone is a mere shell and is situated posteriorly in relation to the upper jaw, and the alveolar processes of the upper and lower jaws contact only duringsuckling**.** The angle is 175˚. The alveolar process measures 8.5 mm in height, and the mandibular body measures 3-4 mm (3-5 mm in adults). The alveolar process houses the incisor, canine, and two molar tooth sockets. In newborns, the mandible ramus is short, resembling a square, and represents 35% of the length of the mandible. The articular fossa is flat. After birth, the two segments of the bone become joined at the symphysis, from below upward in the first years, but the separation trace may be visible in the beginning of the second year, near the alveolar margin. The coronoid process iscomparatively large in size and projects above the level of the condyle. The mandibular canal is large in size and runs near the lower border of the bone. The mental foramen opens beneath the socket of the first deciduous molar tooth. The condyloid portion is nearly in line with the body.

The ramus of the mandible in 3- to 4-year-old children is twice as narrow as that of adults. To accommodate the teeth, the mandible body is elongated along its entire length, but especially behind the mental foramen. The mandibular canal after the second dentition eruption is situated just above the level of the mylohyoid line, and the mental foramen usually occupies the same position in adults. The distance from the oblique line to the mandibular foramen is 8-9 mm at 5-6 years, 10 mm at 11-13 years, and 11-13 mm at 13-15 years. The mandibular angle is obtuse, ranging between 140° and 150°. In adults, it drops to 104-110°. On the vestibular side of the mandible, the mental foramen is close to the alveolar process at the level of the canine tooth bud. In infants and young children, the mental foramen is located at the middle level of the body between the alveolar process and the lower margin of the mandible, at the first premolar tooth bud.

The mandibular condylar cartilage is the center of greatest growth in the craniofacial complex. The condylar process occurs in a wide range of directions, from anterior-superior to posterior, resulting in highly diverse mandibular growth and morphology. The condylar cartilage is a heterogeneous tissue containing fibroblasts, osteochondral progenitor cells, and chondrocytes. Type I collagen, which is derived from progenitor cells, has some cartilage characteristics, and type II collagen is colocalized in the cartilaginous cell layer. The colocalization of both collagen types is an adaptation feature to the complex biomechanical environments of the condylar cartilage. The peripheral condylar cartilage contains chondroid bone, a specialized calcified tissue with morphological properties intermediate between those of bone and cartilage. The hybrid tissue plays an important role in regulating different rates of bone formation in intramembranous and endochondral ossification, allowing for highly diverse growth directions and condylar and maxillofacial morphology. The growth of the mandible is due to endochondral ossification, which starts with the condylar process. During the entire longitudinal growth period of the mandible, some remodeling changes in osteogenesis are observed in the mandibular ramus: anteriorly, the resorption remodeling, and posteriorly, the periosteal osteogenesis. Therefore, the ramus and the body of the mandible gradually grow longitudinally.

Between 9 months and 1,5 years, the mandibular foramen is located 5 mm below the mandibular alveolar process. At 3-4 years, the mandibular foramen is located 1 mm below the lower mastication surface of the inferior molars. At 6-9 years, the mandibular foramen runs up to 6 mm above the inferior molars, and at 12 years, it reaches 3 mm above the same surface. The mandible grows intensively in the following age groups: 2,2-4 years, 9-12 years, and 15-16 years. The mandibular ramus grows intensively at 3-4 years and 9-11 years. It continues up to the eruption of permanent teeth (15-17 years).

During childhood, height growth is relatively stable up to the age of 4 years. Girls grow slightly faster than boys, then their growth has an average speed of 5-6 cm and 2.500 kg per year until puberty. From early childhood to late adolescence, bone formation predominates over bone resorption, with a steady accumulation of skeletal mass, which increases approximately by 70-95g at birth to 2400-3300g in young women and men, respectively.

In children, most of the skeleton is composed of cancellous bone. Cancellous bone is highly vascularized and contains red bone marrow. As children grow up, the cancellous bone is slowly converted into cortical bone, and the bone marrow gradually decreases in quality and is converted into fatty yellow tissue.

In children, the cortical bone is characterized by strip, a Haversian system that has a large central canal. The architectural changes of the mandible structure depend on the child's age, functional loading, tooth eruption, systemic and local factors, general condition, etc. In newborns and infants, the structure of the mandible, both in the body region and in the ramus, is radiologically well observed and clearly visible; at the level of the lines of force, the trabecular picture is not marked. The spongy substance in a 6-month-old infant is located at the level of the primary molar buds, being absent in the alveolar process. The amount of the spongy substance is very small, having an undifferentiated structure. The spongy tissue grows intensively from 6 months to 3 years, i.e., during the period of primary tooth eruption.

There are structural changes between the ages of 2 and 3 years, occurring due to the activity of mastication and functional loading. The upper and lower jaws grow significantly, and the bone architecture becomes dense. The trabecular bone morphology is clearly visible along the mandibular body andthe alveolar process. Between the ages of 3 and 9 years, the cancellous tissue is remodeled. Bone trabeculae are aligned toward the mechanical load distribution.

The alveolar process in children depends on tooth development and eruption. During the physiological exfoliation of the primary teeth, the tip of the interdental septum is erased, and the teeth seem situated in bags, which radiologically are highly opaque with irregular contours. Their anatomical contour is restored when teeth erupt. The interincisal septum is round or sharp, and the cortical layer is thick. Normally, interdental segments are located in the enamel and dentin. The development of the alveolar crest ends between the ages of 8 and 9 years.

The salivary glands are developed at birth. In comparison to the adult glands, which weigh 43g, 4g, and 6g, the parotid gland weighs 1.8 g, the submandibular gland 0.84 g, and the sublingual gland 0.4 g. At the age of 3 months, their weight doubles; at 6 months, it triples; and at the age of 2 years, it exceeds 5 times the initial weight. In newborns, the secretion of the salivary glands is diminished, resulting in a mild dryness of the oral mucosa. At 5-6 months, the function of the salivary glands is activated, causing physiological hypersalivation. If the extra saliva begins to accumulate, it may begin to drip out of the mouth unintentionally. The saliva volume increases in oral infections and decreases in such conditions as diarrhea, high temperature, cachexia, and suckling disorders. The salivary duct of the parotid gland in young children has an irregular pathway, and the Stenon`s orifice is located 0.8-1cm above the masseter muscle. The parotid gland has a round shape, is located anteriorly to the ear, and extends up to the mandibular angle. The branches of the facial nerve pass through the parotid glands superficially.

1.3. Peculiarities of the development of systems in children

Nervoussystem. The primary function of the nervous system is to provide the best adaptation of the organism to the influence of the environment and to produce its overall reaction. The development of the nervous system in early childhood is decisive for the rest oflife, namely the ability to carry out creative activities, to study, and to regulate emotions. In the first three years of life, the nervous system has the ability to develop and restore neurological functions. After 10 years, this potential is critically limited. The first 18 months are the critical period for unfavorable influences. The combined reasons are the most dangerous for the development of the child's intellect, namely the absence of proper care and nutrition, unfavorable hygienic and environmental conditions, the presence of family stress, a lack of medicine, etc. The development of the nervous system and the correction of neurological disorders directly depend on the timeof treatment or prevention.

Brain accounts for 10% of the body mass in children. At the age of 16-17 years, it is 2,5% of the body mass with a large amount of water. The oxygen requirement of the brain cells is 20 times greater than that of the muscular tissues, so there is an increased sensitivity of the brain cells to oxygen starvation, the action of toxic substances, and high intracranial pressure. Chronic hypoxia and intoxication lead to the degeneration and atrophy of the nerve cells and even death. At the age of three, the process of nerve cell differentiation is complete. Up to the age of eight years, there is a higher permeability of the blood-brain barrier, and the gray substance of the brain cortex is not separated from the white one. At the age of eight years, the cortex is slightly different from the cortex of adults. The immature nervous system is characterized by functional weakness of the nervous system. The organs of vision and hearing are developing. Photophobia, heterotropia, nystagmus, the absence of pupils widening under strong painful irritants, and low visual acuity are observed in newborns. The organs of touch are differentiated in the first months of life. Skin irritation causes a general reaction and discomfort in children. Pain and irritation cause local and general reactions in babies.

The cellular structure of the cerebellum is fully developed by 7-8 years. Vegetative reactions are well expressed in newborns, providing the functions of breathing, blood circulation, digestion, etc. The undeveloped nervous system in children creates favorable conditions for the generalization of the odontogenic and non-odontogenic purulent processes in a short period of time. Therefore, the general clinical signs (vomiting, diarrhea, restlessness, fever, etc.) manifest first. Local clinical manifestations are wiped out due to abundant fat and the fact that children can hardly determine the location of the purulent focus. Facial asymmetry is the first sign observed by parents. The respiratory system and the unstable vegetative nervous system respond to damaging factorsthrough breathing disorders.

Children's psycho-neurological responses to treatment are inappropriate. As a result, phobia and stuttering are very often observed in children treated by force without any remedies to protect the nervous system or with inadequate anesthesia.

The cardiovascular system in newborns is the most developed comparedto other systems. Heartbeats are more common than in adults, and blood pressure is lower. The blood volume in children is higher and constitutes 80-100 ml/kg, compared to 60 ml/kg in adults. The blood circulation speed is twice as high as in adults. Most of the bloodstream circulates through the central vessels of the internal organs and the peripheral ones; a much smaller amount circulates in newborns and young children. Children are less resistant to hemorrhage and orthostatic disorders due to the low sensitivity of baroreceptors. Blood loss of up to 50 ml is equivalent to 600-1000 ml of blood in adults. Due to this, even minor bleeding requires treatment.

Respiratory system. In the first year of life, the respiratory system is underdeveloped. The pulmonary breathing area per kg is small, and the need for oxygen is high. In the case of tympanites and aerophagia, diaphragmatic respiration predominates over thoracic respiration, and breathing disorders are frequently caused by compressions. The thoracic bones of the chest are located horizontally.

The respiratory tract in young children is narrow, the tongue is relatively big, and the pharyngeal mucosa is more responsive to the development of edema. Respiratory infections, adenoid vegetations, and tonsils often affect young children and cause impermeability of the respiratory tract twice as often as in adults. The insufficiency of the respiratory epithelium and reflective drainage function is a common reaction in young children, especially when anesthesia preparations are inhaled. Therefore, during inhalation anesthesia, the mucoid fluid rapidly accumulates in the respiratory tract, accompanied by breathing disturbances.

Lymphatic system. The lymphatic system is part of the immune system and fights diseases and infections. The lymphatic tissue grows steadily until puberty. Before birth, the fetus relies on the mother's immune system for protection against infections. At birth, the lymphatic system of newborns begins to respond to their frequent exposure to microorganisms. The lymphatic system undergoes numerous changes as a child grows and develops. Children are constantly fighting off new germs and infections, and their lymphatic system quickly responds. For this reason, it is common for children to have slightly enlarged lymph nodes. However, changes in lymph nodes can indicate certain conditions or diseases that need special treatment. In newborns, lymph nodes are characterized by low connective tissue content and an incomplete development of the cellular system. In the first year of life, lymph nodes are not able to defend the body against possible infections, which accounts for the increased probability of generalizing infectious processes. Lymph nodes have full barrier function at the age of 7-8 years.

The gastrointestinal tract is fully formed at 20 weeks of gestation, but it is not fully functional. Infants with an immature gastrointestinal tract have feeding intolerances right after birth. These include vomiting food, stomach bile, and abdominal distension. The baby's abdomen appears abnormally large, with reduced or absent bowel sounds. Children up to one year old have a long evacuation time. The emptying of stomach contents in most children takes 8 hours. Therefore, the risk of vomiting and aspiration increases, especially during sleep and general anesthesia. The insufficient function of the cardiac sphincter and the spasm of the pyloric aperture may also cause vomiting and aspirations.

Endocrine system. Endocrine glands release hormones into the bloodstream, which reach cells in all parts of the body. In children, the hormonal system is different from that in adults. Each age has its own level of hormonal regulation. The endocrine system keeps control of mood, growth and development, the way organs work, metabolism, and reproduction. Under unfavorable conditions, the child's mechanism of endocrine compensation is turned off to help overcome the environmental impact. The development of the endocrine glands and their functioning begins in prenatal development. During the period of a child's development, the thyroid, pituitary, and adrenal glands are of the greatest importance. In the period of 10-12 to 15-17 months of intrauterine development, all endocrine glands are active.

The pituitary gland has the greatest importance in the process of human development. The hormones control many other endocrine glands, but growth control is considered the main function of the pituitary gland. The weight of the pituitary gland in newborns is 0.3 grams, while at the age of 10 years, the weight is 0.7-0.9 grams.

The functions of the pineal gland are most active before the primary school period. By the age of 3-4 years, the activity of the pineal gland decreases. The hormones that inhibit sexual development are produced in the pineal gland.

The parathyroid gland is formed after two months of intrauterine development. Immediately after birth, it weighs 5 grams, and during the child's growth, it increases 15-17 times. The period of greatest activity of the parathyroid gland is up to the age of 2 years, with activity being maintained at a higher level upto the age of 7 years. The parathyroid gland releases the parathyroid hormone, which controls the level of calcium in the blood.

In newborns, there are low levels of calcium and phosphorus in the blood, which leads to the cyanotic color of the skin and hyperactivity of the muscles. The parathyroid hormone, which is released by the parathyroid gland, helps to increase the amount of calcium in the blood, assimilates calcium in the intestines, and increases the concentration of phosphorus in the urine. By the end of the growth period, the level of parathyroid hormone in the plasma is higher than in adults.

The thymus is located in the inner part of the chest and produces white blood cells called T-cells. The thymus is the largest and most active during the neonatal and pre-adolescent periods. By the early teens, the thymus begins to decrease in size and activity, and the tissue of the thymus is gradually replaced by fatty tissue. Nevertheless, some T-cell development continues throughout adult life.

The thymus can become quite enlarged (thymomegaly) in conjunction with adrenal gland hypofunction, resulting in the thymo-lymphatic condition characterized by hypotonia, skin pallor (paleness), and lymphatic diathesis. Thymo-lymphatic conditions occurring during general anesthesia or surgery may lead to death.

The thyroid gland is one of the main endocrine glands and playsa big role in the growing body. The thyroid gland produces thyroid hormones, triiodothyronine and thyroxine, that have a wide range of effects on the child's development. These include metabolic, cardiovascular, and developmental functions. Thyroid hormones increase basal metabolic energy, increase the absorption of substances, stimulate glucose, fat, and cholesterol, and have effects on all body tissues. The hormones increase the rate and strength of the heartbeat. They increase the rate of breathing, oxygen intake and consumption, blood flow, and body temperature. Thyroid hormones are important for normal developpment. They increase the growth rate of young people and their brain development and play a particular role in brain maturation during fetal development and the first few years of postnatal life. The child's physical and mental development is impaired by the thyroid disorder, which also causes dental hard tissue damage, ossification retardation and bone growth arrest, delayed dental eruption, low body temperature, and sluggish skin metabolism.

Thermoregulation is the ability to balance heat production and heat loss in order to maintain body temperature within a certain normal range. The thermoregulation mechanism is immature in infants and neonates. The body temperature of children tends to be higher than that of adults. Body temperature decreases towards adult levels at the age of 1 year and slowly declines to normal body temperature throughpuberty, becoming stable at 13-14 years in girls and 17-18 years in boys. Thermoregulation instability is accounted for by the high conductivity of adipose tissue, poorly developed muscle tissue, insufficient sweat glands, and a relatively large body surface. Atropine demand combined with low or high temperatures results in hypo- or hyperthermia.

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2. OUTPATIENT AND INPATIENT ANESTHESIA IN PEDIATRIC DENTAL SURGERY

2.1. Stages of emotional and cognitive development in children

In dentistry, local anesthesia is the introduction into the body of certain chemical substances that temporarily prevent pain in a specific area of the mouth, the perception or transmission of pain sensitivity, accompanied or not by analgesic sleep, stabilizationof the autonomic nervous system, and muscular relaxation. Anesthesia in pediatric dentistry is different from that in adults, and there are difficulties arising from the specific anatomical and physiological structure of the body due to emotional instabilityand autonomic nervous systems, excitement, a lack of collaboration, and increased psychomotor agitation. The child's body is a complex biosocial system in which all organs and systems are physiologically unstable, and even minor pains are unbearable. Negative emotions can have a bad impact on children's behavior and may turn into an extreme manifestation, the affective state.

Anxiety is a feature of a defined personality and a disorder of affectation manifested through anxiety, fear, and unmotivated worry. It is defined as a reasonless fear. Phobia is an emotional state responsible for the recognition, usually conscious, of external dangers and threats. In contrast to anxiety, phobia is a conscious fear (for example, dento-phobia, or fear of dentists). Anxiety and fear are accompanied by a similar psychological responsibility, which may be considered patho-logic if it disturbs the responsibility of emotional control.

At the age of two years, the ability of children to communicate differs a lot depending on their vocabulary development. At this age, children use almost 12-100 words. They prefer playing alone and are not used to playing with other children. Children do not interact with the doctor verbally but rather through games and toys that they play with, hold in their hands, or touch. The blinding lights and the sparkling tools may cause fear, manifested by anxiety attacks (sudden, unexpec-ted movements). The child tries to get out of the dental chair, turns around, and makes striking, sudden movements with his hands. The forced separation of children from their parents may cause panic attacks. That is why children are examined and treated only in their parents` hands.

At the age of three years, children are ready to communicate easily with dentists. They are very keen on telling different stories. The interest of the physician in children's problems, the kind attitude, and the gentle talk of the medical staff set the stage for a peaceful collaboration between the physician and the young patient. But sometimes, despite being kind, he/she refuses any manipulationsin the oral cavity.

At the age of four years, children listen attentively to the dentist`s explanations. Usually, they are obedient and communicate with pleasure. In some situations, they may become aggressive. Stress manifested at the age of 2-3 years becomes less expressed at the age of 4 years. At this age, physical injury is the cause of emotional lability. Minor injuries, injections, or hemorrhage may have an impact on the child, producing different obsessional and phobic states.

At the age of five years, children have rich experience in activity and communication. They have heard some negative things about dental treatment from their friends. Providing the child with an adequate education makes the child less stressed, althoughthe treatment takes place in the absence of the parents. The comments regarding their personal interests (clothes, music, movies, etc.) may change children's attitudes towards what is happening in the dental office, arousing their interest and encouraging mutual collaboration, which makes them proud of their behavior until the end of the procedure.

At the age of 6-12 years, children have an interest in their environment and become independent. They form groups of close friends, regrouping in clubs or teams. They research methods for achieving their goals in collaboration with others and adapt to social norms. At this age, they can overcome the stress caused by dental procedures if the dentist explains the essence of the manipulations to be made. Children learn to be tolerant of stressful situations.

When describing the child's behavior, the interest or emphasis is laid on behaviors that dentists find difficult to deal with or that are inappropriate in some way. However, there are other behavioral aspects that can sometimes be important, and dentists may need to consider them as well. Children's behavior in the dentist's office can be used to examine children’s environment, children's reactions to different situations, and the way they express fears prior to and during situations of aggression. In children, the most common types of emotional distress during dental treatment are anxiety and fear, originating from a previous traumatic experience in the dentist's office or during hospitalization for other reasons, or due to the impact of the dental atmosphere on the child's emotional and behavioral intentions. Due to a lack of psychological or emotional maturity and/or mental or physical disability, the challenge of treating children who are unable or unwilling to cooperate has led to the development of a variety of behavior management techniques. Painful procedures cause distress in children and their parents. The stress response to pain is associated with metabolic and hormonal changes.

In the dental office, children are examined in order to assess their psychological state and their ability to bear dental fears. Thus, three types of behavior have been determined:

1. cooperative;
2. unable to cooperate;
3. potentially cooperative.

Cooperative behavior. These children present a „reasonable level” of cooperation, which allows the dentist to function effectively and efficiently. They seldom require pharmacologic adjuncts to help perform their treatments.

Children with cooperative behavior present a minimal stress risk. The collaboration between the dentist and the child is kind, and they assess the situation positively. The dental treatment has an obvious effect.

Unable to cooperate. In contrast tocooperative children, these children lack cooperative ability. This could include very young children (below three years of age) with whom communication cannot be established. Comprehension cannot be expected. If their treatment needs are urgent, they can pose major behavioral problems. Pharmacologic adjuncts may be required for their treatment. MacDonald (1969) referred to these children as being in the pre-cooperative stage. The children's behavioral issues are usually resolved with time. As children grow older, they develop into cooperative dental patients, and treatment is provided with behavior modification.

Another group of children who lack cooperative ability is those with specific debilitating or handicapping conditions. The severity of their conditions often interferes with their cooperation in the usual manner. Obtaining information on children`s intellectual development can provide valuable data about the expected level of cooperation. At times, special behavior management techniques, such as body restraints or sedation, are employed to control body movements. While treatment is being performed, major positive behavioral changes cannot be expected.

In most western societies, thrust in intellectual impairment services is community-oriented, and as large institutions for the mentally challenged are phased out, more children with special needs are being treated in dental offices today. More and more, these children and adults are living in group homes and private homes within residential communities. Many dental faculties have recognized this societal change, and programs have been established to prepare undergraduate and post-graduate students to meet the foreseeable demand.

Potentially cooperative behavior. Until recently, the nomenclature applied to a potentially cooperative child was „behavior problem”. The child may be healthy or disabled. There is, however, a distinction to be made between the potentially cooperative child and the child who lacks cooperative ability. A potentially cooperative child has the capability to behave well. It is an important distinction. When characterized as potentially cooperative, the rationale is that the child’s behavior can be modified. The child has age-related cognitive capacities to learn to deal with dentistry and can become cooperative.

Perhaps one of the most challenging issues for the clinician is to determine what behavior can be expected from the new patient. There are children who can cry or scream when they approach the dentist's office. Their behavior is apparent. Conversely, there are children who are quiet, shy, or withdrawn. These children can be hard to read. They may or may not be difficult to treat. Behavioral science researchers in dentistry and allied professions have made efforts to predict children's behaviors before their arrival at a dental clinic.

Children with potentially cooperative behavior are those with different forms of cooperation.

Aggressive behavior is manifested by children aged 3-6 years. The negative reactions appear even at the reception or when the dentist appointment is made. It is characterized by emotional lability, aggression towards himself/herself and the dentist, and negative emotions. These children need a psychological correction of varying duration.

Negative cooperation is characteristic of any age, but more typical of scholars. Some children have controlled behavior, repeating: „I don't want…", „I will not…". These are typically the children who are coerced into receiving dental treatment against their will. They are considered misfits in the dental service and need additional preparation by the psychologist and neurologist.

Teenagers have passive behavior and refuse to communicate verbally. Attempts to engage them in conversation during the examination or treatment are futile. When the physician tries to examine the teenager, he keeps his mouth closed.

Passive cooperation. Usually, these children come from villages with a small number of inhabitants or from isolated regions where the children rarely communicate with foreigners. For them, their parents are the shields behind which they can hide. They do not present physical resistance, but they are emotionally unstable. Children need correction for their behavior. For these children, dental treatment will last a long time. The incorrect attitude of the medical staff and parents towards children may exacerbate tension.

Stressful cooperation. Initially, children accept dental treatment, but during the treatment they become extremely stressed, which is manifested by head and hand tremors, especially while speaking, and sweating. These children keep control of their emotions, and the dentist may not observe their emotional state.

The specific cooperation of children combines the specificity of the last two categories. Children allow the dental examination and manipulations, but the feeling of fear is present during the entire period of examination and treatment. They often complain of pain. Their crying is controlled, constant, and not exaggerated.

There are three degrees of behavior in relation to psychomotor excitation:

I. Prevalence of excitationand aggression. Children's behavior is aggressive and bully-like. Usually, they are the leaders of their groups.

II. Prevalence of emotional depression. Children are depressed and less interested in their environment. They have autonomic nervous system instability (sleep and appetite disorders).

III. Prevalence of autonomic nervous system disorders and asthenia (unstable and unbalanced excitation and inhibition). Children are scared and divergent. They cannot concentrate and usually get tired quickly.

Numerous systems have been developed to classify children’s behavior in the dental environment depending on the central nervous system and the children's reactions to dental interventions. One of the most widely used systems was introduced by Frankl et al. in 1962. It is referred to as the Frankl Behavior Rating Scale. The scale divides the observed behavior into four types, ranging from definitely positive to definitely negative. A detailed scale description is provided, in which four phobiaratings are determined.

Rating 1 Definitely positive. Good rapport with the dentist; interest in dental procedures; and ability to laugh and enjoy the situation. Voluntary interaction of the child with the dentist. Children bear dental treatment well.

Rating 2 Positive. Acceptance of treatment, at times cautious; willingness to comply with the dentist, at times with reservation; but following the dentist's directions cooperatively and positively, with mild dentophobia, manifested by the child's passive refusal: "I don't have pain anymore", „I`d better come tomorrow". The facial expression shows strain. The child looks around with caution and attention; any object in the office or any movement of the medical staff attracts him. He sits in the dental chair willingly but cautiously, following all of the dentist's instructions.

Rating 3 Negative. Reluctance to accept treatment, uncooperative behavior, evidence of a negative attitude but not pronounced (i.e., sullen, withdrawn); medium dentophobia, expressed by emotions, aggressiveness, and panic elements. Children actively refuse dental treatment, manifesting motor excitation and tremor, respiratory disorders and pulse changes.

Rating 4 Definitely negative. Refusal of treatment, forceful crying, fearfulness, or any other overt evidence of extreme negativism. Severe dentophobia: refusal expressed by aggressiveness and neurasthenia (continuous cries and screams). The child presents withreactive neurosis (he turns his back to the physicians, either cuddling his parents or hitting and biting), and physically resists at the entrance of the dental office, sometimes making the entrance impossible. Pulse, abundant transpiration, mydriasis, vomiting, convulsions, paroxysmal cough, and sudden enuresis are determined at this moment.

2.2. Types and techniques of local anesthesia in children and adolescents

Local anesthesia forms the backbone of pain control techniques in dentistry and has a major role in pediatric dentistry. There is an ongoing search for ways to avoid the invasive and often painful nature of the injection and to find a more comfortable and pleasant method of achieving local anesthesia prior to dental procedures. Despite recent advancements, injection remains the preferred method of providing local anesthesia. Anesthesia is classified into local-regional and general.

Local-regional anesthesia

I. Non-injectable surface/topical (terminal) anesthesia:
- Physical (by refrigeration or electrical current);
- Chemical (anesthesia through application);
- Iontophoresis.

II. Injectable local anesthesia:
- Local infiltration (submucosal, paraperiosteal, subperiosteal infiltration, field block, intraosseous and infiltration anesthesia);
- Nerve block anesthesia (peripheral nerve block, basal, through acupuncture);
- Local soft tissue infiltrative anesthesia (serpiginous, Visnevski type).

III. Local anesthesia with pre-anesthesia:
- Psychological preparation;
- Psychological distraction;
- Conscious sedation.

IV. Local anesthesia combined with general anesthesia.

General anesthesia

- Inhalation;
- Intramuscular
- Intravenous;
- Mixed;
- Tracheal intubation.

Local anesthesia is a drug-induced, reversible local blockade of nerve conduction in a specific part of the body that does not alter consciousness.

Non-injectable (topical) terminal anesthesia has a limited effect on the free nerve endings by the application of ointments or solutions through contact with the substance, interrupting the pain sensation in the superficial layers.

The indications for topical anesthesia are as follows: a) extraction of primary teeth during the period when they fall out and present second or third degree of mobility; b) painless insertion of the needle and it is an important part of injectable local anesthesia, aiming to minimize the pain sensation at pricking the mucosa; c) it is recommended as a preparing stage of injectable anesthesia, aiming to distract the child's attention; d) drainage or puncture of gingival abscess; e) anesthesia of the mucosa in case of acute stomatitis; f) incisions of the gingival mucosa in teeth with difficult eruption; j) impression and fixation of orthodontic devices; h) anesthesia of the buccopharyngeal mucosa to avoid vomiting reflex during dental examinations or manipulations.

The local analgesic drugs used in topical anesthesia are as follows: dicaine solution (0,5% - 1%) is a strong anesthetic that is 10 times more toxic than novocaine. Due to its increased resorption capacity, it is indicated for children aged 10 years and older. Trimecaine is more efficient in solutions with a concentration of 2%-5%. Lidocaine and xylocaine may be used in terminal anesthesia as solutions (2%-5%). They are useful as solutions of 10%. It is applied last to pirocaine as a 5% ointment or in combination with 5% methyluracil. Piromacine is also successfully used in dentistry as an ointment of 5% and as an ointment of 5% plus methyluracil. It has a faster reaction time (1-2 minutes), a sufficiently deep penetration, and a lasting action (30-40 minutes). The solution is applied in the form of 5% emulsions or ointments**.** Lidocaine in topical anesthesia is used as 2%-5% solutions and sprays, packed in special boxes (lidocaine 10%), or as an ointment with xiline, contralgin, and contralgan.

The anesthetic is applied to the dry mucosa with sterile gauze and left for about one minute, or as needed. To avoid anesthetic solution leaching onto the oral mucosa, as well as solution absorption, swallow-wing, or aspiration, the gauze must be squeezed. Sometimes, the topical anesthesia is applied to the buccal mucosa through mouthwashes, instillations, or massaging the solution for about 1 minute. The solution should not be rubbed onto the mucosa because the buccal microflora can easily spread through the mucosa, causing ulceration. The anesthetic effect occurs within 1-2 minutes and lasts approximately 10-15 minutes. Because highly concentrated drugs are used in topical anesthesia, they are administered in small amounts in order to avoid overdosing.

Intraosseous or *intraligamentary anesthesia* is fast, deep, and long-acting. It is recommended in the primary dentition, namely in post-extraction complications (root fracture), difficult tooth extractions (tooth position abnormalities), and lasting tooth extractions.

The intraligamentary injection is given into the periodontal ligament using a specially designed syringe. Intraligamentary injections can also be given with a conventional needle and syringe. The needle is inserted into the mesiobuccal side of the rootand advanced into it for maximum penetration. The needle does not penetrate deeply into the periodontal ligament but is wedged at the crest of the alveolar ridge. A 12 mm, 30-gauge needle is recommended, and the bevel should face the bone, although the effectiveness is not influenced by a differentorientation. Because of its variable duration, intraligamentary anesthesia has limitations as a primary method of anesthesia, but it has been used to supplement or replace failed conventional methods. Intraligamentary injections produce significant bacteremia and therefore should not be given to a patient at risk of infective endocarditis unless appropriate antibiotic prophylaxis has been provided.

Special short and thin needles are used for anesthesia and extra small needles in small children, which are pricked in the center of the interdental papillae (mesially and distally of the tooth) or in the periodontal ligament, perpendicularly to the necessary tooth. The needle is deepened 2-3 mm at an angle of 40-50° to the gingival mucosa. The anesthetic solution is left to act until the mucosa whitens. Then the tip of the needle is introduced into the periodontal space and the spongy bone up to 1,5-2 mm, injecting under pressure 0,25 ml of anesthetic solution.

Intraligamentary anesthesia plays a role in local anesthesia in modern dentistry, but it does not fulfill all the requirements for a primary technique. Alternative methods, like traditional methods of obtaining oral local anesthesia, are generally safe if the practitioner understands the principles of their use. Alternative techniques for the delivery of local anesthesia may be considered to minimize the dose of anesthetic used, improve the patient's comfort, and/or achieve successful dental anesthesia. Some of these techniques are desirable, especially in infants, children, adolescents, and patients with special health-care needs, since specific teeth may be anesthetized with less residual anesthesia (i.e., to avoid discomfort and potential self-mutilation of block anesthesia). A child's mandibular bone is usually less dense than that of an adult, allowing for more rapid and complete anesthetic diffusion.

The intraligamentary injection reduces the risk of postoperative bleeding of the vessels within the soft tissue in patients with bleeding disorders.

Intraosseous techniques may be contraindicated in primary teeth due to the potential for damage to developing permanent teeth. The periodontal ligament injection or intraosseous method is contraindicated if there is inflammation or infection at the injection site.

Electronic dental anesthesia. The concept of electronic dental anesthesia (EDA) involves the application of an electric current that loads the nerve stimulation pathway to the extent that the pain stimulus is blocked. The literature review shows a significant reduction in pain observed during all the dental procedures conducted under TENS (Transcutaneous Electrical Nerve Stimulation). Thus, TENS should be considered a useful adjunct in the treatment of pediatric patients during various minor dental procedures. There are medical contraindications to the use of EDA: patients with a pacemaker or cochlear implant, heart disease, seizure disorders, or cerebrovascular disease, head tumor, neurological disorders involving the head and neck (e.g., Bell's palsy, trigeminal and postherpetic neuralgia, multiple sclerosis, or Tourette's syndrome), skin lesions or abrasions on the face, and patients with abnormal bruising or bleeding disorders.

Local injectable anesthesia is a temporary loss of sensation or pain in one part of the body produced by injected agents due to the depression or excitement of nerve endings or an inhibition of the conduction process without depressing the level of consciousness. There is no perfect technique that guarantees success in anesthetizing all children. However, there are a few key procedures that may be valuable to the success of all techniques. The basic criterion for effective dental treatment is establishing trust and collaborative relationships between the physician and the child. In order to prevent the failure of local anesthesia, do not wavethe syringe in front of the children. Moreover, the practitioner should have control over the child's head and a good finger rest to control the syringe in case the child moves or resists. The dental assistant should be prepared to restrain the child's hand gently but firmly. A short (20 mm) or long (32 mm) 27- or 30-gauge needle may be used for most intraoral injections in children. An extra short (10 mm) 30-gauge needle has been suggested for anterior maxillary injections. Long needles are recommended for inferior alveolar nerve block anesthesia. However, many dentists have shown that shorter needles are adequate and safe for young, difficult-to-manage dental patients. The dosage of the local anesthetic must be appropriate for the child's age, the patient's physical status, the area to be anesthetized, oral tissue vascularization, and the administration technique. It is difficult to recommend a maximum dose for children because the dose varies depending on the child's age and weight. For pediatric patients under 10 years of age who have a lean body mass and normal body development, the maximum dose may be determined by the application of one of the standard formulas (Clark's rule). In any case, the maximum dose should not exceed 7 mg/kg of body weight for lidocaine with epinephrine and 4.4 mg/kg for lidocaine with adrenaline. During the anesthesia, the physician must follow the child's behavior andconsider the way he/she answers the questions (the answers have to be clear), the tone of the speech, the color of the skin, and the temperature of the skin. The pulse and respiration should be monitored properly. Local anesthetics should always be injected slowly, preceded by aspiration, to avoid intra-vascular injection and systemic reactions to the local anesthetic solution or the vasoconstrictor.

Local Infiltration Anesthesia – the solution spread to nerve endings. This type of anesthesia is the choice for anesthetizing the maxillary and mandibular teeth successfully in children. For buccal infiltrations, it is recommended to stretch the mucosa at the injection site and gently pull on the obliquely placed bevel of the needle. There are two anatomical landmarks for the infiltration technique: the mucobuccal fold and the mucogingival junction (Sweet's line). The puncture site should be2 to 3 mm above the mucogingival junction, and the depth should be no more than 2 to 3 mm towards the roots of the teeth. A small amount of solution has to be injected into the superficial mucosa. After a few seconds, the needle can be slowly advanced 1-2 mm, and another small amount of solution can be injected. This should be repeated until the remaining anesthetic solution is completely injected. The alveolar bone in children is more resilient than in adults, and even 0.5 ml of solution may be sufficient to produce dental anesthesia.

Anesthesia of the primary mandibular molars can usually be achieved by infiltration in children up to the age of five years. Mandibular buccal infiltration anesthesia is as effective as inferior nerve block anesthesia for some operative procedures. There are no significant differences between infiltration and nerve block. In addition, the location of the tooth, the patient's age, or the type of anesthetic used had no significant impact on anesthesia quality. The anatomical-morphological peculiarities of the bone tissue in the maxillofacial area in children, characterized by the predominance of spongy bone and thin jaws, crossed by a large number of nerves and blood vessels, which anastomose largely with those from the periosteum and soft tissues, the presence of the dental buds, the predominance of organic substances in relation to non-organic ones, and the large Haversian channels, allow a high permeability on the entire surface of the alveolar process. In young children, infiltration anesthesia is closer to the intraosseous one.

The indications for infiltration anesthesia in the dental office are as follows:

- minimally invasive and short surgeries with minor hemorrhages;
- lip or tongue frenuloplasty;
- surgery of the alveolar processes in all primary teeth;

- extraction of teeth with root resorption of more than a third of their length;
- extraction of the primary anterior teeth;
- tooth extraction due to apical resorption.

The position of the physician when performing anesthesia must be comfortable in relation to that of the child, and the preanesthetic and anesthetic manipulations must be done out of the child's sight. The alveolar processes should be well fixed with the fingers of the left hand in order to avoid any sudden movements of the child. When extracting primary and permanent teeth, it is necessary to inject the submucosal layer. The prick is made in the vestibular region of the oral cavity, on the transition plica, at the level of the tooth apex, in the mobile mucosa, over the mucogingival line, with a delicate and short needle, the bevel being directed towards the bone. The mucosa is pricked and a small papule is made, then it is pierced deeply with the needle, pushing the piston continuously and slowly in order to avoid the necrosis of the fibromucosa. Then, the needle is directed in parallel with the gingival edge, crossing the mucosa and the submucosal tissue, and injecting the anesthetic solution in the vestibular region, closer to the apex, at 1 cm, orienting to the root apex. One prick may anesthetize two or more teeth. The anesthetized area covers the tooth, the periodontium, the alveolar bone, the vestibular gum, and the interdental papillae.

In plexus anesthesia, the doctorshould avoid the introduction of the needle through the gingival mucosa, the prick of the needle with the bevel outside and the tip toward the bone, the subperiosteal injection of the anesthetic solution, and the injection of the solution into loose connective tissue.

As the blood vessels and the nerves are located superficially, they can be injured during surgery. As a result, at the primary level, 1 cm from the apex of the milk incisor roots, the infraorbital foramen is situated, and the menton foramen, at the age of 4-6, is located closer to the root apex of the first primary mandibular molar.

Nerve Block Anesthesia and Field Block Anesthesia are less commonly used in children. The anesthetic agent is deposited near the main nerve trunks in the nerve block and around the principal terminal branches in the field block.

Indications for block anesthesia are as follows:

- dental treatment;
- removal of primary teeth with formed roots or root resorption of less than one-third of their original length;
- odontogenic subperiosteal abscesses; removal of several teeth.

In children, block anesthesia is used in the palatine, incisive, and mandibular foramina (directmandibular anesthesia, La Guardia or Akinosi type). Anesthesia in the maxillary tuberosity is not indicated in children.

The inferior mandibular nerve block is the local anesthesia technique of choice when treating primary or permanent mandibular molars in children. The depth of anesthesia is the primary advantage of this technique. For the inferior alveolar block, the child is asked to open his mouth as wide as possible while the operator positions the ball of the thumb on the coronoid notch of the anterior border of the ramus. The needle is inserted between the internal oblique ridge and the pterygomandibular raphe. The position of the foramen changes with the child's age. In young children, the foramen is located on the occlusal plane. but in young children (4 years old and younger), the foramen is sometimes located below the occlusal plane. As the child matures, it moves to a higher position.

The barrel of the syringe overlies the two primary mandibular molars on the opposite side of the arch and is parallel to the occlusal plane. In this case, a small amount of solution should be injected, and, after a negative aspiration, the needle should be advanced until bonecontact is made, very gently and slowly. When the inferior alveolar nerve block does not adequately anesthetize the teeth, long-term buccal anesthesia is required. This is achieved by infiltrating a few drops of the anesthetic into the buccal sulcus just posterior to the molars.

In children, anesthesia in the upper jaw is done very carefully, as the hard palate of the child is a flat and vulnerable bone that separates the oral cavity from the nasal cavity by a thin membrane. The inferior wall is located only 1.5 cm below the inferior wall of the orbit, where the dental buds are located. The hard palate does not have loose connective tissueexcept in the area around the palatine and incisive foramen.

Incisive anesthesia is used in different types of surgery in the anterior part of the jaw:

- extended jaw surgery on the alveolar gums;
- treatment of dental caries and its complications; treatment of dental trauma (luxation and fractures, compact osteotomies, removal of supernumerary teeth);
- root cysts;
- abnormalities of the upper lip membrane fixation.

The location of the incisive orifice can be easily determined visually. Taking into consideration that the area is highly reflectogenic, the mucosa is punctured 0.2-0.3 mm laterally from the incisive papilla. The needle should not penetrate deeperthan 0.2-0.3 mm.

Anesthesia at the level of the *palatine foramen* is indicated for surgeries performed at the level of thelateral jaw alveolar processes, the extraction of primary molars with root resorption not longer than a third of their length, odontogenic cysts, and odontogenic infections.

The projection of the palatine foramen changes asthe child grows. Horizontally, it remains constant at the junction of the alveolar process with the palatine one, and in the sagittal plane, it is located at the distal level of the last tooth crown (the fourth, then the fifth, the sixth, and the seventh primary ones).

The inferior alveolar nerve block (spix's spine) or direct inferior alveolar nerve anesthesia is administered at the mandibular orifice in children. It is indicated for:

- removal of primary teeth with root resorption of less than one-third of their length;

- odontogenic infections in the alveolar processes;
- odontogenic apical cysts;
- dental treatment;
- removal of dental buds.

The mandibular bone in children aged 3-4 years is twice as small as in grown-ups, and there is a small pterygomandibular space. As the fossa is small and packed with a large quantity of loose connective tissue and the nerves (inferior alveolar, buccal, and lingual) crossing the mandible space are located close to each other, the anesthetic solution spreads fast onto the space. The orifice of the inferior alveolar canal changes location once the child grows.

The direct technique of mandibular anesthesia is a type of inferior alveolar nerve block indicated forchildren. Keeping the mouth open, the operator's thumb is placed over the anterior border of the ramus, which helps to mildly retract tissues. The imaginary midpoint between the upper occlusal plane and the lower occlusal plane, posterior to the anterior border of the ramus, is the first site of the needle insertion. The syringe barrel is placed between the canine and premolar teeth on the contralateral side of extraction, and the needle is inserted into the selected site of needle insertion. The needle is advanced till it hits the bone, which is on the medial side of the ramus, behind the anterior border of the ramus. The ramus of the mandible is tightly held intraorally and extraorally (anterior and posterior border of the ramus) with the pollex and index fingers to avoid unexpected movements of the head during injection. The syringe barrel overlies the two primary mandibular molars on the opposite side, and the needle should advance anteriorly and posteriorly until bone contact is made, where the solution is deposited gently and slowly.

The La Guardia (V. Akinosi) nerve block type of anesthesia is a variation of direct mandibular anesthesia that is indicated for non-cooperating or potentially cooperating children. It is almost identical to the direct mandible technique. The mandible is held intraorally tothe anterior border of the mandibular ramus and extraorally to the posterior border of the mandibular ramus. The cheek soft tissue is retracted, and the mouth is closed. The imaginary midpoint between the upper occlusal plane and the lower occlusal plane, posterior to the anterior border of the ramus, is the first site of the needle insertion. The syringe barrel is placed parallel to the occlusal plane on the same extraction side. The needle is advanced in the anterior and posterior directions, keeping contact with the medial surface of the ramus. The syringe is parallel to the occlusal plane of the vestibule of the mouth, and the needle is advanced anteriorly-posteriorly by approximately 1-1.5 cm, maintaining contact with the internal surface of the mandible where the anesthetic is deposited. At the end of anesthesia, the child often opens his mouth, allowing the doctor to end it like a direct mandibular anesthesia.

Needle-free anesthesia through injection. This instrument was developed to achieve local anesthesiafor dental procedures without the use of a needle. This is accomplished by delivering the anesthetic solution under a high compressive force. A number of uncontrolled studies of needleless devices haveexamined both adult and pediatric patients, typically focusing on the anesthetic properties of the device used. The Bi-8 device has a high-pressure chamber (200 atm) and an orifice diameter of 0.15 ml, which is smaller than any needle used to inject the anesthetic. The anesthetic injection speed is 700 cm/hour, and 0.1-0.2 ml of solution are deposited. The indications for anesthesia are as follows:

- removal of primary teeth with root resorption no longer than one-third of their length;
- minor alveolar process surgery;
- minor soft tissue surgery;
- anesthesia in a pricking place.

The technique of anesthesia is simple: the orifice of the Bi-8 is fixed perpendicularly to the anesthetizing surface, and afterwards the trigger piston is pushed. It appears on the mucosa as an inflammation with a diameter of 1 cm and a small hemorrhagic lesion on the mucosa.

It is used to extract a tooth by injecting 0.8-1.2 ml of anesthetic into the region of the tooth root projection–two in the oral region and three in the vestibular region.

2.3. Composition of local anesthetics and maximum doses in children

1. *Novocaine* is a solution with a mild action. Its efficiency does not exceed 75%, and it is less active in an acid environment (in inflammatory processes) and in wounded tissues. Novocaine, in concentrations ranging from 0.25 to 0.5%, is used in children as young as one year old. At the age of 1-5 years, the solution of novocaine 0,5% is used in surgeries on soft tissues; novocaine 1% in jaw surgeries. After the age of 5 years, novocaine 2% is used in tooth extraction removal and jaw surgery. The dose of novocaine must be adjusted according to the child's age, and it must not exceed 1-1,5 ml (2%) at the age of 2-3 years. The dose of novocaine 1% must be doubled. At the age of 6-12 years, a dose of 4 ml (2%) of novocaine is allowed.

2. *Trimecaine* (0,25%, 0,5%, 1%, 2%) exceeds twice the efficiency of novocaine, 2-2,5 times the depth of anesthesia, and 3 times the duration of anesthesia. It has a sedative effect. A 1% anesthetic solution is sufficient for children under the age of 12.

3. *Lidocaine hydrochloride (HCl)* isa solution of the amide group. Its anesthetic efficiency is 90%. It has a strong vasodilation effect. Due to this, the effect of anesthesia is maintained only in association with solutions with a vasoconstriction action. Lidocaine hydrochloride (HCl) 2% with 1:100,000 epinephrine is preferred because of its low allergenic characteristics and greater potency at lower concentrations. The maximum dose of lidocaine and mepivacaine without vasoconstrictors recommended for children is 4.4 mg/kg per body weight and 7 mg/kg per body weight for lidocaine with vasoconstrictors. Anesthesia takes effect 5-10 minutes after administration. The average duration of pulpal anesthesia is 60 minutes for lidocaine 20% with 1:100,000 epinephrine, 50 minutes for mepivacaine 2% with 1: 20,000 levonor-

defrin, and 25 minutes for mepivacaine 3% without a vasoconstrictor. Due to local anesthetic agents used, the soft tissue anesthesia is greater than the pulpal anesthesia. Many attempts have been made to find agents that would reduce the duration ofsoft tissue anesthesia. However, no such reduction has been observed. Thus, it is recommended to use lidocaine 2% with 1:100,000 epinephrine when administering local anesthesia to young children. Solutions at a concentration of 0.25% or 0.5% are used only in the infiltrative anesthesia of soft tissues; solutions of 2% are used in the infiltrative anesthesia of soft parts. The solutions identical to lidocaine are mepivacaine, bupivacaine, etidocaine, and prilocaine. Lidocaine resembles trimecaine in its structure, but it is more efficient in the anesthesia of inflammatory diseases. There are rarely allergic reactions. The concentration of the solution and its quantity are indicated according to the age of the child. Up to the age of 5 years, 1ml (1%) of lidocaine solution is used for regional anesthesia, then 2 ml (2%).

If a local anesthetic is injected into an infection area, its onset will be delayed or even prevented. The inflammatory process in an infection area lowers the pH (normal value - 7.4) of the extracellular tissue to 5-6 or lower. A low pH inhibits the anesthetic action because little of the free base form of the anesthetic is allowed to cross into the nerve sheath to prevent the conduction of nerve impulses. Insertion of a needle into an active site of infection can also lead to the possible spread of the infection.

4. *Ultracaine* was introduced into medical practice in 1976. It is a derivative of thiophene, a weak base. It is neither water-soluble nor liposoluble. After solution hydrolysis, the formation of a liposoluble base that passes through the phospholipid membrane and reaches the nerve endings or branches causes the local anesthetic effect. The decreased liposolubility of the solution determines its minimal resorption in the blood flow and, hence, a decreased systemic toxic action. That is why it is used in children, the elderly, and pregnant women, as well as in patients with liver disorders. Its anesthetic efficiency is five times higher than that of novocaine and two to three

times higher than that of lidocaine and trimecaine. The anesthetic action starts 1-2 minutes after administration. The action time of ultracaine is 60 minutes without adrenaline and 2.5-3 hours with adrenaline. It has a high penetration capacity, allowing a wide range of indications. The anesthetic efficiency is 94%-100%. One in every 400,000 cases had a negative effect: hypotension – 0.26%, headache – 0.15%, nausea – 0.13% of cases. Ultracaine is used at a concentration of 4% with adrenaline (1:100,000). The maximum dose in grown-ups is 7 mg/kg (in patients with a body mass of 70 kg – 12.5 ml or 7 ampoules), in children aged 4-7 years, it is 5 mg/kg. Identical articaine-based anesthetic solutions are alifocaine, septanest, ubestezine, and ultracaine.

Table 1

Maximum recommended doses of anesthetics in children

Lidocaine 2% (Xilocaini, Octacaini) 20 mg/ml, 2.0 mg/1b 300 mg max
Lidocaine 2% with 1:100 000 epiphedrine20 mg/ml, 3,0 mg/1b 450 mg max

Body mass (kg)		Lidocaine 2% with 1.100000 epiphedrine
20	60 mg	1.5 ampoule max
40	120 mg	3.0 ampoule max
60	180 mg	5.0 ampoule max
80	240 mg	6.5 ampoule max
100	300 mg	8,0 ampoule max
150	450 mg	12,5 ampoule max
Mepivacaine 3%, prilocaine30 mg/ml 2.0mg/1b 300 mg max Mepivacaine 2% with 1:20000 Neo-Cobefrin20 mg/ml 3.0mg/1b 400 mg max		
Body mass (kg)	Carbocaine 3%	Carbocaine 3% with 1:20000 Neo-Cobefrin
20	40 mg 0,75 ampoule max.	60 mg 1,75 ampoule max.
40	80 mg 1,00 ampoule max.	120 mg 3,00 ampoule max.
60	120 mg 2,25 ampoule max	180 mg 5,0 ampoule max.
80	160 mg 3,00 ampoule max	240 mg 6,50 ampoule max.
100	200 mg 5,50 ampoule max	300 mg 8,00 ampoule max
150	300 mg 5,50 ampoule max	400 mg 11,00 ampoule max
Prilocaine 4% (Citanest) (40mg/ml) 3.0mg/ml3.0mg/1b 500 mg max Prilocaine 4% (Citanest Forte) (40mg/ml) 4.0mg1b 600 mg max		

Body mass (kg)	Citanest 4%	Citanest Forte 4%
20	60 mg 0,75 ampoule max.	80 mg 1,00 ampoule max.
40	120 mg 1,50 ampoule max.	160 mg 2,00 ampoule max.
60	180 mg 2,50 ampoule max.	240 mg 3,25 ampoule max.
80	240 mg 3,25 ampoule max.	320 mg 4,25 ampoule max.
100	300 mg 4,00 ampoule max	400 mg 5,50 ampoule max.
150	450 mg 6,00 ampoule max	600 mg 8,00 ampoule max.

The dose of the local anesthetic depends on the patient's physical condition, the area to be anesthetized, the vascularization of the oral tissues, and the technique of administration. It is difficult to recommend a maximum dose for children because the dose varies with age and weight. For pediatric patients under 10 years of age with lean body mass and normal body development, the maximum dose may be determined by applying one of the standard formulas (Clark's rule). In any case, the maximum dose should not exceed 7 mg/kg per body weight for lidocaine with epinephrineand 4.4 mg/kg for plainadrenaline.

*Clark's Rule.*This method is used when either the manufacturer has not recommended dosagesfor children or the prescriber has requested that it be used. This can be accounted for by the fact that children vary in weight, size, tolerances, etc. Clark's Rule uses weight in Lbs, never in Kg.

- Adult Dose X (Weight ÷ 150) = Child's Dose
- *Example:* 11-year old girl/70 Lbs 500mg X (70÷150) = Child's Dose 500mg X (.47) = Child's Dose 500 Mg X. 47 = 235mg

The efficiency and duration of the local anesthesia may be prolonged through the administration of adrenaline solutions. Adrenaline has a vasoconstriction action, increasing the blood pressure and stabilizing the glucose. A drop of adrenaline, 0.1% per 5-10 ml of anesthetic, increases the duration of anesthesia. The predominance of sympathetic innervation in children aged up to five years does not allow the use of adrenaline solutions. The administration of adrenaline solutions at this age may have harmful effects, causing an increased pulse, high blood pressure, heart rhythm disorders, ventricular fibrillation, and stenosis of blood vessels in the abdominal organs and skin, manifested by tremors, wanes, stickyand cold sweats, and fainting. Adrenaline is not recommended for children under the age of five. In children above the age of five years, it may be administered at a concentration of 1:1000, with a peculiar precaution, as the abundant vascularization of the area increases the absorption capacity and an overdose can produce the above-mentioned effects.

2.4. Pain management in children

Unfortunately, neither the various methods of anesthesia nor the numerous anesthetic solutions radically solve the problem of anesthesia in pediatric dentistry because they fail to completely remove or depress the child's emotional state. In non-cooperating children, local anesthesia does not respond adequately. Confidence and mutual collaboration between the little patient and the dentist should be based on a pre-surgical sedative preparation that includes physiologicaldistraction and psychotherapy.

Psychotherapyis indicated for all children requiring dental care. The psychological preparation of the child should begin with his/her appointment with the dentist. The parents are the first to inform the child about the need for dental treatment. The first session should involve the diagnosis, allowing the child to get acquainted with the staff and the environment. The collaborators must be gentle, calm, and smiling, paying particular attention to the littlepatient. During the conversation with the dentist, the collaborators must take anactive part, and the movements within the space related to the preparation of the tools must be minimal and well hidden. The discussions between the staff should be avoided. Verbal communication should focus on different subjects, such as clothes, friends, siblings, and children's favorite TV series. Nonverbal communication in young children consists of playing games with a soft and friendly voice until the child smiles and becomes confident. The treatment in teams with other children, especially with cooperative children, and the treatment performed by a single physician have a good psychotherapeutic effect.

Parents are not required to accompany their children during dental treatment because they may impose or coerce behavior on the child that interferes with direct communication between the dentist and the child. Sometimes, the physician's voice may be considered an offense by the parents, and the child shares his attention between the parents and physician. Parents should collaborate with the physician, or if their presence modifies the child's behavior in some way, especially in the case of children under the age of three, the child should be accompanied to treatment. As the child becomes more interested in the words of the parents, physician, and assistant, the parent should gradually step aside, and the assistant should sit between the child and the parent to continue communication.

The physiological distraction, one of the psycho-behavioural approaches used in medical and dental treatment situations, is defined as a non-aversive approach used to modify a child’s discomfort by disrupting his/her attention away from the main task to accomplish successful treatment with high quality.

The physiological distractionis based on the formation of a dominant focus in the cerebral cortex, realized through the action of factors on the visual and auditory analyzers. For this purpose, the dental office should look like a home.

Distraction during dental treatment is beneficial to patients by reducing their distress and, in turn, decreasing their perception of pain sensation, especially during injections with local anesthesia. It has been stated, however, that the ideal distractor ought to possess various abilities such as visual, auditory, and kinesthetic modalities (i.e. physical movements) to provide the full capacity to harness the child’s concentration and attention, in turn minimizing the child’s anxiety. The use of audiovisual distraction not only leads to full involvement in scenes (visual and auditory), but it also induces a positive emotional reaction, resulting in a relaxed experience. For this purpose, the dental office should look like a home; the walls in the waiting room should be painted in bright colors with pictures of fairy tale heroes. Books, toys, colored floor mats, musical instruments, a nature corner (decorative fish, birds, etc.), audio and video devices, a collection of music and movies, and child-friendly setups should be available in the waiting room. Distraction is a useful tool to decrease distress and dental anxiety during dental treatment.

The dentists must do their activity with precision, taking care that young patients do not wait too long and do not hear any crying or screaming from the treatment ward. Children subject to treatment should not meet children awaiting treatment. Therefore, both the entrance and the exit must be isolated.

*Pre-Anesthetic Medication*aims at preventing and removing the complications and adverse reactions caused by anesthesia and surgery, diminishing anxiety, and modifying children's behavior during dental care, creating a calm atmosphere between the child and physician, facilitating the child`s induction during general anesthesia, decreasing the concentration of toxic substances, preventing the unwanted exaggerated reflexes during anesthesia and surgery, and diminishing the secretion of the salivary glands and bronchi.

The response of children to pre-anesthetic medication may be different. Some children become sleepy, indifferent, unresponsive, or indolent depending on their psycho-neurological characteristics, while others become excited, nervous, steered, or roused. Serious complications, such as central nervous system depression leading to bradycardia, hypoxia, and respiratory arrest, can occur in rare cases.

To prevent the above-mentioned states, there are some general principles for administering the pre-anesthetic medication: 1) the dentist requires special training in order to use sedative preparations and provide emergency airway assistance 2) the presence of necessary equipment in the case of acute cardio respiratory failure; 3) medical history; 4) compulsory monitoring of vital organs during the pre-anesthetic medication; 5) the administration of doses according to the child's age.

The indications for pre-anesthesia medication are as follows: children aged up to 5 years; children requiring therapeutic or surgical treatment from I and II ASA groups, accompanied by parents; children with developmental disorders, mental and physical retardation; non-cooperative children and those who poorly adapt; children with dent phobia and 2^{nd} and 3^{rd} degree behavior; and children requiring long-lasting treatment. The contraindications to pre-anesthesia are as follows: children with organic disorders at the stage of decompensation and children below the age of 1 year.

Medication may be conscious or unconscious. *Conscious Sedation* is a medically controlled state of depressed consciousness that allows protective reflexes to be maintained, retains the patient's ability to maintain a patent airway independently and continuously, and permits an appropriate response by the patient to physical stimulation or verbal command.

In dental offices, conscious sedation is less dangerous as it reduces the level of consciousness depression, allowing the patient to maintain respiration independently, react to physical agents, and accomplish verbal orders.

The medication used in conscious pre-anesthesia can result in minimal or moderate sedation. *Minimal Sedation (Anxiolysis)* is a drug-induced state during which patients respond normally to verbal commands. Although cognitive function and physical coordination may be impaired, airway reflexes, and ventilatory and cardiovascular functions are unaffected. *Moderate Sedation/Analgesia („Conscious Sedation")* is a drug-induced depression of consciousness during which patients respond purposefully to verbal commands, either alone or accompanied by light tactile stimulation. No interventions are required to maintain a patent airway, and spontaneous ventilation is adequate. Cardiovascular function is usually maintained.

Patients under conscious sedation must be monitored continuously clinically, which includes: the patient's response to physical stimulation and verbal commands; observing breathing, movements of the thorax, passage of the air stream, respiratory frequency, observing skin colour.

Various drugs in low doses can be used for conscious sedation (lightly sedated patient, who is awake, cooperative on demand, amnesic, and free from anxiety and fear to meet these). Among these are phenothiazines, butyrophenones, barbiturate and non-barbiturate hypnotics, benzodiazepines, and the hypno-analgesic, ketamine. Diazepam has been the 'gold standard' of sedation, but the more modern benzodiazepines, particularly midazolam, are now more commonly used. Because benzodiazepines offer both sedative and profound amnesic and anxiolytic effects, these drugs are used for conscious sedation worldwide.

Deep sedation or analgesia is a drug-induced depression of consciousness during which patients cannot be easily aroused but respond purposefully following repeated or painful stimulation. The ability to independently maintain ventilatory function may be impaired. Patients may require assistance in maintaining a patent airway, and spontaneous ventilation may be inadequate. Cardiovascular function is usually maintained. This type of sedation produces a strong and long-lasting depression, thus requiring bed rest and the physician`s surveillance for 2-3 hours, and it is indicated mainly on an inpatient basis. It is achieved by the administration of a „lytic cocktail", which is a mix of medications, each of which has a different pharmacological effect.

2.5. General anesthesia in children. Complications of local anesthesia

General anesthesia in dentistry temporarily suppresses sensitivity through a reversible, controlled inhibition of the central nervous system and may be administered by inhalation, intravenously, or intramuscularly.

In dentistry and oral and maxillofacial surgery, there are multiple peculiarities and risks associated with general anesthesia. The main difficulties and risks for the anesthesiologist are outpatient conditions, patients' sitting position, difficulties in surveilling eyeball movements, palpebral and pupillary reflexes, dental manipulations in the oral cavity, which may compromise airways (secretions, clots, foreign bodies such as dental dust, teeth, filling material), gauze sliding into the pharynx, the tongue falling posteriorly into the pharynx, and the posterior position of the mandible.

The classification of the child's physical status according to the American Society of Anesthesiologists (ASA) contributes to general anesthesia indications in children: in the first group, there are no organic, psychological, or biochemical disorders. They are outside- the-system diseases. Planned surgeries are local and do not enter the category of systemic diseases. Children in outpatient conditions may be operated on without any restrictions. The second group includes moderate systemic disorders caused by the conditions under which children must be operated on or due to some pathomorphological processes. Outpatient surgery can be performed only after the examination of systemic diseases. The third group entails severe systemic disorders or diseases caused by any of them, although it is not possible to determine definitively the degree of disability. Patients have some restrictions on daily physical activities. Surgery is performed after a thorough examination and under special post-surgical conditions; the fourth group includes patients with severe systemic disorders in compensation status with permanent treatment; the fifth group comprises patients with extremely severe health conditions, in decompensation, in emergency situations, or who are moribund.

The indications for general anesthesia in children are as follows**:** major medical issues, congenital heart defects, hematological diseases (hemophilia), or other conditions requiring urgent dental treatment; allergic reactions to local anesthetics; young age (children as young as three years old); neurological and mental disorders; multiple caries requiring multiple treatment sessions; behavioral disorders, third- or fourth-degree dentophobia, anxiety, poorly adaptive and non-adaptive (aggressive and negative behavior) children in whom the treatment with local anesthesia failed; dental and extended oro-facial trauma; children from rural areas where dental care is not accessible and there are transport problems; infants and children with associated craniofacial anomalies; children and teenagers neglecting the dental treatment.

Outpatient general anesthesia is indicated for children in the ASA's first and second groups with serious dental diseases whose parents are capable of providing presurgical and postsurgical care. Outpatient general anesthesia is indicated for children in the ASA's first and second groups with serious dental diseases whose parents are capable of providing presurgical and postsurgical care. Patients of the 3rd, 4th, and 5th groups (ASA) are candidates for inpatient general anesthesia.

The contraindications to general anesthesia are relative and temporary. The contraindications to outpatient general anesthesia are as follows: acute contagious diseases, acute herpetic stomatitis, acute viral respiratory infections; constitutive abnormalities, external airway obstruction (deviated nasal septum, adenoid vegetations, chronic rhinitis); children of the 3rd, 4th, and 5th groups (ASA).

General anesthesia in the dental office. The main prerequisites for administering general anesthesia to children are that the dental office be located in a regional, district, or republic outpatient department and have a radiological office and trained nurses. The department of anesthesiology must be located in at least two offices. One must be intended fordental treatment and the other for rehabilitation after surgery or dental treatment under general anesthesia.

The medical staff must include a pediatric dentist, anesthesiologist, and trained nurses, who have to meet the highest professional requirements.

The dentist has to be highly qualified and skilled in endodontics and have surgical knowledge (dental extractions and emergency care for inflammatory processes). During the manipulations under general anesthesia, the dentist faces some difficulties, such as the lack of a patient’s reaction during the dental treatment, the need to treat in a single session, and the impossibilityof taking radiological pictures during the treatment.

Complications of loco-regional (block) anesthesia in children. Fear-related behaviors have long been recognized as the main issue in patient management and can be a barrier to good care. Nerve block anesthesia may produce local or general, mild or severe, primary or secondary, transitory or permanent complications.

Complications of local anesthesia.

Soft tissue injury is a clinical complication following local anesthetic use in the oral cavity and its self-induced soft tissue trauma. Most lip- and cheek-biting lesions are self-limiting and heal without complications, although bleeding and infection are likely to arise. They may appear after mandibular anesthesia because children may intentionally or accidentally bite their lips or cheeks. Clinically, a swelling of the lower lip or cheek may appear on the anesthetized side with oral mucosa erosions. To avoid this, protective measures or gauze application on the lower lip during the entire postanesthetic period are necessary. For this reason, long-acting local anesthetics (i.e., bupivacaine) should not be used on children or physically or mentally disabled patients due to their prolonged effect, which increases the risk of soft tissue injury. Caregivers responsible for postoperative supervision should be given a realistic time frame for the duration of numbness and informed of the possibility of soft tissue trauma.

*Paresthesia*is persistent anesthesia beyond the expected duration. Injuries to the inferior alveolar nerve and lingual nerve can be caused by local analgesic block injections. A nerve injury may be physical (from the needle) or chemical (from the local anesthetic solution). The patient may experience an „electric shock” in the involved nerve distribution area. Paresthesia can also be caused by hemorrhage in or around the nerve. Reports of paresthesia are more common with articaine and prilocaine than expected from their frequency of use. Most cases resolve in eight weeks.

*Needlebreakage*during anesthesia is a dangerous accident and is usually common in anxious children. Once a child has grabbed the syringe or bumped the operator's hand and driven the needle into the bone tissue, it may be too late to respond, and a lasting impression has been made in the child's mind with regard to the pain associated with the local anesthetic injection. In such cases, the needle can be broken and slipped into the pharyngeal mucosa, or it can be aspirated, causing swelling of the nasopharyngeal mucosa and asphyxia. In order to avoid this, the doctor should be assisted by a skilled nurse. The practitioner should have control of the child's head and a good finger rest to control the syringe in case the child moves or resists. The dental assistant should be ready to restrain the child's hand gently but firmly.

Overdose reactions are a particular risk when treating children. Overdose reactions are determined by the amount of vasoconstriction (adrenaline) and anesthetic solution. The dosage of the local anesthetic depends on the physical status of the patient, the area to be anesthetized, the vascularity of the oral tissues, and the technique of administration. Toxicity occurs primarily in the cardiovascular, respiratory, and central nervous systems. The toxic reaction could stimulate or depress the central nervous system.

Stimulation of the central nervous system can cause a toxic vasoconstrictor reaction, and the signs and symptoms are tachycardia, apprehension, sweating, and hyperactivity. Depression of the central nervous system may follow, causing bradycardia, hypoxia, and respiratory arrest.

Local anesthetic toxicity can be prevented by careful injection technique, watchful observation of the patient, and knowledge of the maximum dosage based on weight. Practitioners should aspirate before every injection and inject slowly. Early recognition of a toxic response is critical for effective management. When signs or symptoms of toxicity are noted, administration of the local anesthetic agent should be discontinued. Additional emergency management is based on the severity of the reaction.

The toxicity of vasoconstrictor solutions is manifested by palpitations, tremors, headaches, dizziness, restlessness, and breathing difficulties. Anxiety, pallor, „chicken skin”, cold sweats, vomiting, tachycardia, hypertension, and loss of consciousness are all symptoms of high doses.

Epinephrine is contraindicated in patients with hyperthyroidism. Its dose should be kept to a minimum in patients receiving tricyclic antidepressants since dysrhythmias may occur. Its dose should be kept to a minimum in patients taking tricyclic antidepressants because dysrhythmias can occur.

Levonordefrin and norepinephrine are absolutely contraindicated in these patients. Patients with cardiovascular disease, thyroid dysfunction, diabetes, or sulfite sensitivity, or those receiving monoamine oxidase inhibitors, tricyclic antidepressants, or phenothiazines, may require a medical assessment to determine whether a local anesthetic without a vasoconstrictor is necessary.

These complications can be prevented by considering that emotions and pain determine the overdose of adrenaline on the one hand, and the sympathetic system predominates up to the age of 5 years on the other hand. That is why it is not recommended to use adrenaline in local anesthesia up to the age of 5. After the age of 5, adrenaline is administered at a concentration of 1:1,000. Although children at that age tolerate this concentration of adrenaline better, it should be administered with caution.

The treatment consists of inhaling amyl nitrate (5-10 drops), papaverine or miofilin, diazepam, or short-acting barbiturates, administered by injection.

Toxic shock is caused by the overdose of the anesthetic solution in block and infiltration anesthesia. Extremely abundant vascularization causes fast resorption, sometimes with serious consequences.

The first clinical signs are manifested by the pallor of the teguments, sweating, mydriasis, nausea, imminent vomiting, cyanosis, the presservation of consciousness, an accelerated pulse, low blood pressure, superficial respiration, and a low temperature. After a short period of excitation of the central nervous system, there is inhibition, leading to consciousness, a state of collapse, and convulsions. In severe cases, cardiorespiratory failure may occur.

When there are signs or symptoms of toxicity, the administration of the local anesthetic solution should be discontinued. After the airways have been cleared, the patient is placed in a lateral position, with the head extended and the mandible in the anterior position.

Allergic reactions to local anesthesia are rare. The local anesthetic agent with the highest incidence of allergic reactions is procaine. Its antigenic component appears to be para-aminobenzoic acid (PABA). Cross-reactivity has been reported between lidocaine and procaine. Clinical symptoms of allergy include urticaria, where the anesthetic is injected, dermatitis, angioedema, fever, photosensitivity, or anaphylaxis, intense itching, skin rash, rhinitis, and asthma. Angioneurotic edema (Quincke's edema) can manifest with swelling of the lips, tongue, pharynx, and eyelids and can compromise the airways rapidly, potentially leading to a life-threatening situation.

Patients with a history of allergy to a local anesthetic who cannot identify the specific agent used present a problem. The patient should be referred for evaluation and testing, which will usually include both skin testing and provocative dose testing (PVT). For patients who have an allergy to bisulfates, the use of a local anesthetic without a vasoconstrictor is indicated. Local anesthetics without vasoconstrictors should be used with caution due to their rapid systemic absorption, which may result in overdose.

Anaphylactic shock, also called anaphylaxis, is triggered by contact with the host with a very small amount of allergen, contact that can be achieved by ingestion, inhalation, injection, or by simply approaching the allergenic substance. The most severe type of allergy is anaphylactic shock, which occurs as a result of a strong immune response related to the release of high amounts of immunological mediators (histamine, prostaglandins, etc.) from mast cells and basophils. The effects of these mediators have adverse consequences on the host: peripheral and central systemic vasodilation, which leads to a sudden decrease in blood pressure, and edema of the bronchial mucosa, which causes bronchospasm. The causes of anaphylactic shock can be certain drug substances, some food products, and insect bites (bees, wasps, and red ants). There are also factors whoseanaphylactic potential is reduced, and the incidence of an allergy in the case of exposure to them is minimal, like latex, acetylsalicylic acid (aspirin), and non-steroidal anti-inflammatory drugs (ibuprofen, naproxen). The entire range of symptoms is due to the release of histamine-type mediators as a result of immunoglobulin E activity.

Anaphylactic shock may develop as: a) dermal (skin rash, itching, skin hyperemia, Quincke's edema); b) neurological (acute headaches, hyperesthesia, vomiting, paresthesia, convulsions); c) asthma (asphyxia); d) cardiogenesis (myocarditis, myocardial infarction); e) abdominal (involuntary urination, polyuria, abdominal pain).

The first symptom is general malaise. Pulmonary edema, dyspnea, bronchospasm with breathing difficulty, dysphonia, palpitations, low blood pressure, dysphagia, abdominal cramps, vomiting, diarrhea, polyuria, encephalitis, urticaria, itching of the lips, palate, hands, eyes, and feet, redness of the skin (due to stress), and collapse are all symptoms of anaphylactic shock.

Anaphylactic shock occurs from a few seconds to 2 hours after exposure to the allergen. The faster the reaction, the worse the child's condition becomes. There are three levels of anaphylactic shock severity, according to this criterion. The first degree develops from a few minutes to two hours after the contact of the host with the allergen and is characterized by hypotension, tachycardia, hyperemia, itching (especially inthe mouth, lips, and throat), facial swelling, periorbital edema, skin rash, mild rhinorrhea, and wheezing.

The second degree involves the response appearing within minutes of contact with an allergen. The anaphylactic reaction is systemic, i.e., it is not limited to one area of irritation. Clinical events are more widespread. Sudden drops in blood pressure cause tachycardia, rapid pulse, weakness, arrhythmia, chest pain, dizziness, pallor, and mental confusion. Vomiting, abdominal cramps, bloody diarrhea, involuntary urination, and polyuria can sometimes occur. Anxiety, dizziness, extreme tiredness (lethargy), and vision disorders can also occur.

The third degree (fulminant) appears immediately following allergen factor contact. Suddenly, collapse appears (pallor, cyanosis, decrease in blood pressure, imperceptible pulse), followed by an agonal state (pallor, mental confusion and unconsciousness, mydriasis). Thus, in the absence of treatment, the impossibility of breathing can lead to death.

A history of allergic status and chronic conditions is taken as part of the prophylaxis. Treatment must be prompt and effective. The administration of the anesthetic should be immediately discontinued. The medical treatment consists of intravenous H1 and H2 histamine blockers, parenteral and inhalational corticosteroids, beta-2 mimetics like albuterol or salbutamol, and nebulized or topical epinephrine. In severe cases, parenteral epinephrine should be considered, and the child should be taken to the nearest hospital's emergency department. Meanwhile, cardiorespiratory resuscitation should begin immediately. The child's tight clothes have to be taken off. The child should be calmed, as he is probably scared and does not understand what is happening. The child is held upright, or as upright as possible, to help him breathe. In the case of vomiting, the child is put on one side. The superior airways are cleared of saliva, clots, and foreign bodies, maintaining their permeability; the abdomen and the extremities are intensively massaged. The blood pressure is takenevery 3-4 minutes, and continuous oxygen is administered. If the child loses consciousness and stops breathing, mouth-to-mouth resuscitation is performed. Locally, solutions that reduce the absorption capacity of the anesthetic are administered: adrenaline solutions (0,15-0,75 ml in 2-3 ml of physiological solution), local mucosal or skin washes with weak antiseptic solutions. Adrenaline solution (1:1000) is given intramuscularly or intravenously (0,1-0,3 ml), which helps to open the airways and improve breathing, raises blood pressure, and reduces allergic reactions in the body. Hormones are administered intravenously (prednisolone 3%, 2-5 mg/kg, dexamethasone 0,004 g, 0,05-2 mg). Plasma restitution preparations (polyglucinum, rheopulyglucinum, 10-15 ml/kg) are administered to increase circulating blood volume. In the case of tachycardia, cardiac preparationsare indicated (strophantinum 0,05%-0,06%, 0,4-1,5 ml or corglycon 0,06%, 0,1-0,075 ml). In bronchiolitis, antihistamines (dimedrol 1%, 0,1-1,5 ml, suprastin 2%, 0,1-1 ml), ephedrinum hydrochloridum 5%, 0,1-1 ml) are given. In glottic and cerebral edema, manifested by seizures, patients are given fast-acting diuretics (furosemide 2%, 0,03-0,02 ml/kg).

Lipothymia is characterized by short-term cerebral vascular failure, most often caused by the hypotensive action of anesthetics. It often occurs during adolescence due to vasomotor instability and hypotonia in the case of dentophobia. Lipothymia can be caused by a variety of factors, including a sudden shift from horizontal to vertical position, prolonged bleeding, non-ventilated rooms, the action of hypotensive preparations, hyperglycemia or hypoglycemia, and vagus nerve irritation caused by neck constriction. Clinically, it is presented as a loss of consciousness, decreased breathing, and low cardiac activity. It may be preceded by the tendency to get sweaty, facial paleness, and sometimes nausea. When it suddenly occurs, it causes a partial loss of consciousness, a pale face, cold sweats, and weakness. The initially increased pulse becomes weak and irregular. Blood pressure drops, breathing becomes shallow with a diminished rhythm.

Syncope is a temporary state of vascular and heart failure. It is a rare accident that occurs suddenly or is the continuation of lipothymia. Loss of consciousness is total. Syncope can appear in blue (respiratory) form, with cyanosis caused by stopped breathing; the heart continues to beat; and the pulse is imperceptible. White (cardiac) syncope is characterized by sudden cardiac arrest, the absence of pulse and cardiac noise, a drop in blood pressure, general pallor, and capillary cyanosis.

The medication is given urgently in order to ensure cerebral oxygenation and the activity of vital organs. The injection of the anesthetic is stopped immediately. The patient is placed horizontally, with the head lower and the neck in hyperextension. In order to ensure better irrigation of the brain centers and to facilitate breathing, the clothes from the throat, chest, and abdomen are removed. The face is splashed with water, and amyl nitrate (5-10 drops) is inhaled. In severe lipothymia, oxygen and heart medications are administered (coffeinum natrii benzoas 10%, cordiaminum 0,1 ml orally).

Cardiorespiratory resuscitation should be started as early as possible, prior to the developmentof organic lesions, correctly and in order of priority. Resuscitation measures are carried out in three stages: 1) ensuring the freedom of airways, as well as breathing and circulation; 2) maintaining vital functions throughtreatment, electrocardiographic monitoring, and defibrillation, or electrical stimulation; 3) supporting vital functions by assessing the general condition and the cause of cardiac arrest, identifying indications for therapy continuation, providing neuronal protection and restoration, and using intensive therapy to improve the immediate effect of resuscitation.

Learning objectives

1. Use of topical, infiltration and regional anesthesia in children. Techniques.
2. Use of general anesthesia in outpatient children with odontogenic problems.
3. Indications for medication on an outpatient basis in children with odontogenic problems. Medications usedon an outpatient basis in the dental office.
4. Direct inferior mandibular nerve block anesthesia. La Guardia (Akinosi) anesthesia, indications.
5. Complications of local anesthesia. Anaphylactic shock. Clinical manifesttations, treatment.
6. Syncope. Clinical manifestations, treatment.
7. Anatomical and physiological peculiarities of the upper and lower jaws that allow for infiltration anesthesia in the pediatric population. Indications for infiltration anesthesia. The technique of infiltration anesthesia in the pediatric population
8. Peculiarities of the pterygomandibular fossa in children depending on age and their role in inferior mandibular block anesthesia.
9. Anesthetic overdose in children. Clinical manifestations of toxic shock and overdose toxicity.
10. Anatomical landmarks of infraorbital, mental, and palatal orifices in children according to the age of development. Peculiarities of tuberal anesthesia in children.

TESTS

1. SC. In children, the sympathetic nervous system predominates at the following ages of development:

A. under 5 years old;
B. up to the age of 3 years;
C. 7-12 years;
D. 3-7 years;
E. after 12 years.

(A)

2. SC. The inferior alveolar mandible foramen, located 6 mm above the masticating surfaces of inferior molars, is characteristic for the following age periods:

A. 1-3 years;
B. 3.5-4 years;
C. 9 months – 1.5 years;
D. 12 years;
E. 9 years.

(E)

3. SC. What factors can cause toxic shock:

A. food ingredients;
B. anxiety;
C. sharp pain;
D. overdose of the anesthetic solution;
E. immature lymphatic system.

(D)

4. MC. In which medical conditions should general anesthesia be used inthe dental office:

A. mental retardation;
B. compensated hereditary or acquired cardiac disorders;
C. diabetes;
D. allergy condition of the host;
E. contagious diseases.

(A, B, D)

5. MC. What are the clinical manifestations of anaphylactic shock:

A. breathing difficulties;

B. B.low blood pressure;
C. nausea, vomiting
D. unresponsiveness
E. mydriases.

(A, B, C)

6. MC. In whichemergencyconditions should general anesthesia be used in the dental office:
A. frenuloplasty of the upper lip;
B. osteoplasty;
C. arthroplasty;
D. acute dental trauma;
E. acute odontogenic infection.

(D, E)

7. MC. Which side effects arise from adrenaline overdosage in children up to the age of 5 years:
A. dry mouth;
B. agitation;
C. hypertension;
D. tachycardia;
E. dysrhythmia.

(B, C, D, E)

8. MC. What are the time periods of intensive physical growth of the mandible in children:
A. the first month of life - up to the age of 12 months;
B. 1 to 2 years old;
C. 9-12 years old;
D. 15-17 years old;
E. 3-7 years old.

(B, C)

9. MC. What is the goal of premedication in the pediatric dental office:
A. to reduce inapprehension;
B. to prevent aspiring extracted teeth, root remains;
C. to decrease oral secretions;
D. to prevent exaggerated reflexes during anesthesia;
E. E.to eliminate anxiety.

(A, C, D)

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3. DENTAL EXTRACTIONS IN PERMANENT AND PRIMARY DENTITION

A dental extraction is a procedure that is performed with the purpose of removing a tooth from its alveolus, which can no longer be recovered by conservative treatment methods.

This type of surgery is performed frequently in dental practice. In children, the principles of extraction of a permanent and a deciduous tooth are the same. But care must be taken, keeping in mind the anatomical and physiologic differences between an adult and a child. However, it would be a mistake to consider it only a minor intervention, which can be carried out without any serious preparation and without knowing its indications, contraindications, and technique. Prior to dental extraction, a general and local thorough examination based on the modality of the dental extraction surgery chosen is performed, followed by post-extraction assistance.

3.1. Peculiarities of dental extractions in children

Dental extractions are performed in relation to the child's age, with the periods of primary tooth root formation, apex closure, and tooth resorption and exfoliation. The same tools are used in dental surgery on children as in elderly patients, but the tools are smaller to accommodate the size of the oral cavity. The surgical instruments designed for procedures on adults can be used effectively on children, although forceps specifically designed for a child's mouth are also available. They are small, easy to conceal, and less frightening. Forceps such as cowhorns and elevators that, by virtue of their design and function, threaten the underlying, developing permanent teeth, should be avoided. Narrow root picks, mosquito forceps, and dental excavators are the instruments of choice for root removal to prevent extraction of neighboring teeth or neighboring dental buds.

During the extraction, care must be taken. The dentist should explain to the child the sensations and feelings before the administration of the local anesthetic. The child should be briefed on the extraction procedure in simple language so that he or she is prepared and ready for the procedure. The uninformed child can interpret the sensation of pressure from the forceps during the extraction procedure as pain, and extraneous noise and osseous sound conduction associated with luxation may aggravate anxiety.

*Dental extraction*s are carried out in six stages: two additional and four basic: 1) syndesmotomy; 2) application; 3) insinuation; 4) fixation, 5) dislocation, 6) removal, 7) curettage of the alveolar wound. In order not to damage the permanent buds during the extraction of the primary teeth, syndesmotomy and deep insertion are not indicated (only up to the cervical part of the teeth).

Simple extraction of the milk tooth. The extraction of the milk tooth by means of the clamps is done by the doctor after adequate anesthesia has been performed, which would allow a painless tooth extraction. In order to stabilize the mandible preoperatively, the thumb and the index finger are placed on the alveolar processes at the level of the tooth to be extracted, fixing it firmly but gently and at the same time providing support under the mandible with other fingers. In this way, the movements of the child's head during manipulations will be avoided. When scheduling multiple dental extractions, the teeth must be marked to avoid errors.

Primary incisors and canines can be luxated slightly with the periosteal elevator, especially if the tooth is mobile. Firm apical pressure is maintained with the forceps. Gently direct the initial luxation lingually or palatally and carefully towards the labial side. A rotational force is applied along the long axis of the tooth, delivering it through its path of least resistance. Mold the labial, lingual, or palatal plates of the alveolar bone into normal conformity with digital pressure. Fold and place a sterile gauze sponge over the wound to help establish hemostasis. Do not allow the patient to leave until the bleeding is over.

Primary molars can be luxated slightly with the periosteal elevator, especially if the tooth is mobile. Firm apical pressure is applied with the forceps. The initial luxation movement is toward the buccal side. Hold pressure momentarily, permitting buccal alveolar plate expansion. The luxating force should be returned lingually and palatally, as well as slightly rotating the maxillary teeth, vestibularly and lingually, and slightly rotating the mandibular teeth. Hold pressure to permit lingual alveolar plate expansion. Alternate the buccal and lingual/palatal movements to further expand the cortical plate. When there is adequate freedom of movement, deliver the tooth to the buccal side, exercising slow, firm, continuous pressure. Labial and palatal cortical plates should be molded so as to conform to finger pressure after extraction.

Hemostasis of the alveolar wound after extraction is usually performed with a gauze sponge. Post-extraction sutures are performed for hemostatic reasons in children with mental disorders and multiple extractions, as well as in very young children and those from rural areas.

The technique of dental extraction in milk dentition according to the type of root resorption. In order to avoid possible complications during the extraction of milk teeth, the breaks of the extraction forceps are inserted in relation to the root resorption. There are two types of resorption: (physiological and pathological) and three types of physiological resorption: 1) uniform root resorption; 2) vertical resorption (external); and 3) horizontal resorption (internal).

External resorption is more common than internal resorption and can affect any part of the outside of the root, from the roots to the cementum on the outside. On the outside, teeth look like they have deep holes or chips. Resorption affecting the roots of the tooth can be seen on X-rays as a shortening of the root length and a flattening of the root tips. In primary dentition, the radiological picture shows resorption of one root in a vertical plane, with the other remaining complete, and the dental buds located beneath the resorbed root.

Internal resorption affects the inside of the tooth. It is much less common than external resorption. On X-ray, a tooth will show a dark spot where internal tissue is missing. Internal resorption on X-ray pictures in the primary dentition shows that the dental buds are located at the bifurcation of the milk teeth. In both cases, the implantation of the milk teeth is firm, but the breaks must be inserted only up to the cervical part of the tooth and located on an unresorbed root. Root fracture, contusion, injury or removal of the tooth bud, and injury of neighboring teeth are common complications of milk teeth extraction.

Specific problems of dental extraction in children

Natal and neonatal teeth. Teeth that are present in newborns and teeth that appear in the first month of life are called *natal* or *neonatal* teeth. 5% of these teeth are supernumerary. 85% of the natal and neonatal teeth are located in the jaw, in the lower incisors. They appear in pairs, symmetrically. Natal and neonatal teeth are characterized by enamel dysplasia, unformed roots, and poor implantation. Therefore, they are movable and can fall by themselves. The tooth is a source of mechanical irritation, causing gingivitis and ulceration on the ventral surface of the tongue and frenulum (Riga-Fede disease).

When the prenatal teeth cause ulceration with their sharp edges, they should be gently ground with a stone or diamond. If no improvement is obtained, they must be removed. The marked mobility of these teeth is an indication of their removal. It is usually recommended to remove them 8 to 10 days after the baby's birth to prevent oral injuries.

Atypical resorption of primary teeth. Vertical or horizontal resorption is often the cause of the apex fracture during extraction surgery. If the apex is visible, it is carefully removed. If the erupting tooth is located very close to the fractured apex, it is left in the bone. Gradually, it can appear, and sometimes it erupts with the permanent tooth. If it fistulates until the permanent tooth erupts, it will be removed. Sometimes, its removal can damage the permanent dental buds, manifestingas hypoplasia and dilaceration of the crown.

Ankylosed deciduous teeth. Ankylosis is caused by the accretion of the cementum and the alveolar bone and the obliteration of the periodontal space. The tooth that is 4 mm in infraocclusion has extensive osteoid tissue at the bifurcation with minimal osteoclastic activity. Clinically, the tooth with ankylosis is incompletely erupted, and the dental crown is below the occlusal plane. Radiologically, the membrane of the periodontium is missing, either completely or partially, in the ankylosed tooth.

Ankylosis of the molars is often accompanied by a congenital lack of the tooth bud. An ankylosed tooth can serve up to 20 years when the replacement tooth is missing. In cases where the tooth is below the occlusal plane, at a short distance, it can be restored to the occlusal plane by orthodontic or therapeutic means.

The ankylosed tooth is easily removed with pliers if the tooth surface is small or if there are root resorption elements. In other situations, it is extracted by dissecting the crown into two sections. Sometimes, it is necessary to create a mucoperiosteal flap to facilitate operative access. Occasionally, removing the ankylosed tooth is difficult. In such cases, it is recommended to remove the dental structures while keeping the alveolar bone. The presence of the tooth bud at the ankylosed tooth level will indicate complete removal of the tooth (dental debris may affect permanent tooth eruption). Sometimes, the tooth buds of the permanent teeth are located immediately below the apex of the ankylosed tooth.

Permanent tooth ankylosis. The permanent tooth is considered ankylosed when the maximal orthodontic forces are not able to move the tooth into occlusion or when it is located in the bone. It is removed only after the covering of the bony portion is removed. For the anterior teeth, corticotomy, or the removal of a covering bone portion, is performed in order to facilitate their orthodontic movement in the occlusal plane.

3.2. Indications and contraindications of dental extractions in children

Indications of dental extraction in children can be divided into two groups:

1. extraction of healthy teeth in the primary, mixed, or permanent dentition;
2. extraction of teeth with complicated caries in the primary, mixed, or permanent dentition.

I. Extraction of milk and permanent teeth, healthy and treatedteeth, in order to direct the growth and development of the dental-maxillary area and to create optimal conditions for dental occlusion.

The number, shape, and size of the teeth, their correlation with the face type, the volume of the jaws, their location, and the physiological changes have a dominant effect on the growth and formation of the dento-maxillary system. Therefore, the main objectives of pediatric dentistry include the control of the number of teeth present on the dental arch, the order and timing of the physiological changes in relation to occlusion, and the individual developmental features of the dento-maxillary system.

1. *Dental extractions during the period of physiological exfoliation*. The periods of exfoliation are extremely varied; therefore, extractions mustbe performed when the second or third degree of dental mobility is detected, which embarrasses the child's feeding and speech. The diagnosis of the exfoliation of deciduous teeth does not present difficulties. Clinically, it is characterized by visible dental mobility. The dental mobility of the primary teethduring the early period of exfoliation can be caused by trauma, which can lead to the early resorption of the injured tooth or the development of a cyst, tumor, or systemic disease. Over-retained deciduous teeth are characterized by firm implantation in the alveolar process over the period of exfoliation. This can be caused by supernumerary or crowded teeth, primary anodontia of permanent teeth, ectopic teeth, tumors, etc. In these cases, dental extractions must be indicated after a radiological examination.

2. *Extraction of permanent or primary teeth for orthodontic purposes*. Dento-alveolar abnormalities (maxillary or mandibular prognathism, dental crowding) are indications for dental extraction in the permanent or primary dentition, which must be performed only after clinical, radiological, and biometric examinations.

3. *Dental extraction in the early period of the exfoliation of the primary teeth* is generated by the lack of spaces in the dental arches and is performed with the purpose of creating these spaces for permanently

erupting teeth. Most often, tooth extractions are performed in the frontal group of the jaws. For example, the lack of space for the eruption of the central incisors is an indication of the extraction of the lateral incisors. Often, symmetrical extractions are indicated.

4. *Permanent tooth extractions in mixed and permanent dentition* are indicated if the tooth is located outside the dental arch and doesnot affect its shape, the interdental contacts are maximally preserved, and the removal of the tooth does not cause changes in the temporo-mandibular joint. Occasionally, the space for the erupted tooth outside the archway is made by extracting permanent teeth with a lower functional value or teeth affected by complicated caries. Permanent tooth extraction is indicated in the case of a 6 mm dental arch deficit in each hemiarch.

5. *Extraction of permanent dental buds* is a rational and efficient method of correcting occlusion and directing the growth of the jaws. Permanent dental bud extractions favor intraosseous migration of neighboring buds and correct tooth eruption in the dental arch, preventing dento-alveolar abnormalities.

II. Dental extractions related to abnormalities and developmental defects, dental disorders, and neighboring tissues.

1. *Developmental defects of the teeth* are extremely varied and affect the structure of the dental tissues, their shape and position, as well as their number and development. They cause facial appearance problems, a decrease in functional value, and occlusal anomalies.

2. *Teeth with abnormalities* of the hard tissue structure present a considerable vicious appearance in shape, number, and root structure. Radiological confirmation is required before indicating the removal of these teeth.

3. *Anomalies in the shape of the hard dental tissues* clinically represent anomalies of enamel development. They arrest the eruption of permanent teeth and cause alveolar abnormalities, delayed tooth eruption, and ectopic teeth. Since endodontic treatment and aesthetic

restoration of the teeth do not provide the necessary functional and cosmetic results, extraction is recommended.

4. *Supernumerary teeth* are a frequent anomaly observed in children during the period of exfoliation, especially in the region of the upper incisors. Supernumerary teeth can cause delayed exfoliation of deciduous teeth and can divert the eruption of a permanent tooth from its normal path, deforming and impactingdental archesand causing crowded teeth or over-retained deciduous teeth. Impacted dental arches can cause crowded teeth or over-retained deciduous teeth. Extraction of supernumerary teeth must be performed after establishing the clinical and radiological diagnosis.

5. *An impacted tooth* may be a supernumerary tooth, a malformed tooth, or an unerupted, ectopically placed, crowded tooth. It can create insufficient space for teeth that erupt on the dental arches and generate alveolar abnormalities. The treatment consists of reducing the number of teeth through dental extractions. Extraction of impacted permanent teeth is indicated for those teeth that cannot be aligned in the dental arch using orthodontic methods; teeth affected by caries with extensive crown and root destruction; teeth with apical infection; pulp necrosis, in cases where endodontic treatment has failed; teeth with periodontal disorders; or teeth with low functional value. Impacted teeth may be the cause of tooth retention. In most cases, the retentions are located at the level of the canine, premolar, and third molar. Cysts, infections, or anomalies in the position of the neighboring teeth are indications for the extraction of retained teeth.

III. Dental extractions with pulp necrosis and their complications.

1. Extraction is indicated for *chronic apical infection of the deciduous tooth* in the following cases:

- two years before exfoliation;
- infectious or allergic diseases;
- exacerbation of apical odontogenic infections after root filling;
- a pathological and unusual resorption of a deciduous tooth root that extends beyond one-third of its length;

– teeth with resorption or perforation at the bifurcation;
– resorption of the cortical plate at the apex of the primary tooth;
– infected odontogenic apical cysts;
– exacerbation of chronic odontogenic infection;
– chronic odontogenic infection in the regional lymph nodes;
– a persistent infection between the tooth bifurcation and the permanent dental bud;
– if the primary teeth show radiologically evident periapical destruction following endodontic treatment, with infection spreading from root apexes to permanent dental buds.

Indications for the extraction of milk teeth with pulp necrosis widen in children who are unable to cooperate with the doctor, in parents who categorically oppose treatment under general anesthesia, in children who live far from dental offices and lack transportation, and in the case of multiple treatment sessions.

There are few indications for the removal of permanent teeth with pulp necrosis. They must be removed only when the crown is completely damaged and does not present anatomical or functional interest, and the root cannot be used for prosthetic work or when preservative endodontic treatment can be applied.

2. *Acute and chronic odontogenic periostitis.* Primary pluriradicular teeth that are the cause of acute purulent periostitis must be extracted. Endodontic treatment is required in the case of monoradicular primary teeth during the period of root formation in children in the first and second ASA categories. The effectiveness of the treatment is conditioned by the straight and wide roots, which allow sufficient drainage. However, if satisfactory results are not obtained in the first few days of treatment, the teeth must be removed. Primary and permanent monoradicular teeth, which are the cause of chronic hyperostosis periostitis, must be removed.

3. *Osteomyelitis, odontogenic abscesses, and phlegmons*. Primary and permanent pluriradicular teeth, which are the causes of acute and chronic odontogenic osteomyelitis, phlegmon, and odontogenic

abscesses, are indicated for extraction. The permanent monoradicular teeth mustbe removed only in cases where they do not have any functional or anatomical value. The permanently seizeddental buds must be removed.

IV. Dental extractions in dental and maxillary trauma.

1. *Extractions of the primary teeth in dental trauma* are characterized by complete and incomplete dislocations, less frequently by coronal and radicular fractures. Based on the peculiarities of the dento-alveolar system, primary teeth in dental trauma must be removed in the following cases: total dislocations, incomplete dislocations with axial displacements, and those with labio-vestibular and mesio-distal displacements. Poor implantation in the alveolar apophysis of the primary teeth leads to a short survival time ofthe tooth.

2. *Extraction of permanent teeth.* Multiple fractures, comminuted fractures, longitudinal root fractures, oblique fractures, fracture of half the root length, and crown fractures at the neck level are all indications for permanent tooth extraction in the event of trauma. Teeth with a fracture at or below the neck must be removed only after an orthopedist has examined them. Root fractures must be subjected to apical surgery. If the fractured apex fragment is too short, it is left in the alveolus because it will undergo spontaneous resorption.

3. *Indications for the dental extraction of primary and permanent teeth* from the fracture foci of the jaws include: primary and permanent teeth projected into the fracture line with complicated caries and septic processes; healthy permanent teeth; and dental buds that can be an obstacle in reducing fragments.

V. Indications for dental extractions with tumor processes.

Tumors are the primary cause of dento-alveolar complex destruction, manifesting as radicular resorption, dental mobility, and functional disorders. They must be removed along with the adjacent bone tissues. Removing a single tooth from the tumor region is dangerous because it may cause tumor growth or bleeding.

Contraindications to dental extraction surgery. Dental extraction has no absolute contraindications. However, serious health problems in children can be associated with severe complications after dental extraction. Some health conditions require involving physicians, delaying extraction, performing laboratory tests before surgery, and keeping patients in the inpatient department before and after the extraction.

General factors that require careful timingof dental extractions include:

1. Hemorrhagic syndrome is an increased risk of bleeding due to a deficiency of coagulation factors that are dependent on the level of vitamin K in plasma. It can be detected in the blood in normal quantities in the form of dysfunctional molecules called PIVKA (protein induced by vitamin K absence). The hemorrhagic syndrome is due to biochemical liver dysfunction. Liver disorders can lead to hemorrhagic diseases. It must be taken into consideration that vitamin K is produced by intestinal microorganisms. Because of the lack of prevention of vitamin K deficiency after birth in hospitals, hemorrhagic syndrome frequently progresses as a hemorrhagic disease of the newborn. In some diseases, hemostatic disorders are associated with disseminated intravascular coagulation, infectious-septic, immune, destructive processes, and tumors. For these reasons, in children with liver, spleen, and intestinal disorders and symptoms of bleeding manifested after birth, it is mandatory to confirm this by laboratory tests and medical history in the preoperative period (examination of the bleeding and coagulation rate constants, sometimes also the coagulogram). Because of clinical manifestations and changes in blood constants, a well-trained general dentist, pediatric dentist, or oral surgeon must perform the necessary measures during tooth removal in an inpatient department in collaboration with a hematologist.

2. Compensated heart failure does not require delayed dental extraction. In these cases, pre-anesthesia is preferable in order to decrease the amount of anxiety and obtain sedation of the nervous system. Acute decompensated heart failure is a sudden worsening of the signs and symptoms of heart failure, which typically includes difficulty breathing

(dyspnea), leg or foot swelling, and fatigue**.** It leads to severe acute respiratory distress. Preoperative and postoperative preparation should be performed together with the cardiologist in order to prevent any specific accidents. Patients with valvular prostheses require special care, and the surgery mustbe performed under the supervision of a cardiologist.

3. Leukemia is a type of cancer of the blood cells that involves disorders of the immune system, which protects the host from invasion by bacteria, viruses, and fungi. Due to the risk of bleeding, infection, and necrosis, dental extraction must be performed in the inpatient departmentfollowing laboratory tests. Physicians must be involved in preparing patients for extraction to achieve a successful post-operative recovery. In children, dental extraction must be performed following the decrease in the acute period, control of hemostasis, and antibiotic administration.

4. Diabetes is a group of metabolic disorders characterized by a high blood sugar level over a prolonged period of time. Complications are related to damage to blood vessels, cardiovascular disease, and coronary artery disease. Diabetes increases the risk of hemorrhage after tooth extraction. Some complications can occur after extraction due to damage to small blood vessels, such as infections, surgical wounds that heal poorly, and severe postoperative pain. In such cases, tooth extraction surgery should be performed in an inpatient setting, with laboratory tests before and after the surgery and the administration of antimicrobial drugs.

5. Kidney failure occurs when the kidneys lose their ability to filter waste from the blood and the body becomes toxically overloaded. Acute glomerulonephritis may worsen dental extractions because of wound infection if exodontic therapy is not performed with antibiotic administration. Patients with elevated azotemia usually have increased coagulation and bleeding time, requiring special management of post-extraction wound healing.

6. Rheumatic disease is an autoimmune disorder that affects the bones, joints, and muscles. The most frequent childhood chronic disease is arthritis. Rheumatic disease of the joints or heart may worsen the healing after tooth extraction. In certain types of rheumatism, bactere-

mia, which occurs after the removal of the tooth and the infectious foci, can have a severe influence on the recovery period.

7. Corticosteroids, used for a long time in the treatment of certain conditions, cause a decrease in the host's defense against infectious factors. Extractions must be performed in the inpatient department after a clinical laboratory examination, along with the administration of antibiotics.

8. In oral infections such as acute necrotizing ulcerative gingivitis, acute herpetic stomatitis, acute dentoalveolar abscess, and other acute oral diseases, tooth removal is definitely contraindicated until the infections are eliminated.

3.3. Complications of dental extractions

Root fracture of the deciduous tooth during dental extraction is the most common complication, caused by the process of uneven resorption, which leads to the formation of narrow and long roots. Fractured root fragments are sometimes located just below the buds of permanent teeth. It is known that the removal of fractured fragments is the main condition for the regeneration of the alveolar wound. In children, however, these root fragments are left in the alveolus. The effort to remove the fragments can result in the accidental removal of the tooth bud. Conditions are needed for the uncomplicated regeneration of the dental alveolus (a wide, superficial, small alveolus with a high drainage capacity). With the eruption of permanent teeth, the fractured roots are spontaneously expelled. Total dislocation of the dental bud is a complication of dental extraction in children if the particularities of the exodontic maneuvers are not respected. Dental buds, once luxated, must be immediately reimplanted into the gum and fixed by suturing the alveolar margins or by applying the orthodontic device. Possible infectious complications should be prevented by the administration of antibiotics. The teeth should be subjected to follow-up imaging at intervals of 3 monthsfor 1 year.

Aspiration of the extracted tooth during the extraction can occur in anxious, mobile, and capricious children. These children are referred to the inpatient ward for examination and treatment.

Learning objectives

1. Indications of dental extractions in primary dentition:
   - tooth with a chronic infection;
   - permanent teeth with a chronic apical infection;
   - primary and permanent teeth that are the cause of subperiosteal abscess;
   - primary and permanent teeth that are the cause of acute odontogenic osteomyelitis and chronic osteomyelitis.
2. Indications for the extraction of permanent dental buds.
3. Complications of dental extractions in children.
4. Stages of dental extraction of primary and permanent teeth.
5. Causes and treatment of post-extraction hemorrhage.

TESTS

1. SC. Indicate the treatment options for pathological rootresorption greater than 1/4 to 1/3 of its length:

A. dental extractionafter radiological examination;
B. immobilization with mobilizable orthodontic devices;
C. dental treatment with root canal filling;
D. dental treatment without root canal filling;
E. left untreated, but it isbeing monitored.

(A)

2. SC. In which cases shouldthe primary teeth with chronic apical infection removed:

A. are left for 2 years, until the physiological exfoliation occurs;
B. resorption and perforation of the dental floor;
C. repeated exacerbation;
D. acute purulent pulpitis;
E. hemorrhage from the root canals.

(A, B, C)

3. MC. In which cases after dental trauma should the permanent tooth beextracted:

A. class 2 crown fracture;
B. root fracture in the cervical area;

C. apex facture;
D. class 3 crown fracture;
E. comminuted fracture.

(B, E)

4. MC. Which type of root resorption of primary teeth carries a higher risk of removing permanent buds during the extraction of primary teeth:
A. the vertical type of root resorption;
B. the horizontal type of root resorption;
C. physiological resorption;
D. delayed resorption;
E. pathologicalresorption.

(A, B)

5. MC. In which clinical cases should the monoradicular permanent teeth be removed in children:
A. when they lose their functional value;
B. they are the cause of acute periostitis;
C. they are the cause of acute osteomyelitis;
D. they lose their anatomical value;
E. with orthodontic purpose.

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4. ODONTOGENIC AND NON-ODONTOGENIC INFECTIONS IN THE ORAL AND MAXILLOFACIAL REGION IN CHILDREN.

4.1. Etiology of oral and maxillofacial infections

Infection is the invasion of disease-causing agents into bodily tissues and their growth, as well as the host's tissue response to infectious agents and their toxins.

Three elements interact to cause an infection: the host, the environment, and the organism. These three are in balance when the body is in a state of homeostasis. The host defense mechanisms are the major factor in determining the outcome of an infection, with the environment and the microbe playing important but usually secondary roles. Human beings are subject to various infections, and the relationship between infectious microorganisms and the host is expressed by a balance on which the pathogenic attributes of the microbes (virulence and quantity) are weighed against the protective mechanisms of the host. Virulence includes invasiveness and a multitude of toxins, enzymes, and other harmful products. The quantity of microbes refers to the number of organisms that initially infect the host. Furthermore, an increase in quantity enhances the concentration of virulence. Under normal conditions, host factors predominate. The more they predominate, the greater the host reserve. If microbial factors increase, protective host factors decrease, or a combination of both, the pathogenic potential increases. As this occurs, host reserve diminishes until microbial factors predominate and clinical infection supervenes.

The host defense system is made up of a multitude of smaller subsystems and factors that relate to and interact with one another to provide coherent, single-purpose protection for the host. The host defense system consists of three major components: local, humoral, and cellular factors. Infectious agents initiate a series of reactions in the host that are collectively called „the inflammatory reaction”. This response results in the generation and release of mediators in microvascular changes and in the mobilization and activation of leukocytes, all designed to eliminate thepathogen and repair tissue injury. These reactions are primarily protective. In addition to initiating the inflammatory reaction, infectious agents or their products also produce myriad effects on the host. These effects include direct injury to the host cells, enhancement of the parasite's invasiveness, and amplification of these effects by neutralization of the host defenses. Other products may trigger systemic effects, some beneficial or benign (fever), others inappropriate and harmful (endotoxic shock). Finally, the pathogen or its products may, in abnormal cases, combine with antibodies or sensitized mononuclear leukocytes to produce harmful immunologic effects called hypersensitivity (allergic) reactions.

Inflammation causes five cardinal symptoms, which were described in antiquity by Celsius and Galen, namely: swelling (*tumor*), edema (*rubor*), hyperemia (*calor*), painfulness (*pain*), and functional injuries (*functio laesa*).

The adult oral cavity has a dense, varied, and indigenous microbiota that consists of protozoa, yeasts, viruses, and more than 20 identified genera of bacteria with numerous species, many of which have not been definitively characterized or classified. During birth and immediately thereafter, the neonate contacts the microbial inhabitants of the birth canal and others from the maternal environment. Nevertheless, the oral cavity usually does not contain detectable microorganisms at birth. About 8 hours following birth, the number of detectable organisms in the newborn's oral cavity increases rapidly. The initial microbial flora consists of strains and species of lactobacilli, streptococci, staphylococci, enterococci, veillonellae, neisseria, and coliforms. The bacterial composition of the oral microbiota varies considerably during the first few days of life, but streptococci are usually among the early oral inhabitants, and they persist throughout life.

The microbiota of the mouth at birth is predominantly aerobic because of a lack of areas that favor the colonization of anaerobes. With the eruption of the teeth, new environmental conditions appear that some microbial species find favorable and tend to localize and flourish at these sites. Bacteria generally increase throughout childhood, and the flora of older children resembles that of adults. However, some bacteria, such as prevotella species and spirochetes, are not common until adolescence.

There is no absolute standard for health and disease, but they are dependent upon the interaction and relationship of several determinants: the physical environment, the individual's constitution, and the biological environment, which comprises such things as the climate, the season of the year, and the composition of food and water. Constitutional factors are those associated with race, sex, inherent immunity, inherent disease traits, and anatomic anomalies. Many of these factors alter with age, as physiology changes with age. External and internal microbiota are biological determinants. The external microbiota varies from region to region and from rural to urban settings. These variations may affect the chances of exposure to certain types of pathogens and therefore the incidence of some infections. Personal and public hygiene measures may further impinge upon this, affecting the balance between health and disease.

Infections of the head and neck are of odontogenic and non-odontogenic origin. Odontogenic infections are more common and arise in the pulp and periapical, periodontal, and pericoronal areas. Other infections arise by invasion through the skin or mucosa or are iatrogenic.

The examination of the anaerobic culture detected the main role of the bacterial associations with non-clostridial anaerobes in 65% -67% of purulent odontogenic processes. White and golden staphylococci, streptococci, escherichia coli, corynebacteria, lactobacteria, fusobacteria, colibacilli, and veillonella also play an important role in triggering the infection. A perimaxillary infectious process is dangerous if a pure culture is present.

In 68-90% of cases, multiple microflora examinations in odontogenic abscesses and phlegmons revealed a polymicrobial character. In purulent collections, anaerobes are found in 28-100% of cultures, and mixed flora (anaerobic-aerobic) in 52-68%. Aerobic-anaerobic associations of 3-4 types were found. In mixed cultures, bacteria exist in synergistic and antagonistic relationships, which aggravate the general and local states.

The mechanisms of pathogen spread in the soft parts are the transosseous, submucosal, and lymphatic ways.

4.2. Soft tissue infections in children

The character of the evolution of the infectious process in children is determined by the *anatomical-physiological particularities* of the maxillofacial area, organs, and systems: large root canals, their open apex due to resorption and formation processes, and intimate contact between teeth and bone marrow. Wide Haversian canals, abundant vascularization, thin bone tissue, and a thickened and elastic periosteum are favorable conditions for the infectious process to enter the periodontal tissues, simultaneously affecting the bone, periodontium, and soft parts. Differential diagnosis of deep caries, pulpitis, and periodontitis in children is difficult.

Infections in children are specific and require special care. The systemic effects of infections are more pronounced in children. The younger the children, the less well controlled temperature regulation is, and rapid temperature elevations may occur with the infection. The normal body temperature in children is higher than in adults. Children are more affected than adults by dehydration brought on by fever and failure to consume enough oral fluids while ill. Hospitalization and rehydration should be considered early in the case of an odontogenic infection in children. The white blood cell count, pulse, blood pressure, and other vital signs and laboratory measurements in children differ from those in adults. The bones of the jaws are less dense, with wider marrow spaces than those in adults. This is thought to be responsible for a more rapid spread of infection. If an infection is not treated vigorously and promptly, it may lead to osteomyelitis, proliferative periostitis (Garre's sclerosing osteomyelitis), involvement of the condyle, and subsequent growth deformation. In children, an infection caused by dentoalveolar abscesses may involve the buds of permanent teeth.

Abscesses with fistula formation on the skin surfaces rather than in the vestibule occur more often in children than in adults because of the relative height of the muscle attachments to the developing alveolar process. Failure to recognize dental problems and treat them properly through pulpal and canal therapy or tooth extraction has resulted in repeated courses of antibiotic therapy and unnecessary surgical procedures. It has been noted that general infections in children involving the upper face (orbits, paranasal sinuses, maxillary teeth, and cheeks) occur most often in younger children with unknown causes. They are likely to have penicillin-resistant organisms. Those of the lower face (mandibular teeth and submental, sublingual, and submandibular structures) occur more often in older children (mean 5.6 years). In the latter group, when a source of infection was identified, it was often odontogenic in nature and caused by penicillin-sensitive organisms. Because children become rapidly dehydrated and the infection may spread readily, early hospitalization may be essential to shorten the duration of the illness and reduce morbidity.

Cellulitis is a diffuse inflammatory reaction in which bacteria are able to overcome or avoid the host's defenses so that the infection is not confined to one area. It may instead progress through surrounding tissues and along facial areas, away from the site of infection. Cellulitis of the neck and face is most often caused by periostitis, osteomyelitis, acute purulent lymphadenitis, or tooth extraction. The clinical pictureincludes the local congestion of the involved soft parts, firm and painful swelling with an extensive progressive tendency, regional lymphadenitis, and general condition alteration: fever, chills, and malaise.

In the maxillofacial region, Streptococcus pyogenes is the most common etiologic agent in adolescents and adults. Haemophilus influenzae cellulitis occurs more often in children, mostly under 2 years of age. Ampicillin is effective against most H. influenzae strains.

Treatment includes endodontic drainage of the tooth or its removal, antibiotic therapy, analgesics, and non-steroidal anti-inflammatory drugs.

Impetigo is a bacterial infection that involves the superficial skin. Clinically, yellowish crusts on the skin, face, arms, and legs are common. Staphylococcus aureus and streptococcus pyogenes are the causes of this infection. Risk factors include attending daycare, poor nutrition, diabetes mellitus, contact spots, and skin breakssuch as mosquito bites, eczema, scabies, or herpes. Impetigo is likely to infect children aged 2-5, especially those that attend school or daycare. Contagious impetigo is the most common form of impetigo. It most often begins as a red sore in the nose and mouth area, which soon breaks, leaking pus or fluid, and forms a honey-colored scab followed by a red mark that heals without leaving a scar. Sores are not painful, but they may be itchy. Lymph nodes in the affected area may be swollen, but fever is rare. Touching or scratching the sores may easily spread the infection to other parts of the body. Antibiotics, either as a cream or by mouth, are the main choice of treatment.

Necrotizing cellulitis is a common and potentially serious bacterial skin infection (*figure 1*). It is an aggressive infection involving superficial fascia with undermining of the overlying soft tissue. Most commonly, it occurs in the fascia of the trunk and extremities, but it can also be seen in the maxillofacial region. Necrotizing fasciitis was historically thoughtto be caused by hemolytic streptococci and Staphylococcus aureus, but modern culturing techniques have revealed that anaerobes and gram-negative bacteria are usually present in the wound. Usually, necrotizing fasciitis occurs after trauma or surgery and is common in chronically debilitated children with diabetes mellitus or obliterative small vessel diseases. The clinical manifestation begins with the involved area becoming swollen and erythematous with a low-grade fever. The site soon worsens with sudden pain, worsening of the erythema, edema, and severe generalized toxicity. The skin overlying the infected fascia becomes dusky with purple mottling, gas formation, and skin necrosis; cutaneous nerves become necrotic; and numbness may occur. Systemic manifestations include signs of sepsis, hemolysis, and intravascular volume depletion. High fever, tachycardia, apathy, weakness, nausea, anemia, jaundice, hemoglobinuria, hypotension, and decreased skin turgor are present. Treatment includes high doses of antibiotics and surgery with debridement.

Fig. 1. **Necrotizing cellulitis**

An abscess is a swollen area within body tissue that is localized, limited, and circumscribed and contains pus. The abscess is composed mainly of a central area of the organism and disintegrating polymorphonuclear leukocytes, surrounded by viable leukocytes and some lymphocytes. The adjacent blood vessels are dilated, and if the infection is of odontogenic origin, the marrow spaces may have an inflammatory cell infiltrate.

The clinical signs and symptoms of an abscess include redness, painful swelling of the soft tissue, warmth, and circumscription within the limits of the perimaxillary fossa or region. The swelling may feel fluid-filled when pressed. The mucosa, or skin that covers it, is tense. The consistency and smell of the pus vary depending on the type of germs and possibly the antibiotic therapy used. The general condition of the patients is „septic condition" with oscillating fever, chills, tachycardia, restlessness, local pain, and curvature.

Treatment includes incision and drainage of the collection, antibiotic therapy, anti-inflammatory pain relievers, and suppression of the causative element.

In children, abscesses of the tongue, parapharyngeal and sublingual areas, upper lip, and hard palate present interest.

Abscess of the tongue in children is a rare entity that is potentially life-threatening as it is capable of compromising the airway and disseminating infection to other regions. Lingual abscesses have become extremely rare since the discovery of antibiotics, despite the relatively frequent exposure of the tongue to bites during mastication and seizures. A lost fish bone may be the cause in many cases.

They are located most commonly on the anterior two thirds of the tongue and the lateral parts. These kinds are easy to diagnose, but those situated on the posterior third may pose a diagnostic challenge. Abscesses at the root of the tongue are rare and sometimes caused by congenital cysts. A tongue abscess is clinically present as a painful swelling that causes protrusion of the tongue, dysphagia, odynophagia, and difficulty with speech. The pain radiates into the pharynx, ear, and submandibular area and produces functional disorders in swallowing, phonation, and chewing. There are impressions of the teeth on the level of the lingual slopes; salivation occurs, and there is edema on the buccal floor. Abscesses of the tongue should be considered acute tongue swellings, especially when the host defenses are impaired.

Differential diagnosis. Tongue abscesses must be differentiated from dermoid cysts and benign tumors.

A parapharyngeal abscess is a deep neck abscess that develops laterally to the superior pharyngeal constrictor muscles and medially to the masseter muscle. It is caused by a bacterial infection that has spread from the tonsils, throat, sinuses, adenoids, and nose, or it can be of odontogenic origin. It is a life-threatening infection that can cause laryngeal edema and upper airway occlusion. Their occurrence may be associated with significant rates of morbidity and mortality because of obstruction of the airway due to abscess rupture into the pharynx or trachea, which can lead to emphysema, mediastinitis, carotid artery erosion, jugular thrombophlebitis, or cavernous sinus thrombosis. Because the clinical signs are less clear and the physical examination poses a challenge, the diagnosis of this infection in children is more difficult. On physical examination, the most prevalent findings are severe fever, head stiffness, bulging of the oropharyngeal wall, and lymphadenopathy of the neck region. Because of the diagnostic challenge, the abscesses are clinical entities with a relevant incidence in the pediatric population. Early diagnosis and surgical treatment lead to good clinical outcomes and a low incidence of complications.

An orbital abscess is uncommon and represents a collection of pus within the orbital soft tissue (*figure 2*). This condition is considered life-threatening because the infection can quickly result in blindness, meningitis, or death. Pre-orbital or preseptal involvement tends to occur in a younger age group (below 5 years of age), whereas postseptal involvement tends to occur in older children. In children, suppuration spreads quickly into the surrounding tissue and spaces (cavernous, pterygoid, and temporal sinus). Periorbital and orbital involvement is fairly common as a complication of the infection spreading from the maxillary dentition and paranasal sinuses, or as a result of trauma to the area. Acute sinusitis, apical infection, or extension of the suppurative process from the surrounding spaces following a furuncle (upper lip, mouth cavities, nasolabial folds) can all cause this process. The clinical manifestations include lid edema, conjunctival chemosis, proptosis, an afferent pupillary defect, visual impairment, restricted ocular mobility, pain, headache, and complete closure (obstruction) of the eyelid.

Fig. 2. **Orbital abscess**

The differential diagnosis should be made between transient orbital cellulitis, which has moderate inflammatory signs, and cavernous sinus thrombophlebitis, which has specific symptoms. Prompt diagnosis, intensive antibiotic therapy, and appropriate surgical drainage lead to favorable outcomes.

A phlegmon is an unbounded inflammation of the soft tissue that can keep spreading out along the connective tissue and muscle fiber. Anaerobic germs (hemolytic streptococcus β, clostridia, and bacterioles) play an important role in suppuration. Bacteria may enter via a scratch, insect bite, or injury to the skin or oral mucosa, especially after dental surgery.

The phlegmon symptoms vary depending on the location and severity of the infection. If not treated, it can spread to a deeper layer and disable the area involved. Skin phlegmons are visible and are red, sore, swollen, and painful. They may also exhibit systemic bacterial infection symptoms such as swollen lymph nodes, fatigue, fever, and headache. The clinical signs of the mouth floor phlegmon are toothache, fatigue, earache, confusion, swelling of the tongue and neck, and difficulty breathing. Because children tend to have a moresubtle presentation and are rarely able to verbalize or cooperate during the physical examination, establishing an accurate diagnosis is difficult. On the other hand, oral phlegmon can spread quickly and be life-threatening. For this reason, more tests, such as blood tests, urinalysis, ultrasound, MRI, or CT scan, may be required to confirm the diagnosis.

The septic process is also exacerbated because the suppuration affects patients with low reactivity, immune system depression from another cause, or a compromised area of preexisting disorders (cachexia, convalescence after previous illnesses that have exhausted the immune system). The treatment of the phlegmon depends on the location and severity of the infection but, in general, involves antibiotics and surgery.

A furuncle is a necrotic, infectious lesion of the hair follicle and the surrounding soft parts (*figure 3*). It is found in adolescents. In recent years, the number of life-threatening complications in adolescents has increased by 2-2.5 times. Until recently, monocultural etiology was considered in 98% of cases and staphylococci and other microorganisms in only 5% of cases. In recent years, the association of pathogenic germs with the predominance of anaerobic microflora has been advocated.

Fig. 3 **Furuncle on the face**

The skin is populated by non-pathogenic and pathogenic staphylococci (S. aureus), being in an inert state in the composition of the normal microbiosis. The seborrheic and sebaceous glands of the skin eliminate secretions that inactivate the microflora of the teguments. By colonizing the entire surface of the skin, staphylococci form a pyogenic base with high invasion capacity.

Poor skin hygiene, particularities of the skin during puberty, adverse weather conditions, disorders of the nervous system and endocrine system, avitaminosis, and intoxication are factors favoring the onset of the purulent process. Carbohydrate metabolism disorders play an important role. 20% of patients with diabetes mellitus are affected by furunculosis.

The region within the upper lip, the commissures, the infraorbital region, and the nose is called the „black triangle". The much larger quantity, compared to other regions of the face, of the seborrheic and sebaceous glands on the upper side and chin, located in the deep planes (subcutaneous layer), the specificity of the angular veins, and the microbial composition, explains the high risk of serious complications (sinus thrombosis) of infections localized in the „black triangle".

4.3. Salivary gland infections in children

Sialadenitis is an inflammatory process of the parenchyma of the salivary glands, occurring in most cases unilaterally. The incidence of infection of salivary tissue is influenced by many factors, including age, general health and immune status, state of hydration, sialogogic drugs, and trauma. In children, parotid glands are affected in 30% of cases. The prognosis of parotid gland infection is targeted and reserved because the disorder in salivary secretion is caused mainly by the immunodepression of neurogenic and neuroendocrine etiology that leads to infection of the salivary glands. Neurogenic sialadenitis occurs after abdominal surgery, cachexia, and others. Infections are caused by the penetration of microorganisms into the parotid glands by odontogenic, hematogenic, or lymphogenic routes. Salivary infection can also be caused by ductal abnormalities, foreign bodies, dental therapy, and systemic granulomatous disease.

Parotitis is classified into three types based on the etiopathogenic factors: 1. *Acute mumps*: a) neonatal parotitis; b) acute viral parotitis: epidemic, caused by the influenza virus or other viruses (Coxsackie, Herpes, Cytomegalovirus, etc.); c) non-contagious parotitis caused by general acute infections, appearing during the postoperative periods, caused by infections that cause trophic disorders (heart failure, cachexia, etc.); lymphogenic parotitis (Ghertenberg), contact parotitis, parotitis caused by the obstruction of the salivary secretion pathways by foreign bodies. 2. *Chronic parotitis*: parenchymatous, interstitial, specific (tuberculous, actinomycetic, etc.). 3. *Sialolithiasis.*

Acute neonatal parotitis occurs very rarely in the neonatal period. The risks involved in its pathogenesis are preterminfants, children with an unfavorable background, congenital associated pathologies, infections, dehydration, and the development of neonatal parotitis, which is determined by mastitis in mothers too. Staphylococcus aureus is the most common bacterial agent causing this condition. Acute neonatal parotitis is diagnosed clinically; it has an acute onset and is more common in the first week of life. It starts with swelling in one or both parotid regions, followed by severe general septic conditions (anxiety, insomnia, chills, and anorexia). The characteristic clinical picture is of a sick preterm infant. The parotid gland area is red, painful, swollen, and tender, and there is salivary duct hypertrophy with purulent secretions. Pus dischargesfrom the duct containing *S aureus* in more than half of the cases. The majority of the cultured bacteria come from organisms present in the oral cavity, which suggests an ascending infection that has spread from the mouth.

Differential diagnosis. Acute neonatal parotitis must be distinguished from hematogenous osteomyelitis, which typically occurs in newborns with a septic background.

The treatment at the onset of the disease includes the administration of antibiotics, desensitizers, detoxification therapy, and, locally, poultices with dimexide solutions and heparin ointments. In advanced stages, the pus must be surgically evacuated.

Acute epidemic parotitis (mumps) is a common viral disease that affects the large salivary glands. Mumps is achildhood infection that is spread by droplets or directly from oropharyngeal secretions that contain paramyxovirus. Children in small groups are affected, rarely adolescents, and very rarely adults. In 70% of cases, it is located in the parotid gland; in 10% of cases, it is located in the submandibular gland; and in 4.9% of cases, it is located in the sublingual gland. In 30%, it evolves asymptomatically. It usually affects children aged 4 to 10 years old.

After an incubation period of 16-20 days, the disease starts suddenly, with an interval of 1-2 days, and causes painful swelling of both parotid glands. The swelling is accompanied by an alteration of the general condition (fever, pain). The disease is characterized by grossly enlarged and modestly tender parotid glands. Parotid stimulation causes pain in the gland and ear. The skin is stretchy and congested, with edema, trismus, and pain during mouth opening, as well as hyperemia and swelling of Stenon`s canal.

Mumps is a benign disease in the vast majority of cases but is occasionally complicated by meningoencephalitis, pancreatitis, orchitis, or deafness, especially in young adults.

The differential diagnosis is made with acute non-epidemic parotitis or exacerbated chronic parotitis, which is explained by unilateral affection, chronic evolution with exacerbation periods, anda specific X-ray picture of a flowering tree.

The treatment is symptomatic and supportive. The patient must be treated in the department of contagious diseases and isolated for 20 days. Rest, oral antiseptic washes, sialogogues, and vitamin therapy must be administered.

The introduction of universal immunization, which began in 1977, has made the clinical disease unusual in developed countries. The child should receive the first measles, mumps, and rubella (MMR) vaccine at age one year and a second at age 4-6 years.

Recurrent parotitis of childhood, or *juvenile recurrent parotitis*, is referred to as repeated episodes of parotid gland inflammation, either non-obstructive or non-suppurative, in which recurring episodes clinically resemble mumps. It is the second most common disease of the salivary glands in children, following mumps. Non-specific chronic parotitis in children is found in 14% of cases. Parotid glands (88%) are more often affected.

The etiology and pathogenesis of juvenile recurrent parotitis remain unknown. It is believed to have a multifactorial cause such as congenital malformation of the parotid glands leading to retrograde infection, allergy and its association with auto-immune diseases, upper airway infections, allergy, immunodeficiency, autoimmune disease, and congenital sialectasis.

The age at onset varies from 8 months to 16 years, and the symptoms normally disappear spontaneously after puberty. Usually, it manifests during the period of exacerbation and evolves into periods of remission and exacerbation. The frequency of exacerbations can be quite variable. The exacerbation period lasts 10-14 days, and the remission, in some cases, lasts up to 1-2 years.

The period of exacerbation is characterized by the alteration of the general condition: chills, fever, malaise, insomnia, excitement; and locally, with unilateral swelling of the parotid gland (rarely bilateral) pain, without alteration of the covering skin, tuberous on palpation, congested Stenon`s duct, and hyperemia. Massaging the gland from back to front produces clear saliva with lots of „snowflakes", or little white curds, that are eliminated from Stenon`s duct. Between acute episodes, parotidomegaly may persist, with viscous and slightly reduced salivary secretion and fibrin plugs.

The diagnosis of recurrent parotitis is usually based on a detailed history and an adequate physical examination. The parotid sialogram reveals multiple peripheral ectatic ducts, which are uniformand 2-3 mm in size without any stones or destructive changes, sometimes having the appearance of a „flowering tree" or „apple tree", to which the irregular expansion of Stenon`s canal is added.

Differential diagnosis. Recurrent parotitis must be distinguished from epidemic parotitis or mumps, manifested by symmetrical involvement of the salivary glands with no periods of remission or exacerbation. It must also be distinguished from sialolithiasis, characterized by salivary colic and calculi in the submandibular gland. Recurrent parotitis must be differentiated from intraparotid lymphadenitis, in which the salivary secretions look normal.

The recurrent attacks are treated conservatively with oral penicillin and analgesics. However, no prophylactic therapy is available. In addition to antibiotics, analgesics, and good oral hygiene, massage of the parotid gland, use of chewing gum, and sialogogic agents may be helpful in reducing the attack frequency. During remission periods, it is necessary to stimulate both local and general immune factors. It is important for the child to be examined by the pediatrician, dentist, and otolaryngologist in order to detect and cure chronic foci, general disorders of organs and systems, and boost the immune system. Only patients with persistent problems should receive more aggressive treatment. This may include parotid duct ligation, parotidectomy, or tympanic neurectomy, depending on the physician`s preference and experience.

Acute non-epidemic parotitis. Parotitis is an acute condition of the parenchyma of the parotid gland, usually occurring unilaterally. Usually, it occurs due to immunodepression caused by local and general unfavorable factors. General unfavorable factors include acute viral infections (chickenpox, measles, scarlet fever), cachexia and dehydration, severe general conditions, poor oral hygiene, postoperative periods, and neurovegetative disorders. Local factors that can cause infections are trauma, mechanical obstruction of the salivary duct (foreign bodies in the salivary duct, lymphadenitis with localization in the parotid glands), or the spread of lymphogenic infection in the neighboring regions. The clinical picture is associated with general and local disorders: insomnia, chills, and sharp pain during feeding; xeroderma; swelling and tenderness over the gland on the side of the cheek; along with pus discharging from the opening of the duct on the inside of the cheek.

Staphylococcus aureus is the most common pathogen associated with bacterial parotitis (80%). Mixed infections, including streptococci, anaerobes, and gram-negative bacilli, can be the cause of bacterial parotitis.

Treatment comprises correction of the lack of fluids (rehydration), antibiotics, and pain relief.

Salivary lithiasis affects children during adolescence; it is mainly located in the submandibular gland. Salivary lithiasis is caused by metabolism disorders, avitaminosis, and the chemical and physical properties of saliva. Salivary lithiasis is characterized by a triad: 1) *Submandibular swelling* occurs while eatingand has a brief remission; 2) *Salivary abscess* is a complication caused by superinfection, with intense pain radiating in the tongue and ear, fever, and chewing difficulties. The Warton channel is congested, open, and has reduced eliminations; there is hemiplegic edema and a prominent sublingual envelope („cock crest”). 3) *Salivary tumor* has a rough surface and a firm texture. It is adherent to deep planes and is tender to palpation.

The treatment must be administered in specialized clinics. The calculus must be removed by surgery. Antibiotics, desensitizers, local physiotherapeutic procedures, massage, and paraffin applications must be applied.

4.4. Rehabilitation of children with odontogenic and non-odontogenic oro-maxillofacial infections

The rehabilitation of children with infectious lesions comprises three stages.

I. *The diagnosis* is the first stage of rehabilitation and is made at the level of emergency assistance and primary medicine based on the child's complaint and a local, general, and radiological clinical examination. A late and incorrect diagnosis is the cause of complications and the development of chronic forms. The odontogenic or non-odontogenic origin of the infectious processes is determined even in the absence of a radiological examination. The diagnosis is made after the tooth receives emergency medical care and is monitored for 24 hours. The disease prognosis is based on some risk factors: 1) health status, physical development, previous chronic and acute illnesses, congenital defects, type of feeding (artificial, natural, or combined), and so on; 2) social hygiene - the level of hygiene culture, oral hygiene, living conditions, rest, and physical activity, and parents' attitude towards dental treatment during acute and chronic periods; 3) follow-up and prophylaxis of oral diseases, primary dental therapy, and consulting the pediatrician only in urgent cases. The clinical setting (outpatient or inpatient) is determined according to general and local conditions. The indications for hospitallization of children with infectious lesions are: a) diffuse suppurative processes and extensive invasive processes (osteomyelitis, phlegmon); b) severe general intoxication; c) children under 5 years of age; d) children's physicalstatus- III, IV, and V (ASA); e) organizational reasons (lack of specialist doctors, children from vulnerable families, children living in remote places). Primary local and general emergency medical care must be provided.

II. *The second stage is performed at the level of specialized clinics*.

The definitive diagnosis is madebased on the differential diagnosis, and specific surgical and general treatments are performed. The second stage ends with the discharge of the child.

III. *The rehabilitation of children inthe third stage* is carried out at the level of primary and specialized outpatient departments in order to rehabilitate them morphologically and functionally. It is carried out in collaboration with the family doctor and the dentist. The necessity of this stage is determined by the intensive growth of the body and the high risk of complications occurring during this period (development and eruption disorders of the dental buds and their necrosis; change of the eruption periods of the permanent teeth in the affected regions; change of the shape of the dental arches, followed by dental-alveolar anomalies; change of the facial shape).

The family doctor should assess the general condition and the degree of health through accessible methods in order to make a prediction of the action of the necrotic lesions on the development of the organism. Chronic diseases, anomalies of constitution, the allergic status, and the frequency and character of previous diseases over the last year should be noted, and then an analysis of the immune system must be made. A clinical laboratory examination is indicated.

The first group – During the period of physiological change, the primary teeth are usually healthy; post-infectious, limited destructive changes in the alveolar process and dental tissue; dental buds with moderate changes in the adjacent soft parts; and satisfactory overall condition.

The second group includes children who have lost thepermanent and primary teeth at an early age, creating conditions for the distribution of the masticatory function and increasing the risk of occlusal anomalies. In these children, there is a risk of affecting the neighboring teeth and the dental buds, organic changes in the alveolar process and the jaws, and swelling of the soft parts, which require the continuation of the treatment in an outpatient setting. Children are typically classified as having the first and second physical status (ASA), with changes in general condition, blood, and urine (erythropenia, low hemoglobin, and accelerated ESR).

The third group is characterized by multiple chronic foci (pulp necrosis). Sometimes the teeth are intact but with pulp necrosis, mortified tooth buds, and destructive changes of the jaws, affecting the growth areas and increasing the risk of abnormalities in the facial and maxillary bone development, as well as changes in the occlusion and the facial shape.

Children falling into the third, fourth, and fifth (ASA) health status groups. There are fluctuations in body temperature and changes in blood and urine tests. Patients in this group require repeated treatment in an inpatient setting.

The schedule of rehabilitation of children in the first group includes the examination of the child 30 days after finishing the treatment, during the acute period, at 3-6 months, and after the eruption of the permanent dentition. During the 30-day period, the oral cavity is rehabilitated and sanitized; physiotherapeutic treatments and myogymnastics are administered. A clinical and laboratory examination of the internal organs is performed. After the 30-day period, when the examination and treatment are completed, the restoration of the affected tissues (bone, dental, and soft parts), which have been involved in the infectious process, is confirmed clinically and radiologically. The dental examination is performed clinically and radiologically every 3-6 months, with special attention paid to the teeth treated for infectious foci. Periodontal changes and an extension tendency indicate that the focus has been maintained. In such cases, additional treatment is required.

The repeated examination during the eruption of the permanent dentition can detect possible changes in the hard dental tissues and their remineralization in neighboring regions.

The children in the second group should be monitored for three months. During this period, the oral cavity must be cleansed and sanitized. Physiotherapeutic rehabilitation, along with clinical and laboratory examinations of the internal organs, must be carried out. The children must be examined every month to ascertain that the infectious process in their teeth, bones, and soft tissue has stopped. The dental status, the vital tests of unaffected teeth in the contaminated area, the condition of the dental buds, the dynamics of the tooth eruption, root development, and root resorption must be examined. The results should be compared with similar teeth on the opposite sides. The breakdown of the periapical tissues and the spread to the neighboring teeth and dental buds, as well as pulp necrosis in the neighboring teeth, indicate a chronic infectious process, requiring endodontic, surgical, and orthopedic treatments. In this case, the children need to continue the treatment and should be examined in 2 months. Orthodontic treatment is indicated in order to ensure the smooth growth of the jaws.

Local changes are examined along with the child's general condition and his / her physicalhealth status. If, in the first month, both local and general changes persist, repeated antibacterial and anti-inflammatory therapy, local physiotherapy, and consultation with medical specialists (pediatricians, neuropathologists, etc.) are indicated.

The examination of the child at the third month ascertains the restoration of the bone structure, the development of the dental bud, the formation of the apex, and the timing of the dental eruption. Sometimes, chronic periapical and bone processes persist, which can be confirmed by pulp necrosis, the sequestrum formation, the sequestration of dental buds, and oral or extra-oral fistulas. In these cases, the child's record should be extended. General and local examinations are necessary. Sometimes, children must be treated in the inpatient department.

The restoration of the overall bone and tooth integrity is assessed after six months, following a local, general, clinical, and paraclinical examination.

The rehabilitation in *the third follow-up group* is more difficult and prolonged until adolescence. In the first 6 months, the children require active clinical, paraclinical, and laboratorymonitoring, as well as repeated surgical and general treatment, until the remission of the infectious process is ascertained. The oral cavity must be cleansed and sanitized.

After 6 months, the degree of involvement of the growth areas, the jaw bone defects, the chronic periapical foci, and the abnormal face shape as a result of the infectious process should be ascertained. Until the jaws grow definitively, orthodontic treatment is indicated in order to make the jaws harmoniously grow and teeth erupt completely on the dental arch. Also, dental sanation, intensive myotherapy, massage, physical therapy, and sometimes even plastic surgery of bone defects and soft parts are necessary.

The general principles of treatment of oro-maxillofacial infections in children are identical to those in adults and consist of surgical and general medical care. The sanation of foci that triggered the infection (odontogenic, rhinogenic, otogenic, etc.) and the drainage of purulent collections are the basis of the surgical treatment. In children, the incisions become large, with wide takeoffsof the periosteum. Removingprimary teeth in odontogenic infections is not discussed. The question of permanent tooth preservation remains debatable. The modern methods of diagnosis and endodontic treatment, combined with the anatomical particularities of the permanent teeth in children, widen the conservation period of both uniradicular and pluriradicular permanent teeth.

The general treatment is indicated according to the individual particularities of the organism and the degree of manifestation of the local and general clinical signs.

In children, antibiotics are indicated according to the particularities of infections in the maxillofacial region, and those that have a wide spectrum of actions should be chosen.

The general antibiotic selection principles include: a) germ sensitivity to antibiotics; b) sufficient antibiotic concentration in the infectious focus; c) definitive inhibition of germ multiplication in the infectious focus.

Antibiotics must be administered only after sensitivity is determined. Problems related to the duration of investigations requireweighing the choice of antibiotic in the first days of treatment. Broad-spectrum antibiotics (penicillin, lincomycin, ampicillin, gentamicin) are indicated before the results of investigations are ready or according to the specific microbial spectrum in various forms of purulent collections.

In infections of nonodontogenic origin, penicillin, oxacillin, and erythromycin are indicated, as are gentamicin, levomycin, and azocillin in severe cases. In infectious processes of odontogenic origin, in relation to the predominance of anaerobic and mixed microorganisms, semisynthetic penicillins are administered, such as ampicillin, carbenicillin, ampiox, erythromycin, metronidazole, and azocillin. In severe cases, gentamicin, clindamycin, metronidazole, rifronamicazine, metronidazole, rifronamide, cefatoxin, ceftriaxon forte, and doxycycline are administered.

In osteomyelitis of the jaws, antibiotics with osteotropic action are indicated: fucidin-sodium, lincomycin; in severe cases, ristamycin and rifampicin.

Synergistic antibiotic combinations (penicillin and gentamicin, penicillin and metronidazole) are particularly effective. Macrolides, tetracyclines, semisynthetic penicillins, doxycycline, and fuzidine act on gram-negative anaerobic microorganisms: bacteroides, fusobacteria, and veillonella. Macrolides are particularly effective in the treatment of maxillofacial infections with S. aureus, and they are eliminated by saliva. They act synergistically with metronidazole, especially on the majority of unsporulated anaerobes.

The facultative anaerobic bacteria are characterized by selective sensitivity. Staphylococci are sensitive to oxacillin, gentamicin, phosphomycin, erythromycin, and ristomycin, and streptococci to penicillin.

Immunotherapy with thymaline and thymogen is indicated for children in the third, fourth, and fifth ASA health groups, in which the purulent process is slow, with acute and chronic evolution.

Staphylococcal anatoxin is administeredin severe infectious processes. Its effectiveness is manifested by lowering the poisoning, normalizing the temperature, improving the general condition, and stabilizing the inflammatory process. Antistaphylococcal gamma globulin has a detoxifying, antibacterial, and antiviral action. It activates phagocytosis and limits swelling.

In order to stimulate phagocytosis, stimulants of the reticulo-endothelial system (methyluracil, nucleinate sodium, pentoxyl) are administered.

Septic processes cause avitaminosis, which accounts for the widespread administration of vitamins C and B (C, B_1, B_2, B_{12}, PP).

Plasma substitutes are used to detoxify the microbial toxins: Dextran70, Dextran40, Polividon, Disol, Holosol, and Dextrose. Children are rarely given parenteral albumin, aminoped, infesol, etc.

The following medicinal preparations are recommended for treating a purulent wound depending on its stage of development: at the first stage (inflammation), preparations with dehydration, anti-necrotic, antibacterial, and analgesic (levomycol, levosin, dioxisole) action are used; at the second stage (proliferation), ointments that contribute to the development of granulation(methyluracil, vinicol, olazole, solcoseryl) are applied; at the third stage (postoperative scarring), ointments that stimulate the regeneration processes (vinylin, polymerol, solcoseryl) are applied.

Physiotherapeutic procedures and laser therapy decreasethe inflammation process and the resorption of toxins from the purulent wound, contributing to the improvement of the general condition.

Learning objectives

1. Definition of phlegmon.
2. Definition of abscess.
3. Clinical characteristics of cellulitis.
4. Etiopathogenesis of oral and maxillofacial infections.
5. Differential diagnosis of mumps.
6. Clinical manifestations of neonatal parotitis.
7. Classification of chronic parotitis.
8. Clinical peculiarities of chronic juvenile recurrent parotitis in children.
9. Differential diagnosis of chronic juvenile recurrent parotitis.
10. General principles of the treatment of chronic, acute, and exacerbated parotitis.
11. Principles of following up children with odontogenic and non-odontogenic oral and maxillofacial infections.

TESTS

1. SC. The submandibular space is limited laterally by:
- A. the inner face of the mandible;
- B. the genioglossus muscle;
- C. the hyoglossus muscle;
- D. Warthon channel;
- E. The lateral pterygoid muscle.

(A)

2. SC. Indicate the type of abscess in which acute pain during chewing, swallowwing, and extremely difficult phonation are present:
- A. Lateral pharyngeal;
- B. Pterigomandibular;
- C. Infratemporal;
- D. Sublingual;
- E. Lingual.

(E)

3. MC. The differential diagnosis of acute sialadenitis is made between:
- A. acute lymphadenitis;
- B. periadenitis;
- C. adenophlegmon;
- D. external otitis;
- E. internal otitis.

(A, B, C)

4. MC. The clinical characteristics of the saliva that passes through Stenon`s canal in juvenile recurrent parotitis are:
- A. low saliva flow;
- B. purulent saliva flow;
- C. absence of saliva.
- D. thick after a thorough massage;
- E. without modifications.

(A, D)

5. SC. Acute colicky pain during meals occurs in:
- A. acute epidemic parotitis;
- B. acute parotitis;
- C. sialolithiasis;
- D. chronic recurrent parotitis;
- E. allergic sialadenitis.

(C)

6. MC. The radiological picture of chronic recurrent parotitis has the following features:

A. opaque spots of various sizes;
B. opaque spots located at the ends of the salivary canaliculi;
C. narrowing of the canaliculi;
D. narrowing of Stenon`s duct;
E. irregular expansion of Stenon`s duct.

(A, B, E)

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5. ORAL AND MAXILLOFACIAL INFECTIONS. ACUTE AND CHRONIC LYMPHADENOPATHY OF THE HEAD AND NECK REGIONS IN CHILDREN

The lymph nodes are an essential part of the body's immune system and, as such, are affected by many infectious, autoimmune, metabolic, and malignant diseases. A varying degree of clinical relevance – from common infections to malignant diseases – demands a clear and thoughtful approach to the child's individual clinical presentation. A pathologic or abnormal lymph node is larger than 1 cm in size and, in the pediatric population, more than 2 cm in size. Acute lymphadenopathy lasts for two weeks, the subacute form for 2-6 weeks. Chronic lymphadenopathy does not resolve insix weeks. Cervical lymphadenopathy is a medical condition affecting 38-45% of healthy children, and 90% of children aged 4-5 years.

5.1. Peculiarities of the lymphatic system in children

Theoromaxillofacial region represents an extremely rich lymphatic drainage system compared to adults. The lymphatic system makes up about 5% of the body's weight, and the lymph nodes make up most of the lymphatic system. 30% of their total number is in the head and neck region (*figure 4*).

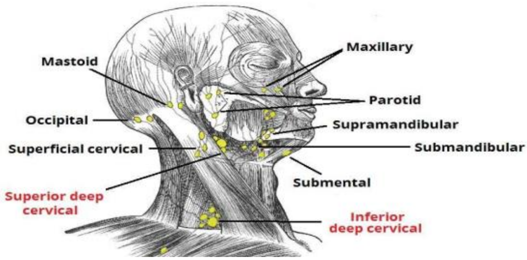

Fig. 4. **The lymphatic system inthe head and neck region**

Lymph nodes play an important role in the immune system. They play a role in the specific and nonspecific defense of the body against physical, chemical, and infectious aggressions, being closely correlated with the process of lymphocytopoiesis and the formation of specific antibodies, as well as filtering, metabolic, and lymphocytic functions.

From a morphological point of view, at the level of a lymph node, three distinct regions can be observed: the capsule, the cortical area, and the medullary area, each of which performs well-defined functions. The lymphatic system in children passes through a period of difficult morphological and functional development. In newborns and infants, the lymphatic system is incomplete, and the immunity is immature. During this period, the lymph nodes are representedby young cells with reduced defense capacity; they are not clinically determined and are not involved in inflammation processes. They develop during the first three years of a child's life. In 3-5-year-olds, lymph nodes develop as an independent anatomical unit, but they are represented by young cellular elements with an imperfect phagocytosis capacity. Probably for this reason, in these children there is a compensatory numerical growth of lymph nodes, located even in the anatomical regions where in adults they are not observed. But the lymph nodes are not able to fix the microbes in circulation, and the young cellular elements can be easily infected. That is why, clinically, adenitis occurs more often in children aged 3-5. Gradually, once the body grows and develops, many lymph nodes atrophy or are replaced by fibrous or adipose tissue.

Lymphatic drainage in the head and neck region is important for surgical diagnosis (*figure 5*).

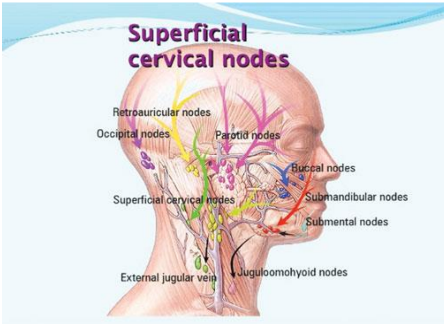

Fig. 5. **Lymphatic drainage of the head and neck**

From a topographic point of view, the cervical-facial lymph nodes are located in groups.

1. The parotid group is well-developed and arranged in two planes in relation to the parotid gland: one superficial, preauricular, located under the fascia, which drains the frontal, temporal, and parietal regions; the other deep, located in the lobe of the parotid gland, which drains the lymph from the tympanic cavity, the nostrils, the hard and soft palatine, the tongue, and the parotid gland. Inconstantly, prior to the tragus, there is a node called the auricle, which drains the lymph from the auricle, the external auditory duct, the conjunctiva, and the eyelid region.

2. Jugular lymph node group – inconsistent, only found in children. It has a single lymph node, located in the following regions: masseter (subcutaneously, at the level of the buccinator muscle along the occlusion line); infraorbital (in the canine fossa); and paramandibular (located on the lateral side of the mandible). Jugular lymph nodes drain the medial portion of the eyelids, nose, upper lip, tonsils, parotid glands, and cheeks.

3. The infraorbital group – located in the canine fossa in the deep and preauricular layers. It drains the lateral portions of the eyelids, the conjunctiva, the jugular skin, and the temporal region of the scalp. These nodules connect with the parotid ones, located in the lateral lobe of the parotid gland.

4. The submandibular group is composed of 5-15 nodules arranged along the lower edge of the mandibular body. In relation to the submandibular salivary gland, they are located anteriorly, posteriorly, superiorly, and inferiorly. They collect lymph at the level of the cheek, submandibular glands, maxillary sinus, nose, upper lip, lower lip, tongue, gums, and teeth.

5. The medial submental group consists of 2-3 lymph nodes located between the digastric muscles. It receives lymphatic afferents from the chin region, ½ ofthe median lower lip, the floor of the mouth, the cheek skin, the bottom lip, and the tip of the tongue.

6. The mastoid group is located behind the auricular concha and posterior margin of the mandibular branch. It is made up of 3-4 lymph nodes that drain lymph from the external auditory duct, the auricular concha, and the temporal portion of the hairy skin of the head.

7. The occipital and posterior auricular groups consist of 2-3 nodules located at the level of the upper cervical vertebrae. They drain lymph from the area of the posterior occipital scalp and the superficial upper-posterior portion of the neck.

8. The posterior cervical lymph nodes collect lymph from the amygdala region, scalp, neck, and the root of the tongue.

9. The lateral pharyngeal group receives afferents from the pterygomaxillary space, the lateral wall of the pharynx, and the deep region of the face.

10. The cervical lymph nodes are located in the deep regions (which extend along the jugular vein, below the sternocleidomastoid muscle) and the superficial regions (the upper and lower regions). The upper portion drains lymph from the posterior regions of the tongue and pharynx**.** The inferior group receives lymph from the deep cervical regions (larynx, trachea, thyroid gland, and esophagus).

The superficial cervical lymph nodes are located above the sterno-cleidomastoid muscle. The anterior lymph nodes (located at the level of the anterior jugular vein) and the posterior ones (located at the posterior cervical triangle) receive lymph from the superficial regions of the neck, the mastoid regions, the postoperative area, and the nasopharynx.

5.2. Classification of cervical lymphadenitis. Etiology, pathogenesis, and diagnosis

Cervical lymphadenitis is classified into four categories depending on the etiology of infectious causes: 1) acute unilateral cervical lymphadenitis, 2) acute bilateral cervical lymphadenitis, 3) subacute or chronic bilateral cervical lymphadenitis, and 3) subacute/chronic unilateral cervical lymphadenitis. A distinction is made between acute (<2 weeks), subacute (2-6 weeks), and chronic (>6 weeks) forms of lymphadenitis.

The lymph node swelling in young patients, from babies to infants and adolescents, is benign in over 80% of cases. The submandibular lymph nodes are affected in 50% of cases and the superior cervical lymph nodes in 25%. Several groups of lymph nodesare usuallyaffected. The infection may spread to the lymph nodes on the opposite side of the body.

Etiology and pathogenesis Many different etiological factors contribute to the enlargement of lymph nodes in children. In some cases, the cause is obvious, in others it is obscure. The enlargement may be focal, regional or generalized; or the first two types may progress to the third one. The enlargement may be the only clinical manifestation of the disease or it may be accompanied by various signs and symptoms, such as pain, pyrexia, exanthemata, changes in the blood or pressure effects on the viscera.

Nonspecific, acute or chronic involvement of the cervical-facial lymph nodes is caused by a remote infection in the drained area of the lymph nodes. The infection can occur either directly or indirectly (hematogenously). The causes may be as follows: chronic and acute dento-periodontal infectious processes (diffuse acute pulpitis, pulp necrosis, periostitis, osteomyelitis), pharyngeal tonsillitis, acute respiratory viral infections, acute bronchitis, skin infections of the face and neck (pyodermitis, furuncle), and contagious diseases (measles, mumps, scarlet fever, etc.).

The etiology of adenitis in children varies according to their age. Thus, at 1-3 years, the causes of adenitis are acute bronchitis, acute viral respiratory infections, and stomatitis; at 3-6 years, the most common are odontogenic causes. Otolaryngological etiology appears more frequently at the age of 4-7 years.

Lymphadenitis is caused by the polymorphic microbial flora (bacteria, viruses, rickettsii, fungi, parasites), which corresponds to the current level of the entrance door. In children older than 3 months, most acute adenitis is caused by *Staphylococcus aureus* and *Streptococcus pyogenes* (group A Streptococci), anaerobes that are normally in the oral cavity. In children up to 3 months, *Streptococcus agalactiae* (group B Streptococci) is common. The likelihood of infections caused by *Staphylococcus aureus* is high in these children. Rarely, infections are caused by gram-negative microorganisms such as *Ecsherichia coli*. Septic status, as an important complication of bacterial adenitis, occurs more often in children up to 3 months.

Acute jugulo-digastric bacterial adenitis is associated with acute tonsillitis. In these inflammations, group A *Streptococcus* is often detected. This syndrome is associated with fever, severe bronchitis, frontal headache, abdominal pain, and severe intoxication. The follow-up examination can detect an increase in the volume of lymph nodes and severe acute tonsillitis.

Microorganisms reach the infected lymph nodes via lymphatic flow from the inoculation sites, by lymphatic flow from adjacent lymph nodes, or by hematogenous spread of systemic infection. Once in the lymph nodes, dendritic cells and macrophages trap, phagocytose, degrade, and present the organisms as antigens on MHC molecules. These antigens are presented to T cells, which leads to the proliferation of clonal cells and the release of cytokines important for the chemotaxis of other inflammatory cells. The B cell is such a cell. B cells, with the help of T cells, are activated, proliferate, and release immunoglobulins that aid in the immune response. This immune response causes cellular hyperplasia, leukocyte infiltration, tissue edema, vasodilation and capillary leak, and capsule distension, which leads to tenderness.

The primary diagnostic tools are anamnesis and clinical examination. The anamnesis must include a past sore throat, earache, or toothache, insect bites, or injuries. Lymph node enlargement is common in infectious diseases (tuberculosis, HIV, parasitoids). The same holds true for symptoms including joint or bone pain, weakness, and anorexia. Any possible contact with animals (cats, rabbits, rodents, tick bites, etc.) and vaccinations must be recorded in the anamnesis. Other environmental factors and noxa (including drugs) are rare causes of lymphadenopathy. The family history not only includes acute infections but also chronic and systemic diseases. The physical exam includes the evaluation of the skin for signs of cellulitis, impetigo, and rash. Examination of the head and neck lymph nodes is the most important part of the differential diagnosis and includes examination of the submental and submandibular lymph nodes at the angle of the mandible and the pre- and post-auricular occipital lymph nodes. These are followed by the group of vertical lymph nodes of the neck along the jugular vein and the posterior cervical lymph nodes, the anterior edge of the trapezius muscles, and the supraclavicular region in the supraclavicular fossa. Examination must include other regions, in particular the axillary and inguinal lymph nodes, liver, and spleen, in order to assess systemic involvement. Palpation of the deep cervical lymph nodes in children is difficult because of the particularities of this area (shot neck, thick fat layer).

The lymph nodes are palpated for tenderness, consistency, and mobility in relation to the surrounding tissue and adjacent lymph nodes; the size - unilateral vs. bilateral, tender vs. non-tender, mobile vs. fixed, hard vs. soft. The location of the lymph node can play an additional role in securing the diagnosis. The occipital lymph nodes are often affected by scalp infections, toxoplasmosis, inflammation of the outer ear, and rubella. The preauricular lymph nodes may indicate infections of the eye, ears, teeth, or parotid gland. The submental or submandibular lymph nodes are often conspicuous in infections of the oral cavity, nose, maxillary sinus, or face. Lymphomas may appear anywhere but often affect the lymph nodes along the jugular vein, occipital, or supraclavicular regions.

If the anamnesis and clinical examination prove inconclusive, or if confirmation of a suspected diagnosis is required, further diagnostic means are available, including serological tests, sonography as the main imaging technique, and, forcertain special indications, MRI and CT. As a means of obtaining histological confirmation, full dissection of a suspicious lymph node should take preference over fine-needle aspiration cytology (FNAC).

Acute lymphadenopathy (*figure 4*) is almost always due to infectious causes and lasts less than 2 weeks. Reactive cervical lymphadenopathy has a viral etiology (rhinovirus, enterovirus such as Coxsackie A and B, and Epstein-Barr virus) and is the most common type. Acute bilateral lymphadenitis is the most common clinical manifestationand is often diffuse, bilateral, and non-tender. Lymph nodes are small, rubbery, mobile, discrete, minimally tender, and without erythema. They are often referred to as „reactive” or „shotty” lymphadenopathy. Coughing, rhinorrhea, and low-grade fever are common complaints.

Fig. 4. **Lymphadenopathy. Acute purulent lymphadenitis in the submandibular area.**

Suppurative bacterial lymphadenitis is the most common lymphadenitis in children older than 3 years of age and is caused by Group A Streptococci and *Staphylococci aureus*. It represents about 57.5% of all cases of lymphadenitis, usually characterized by unilateral lymphadenitis. Common physical findings include erythema or tenderness of the overlying skin in 48.3% of cases and fever in 24.1% of cases. About31% of patients with bacterial lymphadenitis have a phlegmon, infiltrate, or abscess. Management includes oral or intravenous antibiotics, depending on the general appearance and reliability of the child's parents. If an abscess is suspected, a contrast CT and ultrasound are important adjuncts in the evaluation. Common findings of abscess formation are spiking fevers, fluctuance, dysphagia, or airway compromise.

5.3. Clinical picture of acute lymphadenitis

The clinical picture depends on the age of the child and the location of the affected lymph nodes. The local clinical manifestations can be well-appearing or ill-appearing. The ill-appearing children who have acute suppurative cervical lymphadenitis are common in young children; lymph nodes are located in the deep spaces, and the general clinical signs predominate over the local ones. Usually, in these cases, the swelling presents a barely perceptible asymmetry, sometimes painful torticollis, and no skin changes (*figure 5*). For well-appearing children, lymph nodes are located superficially and do not present difficulties in diagnosis. Swollen lymph nodes originating from an infection are painful, soft, and fluctuant if they are abscessed. In the early stages of diagnosis, the general signs are insignificant (slight subfebrility).

Fig. 5. **Purulent non-odontogenic adenopathy in the neck area**

Acute unilateral cervical lymphadenitis in the newborn is caused by *Staph aureus* in most cases. The late-onset group B streptococcal infection *(Streptococcus agalactiae*), that is, „cellulitis-adenitis" syndrome, isanother cause of neonatal acute cervical lymphadenitis. The onset lasts for seven to ninety days. The most affected patients are between 3 and 7 weeks of age; boys are affected in 75% of cases, being febrile and irritable and having a lack of appetite. The examination reveals tender, erythematous facial or submandibular swellings with poorly defined margins. Bacteremia is common. Tularemia is a febrile illness caused by an infection with *Francisella tularensis* that usually occurs following contact with infected animals (e.g., rabbits, hamsters; more than 100 species have been involved), the bite of blood-sucking arthropods, inhalation of organisms in contaminated environments, or ingestion of contaminated water. The ulceroglandular form is the most common clinical type, characterized by a papular lesion in the drainage field of the swollen lymph node within 72 hours of infection and painful ulcerationwithin a few days. Glandular tularemia is similar in presentation, but there are no skin lesions.

Adenitis in the submandibular region should be differentiated from acute submaxillitis and epidemic parotitis with onset in the submandibular salivary gland, characterized by salivary retention, congestion of the sublingual ridge, and Wharton's duct orifice. The bimanual palpation highlights the enlarged submaxillary gland. In acute submaxillitis, radiopaque calculi are found in the Wharton's duct. The clinical picture of acute intra-parotid adenitis is similar to acute parotitis, but it differs in the absence of changes in the salivary secretion.

Acute cervical adenitis must be distinguished from systemic or metastatic lymphonodular processes as well as laterocervical glandular cysts. Lymphadenopathy associated with lymphoma is typically firm, rubbery, and painless. Metastatic lymph nodes are hard and rarely mobile.

5.4. Lymphadenopathy of the oro-maxillofacial and neckregionsin children

Adenophlegmon is a purulent infectious process that has overcome the lymph node barrier and diffusedinto the space. It affects children in early childhood, from 2 months to 7 years. It is located in the genian, submandibular, submental, and parotidian regions. One-third of children with adenophlegmon have acute respiratory infections: bronchitis, acute otitis, pneumonia, and pyoderma. Its evolution is severe in children aged one to three years.

The presence of a thick layer of adipose tissue in the maxillofacial region, the dense vascularization network, anastomoses, and the membranes separating the thin perimaxillary spaces all contribute to the spread of the infectious process in the adjacent anatomical spaces. Swelling located in the regions of the tongue, parapharyngeal, cervical, and buccal floor can be complicated by marked edema, which may be the cause of asphyxiation.

The diagnosis of adenophlegmon is made by anamnesis (nodular onset), the lack of functional disorders (trismus), and the absence of odontogenic foci.

Osteophlegmon, unlike adenophlegmon, developsinto severe forms. Phlegmon worsens the osteomyelitic process andthe general condition. The infectious process in acute osteomyelitis is due to erosion of the periosteum and the direct spread of the purulent process into the adjacent soft parts. Hematogenous osteomyelitis is a severe complicationof the maxilla in infants and newborns that is characterized by the development of retrobulbar and orbital phlegm. Often, odontogenic osteomyelitis generates phlegmon in the perimandibular spaces. The diagnosis of osteophlegmon is made based on the presence of osteoperiosteal foci and marked functional signs, such as trismus caused by the alteration of the mobilizing muscles of the mandible.

Periadenitis is a reaction of the soft parts to the intraganglionary inflammatory process in the phase of crudity, in which there is an enlarged lymph node and increased pain that is spontaneous and palpable, adhering to the superficial and deep planes. The general condition is not significantly affected. Locally, thelymph node is located in an anatomical area with limited mobility, no signs of fluctuation, and an intact covering tegument.

Differential diagnosis. Periadenitis must be distinguished from adenitis, furuncles, and adenophlegmons.

Subacute lymphadenitis persists for 2-6 weeks and is most commonly infectious. The average age of patients is 4.7 years, with a slight female predominance of 53%. The average lymph node size is 3.2 cm. The superior cervical and submandibular regions are most commonly affected. Atypical mycobacteria, cat scratch disease, and toxoplasmosis are the most common causative organisms. To a lesser extent, EBV and CMV are also involved at times.

Toxoplasma gondii is a parasite that causes toxoplasmosis. The consumption of undercooked meat or the ingestion of oocytes from cat feces is the most common route of transmission. Symptoms include malaise, fever, sore throat, and myalgia, and 90% of patients have cervical lymphadenopathy. Adenopathy may be localized or generalized, tender or non-tender, and may persist for many months. This disease is usually benign and self-limited. The diagnosis is made by serologic antibody testing. Complications include pneumonitis and myocarditis. Given the complications, the treatment is absolutely necessary, and antibiotic choices include pyrimethamine or sulfonamides.

Atypical Mycobacterial infections are the most common cause of subacute lymphadenopathy. Most NTM infections occur in immunocompetent children under the age of 5 years. The most commonly involved species include *M. avium-intracellulare, M. haemophilum,* and *M. scrofulaceum*. These infections develop over weeks to months, and in untreated cases, sinus tracts and cutaneous drainage usually develop for up to 12 months. Common findings on physical exams include swollen lymph nodes that are tender and rubbery, as well as a violaceous discoloration of the overlying skin. Diagnosis can be made by acid-fast stain, culture, or even PCR, as described earlier. Historically, surgical excision has been the method of treatment. However, there is some evidence that expectant management may be the best treatment. Atypical infections differ from tuberculous mycobacterial lymphadenopathy in that tuberculous adenopathy is often an ominoussign of disseminated disease and needs to be treated more aggressively.

Cat-Scratch disease (*Benign inoculation lymphoreticulosis-Debre*) is a form of lymphadenitis caused by *Bartonella henselae*. It usually develops in patients under 20 years of age and there is a male predominance. 90% of patients are exposed to a cat bite or scratch. However, fleas and dogs have also been known to carry the organism. The inoculation of *Bartonella henselae* into the skin following a kitten or cat lick, bite, or scratch has been recorded. In 50% to 90% of the patients, there is inflammation at the site of inoculation - a hyperemic papule - occurring 3 to 10 days after inoculation. Adenopathy develops in 1-2 weeks. The disease progresses to spontaneous healing within 2-3 weeks. However, after3-4 weeks, the nodules can grow considerably. In 85% of cases, only one lymph node is affected, located distally from the inoculation site. In 50% of cases, the nodules in the head and neck region are affected. In infants, the infectious process can rarely become generalized. Lymphadenopathy, most commonly in the cervical or axillary regions, can take up to 2 weeks to develop and is usually tender, but systemic signs of malaise and fever are mild and present in less than 50% of patients. Cat-scratch disease may also manifestas Parinaud oculoglandular syndrome, with conjunctivitis and preauricular or submandibular lymphadenopathy following conjunctival inoculation. The diagnosis is made based on serology or PCR, if available. Antibiotic treatment has been traditionally used for patients with severe disease, along with appropriate management. Antibiotics are almost always given to immunocompromised patients due to the risk of disseminated disease, which can be deadly.

Infectious Mononucleosis causescervical adenopathy or even acute polyadenopathy without suppuration. The Epstein-Barr virus is the most common pathogen causing the disease, although Cytomegalovirus has also been found to be involved. Signs and symptoms include fever, a gray-colored exudative pharyngitis, painless and generalized lymphadenopathy, axillary lymphadenopathy with hepatosplenomegaly, leukocytosis, and monocytosis. On peripheral blood smears, more than 50% of patients have lymphocytosis, and morethan 10% of lymphocytes are atypical. The serological examination confirms the diagnosis.

Adenopathy following HIV infection may be a sign of the onset of AIDS. If the local-regional etiological factors are missing and the evolution is prolonged, the HIV serological test should be carried out.

Chronic Lymphadenitis. Once a lymph node has been present for more than 6 weeks, there is a risk of malignancy. The most suspected causes of malignancy in young patients under 6 years of age are acute leukemia, neuroblastoma, rhabdomyosarcoma, and non-Hodgkin's lymphoma. Hodgkin's lymphoma, rhabdomyosarcoma, and thyroid cancer occur more rarely. Nodal metastases associated with nasopharyngeal carcinoma are the most common malignancies.

Hodgkin's disease is the leading malignant cause of neck masses in children and adolescents, beginning at the age of 13 years. The clinical criteria consistent with malignancy are as follows: size over 2-2.5 cm; firm and hard consistency; supraclavicular/axillary localization; posterior edge of the sternocleidomastoid muscle; absence of tenderness; low mobility; progressive courses.

Hodgkin's disease occurs through cervical adenopathy, sometimes associated with axillary, mediastinal, or abdominal adenopathy. It has a masked onset of a characteristic symptomatology: fever, asthenia, night sweats, and generalized itching; the lymph nodes are symmetrical, moderately enlarged, firm in consistency, mobile, and painless. The diagnosis is made by the histopathological examination, which detects the specific Paltauf-Sternberg cells.

Lymphadenopathy secondary to malignancies. The diagnosis can be easily made when the tumor is accessible for clinical examination, but it is extremely difficult when malignant tumors develop without any obvious symptomatology and are not clinically evident. In such cases, the primary tumor is detected following the histopathological examination. Histological examination of neck masses in children shows malignant disease in 11% of the cases.

Cervical lymph node swelling of uncertain origin in childhood can pose a special challenge. Given the spectrum of possible differential diagnoses and malignant diseases, the diagnosis must follow a clearly structured algorithm of diagnostic steps and therapeutic strategies to avoid unnecessary delays in further diagnosis and safeguard against overly hasty, and possibly too invasive, diagnosis and therapy.

Learning Objectives

1. Anatomical peculiarities of the lymphatic system in children.
2. Classification of lymphadenitis.
3. Clinical picture of acute serous lymphadenitis in children.
4. Treatment of serous lymphadenitis in children.
5. Clinical picture of acute purulent lymphadenitis in children and its treatment.
6. Cellulite, periadenitis, adenophlegmon: clinical picture, differential diagnosis, and treatment.
7. Chronic lymphadenitis: clinic, differential diagnosis, and treatment.
8. Treatment of lymphadenitis in children

TESTS

1. SC. The lymphatic system in children fulfills its barrier function in the following age groups:
   A. 1-3 years;
   B. 4-6 years;
   C. 7-8 years;
   D. 9-10 years;
   E. All answers are correct.

(C)

2. SC. Lymphadenitis occurs more often in the following age groups:
   A. 3-5 years;
   B. 6-9 years;
   C. In infants;
   D. In teenagers;
   E. In all age groups, with the same frequency.

(A)

3. SC. The development of the lymph nodes includes the following age groups:
   A. The first 3 years of life;
   B. The first year of life;
   C. The first month of life;
   D. Before the adolescence period;
   E. No answer is correct.

(A)

4. MC. Which type of acute lymphadenitis is irreversible:

A. chronic lymphadenitis;
B. adenophlegmons;
C. paradenitis;
D. purulent lymphadenitis;
E. serous lymphadenitis.

C, E)

5. SC. Surgical treatment and focus drainage must be performed in adenitis in:

A. acute intraosseousphase;
B. acute purulent;
C. hyperplastic chronic;
D. periadenitis;
E. all answers are correct.

(B)

6. SC. Point out the benefits of management of acute lymphadenitis in the neck area in children under 5 years:

A. surgical treatment;
B. treatment with antibiotics;
C. local applications of poultices;
D. suppression of the causal factor;
E. heliomarine cure.

(D)

7. MC. Indicate the groups of lymph nodes present only in children:

A. submandibular;
B. submental;
C. jugular;
D. lingual;
E. paramandibular.

(C, E)

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6. ODONTOGENIC INFECTIONS OF THE JAWS IN CHILDREN.

The odontogenic infection of the jaws is the most frequent disease in children because dental caries remains the most common chronic disease from 1 to 5, 6 to 11, 12 to 18. The mean prevalence of carious lesions in primary dentition is about 33.85%. Caries is estimated to affect approximately 28% of children aged 2 to 5 years and approximately 52% of children aged 6-8 years. These conditions lead to a frequent incidence of odontogenic infections

The onset is apparently associated with a lack of resistance to a particular microorganism that invades the bone and usually arises when normal buccal flora changes from commensal to opportunistic due to a broken balance with the host in certain circumstances. Oral infections can be acute (acute onset, fast evolution, obvious signs and symptoms) or chronic (slow onset and evolution, fewer obvious signs and symptoms). They are classified as odontogenic and non-odontogenic. Odontogenic infections begin as a result of periapical and pulpal necrosis. Non-odontogenic infections start in extra-dental structures (mucous membranes, glands, tongue, etc.) and may begin through wounds, fractures, a hematogenous route, or anything that admits microorganisms into the jaw bone.

Based on the clinical onset and pathomorphology, periostitis is classified into:

1. acute serous periostitis (intraosseous stage of vestibular abscess),
2. acute purulent periostitis (subperiosteal stage)
3. chronic periostitis.

Odontogenic infections are usually localized and respond well to treatment. However, due to children`s unique characteristics, they can spread to remote areas and cause serious processes, even threatening the patient's life. The general anatomical and physiological characteristics of children vary because the age range covered by pediatric dentistry is wide. For instance, the percent of body water and fat, as well as liver enzymes and plasma protein levels, are different in neonates and infants from those of children and adolescents. Therefore, body size and composition, immature gastrointestinal, renal, and immune systems, and nutritional status should be considered when assessing odonto-pediatric patients. In the same way, anatomical features of children's jaws are unique because they presentdental follicles, resorption, and root formation with the permanent open dental apex; maintain intimate communication of the dental pulp with the bone marrow; have large nutritional Havers` channels and a larger amount of less trabeculated cancellous bone but with larger trabeculae; and are highly vascularized with extensive marrow spaces and bigger medullary holes and growth sites. The specifics of the physical (general and local) state of the children affect the extent and rapidity of the spread from one area to another.

The severity of the infection increases in children who have had acute viral diseases or contagious diseases (chicken pox, scarlet fever, mumps, etc.). During the epidemic of viral infections, the frequency of acute periostitis and osteomyelitis increases. A careful history, including a review of systems, physical examination, appropriate laboratory studies, and proper interpretation of the findings, is the basis of patient evaluation.

6.1. Peculiarities of odontogenic periostitis

Odontogenic periostitis is an inflammation of the periosteum caused by infections of the dental and periodontal tissues. In children, acute odontogenic periostitis may develop as a nosological form or as a symptom of some diseases, such as acute purulent pulpitis, odontogenic purulent cysts, and acute odontogenic osteomyelitis. Dento-alveolar abscess is the most common type of odontogenic infection in children (50%). Chronic periostitis manifests as a sign of chronic osteomyelitis of the jaws and chronic apical odontogenic infection.

The etiology of periostitis is typically of dental origin, with a microbial flora and pulp necrosis. The odontogenic infection starts within the dental pulp, located within rigid, calcified walls that contain vascular, neural, and connective tissue.

Invaded by bacteria, inflammation and edema of the pulp rapidly cause venous congestion or avascular necrosis. The lack of collateral blood supply may cause the death of the pulp, which may become a reservoir for bacterial growth. Bacterial reservoirs can easily egress into the surrounding alveolar bone due to the specific conditions in the root and apex areas of the primary and permanent dentition during development.

The microbiological examination showed an association of microorganisms: staphylococci (white and gold, 66%) and β-streptococci (32%), enterococci, fusospiral associations, anaerobes (Clostridium perfringens, oedematiens), and in some cases, Gram-positive and Gram-negative microorganisms (2%).

The jaw periosteum in children, being in an active state of multiplication with high vascularization, reacts even to microbial flora with low virulence. The process of inflammation is extremely complex. Hyperemia is mainly caused by the vasodilation of arterioles and capillaries, increased permeability of venules with slow venous blood flow, the passage of exudates rich in plasma proteins, the escape of leukocytes into the surrounding tissues, and the release of a permeability factor, that allows migration of polymorphonuclear leukocytes and later monocytes into the area.

Periostitis is found in children of all ages but occurs less frequently in children up to 3 years. At 3-7 years, periostitis is caused mainly by the two primary molars (49%), the premolars (30%), the first permanent molar (14%), the milk incisors (4%), and the canines (3%). The number of cases of periostitis increases between the ages of 7-15 years, during the active period of dental physiological change. During this period, periostitis is caused by the dento-periodontal lesions of the first permanent molars (66%), the primary molars (16%), the premolars (6%), the milk canines (3%), and very rarely, the two permanent molars, incisors, and premolars (9%).

Children are not able to explain their acute or chronic pain, and therefore it is left misunderstood, underdiagnosed, and undertreated, especially in the case of toothaches. Many children deny pain because of fear of disappointing caregivers or fear of medical procedures. If pain is not addressed and treated early on, it can greatly impact a child's quality of life by interfering with mood, sleep, appetite, school attendance, academic performance, and participation in sports and other extracurricular activities. If unrelieved, childhood pain can increase a child’s vulnerabi-lity to pain later in life. Repeated exposure to pain causes altered pain sensitivity, anxiety, stress disorders, hyperactivity, attention deficit disorder, impaired social skills, and patterns of self-destructive behavior. Acute or chronic pain can affect children in ways that will follow them throughout their lives. They can develop emotional and psychological scars that can taint future choices concerning their lives and health care. Untreated pain in childhood leads to chronic pain in adulthood and old age.

One of the most difficult challenges, both professionally and emotionally, is learning to handle pain in pediatric patients. It is essential that healthcare providers begin to recognize pediatric pain. There are several categories of pain. One of the most common is that which is associated with a disease state (arthritis, sickle-cell disease) or that which is associated with an observable physical injury (burns, fractures). Some of the most challenging conditions involve pain that is not associated with a well-defined or specific disease, state, or physical injury (tension headache, toothache, etc.). Pain may be caused by habits and behaviors as well as by medical providers.

Pain assessment in children is linked to their developmental stage (neonates, toddlers, and school-age children). Often, children of the same age varywidely in their perception and tolerance of pain. In conditions of acute or chronic pain, the distinguishable alterations in the child's behavior are associated with favoring one arm or leg over the other; a decrease in physical activity; changes in appetite or sleep pattern; avoiding contact with other children; crankiness, irritability, unruly behavior; nonverbal expressions of pain such as gasping, wincing, or frowning; physical cues like eyes, flushed skin, rapid breathing, or sweating.

Accurate pain assessment in children is the main goal in the treatment and management of odontogenic infections. An assessment strategy of the child's pain must be done regarding the level of develop-pment and include several factors that can modify pain perception: age, cognition, previous pain experience, temperament, cultural and family factors, and situational factors. For this reason, there are three widely used categories of behavioral indications of pain: global rating scale (GRS), behavioral observation scales (BOS), and indirect measures. GRS is based on the assessment of predictable behavioral indicators of pain, such as crying, wincing, and screaming. Indirect measures of pain may be assessed by requests for medication. BOS focuses on the documentation of physiologic measures (heat rate, blood pressure).

Neonates may manifest pain by crying, being silent, wiggling, or being still. Fear is a large contributor to the experience of pain in toddlers and school-age children.

Toddlers may become very quiet and inactive while in pain, or they may become very active and manifest their pain fear through aggressive outbursts. Behavior may be difficult due to exacerbating factors such as separation anxiety, the memory of previous painful experiences, or physical restraint.

School-aged children explain their pain more accurately. By the age of 8 years, children can very reliably describe the location of pain. School-age children can exercise self-control when they experience pain. They may not report pain in an attempt to show bravery.

Accurate and early diagnosis is an essential prerequisite for successful treatment and control of dental diseases and developmental disturbances. However, the relatively high prevalence of dental diseases and abnormalities in infants and children eligible for medical careas well as the limited sensitivity of current screening procedures provide strong clinical justification for children receiving diagnostic examinations by a dentist beginning at an early age. For these reasons, the clinical examination of the young patient initially comprises inspection and palpation, both intra- and extra-orally, which are the main steps in establishing the diagnosis.

6.2. Acute serous periostitis

Acute serous odontogenic periostitis begins with acute apical periodontitis or diffuse acute pulpitis. Periostitis in the intraosseous stage is not recognized as a nosological type of inflammation; it is merely a symptom that permanently accompanies the infectious lesions caused bypulp necrosis. At the age of 3-5 years, serous periostitis often manifests as acute pulpitis, regardless of the group of offending teeth. Acute periostitis is a permanent symptom of acute periodontitis or an exacerbation of chronic periodontitis in children. Acute periostitis can cause swelling of the soft parts of the affected tooth that lasts for several days after the tooth is extracted.

The clinical signs of periostitis are determined by the child's age. Acute serous periostitis begins with acute pulpitis, or exacerbated chronic periodontitis. The main complaint is spontaneous pain, or pain caused by touching the offending tooth in the affected part. Usually, pain is caused by distension of the periosteum due to the pressure of trapped pus that has crossed the bone and becomecontinuous and violent. This pain can extend to the ear, neck, and head and may get worse when lying down. Extraoral examination reveals a slight facial asymmetry. The skin color in the swollen area is not altered. The localization of the facial swelling depends on the offending tooth, and it usually extends to one or two areas of the head and neck.

Intraorally, the gum is congested and edematous, with a discrete swelling in the vestibular fold. The edema is located on the alveolar process mucosa, extending to the offendingtooth or within two teeth. It is extremely painful to touch, with a thickening that has no defined limits. The regional lymph nodes are enlarged and tender to palpation. The general condition can be altered by mild fever, insomnia, anorexia, embarrassment, chewing, etc.

Differential diagnosis. Periostitis must be distinguished from diffuse acute pulpitis, acute periodontitis, and acute dentoalveolar abscesses.

Treatment depends on the cause of periostitis. In most cases, infections such as acute pulp infections are localized in the offending tooth. Treatment is indicated according to the degree of root formation and the child's age. In such cases, management of localized pulpal infections in the primary dentition includes root canal treatment, extraction, and space maintenance. Treatment of a spreading acute dental abscess centers on pain control, antibiotics, surgical drainage, and removal of the source of infection, which may include endodontic treatment or tooth extraction.

The antibiotic used for acute oral infections in children is amoxicillin, usually prescribed at a dosage of 20-40 mg/kg/day, in three divided daily doses for five days. For children who are allergic to penicillin, oral clindamycin is administered at a dose of 15-25 mg/kg/day in three or four equal daily doses.

Children who exhibit signs of the dental infection spreading to neighboring areas, the bottom of the vestibule, or other facial regions, or affecting the patient's appearance, require hospitalization and parenterally administered antibiotics.The hospitalization criteria for children who develop dental infections are as follows: general involvement and immune-compromised patient (diabetes, malnutrition, HIV, etc.); rapidly progressive cellulitis; dyspnea, dysphagia; diffusion to deep facialspaces; fever higher than 38˚; intense trismus less than 10 mm; patient who does not cooperate or is unable to follow the ambulatory treatment; initial treatment failure.

The pluriradicular primary teeth that cause acute periostitis are indicated for extraction. The monoradicular teethwith formed roots should be treated. Periostitis in acute diffuse pulpitis should be treated in both the permanent and primary dentition.

6.3. Acute purulent periostitis (dentoalveolar vestibular abscess)

Acute purulent periostitis is a further evolution of acute serous periostitis. The most commonly affected teeth are the primary first molars (34%), followed by the primary second molars (31%).A dentoalveolar abscessresults from neglecteddental caries. These infections are polymicrobial, containing both anaerobic and aerobic bacteria. Approximately

50% of odontogenic infections are caused by anaerobic bacteria alone, 44% involve a combination of aerobic and anaerobic bacteria; and only 6% are caused by aerobic bacteria alone. Left untreated, an odontogenic infection can progress to a life-threatening bacterial infection throughout the entire body. Once the infection has passed the apex of the teeth, the pathophysiology of the given infectious process can vary depending on the number and virulence of the organism, host resistance, and anatomy of the involved area.

An acute odontogenic infection originating in the periapical dental regions progresses by direct extension via the blood vessels and lymphatics to other areas far from the site of the initial infection. Due to the specifics of the dental alveolar system (eruption of primary and permanent dentition) and the general condition of children (immature immune system, undeveloped endocrine and nervous systems), the direct invasion involves movement from one area to another and between facial layers very quickly and easily. In children, the serous form passes to the purulent one fairly quickly, evolving in a few hours. After the infection breaks through, it causes a marked swelling of the face with a marked asymmetry, even in serous periostitis (*figure 6*).

Fig. 6. **Odontogenic purulent periostitis. Extraoral view: facial asymmetry.**

When the pus erodes and exceeds the cortex, it accumulates under the periosteum and forms a well-defined subperiosteal abscess. The subperiosteal purulent collection gradually erodes the periosteum and penetrates submucosally. When the pus reaches the submucosal layer, the general condition improves (subperiosteal stage).

The onset of the acute episode of purulent periostitis is painful, with a continuous, sharp, or throbbing pain; pain when chewing; an antalgic position of the head; and a bitter taste in the mouth.

Extraoral examination usually detects a marked diffuse spread of the infection that may involve considerable areas of the head and neck. The overlying skin appears inflated or even purple in color, sometimes shiny and firm.

Intraoral examination reveals the offending tooth, sometimes with mobility, and red and swollen gums. The examination reveals a firm swelling in the vestibule side of the gum, bulging around three or more teeth, located on one side of the gum (usually the vestibular side), and fluctuance on palpation (*figure 7*).

Fig. 7. **Purulent periostitis. Intraoral view.**

The localization of the dentoalveolar abscesses is related to the anatomic position of the dental root from which they originate: the palpebral parts, nose-lip envelopes, upper lip, and submandibular region. The vestibular abscess, which has as its starting point the upper central incisors, is accompanied by swelling and marked edema of the entire upper lip, called the *tapir lip*. The abscess of the canine region is manifested by the swelling of the lower eyelid and the buccal region; the abscess arises from the second primary upper molar - by marked swelling of the cheek, the infraorbital region, andthe orbital region.

Abscesses caused by mandibular molars are accompanied by swelling of the cheek, which can extend to the submandibular region. The processlocated in the region of the mandibular angle causes trismus.

Suppurative periostitis is accompanied by regional lymphadenitis or periadenitis.

The general condition is altered: insomnia, restlessness, fever (38-38.5° C), general malaise. Blood parameters showpoisoning, with an elevated ESR (30-40 mm), leukocytosis (20.0-25.0.10 9/l), lymphopenia, and traces of protein in the urine.

Differential diagnosis. Purulent periostitis must be distinguished fromfacial furuncles, adenophlegmons, acute odontogenic osteomyelitis, acute odontogenic serious periostitis, and acute diffuse pulpitis. Acute periostitis is diagnosed based on the affected tooth, periosteal reactions with inflammation only on one part of the alveolar process (usually the vestibular one), atthe level of 2-3 teeth adjacent to the affected tooth, and the mobility of the affected tooth. Acute odontogenic osteomyelitis is subsequently diagnosed (*ex juvantus*). In the case of acute purulent periostitis, the child's general condition improves within the first 24 hours after the surgical treatment.

The treatment of odontogenic infections may involve medical, surgical, or dental therapy. In children, surgical treatment is performed only under general anesthesia and in specialized hospitals. Infected primary teeth are indicated for extraction without considering the group of teeth (monoradicular or pluriradicular) and their anatomical and functional values. The permanent ones should be removed only when the dental crown is softened and have no anatomical or functional value. The incisions on the transition envelope must be large, with periosteal detachments at the level of 2-3 teeth. The subperiosteal abscess at the maxilla needs to be opened very carefully, as the thin cortex may be damaged, denuding the tooth bud or affecting the maxillary sinus. The palatal abscess is treated by excision of a mucoperiosteal strip.

The choice of antibiotic therapy for odontogenic infection depends on the laboratory results of culture and sensitive testing. A pragmatic, rational approach to empirical antibiotic selection is acceptable, both ethically and legally, if the choice is based on scientific data and contemporary experience, as well as the microbiology of the oral flora in infections.

Prognosis. After a complete and prompt general and local treatment, the disease subsides in 2-3 days. Neglect of dental treatment, visits during advanced infectious lesions, incisions, and small periosteum takeoffs cause periostitis to develop into acute odontogenic osteomyelitis.

6.4. Chronic periostitis. Etiology. Etiology and pathomorphology

Chronic periostitis is known as chronic sclerosing osteomyelitis, sclerosing osteitis, ossifying osteomyelitis, and local bone sclerosis. In children, during the development of the facial skeleton, periosteal osteoproduction processes take place. Because the periosteum is in an active physiological state of bone production, it responds quickly to pathological periapical infections by increasing osteoproduction. Pulpal necrosis is the source of infection that constantly irritates the periosteum. The last one responds bybone production.

Chronic periostitis is an inflammation of the jaws characterized by a gradual onset and painless bone swelling. The causes may be the lack of endodontic treatment of primary teeth or dental trauma after inadequate endodontic treatment of deciduous teeth. Typically, chronic types of lesions in children are discovered during a prophylaxis examination.

Radiographically, chronic sclerosing periostitis is seen either in a focal or diffuse form. The focal form usually occurs in children and is associated with the infected pulp of the lower primary or permanent molar teeth. It appears as a circumscribed radiopaque mass of sclerotic bone associated with tooth roots. As a result of the body's reaction to irritation, the margins may be distinct or blend into the surrounding bone. Histologically, the lesion consists of a dense mass of trabecular bone with little interstitial marrow. Bone growth represents the deposition of new bone tissue on the cortical surface of the jaws.

Diffuse chronic periostitis is limited to the alveolar process. The lesions are asymptomatic. On radiographs, such lesions are dense, radiopaque masses. When associated with a chronically infected dental pulp, these masses represent true chronic periostitis, whereas masses with similar radiographic characteristics not associated with an infected pulp must be differentiated from tumors.

The differential diagnosis. Chronic periostitis must be distinguished fromchronic productive osteomyelitis and benign tumors: sarcoma, osteoma, fibrous dysplasia, and cementoma. The diagnosis of chronic periostitis is established on the basis of anamnesis, clinical, local, and radiological data analysis, and, in difficult cases, by open biopsy.

Treatment of chronic periostitis consists of endodontic therapy or extraction. Affected primary teeth are removed, and permanent ones are treated. Following the treatment, bone lesions may remodel or remain distinct. In those instances, in which a source of infection cannot be detected, the term „focal periapical osteopetrosis" has been proposed. There is no need to surgically treat focal, asymptomatic sclerotic lesions.

In chronic periostitis, antibiotics are not given because they do not decisively influence the treatment of the infectious process. Complex treatment includes follow-up for several months; ionophoresis, sulfani-lamides, and vitamin intake are recommended. An X-ray examination must be taken during the growth of the child. Sanation of the oral cavity carried out regularly and systematically is the most effective means of preventing acute and chronic lesions of the jaws.

6.5. General characteristics of odontogenic osteomyelitis of the jaws

Osteomyelitis is a necrotic-purulent inflammation of the bone that begins as an infection of the medullary cavity with rapid involvement of the haversian system and extension to the periosteum (affecting the whole bone – cortical and medullary). It is characterized by marked symptoms of suppurative-necrotic intoxication, radiological, clinical, and laboratory changes. Osteomyelitis was a common disease before the advent of antibiotics. Today, osteomyelitis of the facial skeleton is a rare condition, and it tends to occur more commonly in the mandible than in the maxilla.

Different terminologies and classification systems are used based on a variety of features, such as the clinical course, pathological–anatomical or radiological features, etiology, and pathogenesis. According to the mechanisms of penetration of the causative agent, osteomyelitis is classified as odontogenic, hematogenous, or traumatic, depending on the clinical evolution of acute and chronic odontogenic osteomyelitis. The chronic ones are specific or non-specific**.** There are three types of non-specific chronic odontogenic osteomyelitis: destructive, destructive-proliferative, and proliferative. The specific ones are as follows: tuberculous, syphilitic, and actinomycotic. Specific chronic ones are rarely encountered in children. Actinomycotic osteomyelitis is a type of odontogenic osteomyelitis, with dental and periodontal diseases being the pathways of germ entry.

In children, the frequency of odontogenic osteomyelitis is 80% of the total number of cases of jaw osteomyelitis, hematogenous osteomyelitis – 9%, and traumatic osteomyelitis – 11%. Hematogenous osteomyelitis is more common in children under the age of three, and odontogenic osteomyelitis is more common in children aged three to twelve.

6.6. Acute odontogenic osteomyelitis. Etiology and pathomorphology

The development of an infection in the bone is related to the virulence of the organism, the integrity and effectiveness of the host defenses, and anatomical structural factors. Osteomyelitis of the jaws represents 50% of all bone infections. According to Iu. I. Bernadsky, osteomyelitis of the jaw in children accounts for 34.1% of cases.

In 80-87% of cases, the main causes of odontogenic infections in children are milk molars and the first permanent molars. Osteomyelitis is rarely due to suppurative root cysts.

Most cases of osteomyelitis of the jaw are caused by a mixed and diverse association of microbial flora that begins as a result of a periapical infection. The onset is apparently associated with a lack of resistance to the particular microorganism that invades the bone. Staphylococcus aureus has the main role, but most infections are caused by aerobic and anaerobic streptococci. In severe forms, anaerobic streptococci and staphylococci are detected. The incidence of bacterial infections has significantly decreased since the widespread use of antibiotics. These conditions lead to an increase in bacterial infections caused by organisms with low or no pathogenicity. The development of an inflammatory process is caused by pathogenic strains of staphylococci. The infection increases and becomes extensive. Non-pathogenic strains of Staphylococci and Streptococci cause the development of limited bone lesions or mainly soft tissue lesions of the maxillofacial region.

The high frequency of jaw osteomyelitis in children is due to constitutional factors associated with inherent immunity, inherent disease, anatomic anomalies, and the specificity of the development of the nervous system.

The autonomic nervous system (sympathetic/parasympathetic) plays an important role in the development of the infection. Children of different ages regulate their autonomic nervous system and display adaptive strategies. Under adverse conditions, children are vulnerable and may not be able to self-regulate and adapt to difficult circumstances. They show autonomic dysregulation (sympathetic/parasympathetic imbalance) during growth. The classic response of the autonomic nervous system in childhood is sympathetic activation (increased impulsivity, risk-taking, and aggressive behavior) that displays sympathetic dominance with a high sympathetic nervous system and low parasympathetic nervous system responsiveness.

The immune system is relatively immature at birth and has to be involved during the lifetime of exposure to foreign challenges, from childhood through young and mature adulthood. The immune system shows weak bactericidal functions, poor responses to inflammatory stimuli, reduced adhesion of endothelial cells, and diminished chemotaxis. The low serum levels of IgG and complement and the impaired neutrophil functions put the child at risk of bacterial infections. Thus, children may be prone to contracting microbial and viral infections and have an increased risk of developing conditions such as diarrhea, ear infections, chickenpox, food allergies, and croup, and on the other hand, may take more time to recover from them. Frequent infections, such as bronchitis, ear infections, and dental infections, may cause problems in a child's immune system too. The immune system gradually matures during infancy so that the protection provided by the immune response increases and young adults contract fewer infections. Children do not have fully developed immune systems until they are about 7-8 years old.

During childhood, the suppurative processes of the jaw have an increased risk of invasiveness of the microbes with their products (toxins, enzymes) into the bloodstream, especially for young children favored by physical and chronic processes, viral infections, and frequent contagious infections (measles, scarlet fever, etc.). Severe generalized forms of acute odontogenic osteomyelitis in children occur during periods of viral foci.

The high incidence of odontogenic infections is favored by the anatomical dentoalveolar and odontomaxillary specificities during development: constant growth and development of the jaws, active changes during tooth eruption and exfoliation, wide Haversian canals, tender bone trabeculae, myeloid bone marrow instability to infection, vascularization, etc.

There are three hypotheses in the etiology of odontogenic infectious osteomyelitis, namely: Bobrov and Lexer (infectious embolic hypothesis); Derizhanov S.M. (hypothesis of host sensitization); Semencenko G.I. and Soloviov M. M. (hypothesis of neuro-reflective disorders).

The development of the infectious process in the jaws is conditioned by the factors that increase the microbial virulence and decrease the resistance of the organism. The suppurative processes of the jaws have an increased risk of dissemination of germs in the circulation in young children, which is favored by physical overload, chronic processes, viral infections, and frequent contagious infections (measles, scarlet fever, etc.). Severe generalized forms of acute odontogenic osteomyelitis in children occur during periods of winter viral outbreaks. Age-related particularities of the nervous and lymphatic systems, on the one hand, and the dental system, the pathomorphology, and the function of the jaws, on the other hand, favor the spread of the infectious process from one biological level to another and even its generalization.

Acute osteomyelitis begins with a process of suppuration that causes bone necrosis. Microscopically, the infectious process passes through the stages of congestion, suppuration, necrosis, and repair.

The process leading to osteomyelitis is initiated by acute inflammation: hyperemia, increased capillary permeability, and infiltration of granulocytes. The first stage is a reversible inflammation process, characterized by increased congestion with hyperemia and vasodilation, inflammatory, medullary, and periosteal edema. Vascular hyperemia extends to the oral mucosa and the soft perimandibular parts.

When pus, which is composed of necrotic tissue and dead bacteria within white blood cells, accumulates, the intramedullary pressure increases, resulting in thrombosis of the vessels in the marrow with consequent vascular collapse, venous stasis, and ischemia. Pus travels through the haversian and nutrient canals and accumulates beneath the periosteum, elevating it from the cortex and further reducing the vascular supply.

The intensity of the spread of the infectious process in the bone is directly proportional to the physiological changes of the bone during the periods of growth, root formation, eruption, and dental exchange. The insufficient mineralization of the jaws, the predominance of organic substances, the structure of gingival spongy tissue, the intimate relationship of dental root apices with the medullary cavity, and the comparatively thin cortical plate of the jaws facilitate the diffuse spread of the infectious process. For this reason, in children, acute osteomyelitis with localization of the infectious process in the alveolar process is not common. Beginning in the periapical regions of the milk or permanent molars, the infectious process extends for several days before acute clinical signs appear: at the maxilla, towards the anterior wall of the maxilla, towards the dental buds, and at the maxilla-malar suture; and at the mandible, towards the mandibular angle and branch.

Clinical picture of acute odontogenic osteomyelitis. The onset is usually sudden. The clinical picture of acute odontogenic osteomyelitis depends on the age of the child, the onset, the reactivity of the body, and the virulence of microorganisms. The younger the child, the more severe the disease is. The period from the first clinical signs of acute pulpitis to the onset of acute osteomyelitis is 1-2 days.

In young children, general symptoms are severe and frequently show up before local clinical symptoms. Children complain of deep pain, malaise, anxiety, insomnia, anorexia, fever, chills, and weakness. In young children and adolescents, vomiting, convulsions, dehydration, an accelerated pulse, gastrointestinal disorders, and signs of excitation of the central nervous system are often the main clinical signs of intoxication.

Swelling is diffuse and wide-spread and involves extraoral and intraoral anatomical structures and layers. Odontogenic infections are usually secondary to caries, pulpitis, periodontal disease, and postoperative infections. There is marked facial asymmetry, and the localization depends on the cause of the teeth (*figure 8*). The location of the infectious process in the buccofacial area determines the risk of compromising the respiratory tract and affecting the vital structures and organs. Facial asymmetry reveals firm cellulitis of the soft tissue. Purulent processes arising from the maxillary teeth reveal diffuse swelling extending to the paranasal sinuses, orbital area, infraorbital area, cheek area, temporal area, etc. Odontogenic infections arising from the tooth-bearing part of the lower jaw involve deep and superficial spaces around the mandible (submandibular, submental, sublingual, pterygomandibular, and parapharyngeal) and spread to the cervical deep and superficial spaces. In acute infections, lymph nodes are enlarged, soft, and tender.

Fig. 8. **Acute odontogenic osteomyelitis of the upper jaw. Extraoral view.**

Intraoral inspection (*figure 9*) reveals offending teeth sensitive to percussion. Pus exudes around the gingival sulcus from the medullary tissue. The jaw is enlarged due to increased periosteal activity. There is erythematous bulging involving the vestibular fold, and the alveolar process on both the vestibular and lingual surfaces. The mucosa is swollen, hyperemic, diffuse, and extends around 2-5 teeth. Poor oral hygiene with a fetid oral odor, hypersalivation, dysphagia, and trismus are often present. There is pain in the affected teeth; they are mobile, and pusdischarges from the periodontium. The septic process in the temporary teeth may extend and affect the buds of the permanent teeth.

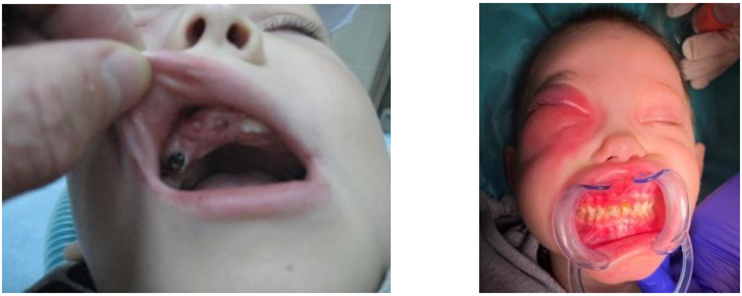

Fig. 9. **Odontogenic osteomyelitis of the upper jaw. Offending teeth sensitive to percussion. Intraoral view.**

In children, due to behavioral features, clinical examination presents some difficulties. Therefore, dental sensitivity to percussion, paresthesia of the lower lip of the affected part of the mandible (the Vincent d 'Alger sign), and hypoesthesia of the infraorbital nerve are difficult to determine. Dental mobility caused by the inflammatory process must be distinguished from that caused by root resorption.

The radiological picture in the first 7 days is not conclusive. At the end of the first week, a demineralization process is observed, visualized by anerased trabecular picture and enlarged medullary spaces. The opacity increases, the trabecular picture is erased, and the cortex is thinned, sometimes eroded.

A complete blood count examination is requested to make diagnostic, prognostic, and therapeutic recommendations. Laboratory tests of acute odontogenic osteomyelitis show a significant increase in the count of leukocytes ranging from 15,000-25,000 cells/mm² with a shift to the left. The erythrocyte sedimentation rate is elevated up to 60 mm. An elevated band neutrophil count above the normal range (70-80%) and lymphopenia (up to 10%) areprognostic signs of an odontogenic infection. Increased numbers of young leukocytes, a lack of eosinophils, and decreased numbers of monocytes are signs of a high degree of bloodstream infection. A severe bloodstream infection (bacterial infection or condition associated with severe sepsis) is characterized by anemia with low hemoglobin levels (83-67 g/l) and erythrocytopenia (3.10-12 g/l).

The diagnosis of acute odontogenic osteomyelitis is made by using plain films, supplemented as needed by computed tomography and magnetic resonance imaging. In the early stage of the disease, the full extent of bone dissolution cannot be determined radiographically for up to 3 weeks after the onset of osteomyelitis. Therefore, in the early stages of the disease, clinical findings constitute the basis upon which the diagnosis is made. The clinical picture (extraoral and intraoral) of acute odontogenic osteomyelitis is based on: 1) the presence of the offending tooth; 2) mobility of the affected tooth and neighboring teeth, even when healthy; 3) extended subperiosteal abscesses, located bilaterally (lingually and vestibularly) on the alveolar process; 4) purulent eliminations fromthe periodontal ligaments of the involved tooth.

Acute odontogenic osteomyelitis mustbe distinguishedfromacute odontogenic periostitis, inflammation processes of the parotid gland, and sarcomas.

The prognosis of acute odontogenic osteomyelitis depends on the location of the process (maxilla or mandible) and the child's age. The smaller the child, the higher the severity of acute osteomyelitis in the upper jaws and the lower the severity in the lower jaws. Meningeal signs (adynamia, drowsiness, inappetence, asthenia, pathological reflexes) can appear even in the early stages of acute odontogenic osteomyelitis. The older the child, the more severe the evolution of acute odontogenic osteomyelitis of the mandible is. In children, acute odontogenic osteomyelitis of the mandible involves the deep and superficial spaces.

The treatment of acute odontogenic osteomyelitis involves medical, surgical, and dental therapy. Once the affected teeth have been identified, endodontic therapy (removal of the infected pulp) must be performed. The method of treatment used for the tooth is a question of judgment. Mainly, it is determined by such factors as the extent of the infection, the patient's health status, the degree of trismus, and the biomechanical necessity of retaining the tooth. Extraction of the involved deciduous teeth is the most rapid method of establishing drainage. Root canal therapy to eliminate the source of infection is indicated for permanent teeth.

Abscesses should be surgically drained properly, especially when deep abscess draining is performed at the same time as dental therapy. An underlying altered host defense is common in many patients with odontogenic osteomyelitis. Careful examination reveals dehydration, convulsions, diabetes, autoimmune disease, severe anemia, leukemia, cancer, and drug abuse. This involves treating anemia with iron or transfusions, managing malnutrition with dietary supplements and vitamins, and controlling diabetes.

The choice of antibiotic therapy for odontogenic infection depends on the definitive laboratory results of culture and sensitivity testing. Sometimes, a pragmatically rational approach to empirical antibiotic selection is acceptable, both ethically and legally, if the choice is based on scientific data and contemporary experience with the microbiology of the oral infection flora.

6.7. Secondary chronic odontogenic osteomyelitis of the jaws in children

Etiology, pathogenesis, clinical, radiological, and morphological pictures. Secondary chronic osteomyelitis is an uncommon clinical finding in children, but the description of its characteristics is important for accurate and adequate clinical management. Acute and secondary chronic osteomyelitis are considered the same disease at different stages. The chronification of the disease reflects the inability of the host to eradicate the pathogen due to a lack of treatment or inadequate treatment, resulting in failure to reestablish the carefully balanced equilibrium between host factors and pathogens found in a healthy oral environment. Systemic diseases and pathological conditions that cause ischemic bone necrosis, as well as changes in host defenses, may have a significant impact on the onset of secondary chronic osteomyelitis.

Odontogenic osteomyelitis develops in three stages: the acute stage (10-14 days), the subacute stage (3-6 weeks), and the chronic stage (2-3 months). The pathological-anatomical onset of osteomyelitis corresponds to deep bacterial invasion into the medullary and cortical bones and takes 3-4 months after the onset of the acute stage. After 4 weeks, a persistent bone infection should be considered secondary chronic osteomyelitis. In children, acute forms turn into chronic forms faster than in adults, and chronic osteomyelitis occurs less rarely, being characteristic of chronic primary forms. In secondary chronic osteomyelitis of the jaw, a new equilibrium is established between the host and the aggressor causing the infection. A newly formed balance is dependent on the host's immunity, supported by medical therapy and causative bacteria.

According to the new balance, clinically and radiologically, there are three types of chronic osteomyelitis of the jaws: 1) destructive; 2) destructive-productive; and 3) proliferative.

The destructive type of secondary chronic osteomyelitis is the consequence of acute odontogenic osteomyelitis. Secondary chronic destructive osteomyelitis usually occurs in children in cases of an underlying general condition (chronic diseases of internal organs, cancer, systemic bone diseases, contagious diseases that lead to weak immunity, rheumatic disease, diarrhea) or inadequate therapy of acute odontogenic osteomyelitis, inadequate dental treatment, delayed tooth removal, delayed determination of microbial sensitivity to antibiotics, and neglect of dental treatment.

Chronic secondary osteomyelitis occurs in children aged 3-12 years and is associated with intensive physiological change. In children, the disease turns from an acute to a chronic stage within 7-10 days from the onset of the disease. The definitive diagnosis is made within 1-1.5 months based on the clinical and radiographic examinations.

Clinical findings are usually limited. The general condition improves; the temperature returns to normal; there is subfebrility, leukocytosis, and eosinophilia; and a decrease in monocytes. The clinical findings are usually limited to fistulae appearing on the mucosa of the alveolar process or on the skin or ulceration in the oral mucosa. The clinical findings reveal an induration of soft tissues and a thickened or wooden-like bone with slight pain and tenderness on palpation. Fistulae can be explored by catheterization with a bland instrument. Sequestrum is mobile and comes in a variety of sizes. Often, dental buds become sequestra, and growth areas are affected. The regional lymph nodes remain enlarged and painful on palpation.

Diagnosis and treatment management can be challenging in children due to the anatomical and physiological peculiarities of the jaws, organs, systems, and behaviorduring the period of growth.

Radiographic studies are not useful in making a diagnosis as early as 10 to 20 days after the onset of the bone infection. Radiographic pictures can be reliable when there is a loss of 20% to 50% of the bone mineral content. However, it is useful in distinguishing osteomyelitis from other diseases such as malignancies (sarcomas). It can reveal soft tissue swelling, periosteal reaction, loss of bone density, and low bone regeneration. Late signs include bone resorption, sequestra, and new bone development in the periosteum. In children, X-ray signs are determined 2-3 months after the acute onset. The sequestrum is an irregular osteosclerosis area of the bone that varies in size and is separated from the healthy part of the bone by a radiolucent area. Permanent tooth buds are frequently sequestra.

A CT scan provides the most detailed imaging of the cortical bone, identifying sequestra and intra-osseous fistulae. It also shows both the periosteal reaction as well as bone marrow and soft tissue involvement at an early stage.

MRI has an advantage in assessing the bone marrow and the surrounding soft tissues, showing associated edema and hyperemia in the very early stages of the disease.

Positron emission tomography (PET) has the highest sensitivity and specificity for delineating lesions with their concomitant inflammatory activity at very early stages. A meta-analysis investigating the accuracy of diagnostic imaging for chronic osteomyelitis showed that fluorodeoxyglucose (FDG) PET has the highest diagnostic accuracy both for confirming and excluding the diagnosis of chronic osteomyelitis.

Individual treatment according to the patient's severity and duration of symptoms, as well as the clinical and radiological response to treatment, is required. In all cases, a combined antimicrobial and surgical treatment, as well as appropriate dead space management and skeletal reconstruction, should be considered.

Treatment should include adequate surgical debridement to remove all pathogens from bone and sequestra, reaching down to healthy and viable tissue. The indications for sequestrectomy in children are as follows: large sequestra, which do not have a resorption tendency, and permanently necrotizeddental buds. In order not to injure the growth areas, the intervention must be performed intraorally with the management of the periosteum, which plays an important role in bone regeneration. A multidisciplinary approach involving radiologists, microbiologists with expertise in infectious diseases, orthodontists, and plastic-reconstructive surgeons is advocated. The follow-up period for children should be as long as five years.

Chronic inflammation and infection processes can result in a variety of complications. Abscesses, sinus tracts, and the involvement of adjacent structures are some of the most common complications. In severe cases, total sequestration of the temporomandibular joint, mandibular ramus and/or body, dental buds, disorders of the mandible development, dento-alveolar deformities, and malocclusion occur during the development period.

The main principles of prophylaxis of secondary chronic destructtive osteomyelitis are appropriate individual management, including surgical and antibiotic treatment, early diagnosis, and medical comorbidities.

The productive-proliferative type of secondary chronic osteomyelitis is the most widespread, being characterized by the establishment of an early new equilibrium between the host and the aggressor causing infection. Most symptoms, such as pain and swelling, are usually less extensive in the chronic stage than in the acute stage. The deep and intense pain that is common in the acute stage is replaced by dull pain. Painful swelling caused by a local edema and abscess in the acute stage is followed by a harder, palpable tenderness caused by a periosteal reaction.

Alterations in bone resorption and bone sclerosis are usually revealed. Sclerosis manifests as radiopaque areas that correspond to multiple extended small sequestra. Osteolysis appears on X-ray pictures as a small radiolucency area among dead bone pieces. Dead bone is exfoliated through drainage incisions, or it can reabsorb on its own.

Proliferative chronic osteomyelitis (Garre's sclerosing osteomyelitis, periostitis ossificans, chronic nonsuppurative sclerosing osteomyelitis, proliferative periostitis of Garre, chronic osteomyelitis with proliferative periostitis) is a rare type that is common at the age of dental eruption, during the period of maximal growth of the jaws (12-15 years). The lower jaw is affected more often. The odontogenic origin of chronic proliferative osteomyelitis has been revealed in 60-70% of cases, but its etiology has not been determined so far.

Garre's osteomyelitis is thought to occur because of a low-grade infection or irritation that influences the potentially active periosteum of young individuals to lay downnew bone. Staphylococcus aureus and Staphylococcus epidermidis have been found in bone specimens that were obtained by biopsy of lesions. The atypical microbial flora with low virulence is due to inadequate use of antibiotics (small doses and short treatment sessions), inadequate endodontic treatment of primary teeth, difficult eruption of primary and permanent teeth, oral trauma, and an immunocompromised host.

Clinical picture. Clinically, it is characterized by a localized, hard, non-tender swelling of the lateral and inferior sides of the mandible. Occasionally, it starts with unexplained pain. Lymphadenopathy, hyperpyrexia, and leukocytosis are usually not found. It is commonly associated with a carious molar, usually the first molar, and a history of past toothache. On occasion, no dental etiology or radiolucency may be found.

The disease is accidentally observed by parents and children during the prophylactic examination or during the outpatient period. The blood picture in chronic osteomyelitis is characterized by normal values of leukocytes and ESR. During an exacerbation, local clinical signs of inflammation may occur.

Radiological picture. Radiographically, a focal area of well calcified bone proliferation may be seen. It is smooth and often has a laminated or onion skin appearance.

The disease develops with a diffuse thickening of the bone in the periosteal stratification, followed by structural changes and **endoosal** osteogenesis. At the onset of the disease, parallel to the basilar margin, a white band appears (the ossifying periosteal layer), which gradually grows wider and wider. Over 2-3 months, signs of young bone tissue without any structure appear between the ossifying periosteal layer and the basilar margin. At the same time, changes in the ossifying periosteal tissue, characterized by the direction change of bone structures, begin. At first, the horizontal orientation manifests, then the orientation of the newly formed periosteal bone tissue in a vertical plane, relative to the basilar margin. The thickness of the periosteum deposits may vary depending on the length of the process. Sometimes the cortical line may be interrupted. After 6-8 weeks, the periosteal layers, which remain porous, converge with the cortical layer, which gradually loses its characteristics of compact bone and becomes porous. The bone structure becomes uniformly trabecular without differentiating the cortical layer. The surface of the bone remains smooth, and the bone has equal thickness and a strict direction. The homogeneous structure of the bone gives the impression that the cortical layer is thin. Bone destruction does not manifest, and inflammation reactions are accompanied by the productive processes of the bone structures and the excess formation of fibrous tissue. The new bone trabeculae are at different stages of development. The radiological image is characterized by replacing the normal architecture with an unusual trabecular picture. At this stage, osteomyelitis resembles fibrous dysplasia. After several months, the bone structure gradually changes, restoring and differentiating into the cortical and spongy tissues. In the case of exacerbation, the radiological image shows small opaque spots and a new periosteal ossifying layer.

Differential diagnosis. Ewing's sarcoma, osteosarcoma, cortical hyperostosis, and osteitis are similar in radiographic appearance and must be differentiated from Rarre's osteomyelitis. When no dental disease exists or when the lesion persists after treatment of dental disease, a biopsy should be considered to establish the diagnosis. The lesion is histologically composed of new bone with fibrous marrow containing chronic inflammatory cells.

The treatment is focused on removing identifiable sources of inflammation. When the involved teeth cannot be restored, extraction is indicated. Endodontic therapy has been successfully employedwith mass resolution. Antibiotics should not be administered unless signs of infection are present. Post-treatment follow-up is essential. Early biopsy is indicated if the lesion continues to increase in size after apparently successful treatment of the infection. The mandible is usually remodeled naturally after successful treatment of the dental disease, but the deformity may persist, necessitating surgical recontouring.

6.8. Hematogenous osteomyelitis

Hematogenous osteomyelitisis a purulent process that settlesin the bone after the infection has migrated hematogenously from distant regions of the body. In 50% of cases, newborns and preschool children are affected. In 77.4% of cases, it is found in children from one month to one year old. From 1 to 3 years, the frequency accounts for 15.24%, and from 3 to 12 years - 7.31%. The upper jaw is affected more often than the mandible. The ratio of girls to boys is equal.

Etiology and pathogenesis. In children, hematogenous osteomyelitis usually develops as a result of a septic state (septicemic form). Primary purulent foci, such as umbilical infections, skin pyoderma, contagious diseases, respiratory infections, otitis, ethmoiditis, mucosal trauma, pharyngeal mucosal lesions, and inflammation in the mother's body, cause hematogenous osteomyelitis in the facial bone region (mastitis).

An unfavorable premorbid picture can be found in children with acute hematogenous osteomyelitis. Predisposing factors may be premature birth, pregnancy pathologies, repeated acute respiratory infections, or contagious infections. In 65-91% of cases of purulent foci, the golden staphylococcus and the white staphylococcus are detected.

The elimination of the hematogenous infection, with its develop-pment away from the primary focus, is explained by several factors: the immature immune system and the uncompleted functions of the endocrine and histiocytic systems in the newborn and early periods. Therefore, the newborn is peculiarly vulnerable to invasive infections with Gram-negative bacilli during the first two weeks of life because of a transient deficiency of globulins. Infants are unusually susceptible to invasive infections with most pyogenic bacteria from about 1-2 months to about 18-24 months of age because there are primary infections occurring in infants without any previously established delayed hypersensitivity and in whom antibody formation is slow. The body's reactivity gradually changes with the maturation of the immune system. The onset of hematogenous osteomyelitis in later periods of child development is explained by the body's predisposition to certain infections and its sensitization.

Hematogenous osteomyelitis of the maxillofacial region. Acute hematogenous osteomyelitis is an infection that affects the fast growing skeleton, involving primarily the most vascularized regions of the bone. Acute osteomyelitis has an incidence of 8-10 per 100,000 in developed countries and up to 80 per 100,000 in developing countries. Osteomyelitis involves the lower extremities more than the upper ones. The head area is involved in 3% of all cases of hematogenous osteomyelitis.

Staphylococcus aureus is the most common organism that affects the bones, followed by the respiratory pathogens Kingella kinger, Streptococcus pyogenes, and Streptococcus pneumonia. Bacteriology has changed as a result of increased virulence of Staphylococcus aureus, primarily methicillin-resistant strains with increased tissue necrosis and neutrophil destruction, which is associated with a higher rate of septic shock, a greater need for surgical interventions, and prolonged hospitalization.

The disease caused by K. kingae affects mostly younger children and involves primarily the musculoskeletal system, giving rise toseptic arthritis, spondylodiscitis, and osteomyelitis. In contrast, Gram-negative organisms such as *Escherichia coli* and group B streptococci are more common in neonates and young infants and, in a series, accounted for 60% of the musculoskeletal infections occurring before 4 months of age.

Osteomyelitis occurs when an infection develops within a bone or reaches the bone from another part of the body. Trauma and infections are the causes of the disease. The perinatal trauma of the oral mucosa from an obstetrician's finger or mucus suction bulb used to clear the airway immediately after birth is the source of infant infection. Contaminated human or maternal nipple infections, umbilical infections, and skin infections can all play an important role in the etiology of hematogenous osteomyelitis.

The pathophysiology of hematogenous osteomyelitis is based on the current paradigm of the metaphysis of long bones, described by

Trueta. Metaphyseal spongiosa contains abundant blood vessels with leaky endothelium and sluggish flow that end in capillary loops. Recent research has found that these vessels are terminal and that bacteria lodge at the junction between the physis and metaphysis. In the first 18 months of life, there is communication between the epiphyseal and metaphyseal vessels. This communication results in the direct extension of metaphyseal infections into the epiphysis. Epiphyseal extension can cause the destruction of the epiphyseal cartilage and secondary ossification center and can affect the cells of the germinal zone of the physis, which ultimately results in a permanent growth disturbance. This age group also has a higher incidence of septic arthritis due to a slight extension into the epiphysis.

The junctions of bone and cartilage in skeletally immature flat bones, round bones, and epiphyseal ossification centers have a structure similar to that of the metaphysisof long bones (metaphyseal equivalents). They are highly vascularized, have sluggish flow, and contain more hematopoietic marrow than the remainder of the adjacent bone. The periosteum is highly vascularized in children, but it is unclear whether it can be the site of infections.

Osteomyelitis affects the fast-growing area more commonly and involves the frontal and zygomatic processes of the upper jaw and the articular processes of the lower jaw. Usually, only one area is affected, but there are multifocal cases when one or more areas, either unilaterally or bilaterally, including those located in other parts of the body, are involved. The following bones have the highest rates of osteomyelitis in all pediatric groups: clavicle 1-3%; thorax-3%; humerus 5-13%; ulna 1-2%; radius 1-4%; spine 1-4%; pelvis 3-14%; hand 1-2%; femur 23-29%; tibia 19-26%; calcaneus 4-11%.

In children, the clinical picture of hematogenous osteomyelitis of the jaw occurs in the first month of life, often up to one year, and rarely at 3-7 years. The onset is sudden, as is the rapid spread of the process. The inflammation of the soft parts is located in the infraorbital region and the lateral part of the nose, or in the external/internal infraorbital and orbital regions, often with purulent conjunctivitis. Clinically, the patient presents with facial cellulitis centered around the orbit. Irritability and malaise precede cellulitis and are followed by hyperpyrexia, anorexia, and dehydration. Convulsions and vomiting may occur. Leukocytosis is present with a left shift.

Inner canthal swelling, palpebral edema, closure of the eye, conjunctivitis, and proptosis may result. A purulent discharge may be associated with the nose or with an inner canthal sinus. Intra-orally, the maxilla on the affected side is swollen both buccally and palatally, especially in the molar region. Fluctuance is often present.

Mandibular hematogenous osteomyelitis occurs with a frequency of 25%, isolated or in combination with other facial or skeletal bone damage. It occurs more frequently between the ages of one month and one year. The local clinical picture is poorer than in the upper jaw, and the disease develops more slowly. A slight swelling in the parotid or submandibular region is present.

Hematogenous osteomyelitis with multiple foci of the facial bones and skeleton often occurs in children in the first year of life. The foci can be present in two, three, or more regions of the skeleton, unilaterally or bilaterally (maxilla, malar bone, ethmoidal cells, nasal bones, orbits, frontal bones, tubular bones, clavicle). In 70.9% of cases, it develops severely as sepsis. In newborns, the progression of hematogenous osteomyelitis with extensive bone damage is extremely severe. Sometimes, although intensive care is promptly initiated, purulent foci attack new regions of the skeletal bones and internal organs. Severe cases include retrobulbar abscesses and septic pneu-monia. Evacuation of the pus or formation of fistulas improves the general condition, but the danger to the child's life disappears only after 3-4 weeks of treatment.

Complicationsof hematogenous osteomyelitis of the jaws include early sequestrum and fistulas located in the oral cavity (on the palate and oral vestibule), at the medial and lateral angles of the eye, and on the lateral side of the nose.

Sometimes the lower eyelid appears to be fixed to the bone, resulting in „pitting” or scarring that affects the appearance of the face. Bilateral destruction and resorption of the bone cortical layer cause the escape of the infectious process in the nasal and sinus cavities. Dental buds that have become sequestered maintain the chronic inflammation for a long time.

The auditory duct may be affected by resorption of the lower wall. Fistulas in the submandibular, retromandibular, or intraoral regions, both buccally and lingually, are uncommon and usually occur following surgical drainage of the purulent process. Inadequate treatment leads to the development of phlegmon in the submandibular, parotid, sub-masseteric, and pterygomandibular spaces. Sequestrum of the temporo-mandibular joint and ramus is a rare complication, occurring several months after the onset of the disease.

The diagnosis of sequestrain young children is difficult. In 35% of cases, sequestra of the primary or permanent dental buds occurs. Occasionally, the affected tooth buds erupt with shape abnormalities and enamel hypoplasia (Turner`s teeth). Disorders in the growing area and loss of the primary and permanent buds may lead to asymmetry, deformity, and disfigurement of the face.

The radiological examination in the first 7 days is poor. On day 7, diffuse foci of bone resorption appear. The sequestra are determined in the third week after the onset of the disease. Conventional radiographs should be the first step in imaging evaluation. Although radiographs are only diagnostic in less than 20% of cases of acute staphylococcal osteomyelitis in children, they can help direct the subsequent imaging evaluation and, more importantly, show whether the symptoms are caused by trauma or a tumor. MRI imaging has become the recommended modality for the evaluation of a child with suspected osteomyelitis. If the child can be evaluated with MRI imaging within hours of the suspected diagnosis of osteomyelitis, it is reasonable to proceed first with this modality. Emergency MRI imaging has become a reality as more centers have MRI schedules that approach 24-hour availability.

Differential diagnosis. Hematogenous osteomyelitis of the maxilla must be distinguished from acute ophthalmic infections and, in the chronic period, from dacryocystitis; in the jaw, from acute and chronic otitis, which becomes especially difficult when the fistulas open in the auditory duct. The diagnosis is based on the radiological examination and computed tomography.

The treatment is carried out in the intensive care department. The surgical treatment includes the removal of the purulent fluid. Sequestrectomy is performed in specialized hospitals in collaboration with the dental surgeon, pediatrician, otolaryngologist, and ophthalmologist.

Acute hematogenous osteomyelitis requires appropriate antimicrobial therapy in all cases, as well as surgical incision and drainage. Whether or not surgical intervention is required, the successful treatment of all forms of osteomyelitis requires appropriate antibiotic therapy. In children, the empiric therapy (children 3 months of age and older) should be antistaphylococcal penicillin (nafcillin and oxacillin) or first-generation cephalosporins (cefazolin). These agents are effective not only against *S. Aureus* but also against other causes of acute hematogenous osteomyelitis, including *S. Pyogenes* and *K. kingae*. Parenteral antibiotics should be given until the child is afebrile and has demonstrated improvement by both physical assessment and laboratory analysis. Surgical removal of devitalized bone and debridement of affected soft tissues are often necessary in children and adolescents with community-acquired methicillin-resistant *Staphylococcus aureus* and osteomyelitis, even with appropriate antibiotic therapy. Surgical drainage should also be considered when a child does not respond to empiric antibiotic therapy. In addition, surgical intervention allows for the collection of tissue, which can be microbiologically evaluated for an unusual etiology of osteomyelitis and histologically examined to confirm the diagnosis.

The results of hematogenous osteomyelitis in children are directly proportional to its acute form. Toxic and septicemic forms in young children have a lethal outcome. With the growth of the body, deformities of the jaws may develop with insufficient development, manifested by facial asymmetries, deformities of the eyelids, secondary anodontia, arthritis, or ankylosis. They are caused by the removal of the sequestrations from the orbital margin, the destruction of the growth areas, and the sequestra of the dental buds.

Learning Objectives

1. Classification of jaw infections and abscesses in children.
2. Pathomorphological picture of the intraosseous stage of the development of dentoalveolar abscesses.
3. Pathomorphological picture of dentoalveolar abscesses.
4. Clinical picture of odontogenicabscesses in children.
5. Diagnosis of dento-alveolar abscesses in children.
6. Pathomorphological picture of chronic periostitis.
7. Diagnosis of chronic periostitis.
8. Clinical picture of chronic periostitis in primary dentition in children. Treatment of chronic periostitis.
9. Evolution of acute and chronic periostitis in children.
10. Treatment of acute and chronic periostitis depending on children`s age.
11. Classification of hematogenous osteomyelitis.
12. Etiopathogenesis of osteomyelitis in children.
13. Anatomical and physiological peculiarities of the onset and evolution of odontogenic osteomyelitis in children.
14. Acute odontogenic osteomyelitis.
15. Destructive chronic osteomyelitis.
16. Clinical picture of chronic hyperostosis osteomyelitis in children.
17. Etiopathogenesis of hematogenous osteomyelitis.
18. Clinical picture of hematogenous osteomyelitis.
19. Hematogenous osteomyelitis of the maxilla.
20. Complications of hematogenous osteomyelitis.
21. Treatment of osteomyelitis according to its forms.

TESTS

1. SC. The vestibular abscess that has the starting point in the upper central incisors is accompanied by:
A. the swelling of the lower eyelid;
B. the swelling of the genian region;
C. a marked swelling of the entire upper lip (*tapir lip*);
D. a marked genian swelling, which greatly deforms the face;
E. the swelling of the cheek that extends to the submandibular region.

(C)

2. SC. Periostitis, which clinically represents the swelling of the cheek and extends to the submandibular region, is caused by the following teeth:
A. premolars and upper molars;
B. lower incisors and canines;
C. premolars and lower molars;
D. upper central incisors;
E. upper canines.

(C)

3. SC. The odontogenic subperiosteal abscess represents:
A. inflammation of the periosteum of the jaws, caused by dental or periodontal infections;
B. an infectious process characterized by the absence of laminate collections and the tendency of septic invasion with marked lysis and tissue necrosis;
C. an infectious-inflammatory process of the maxillary bones, triggered by the penetration of a pathogen into the bone tissue;
D. suppuration circumscribed in the form of a collection with purulent content;
E. marked vasodilatation, serous exudate, leukocyte diapedesis, and cellular infiltration in the soft parts.

(A)

4. MC. Periostitis, in the stage of serous infiltration, is clinically characterized by:
A. congested mucosa;
B. edema and discrete swelling in the vestibular groove on the right side of the affected tooth;
C. swelling in the vestibular groove;
D. thickening with no precise boundaries;
E. extremely painful palpation.

(A, B, C, D, E)

5. SC. The vestibular abscess that has the starting point in the upper central incisors is accompanied by:

A. swelling of the lower eyelid;
B. swelling of the genian region;
C. marked swelling of the entire upper lip (*tapir lip*);
D. marked genian swelling, which greatly deforms the face;
E. swelling of the cheek that extends to the submandibular region.

(C)

6. SC. Periostitis, which clinically represents the swelling of the cheek, and extends to the submandibular region, is caused by the following teeth:

A. upperpremolars and molars;
B. lower incisors and canines;
C. lowerpremolars and molars;
D. upper central incisors;
E. upper canines.

(C)

7. SC. The odontogenic subperiosteal abscess represents:

A. inflammation of the periosteum of the jaws, caused by dental or periodontal infections;
B. an infectious process characterized by the absence of laminate collections and the tendency of septic invasion with marked lysis and tissue necrosis;
C. an infectious-inflammatory process of the maxillary bones, triggered by the penetration of a pathogenic germ into the bone tissue;
D. suppuration circumscribed in the form of a purulent collection;
E. marked vasodilatation, serous exudate, leukocyte diapedesis, and cellular infiltration in the soft parts.

(A)

8. MC. Periostitis, at the stage of serous infiltration, is clinically characterized by:

A. congested mucosa;
B. edema and discrete swelling of the vestibular groove on the right side of the affected tooth;
C. swelling of the vestibular groove;
D. thickening without clear boundaries;
E. extremely painful palpation.

(A, B, C, D, E)

9. MC. Secondary clinical signs in acute purulent periostitis may be:

A. Periadenitis;
B. Adenophlegmon;
C. Painful percussion in 1-2 teeth;

D. Mobility of the affected tooth;
E. Mobility of several neighboring teeth.

(A, C)

10. MC. In children, the rapid spread of the inflammatory process is due to the following anatomical-pathomorphological features:

A. insufficient mineralization of the jaw bones;
B. the predominance of organic substances in relation to inorganic ones;
C. a thin cortical layer of the jaws;
D. tooth root development in progress.
E. Haversian canals

(A, B, C, D, E)

11. MC. The satellite symptoms of acute odontogenic osteomyelitis are:

A. Lymphadenitis;
B. Periadenitis;
C. Phlegms of superficial spaces;
D. Phlegms of deep spaces;
E. Mediastinitis.

(A, B, C, D)

12. SC. Long-term complications of chronic destructive osteomyelitis may be:

A. disorders of the mandibular shape and size;
B. suppuration of soft facial parts;
C. progression of the process, regardless of the surgical and therapeutic treatment;
D. necrosisof dental buds in the focus;
E. septicopyemia.

(A, D)

13. MC. The focus of hematogenous osteomyelitis of the maxilla is in:

A. the alveolar apophysis;
B. the median suture;
C. the frontal apophysis of the jaw;
D. the zygomatic apophysis of the jaw;
E. the pyriform apophysis.

(C, D)

14. SC. The blood formula that corresponds to acute odontogenic osteomyelitis is:

A. ESR up to 7-10 mm, leukocytes 6.7-10 9/l;
B. ESR up to 40 mm, leukocytes 20-30 9/l;
C. ESR up to 15 mm, leukocytes 11-10 9/l;
D. All answers are correct;
E. None of the answers are correct.

(B)

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7. TRAUMA OF THE SOFT TISSUE IN THE ORO-MAXILLOFACIAL REGION IN CHILDREN

7.1. Etiology and pathogenesis

Children are energetic, curious, and often awkward. They commonly sustain soft tissue injuries to the head and neck region. The face is the most important part of the body and thus plays a major role in the development of the child's self-esteem and the manner in which he interacts with others. The face is the child's window to the outside world and the subject of peer interaction.

Facial and neck traumais common in children; however, because of their small face size, skeletal flexibility, and increased facial fatty tissue, most of these traumas result in soft tissue injuries (cuts and bruises). The face and head are more often exposed to trauma than other parts of the body. According to V. N. Siraкova, the frequency of trauma in children is 10:1000 of all traumas in children. Fortunately, severe facial and neck fractures are uncommon. On the other hand, due to the morpho-functional complexity of the regions in which the ears, nose, throat, eyes, stomatognathic system, and nervous system are located in the small area, the traumatic lesions have their own characteristics regarding the clinical aspects, the evolution, the complications, and the treatment approach.

Despite the numerous types of minor and severe injuries, the majority of lacerations are caused by:

1. Blunt trauma. It refers mostly to injuries resulting from a physical attack or an impact from an object (big or small). A good example is a physical confrontation where punches have been thrown and have landed on different parts of the body, resulting in cuts and split lips. Blunt trauma can also be caused by a major blow to any part of the body, like being hit by a baseball bat. A collision has the heaviest impact and is another blunt trauma that causes lacerations.

2. Birth trauma. It mainly refers to the laceration of the perineal skin. The laceration occurs in the birth canal and can be mild or severe.

3. Fall. There are two ways that a fall can cause lacerations. First, it can occur as a result of a fall in which the person is cut on the way down, such as when he falls from a tree and hits several branches before reaching the ground. Second, a person may get a laceration when he hits the ground, like in the case of falling from a bike, and he scrapes his knees, legs, or palms.

4. Sharp-edged objects. These are the most common causes of lacerations because it is easy to get cut, especially by knives, scissors, and broken glasses.

The distribution of etiological factors in craniofacial traumas is directly proportional to the country's socioeconomic development, geographical location, and even religious beliefs. Due to this, the information about the etiological factors and incidence differs from one source to another.

The home is a potentially dangerous environment for children, especially during the preschool years, because of the hard, acutely angled surfaces of housing structures and furniture around children's activity zones.

There are multiple factors associated with trauma, such as: indoors-78.9%, outdoors - 17.8%, motor vehicle accidents - 3.3%. Isolated soft tissue lesionsare detected in approximately 13.9% of cases, fractures of the alveolar apophysis and dental trauma are revealed in 3.3%, isolated mandibular fractures are found in 50.7%, fractures of the upper jaw are detected in 3.3%, fractures of the nasal bones - 6.5%, extensive lesions combined with bone fractures - 20.4% of cases. The need for hospitalization of children with oral-maxillofacial lesions accounts for 18%.

Foreheads (26.4%) take the brunt of the most frequent injuries, followed by the eyelids (20.6%), eyebrows, including the glabella (19.7%), and chin injuries (15.7%).

Overall, 48.9% of injuries occur outdoors, and 45.1% occur at home. By age, the frequency of trauma is as follows: up to 1 year - 1.9%; 1-2 years - 23.6%; 3-7 years - 4.5%. The soft tissue injuries account for 98%, the fractures of the facial skeleton - 1.5%, and the dental-alveolar injuries - 0.5%. Only 6.0% of injuries occur in schools or kindergartens. Injuries that occur in schools or kindergarten increase with the age groups (from 2.3% for 0- to 3-year-olds to 19.1% for 13- to 15-year-olds).

The etiology, circumstances, and frequency of trauma have been studied in the Republic of Moldova. The highest frequency of trauma isat the ages of 3-7 years (40%), 7-12 years (25%), 1-3 years (21%), and 12-16 years (11%).

In the preschool period, traumas occurring at home and outdoors are caused by being struck. Slips, trips, falls, and bumps (32.5%) are common in childhood, followed by slipping down (31.5%) during games.

In children aged 0-3 years, thetraumas caused by accidents in the home predominate; accidents involving sharp edges of furniture and stairs account for 9%. Dog bites occur in 11% of cases, with severe consequences.

The circumstances in which traumas occur at 3-7 years of age are different from those below 3 years of age. Outdoor accidents increase with age when compared to those at home: 8% from 3 to 7 years of age and 25% from 7 to 11 years of age. Injuries caused by motor vehicle accidents have a frequency of 3%, injuries occurring in schools - 4.5%; traumas in kindergartens - 9.4%, and animal kicks (dogs, horses, cattle) - 10 %.

In children, facial lacerations occur more frequently in the 3-7-year-old age groups. It is worth noting that the peaks of maxillofacial bone trauma occur between the ages of 7 and 12 years and are caused by car accidents, animal kicks, and fights. Soft tissue injuries are found in 85% of cases, injuries to the lower jaw in 4.4%, the teeth in 6%, and the facial bones in 2%.

7.2. Classification and peculiarities of soft tissue injuries in children

Soft trauma predominates in children up to 7 years old. The lesions, without continuous solutions, are accompanied by markededema, which can mask the trauma of the maxillofacial bones. They are caused by the peculiarities of the maxillofacial area: the elasticity of the skin, the well-developed fat layer, the rich network of vessels and lymph nodes, and the accelerated and unstable metabolic changes. Soft-tissue injuries in children are frequently associated with dento-alveolar and facial bone traumas. Post-traumatic edema often develops into infected infiltrates, which can persist for a long time.

Injuries caused by animal bites are common. Usually, they are located in the extreme regions of the face - the nose, ears, and lips. Anti-rabies medication should be used to treat animal bites.

In children, characteristic intraoral wounds are related to the mucosa and different planes of the walls of the mouth and the tongue. Sharp objects (pencils, sticks, toys) held in the mouth during falls and accidentally penetrating through the mucosa cause stinging wounds. Located in the palatine vault, they can establish oral and nasal communication. Tongue and mouth injuries cause heavy bleeding. Often, foreign bodies are found when inspecting injuries. Oral lesions in young children may be complicated by extensive edema, which may cause asphyxia.

Due to the abundance of fat tissue, the presence of the well-developed Bisa bubble, the high elasticity of the skin, and the small body weight (up to 3-5 years), although the children fall frequently, the lesions are without continuity, but accompanied by marked edema.

Classification. An injury is a general classification of any type of damage to the body. The most common types of injuries are wounds, bruises, burns, dislocations, fractures, sprains, and strains.

Traumatic lesions of the oro-maxillofacial area in children are classified as:

1. lacerations, contusions, and excoriations of the soft tissues (lesions of the muscle and tongue, mucosa of the oral cavity, salivary glands, etc.);
2. traumatic dental injuries (crown and root trauma);
3. jaw injuries;
4. trauma to the facial bones (fractures of the nasal bone and zygomatic bone);
5. combination of soft and hard (dental) tissues;

6. traumatic lesions of the maxillofacial area associated with closed brain trauma;

7. traumatic oro-maxillofacial lesions associated with traumatic lesions in other areas (extremities, chest, abdominal cavity, spine, etc.)

Although the diversity of traumatic lesions of the soft layer does not allow a relevant classification, according to the clinical signs, there are the following types: 1) closed wounds, without skin continuity solutions: abrasions, contusions, hematomas; 2) open wounds, with skin continuity-damage:excoriations, lacerations of different types (soft tissue loss, dog bites (*figure 10*)) that can extend only to the skin or only the oral mucosa or can encompass all soft tissue layers, penetrating or not the walls of the oral cavity or nasal cavity, and associated or not with maxillofacial bone fractures with traumatic dental-periodontal lesions. They can result in a secondary facial deformity with severe functional disorders.

Fig. 10. **Dog bite laceration withsoft tissue deficiency**

Soft tissue lesions can be classified based on the following etiological factors:

1. A cut wound caused by a clean, sharp-edged object like a knife or glass splinter.

2. A laceration appears more jagged and is typically caused by blunt trauma.

3. A gash is a term used for a wound that appears longer or deeper than a cut or a laceration.

4. An avulsion is a wound where tissue is not just separated but torn away from the body. There are also other types of open-wound injuries that may be classified based on their causes.

5. An „abrasion” or „graze” refers to a wound where the top part of the skin is scraped off, like through friction or scrubbing.

6. Puncture wounds are named as such because they cause perforation (or holes) in the skin, like a nail or a needle.

7. Penetration wounds are like puncture wounds, where they cause a hole, but the wound goes deeper and may be jagged or irregularly shaped, like in stabbing injuries.

8. Gunshot wounds are caused by bullets that can enter and exit the body, leaving holes on both ends. Sometimes, there is only an entry wound from the bullet getting lodged inside the body.

The clinical manifestations of the wound are as follows: 1. skin pain, bruising, and swelling; 2. bleeding (minimal when it is just a surface wound, minor and profuse bleeding when it involves the face, mouth, or head, as these areas contain several blood vessels; 3. numbness and tingling (an abnormal feeling or loss of sensation); 4. loss of function (hypersalivation, masticatory disorders, phonation, swallowing, and respiratory disorders); 5. general disorders: fever, agitation, restlessness, hypovolemicshock, mental disorders.

Soft-tissue hematomas are closed lesions manifested by the rupture of dermal or subdermal, small and large blood vessels as a result of blunt injury with no continuityof the skin or mucosa. Often, a hematoma is indicative of a more significant underlying bone injury.

Clinically, it is manifested by discoloration of the skin from a purple, blue, or dark spot to a dark blue, or black area, swelling, tenderness, and pain. Hematomas may be the cause of difficulty moving the lower jaw and difficulty swallowing. Small hematomas usually heal on their own or are treated with warm compression and conservativemanagement. However, large hematomas may require aspiration with a fine needle for 7 to 10 days if liquefaction occurs.

Abrasion is a partial thickness wound of the skin caused by rubbing a hard plane against a rough surface, resulting in bleeding, edema, and bruises. A superficial abrasion involves only the epidermis; it does not bleed or scar because the dermis is left intact. Sometimes, after cleaning, the affected area is left uncovered to allow the crust to form, which will protect the wound until epithelialization. Deep abrasionsinvolve the deep dermis, disrupt the normal dermal structure, and may lead to the formation of scar tissue. Avulsion is a more traumatic abrasion that removes all layers. Treatment requires the removal of foreign bodies from the surface of the lesions, which are cleaned with tap water and mild soap and adequately covered with a non-adhering dressing (petrolatum-based antibiotic ointment). Deep abrasion after wound debridement and irrigation with sterile saline solution leads to primary closure, which should be performed when possible.

Laceration is the tearing of the skin that results in an irregular wound and may be caused by injury with a sharp object or by impact injury from a blunt object or force. Lacerations can be classified ascut lacerations, degloving lacerations, perforated lacerations, split lacerations, stretch lacerations, and torn lacerations. A facial laceration may involve multiple nerves, parotid ducts, lacrimal ducts, etc. A severe laceration may extend through the full thickness of the skin and into subcutaneous tissues, including underlying muscle, internal organs, and bone. A severe laceration is often accompanied by significant bleeding and pain.

They are characterized by the interruption of the skin or mucosa, irregular and loose margins, pain, bleeding, abundant salivation, mastication and swallowing disorders, as well as respiratory and phonation disorders.

Animal bites are a significant cause of facial trauma in the pediatric population. The face is a common site of injury because animals are often the same height as children and they reach directly for the face with their bites. Medical management should include prophylaxis for tetanus and rabies as well as prophylactic antibiotics. Wild animals must be considered rabid, and if possible, the brain of the offending animal should be examined for the presence of antibodies to rabies. If possible, the patient should be treated with human rabies immune globulin (HRIG) and human diploid cell culture rabies vaccine human cell vaccine (HDCV). If the bite is caused by a non-immunized or questionably immunized domestic animal, the animal may need to be quarantined for 10 days. There is no need for treatment if no illness orold behavior is observed during this period.

Thermal and electrical burns are common in the pediatric population. Electrical burns (*figure 11*) are usually the result of placing an electrical wire in the mouth, producing the classic injury to the lateral commissure.

Fig. 11. **Electrical trauma to the lower lip.**

Facial trauma in newborns is caused by pressure during delivery. The nose at birth may be stuck to one side (subluxed) as a complication of being in the womb or through pressure during delivery. This problem may be corrected on its own; however, if there are breathing problems or the nose is markedly deformed, it can be moved back into its normal position by an experienced physician.

Injuries involving the palate (roof of the mouth) are common in children. These usually occur when a child is running or playing with something in the mouth, like a pencil, toothbrush, or stick. Other mouth injuries include cuts or tears to the tongue, tonsils, and/or inside the cheek.

Cuts on the tongue, if small, usually heal on their own. However, if there is extensive swelling or a risk of airway obstruction, hospital observation and a possible tracheotomy may be indicated.

7.3. Complications of soft tissue injuries in children

1. Immediate, severe, life-threatening complications. They occur within minutes or hours of the trauma and include: shock (which is less common in some severe traumas with large bleeding); bleeding (which may be abundant and severe due to injury to large vessels); asphyxia (caused by clogging the airways, falling back of the tongue, blood clots, foreign bodies, aspiration of gastric contents, hematoma, para-pharyngeal edema); and concussion.

2. Secondary complications develop a few days after the trauma. Due to oral septicity and often inadequate oral hygiene or contamination by foreign bodies, most significant situations consist of severe wound infection, bacteremia, and expanding hematoma formation. 3. Early complications include suppuration in the superficial or deep layer of the soft tissue, chronic fistulas, thrombophlebitis, septicemia, pulmonary, gastrointestinal, and renal complications, secondary hemorrhages, Salivary fistulas, etc. 4. Late complications include widespread scars, hypertrophic scars, hypo- or hyperpigmented scars, disfigurement scars, soft tissue defects, mouth incontinence, trismus, chronic salivary fistulas, and facial paralysis.

7.4. Treatment of traumatic lesions of the oro-maxillofacial region in children

The treatment includes several stages that should be undertaken concurrently, depending on the possibilities of resolution and the time elapsed since the accident occurred.

The general principles of treatment of traumatic lesions in the OMF area depend on the anatomical-morphofunctional features of the area.

✓ Life-threatening injuries (shock, asphyxia, hemorrhage) and complications that can occur in the days following trauma (trauma of the abdominal organs, thoracic organs, and central nervous system injuries), which may compromise the function of the biological system, should be prioritized.

✓ The goal of therapeutic management is to recover the morphological, functional, cosmetic, and psychological integrity of the tissues, organs, and systems.

✓ Oral hygiene must be carried out prior to any therapeutic management, and dental hygiene should be maintained throughout and after the treatment.

✓ Facial lacerations must be thoroughly irrigated, inspected, and properly debrided. Facial debridement should be minimal. Devitalized and shredded tissue should be removed to create a viable wound margin. Wound closure should be preceded by beveling or a slight undercutting of the skin margins to counter scar separation and depression. Muscular tissues, facial layers, and other subcutaneous tissues must be well-approximated. Injuries that extend across margins such as the lips, nostrils, eyelids, or external ears require special care, proper planning, and alignment.

✓ The wounds must be sutured after the reduction and immobilization of the bone and dental-periodontal lesions. Well-approximated deep layers are the key to a well-healing superficial wound. Meticulous attention to hemostasis and copious irrigation are crucial before final epidermal closure in order to prevent infections and hematomas.

✓ Delicate facial closure with synthetic absorbable sutures for well-approximated deep layers and re-approximated margins of the epidermis with interrupted sutures facilitates precise anatomic orientation and is the key to a well-healed superficial wound.

1. Treatment of life-threatening complications.

a. Foreign body aspiration occurs when milk or permanent teeth enter the airway through the mouth after the trauma and cause choking. In children aged 6 months to 3 years, aspiration of foreign bodies is due to their tendency to place and hold the objects in the mouth. Because children are often exposed to trauma by falling, it can lead to foreign body aspiration. Foreign bodies can be nuts, hard candy, beans, popcorn kernels, and pencils. In children, latex balloons, which can conform to the shape of the trachea and block the airway, can be a choking hazard.

Clinical signs of foreign body aspiration are abrupt in onset and include coughing, choking, and wheezing. Partial obstruction includes choking with drooling and stridor. The patient maintains the ability to speak. Complete obstruction includes choking and the inability to speak or cough, along with respiratory distress (cyanosis). An increased respiratory rate may be the only sign of foreign body aspiration in children who cannot verbalize if they have swallowed a foreign body. Efficient and effective treatment must be initiated as life-threatening problems are identified, often without the benefit of a definitive diagnosis.

The treatment of foreign body aspiration depends on the severity of the obstruction of the airway involved. In partial obstruction, the child can clear the airway by coughing orby laying hisface down over the rescuer's arm. In cases of complete obstruction, cardiopulmonary resuscitation is required. If adequate ventilation cannot be restored and patients are unable to respond to non-invasive airway clearance techniques, laryngoscopy, intubation, tracheotomy, or needle cricothyrotomyshould be performed. The tongue is drawn anteriorly and caught between the fingers, and aspiration of the saliva and blood from the mouth should be done. In conscious children, the tongue must be kept outside by a thread passed through the tip of the tongue and tied to a buttonhole.

b. *Hemostasis* – Loss of blood beyond a certain limit is potentially life-threatening. Bleeding is very disturbing, both for the patient and the surgeon. Extensive blood loss can cause hypovolemia, which may progress to hypovolemic shock and eventually lead to death. Hemostasis management after trauma in children is a complicated procedure that needs to be handled in a hospital. Several conventional hemostatic techniques are used to minimize blood loss (mechanical, thermal energy-based, and chemical (pharmacological) methods). They are used according to the stage of evacuation from the site of the trauma to the specialized department. Mechanical methods of hemostasis include manual pressure, ligation of blood vessels by cautery, suturing, and the application of a tourniquet. Sealing of bleeding vessels can be achieved by thermal methods such as electrocauterization or laser cauterization. Chemical hemostatics include Ankaferd blood stopper, chitosan, Floseal thrombin-gelatin matrix (Tissel), Gelfoam and tranexamic acid. A combination of these methods is required to achieve adequate hemostasis.

The application of a Kerlix compression bandage can facilitate adequate hemostasis.

Nasal bleeding will be stopped by an anterior, posterior, or associated nasal packing. In the case of extensive bleeding, blood loss will be compensated by transfusions of blood components. As children are more susceptible to blood loss, hemostasis will be performed in a short period after the trauma.

c. Shock is a life-threatening condition of circulatory failure causing inadequate oxygen delivery to meet cellular metabolic needs and oxygen consumption requirements, producing cellular and tissue hypoxia. Shock is initially reversible, but quickly becomes irreversible, resulting in multiple organ failure and death. Perhaps a more important change is the frequent onset of disseminated intravascular coagulation. Traumatic shock can be caused by any injury, such as severe tissue damage, severe contusions, or multiple fractures. The signs and symptoms of shock include a rapid, weak, or absent pulse; an irregular heartbeat; rapid, shallow breathing; lightheadedness; cool, clammy skin; dilated pupils; lackluster eyes; chest pain; thirst and dry mouth; low blood sugar; and a loss of consciousness. Pain relief, immobilization, anti-inflammatories, and surgery are the main methods of treatment. In severe cases, the doctor must lay the child down, elevate his feet, treat obvious injuries, and keep the child warm and comfortable. The most important part is to assess the magnitude of ongoing blood loss. Patients must be brought directly from the accident scene to the regional trauma center, even if it means bypassing closer hospitals.

2. *Treatment of severe associated injuries*. The life-threatening injuries must be treated as soon as possible (in order of priority or concurrently, under general anesthesia, in mixed, multidisciplinary

teams). In the following hours or days, they must not be delayed due to secondary and irreversible complications. The surgeon may treat facial wounds at the same operating stage.

3. *Treatment and management of facial wounds*. A soft tissue injury in children requires early, appropriate wound management and handling of the injured and frightened child, as well as the concerned, anxious, angry, and often guilt-ridden parents. The first contact with the child is made at the accident site. It is necessary to calm the child, and if it is not possible to gain the cooperation of the patient, which is common in preschoolers, then procedural sedation should be considered. In the same time frame, pressure with a clean cloth or bandage must be applied forseveral minutes to 10 minutes. Once the bleeding has stopped, adequate cleaning is essential for wound care, and this is usually achieved by irrigation with sterile saline, although there is evidence to suggest that potable tap water is not inferior for this purpose. This stage can include mechanical exploration and cleaning of foreign bodies, bone fragments, and tooth fragments, temporary immobilization of jaw fractures; and finally the application of an adhesive bandage or gauze on the wound. Most simple wounds can be handled by clinicians in the office or by trained emergency medicine clinicians. Relatively painless and rapid in application, tissue adhesives (steri-strip, cyanoacrylate adhesive) are ideal for pediatric patients who sustain an uncomplicated wound that is less than 5 cm in length. If the wound is deemed to necessitate hospitalization, the patient must be transferred to the hospital for closure. The indications for hospital-lization are as follows: wounds that are bleeding heavily and do not stop after five to ten minutes of direct pressure; deep wounds that are longer than 5 cm; wounds located near the eyes, lips, nose, or cheeks; puncture wounds caused by a dirty, rusty or sharp object, wounds embedded with debris such as dirt, stones, or gravel; wounds with ragged or separated edges; wounds caused by an animal or human bite; excessively painful wounds; wounds showing signs of infection (increased warmth, redness, swelling, or drainage).

In the case of severe wounds, during transportation to the hospital, the child should lie down on the lateral shoulder and receive pain relief medication, antibiotics, the tetanus vaccine, and the rabies vaccine.

The definitive treatment of the wounds is carried out in the specialized medical care services, which are well equipped and where collaboration with other specialists can be ensured (neurosurgeons, otolaryngologists, ophthalmologists).

When the patient is first seen, the goal is to prevent immediate complications and long-term disfigurement. Given the fact that the face is an integral part of personal presentation and expression for growing children, considerable attention must be given to the injury, including parents` preparation for the healing process, possible complications, and the potential need for future revisions. On initial presentation, maximum attention must be given to primary repair with exploration and preparation of the wound borders and meticulous closure.

It is necessary to choose adequate anesthesia for thorough treatment of all soft tissue wounds. Children aged 3-5 years with maxillofacial lesions must be handled in hospitals under general anesthesia.

Management of facial wounds in children. Primary wound closure is the fastest type of closure and is also known as healing by primary intention. Wounds that heal by primary healing have a small, clean defect that minimizes the risk of infection and requires new blood vessels and keratinocytes to migrate only a small distance.

Simple repair is used when the wound is superficial, primarily involving the epidermis, dermis, and subcutaneous tissues without significant involvement of deeper structures where only one layer of closure is necessary using sutures, tissue adhesives, or other closure materials. *Intermediate repair* includes the repair of a wound that, in addition to the requirements for simple repair, involves a layered closure of one or more of the deeper layers of subcutaneous tissue and superficial (non-muscle) fascia in addition to the skin (epidermal and dermal) closure.

The single-layer closure of a heavily contaminated wound that requires extensive cleaning or removal of particulatematter may also be considered an intermediate repair. *Complex repair* includes repairs that require more than a layered closure, such as scar revision, debridement of traumatic lacerations or avulsions, extensive undermining, stents or retention sutures, debridement and repair of complicated lacerations or avulsions.

Primary surgical closure of the wound and *laceration* is an emergency care safely closed by primary intention. It can beinfluenced by the mechanism of the injury, the anatomic location, and the level of contamination. The period of closure can range from 48 hours to 72 hours after injury in the pediatric population because of a highly vascular face and scalp. Although any injury, regardless of the time elapsed after injury, that can be converted to a fresh appearing clean laceration of the face, slightly bleeding, non-devitalized, with no visible contamination, can be considered for primary closure.

Treatment must begin only after a complete clinical and radiological investigation. Lacerations must be thoroughly irrigated, inspected, and properly debrided. Sterile saline is widely used as an irrigation fluid. Wound exploration of the face requires thorough inspection and direct exploration from the superficial layers to the deep layers. It is important that wound closure be preceded by beveling or a slight undercutting of the skin margins to counter scar separation and depression. Facial debridement in children should be minimal. Muscular tissues, facial layers, and other subcutaneous tissues may be well approximated with plain or chromic catgut or with synthetic absorbable sutures such as Dexon or Vikryl. Injuries that extend across margins such as the lips, nostrils, eyelids, or external ears require particular attention, planning, and alignment. If there is any suspicion of eye injury, an early ophthalmologic examination is imperative.

Deep wounds, contaminated wounds, and bites need especially copious irrigation prior to closure. Debridement of facial wounds must always be minimal; devitalized and shredded tissue should be removed to create a viable wound margin for closure. Irregular margins may be used to create a broken line for closure and reduce the need for unnecessary debridement of viable tissue. Cheek lacerations may represent damage to the facial nerve or may penetrate the oral cavity. If the nerve is lacerated, a primary anastomosis should be performed before closure. Deep cheek lacerations require a multilayered closure and those penetrating the oral cavity necessitate a separate row. In cases of gravel rash-type abrasion, dirt can be ground into the skin, with the potential for a tattooing effect. These wounds require scrubbing with a brush after appropriate anesthesia. Sharp debridement with a scalpel or sharp tissue scissors is required to remove non-viable or irreversibly contaminated tissue from the wound edge and to leave a clean, even-edged wound free of devitalized tissue. Full-thickness loss of the skin and all underlying tissue (avulsion or loss of soft tissue) in the pediatric population requires special consideration. Keeping in mind age relative to puberty avoids creating a secondary deformity on the face. Many tissue defects on children's faces can be repaired with a rotation or transposition flap taken from adjacent areas. When skin grafting is required, special consideration must be given to the choice of donor site.

Management of thermal burns on the face includes cleaning and debridement, tissue grafting, and reconstruction. After resuscitation, the wound is allowed to demarcate before being thoroughly debrided. First-degree burns are best treated conservatively with localized care and bactine ointment. Second-degree burns of the face are best treated with antibiotic ointment and subsequent coverage with a moist, semi-occlusive dressing. Although most facial burns may be dressed with bacitracin or occasionally silver sulfadiazine, second- and third-degree auricular burns should be treated with Sulfamylon, which penetrates down to the cartilage and gives excellent coverage for Pseudomonas infection. Noninfected third-degree facial burns may be initially treated with the application of a bilaminated layer margin such as Integra. This will facilitate the development of a granulation bed onto which a graft may eventually be placed.

Wounds with suspicion of a retained foreign body, especially in the case of retained organic material, warrant careful consideration of referral, as removal of these can be a technically difficult procedure. The edges need to be retracted to explore the depth of the wound, which may include embedded foreign bodies, hematomas, or fractures. Foreign bodies (clots, sand, soil, tooth fragments) can be manually or instrumentally removed from dirty wounds, or the wounds can be irrigated with large amounts of warm, sterile saline. Reduction and immobilization of jaw fractures and dental-periodontal lesions should be performed until the wound is definitively closed.

All wounds should be visibly clean prior to closure. Wounds at high risk include those around the medial canthus, with its associated proximity to the lacrimal apparatus, and those overlying the parotid or major branches of the facial nerve. The continuity of the lines of the eyebrow and the vermillion border are of particular cosmetic importance.

Suture size is determined by the wound location: a size of 4.0 is ideal for deep sutures in many non-weight-bearing areas; 3.0 is reasonable for most deeper sutures on the trunk and limbs; and 5.0 is suitable for most facial and other superficial repairs, provided good opposition has been achieved in the deeper layer. Excessive tension on the superficial sutures can lead to poorer cosmetic outcomes, probably to a greater degree than the size of the sutures used. The choice of suture is multifactorial.

Immediate common complications of primary surgical treatment in the pediatric group include infection, bacteremia, expanding hematoma, and secondary bleeding. Patients who are immunosuppressed or who have sustained blunt trauma, crush injuries or dirty wounds, or penetrating injuries to the oral or nasal cavities have a higher risk of infection. For this reason, meticulous hemostasis, well-approximated facial layers, and antibiotic management are the best prevention procedures.

Late common complications in the pediatric age group include widespread scars, hypertrophic scars, hypo- or hyperpigmented scars, visible suture marks, facial disfigurement, and facial nerve paralysis.

Hypertrophic scars tend to resolvewith time. A keloid may significantly increase with time, causing a significant itching or burning sensation. Kenalog injections and pressure dressings are the principal postoperative treatment modalities in this case. Secondary repairs should be delayed for a minimum of 6 months, if not more than 1 year. There is no need to wait until adulthood to repair facial deformities, which may carry significant psychological implications and potentially have deleterious effects on development.

Secondary closure (secondary intention). Skin infections, ulcerations, abscess cavities, punctures, small insignificant animal bites, and partial thickness (abrasions, second-degree burns) tissue loss are left to heal by secondary intention. Wound care entails thorough cleansing, irrigation, and debridement of devitalized or contaminant-impregnated tissue. Usually, these wounds are not closed with sutures and are allowed to heal gradually by granulation and eventual re-epithelialization.

Tertiary closure (delayed early and secondary sutures). Delayed early suture is performed 10-12 days after the accident, and secondary 20 days after the accident. High-velocity force injuries (bite wounds, lacerations, road traffic accidents, sport-related accidents, concomitant injuries) are an indicationfor delayed closure. Delayed wounds can be converted to fresh ones by cleansing, irrigation, and debridement. A shredded, ischemic, or blue-black wound requires debridement. Simple excision, wound edge revision, or full wound excision techniques can be used for debridement. Oral antibiotics are administered after initial care before delayed closure.

Delayed secondary suture healing treatment includes sequelae and is applied only after all the lesions have completely healed and the tissues have regained their normal plasticity (usually after a period of more than 6 months).

*Scar Management. B*ecause children tend to heal by scarring, it is important to guide the wound during active healing. All permanent sutures should be removed in 3-5 days and wound support dressings applied for 10-14 days to remove tension from the wound bed, which would increase collagen deposition. During this period, any irritating influences and encrustations should be removed from the wound, and the area should be kept moist and covered. Topical antibiotic ointment should be discontinued after 7 days to prevent tissue reactions. When the wound is well epithelialized, usually in 7-10 days, silicone sheets or topical scar gels can be applied for several weeks. These agents keep tension off the wound and maintain a slight pressure on it to help reduce excessive collagen deposition in the scar. During this period, it is important to avoid excessive wetting, drying, heat, or irritating agents that might exacerbate the inflammatory response. The patient should use sunblock with a high sun protection factor while outside and wear a wide-brimmed hat to cover the face, if possible, for up to a year after injury to avoid ultraviolet stimulation of melanocytes in the wound bed and subsequent hyperpigmentation.

Learning objectives

1. Peculiarities of the skin development in children according to their age.
2. Etiology of traumatic soft tissue lesions of the oral and maxillofacial region.
3. Clinical manifestations of soft tissue laceration of the oral cavity in children.
4. Frequency of traumatic lesions of soft tissues in children in relation to age, traumatic dental injuries and facial bones.
5. Clinical manifestations of soft tissue lacerations.
6. Type of surgical treatment of soft tissue injuries according to the length of time since the accident.
7. Particularities and stages of primary surgical treatment of traumatic soft tissue lesions.
8. Complications of soft tissue injuries in children.
9. Prophylaxis of traumatic soft tissue lesions.

TESTS

1. SC. Which period of time following the accident is considered appropriate for primary surgical treatment of lesions in children:

A. 12-24 hours;
B. 24-24 hours;
C. 28 hours;
D. 10-12 days;
E. 20 days.

(B)

2. SC. Which period of time after the accident is considered appropriate for delayed primary suture:

A. up to 24 hours;
B. after 10-12 days;
C. after 20 days;
D. after 24-28 hours;
E. all answers are correct.

(D)

3. MC. Indicate the clinical signs of soft tissue lesions of the oral cavity:

A. abundant bleeding;
B. deep wounds;
C. irregular edges;
D. tissue defects;
F. regular edges.

(A, B, C)

4. SC. Indicate the type of closed lesion of the soft tissue with edema, diffuse swelling, ecchymosis, or hematoma as clinical manifestations:

A. cut wounds;
B. penetrating wounds;
C. excoriations;
D. contusions;
E. contused-crush wounds.

(D)

5. MC. Indicate the common signs of soft tissue injuries:

A. breathing disorders;
B. pain;
C. hemorrhage;
D. hypersalivation;
E. interruption of skin continuity.

(B, C, E)

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8. PEDIATRIC MAXILLOFACIAL TRAUMA OF THE UPPER AND LOWER JAWS, AND FACIAL BONES

8.1. Etiology and peculiarities of facial bone fractures in children

Facial fractures are rare in children. Approximately 5 to 15% of all facial fractures occur in children. The prevalence of pediatric facial fractures is lowest in infants and increases progressively with increasing age. Only 1.0% of facial fractures occur in children younger than 5 years, whereas 1.0 to 14.7% occur in patients older than 16 years. Two peaks are typically observed in the frequency of such fractures. The first, at the age of 6 to 7 years, is associated with the beginning of school attendance. The second, between 12 and 14 years, is related to increased physical activity and participation in sports during puberty and adolescence.

In children, there is a much greater incidence of skull fractures than facial fractures. The cranial-to-facial proportion is approximately 8 to 1, compared to 2 to 1 in adults. There is a much greater cranial volume-to-facial volume ratio in children than in adults. Because of the cranial-to-facial proportion, head injuries in the pediatric population make up about 60%.

In the adult population, nasal bone fractures are the most frequent facial fractures. Mandibular fractures, on the other hand, account for the highest percentage of bone injuries in children (31%). As children get older, more fractures are seen.

Of all facial injuries, mandibular fractures occur in more than 60% of cases. Mandibular trauma commonly occurs between the ages of 7 and 14 years, with the highest frequency. Its incidence in children under the age of six is low, but it is severe, and it is associated with trauma (closed craniocerebral).

Refractory fractures are common in children. Condylar fractures, either unilateral or bilateral, caused by chin trauma are the most common refractory fractures. The clinical form varies according to the child's age. The incidence of condyle fractures decreases, and the incidence of body and angular fractures increases with age. The incidence of condyle fractures is higher in children under 6 years (43.4%), and it decreases to 7% in children aged 13 to 18 years.

Greenstick fractures are more common in children than in adults because children's bones seem to be more elastic. A large cartilaginous growth center contributes to facial bone flexibility. The low facial-to-cranial ratio increases the potential that force from an impact may be directed to the cranium, therefore decreasing the direct force to the face. A greater cancellous-to-cortical ratio in children's bones, a high tooth-to-bone ratio, and a thinner cortical bone with ill-defined margins between the medullary and cortical bones lead to greenstick injuries. Stability is increased by the presence of developing tooth buds within the jaws and the lack of sinus pneumatization. A thicker layer of adipose tissue covers the more elastic mandible and midface bones, and suture line flexibility makes facial bone fractures in children more minimally displaced. An intensive vascularization, a thick layer of periosteum, and a wide Haversian system show great elasticity of the bones. A well-developed Bichat's fat pad and a thicker layer of fat on children's faces lead to less severe trauma, most of which result in greenstick injuries. The mandibular condyle in children is short, stout, and highly vascular with a thin cortical plate. In younger children, the inferior alveolar nerve travels adjacent to the lingual cortical surface, close to the inferior border of the mandible.

In addition, high energy is required to produce fractures of the mandible and midface. Children with mandibular and midface fractures have a higher incidence of associated injuries (chest, abdominal, extremity, and cervical spine).

Although low-impact or low-velocity forces often (25-75%) lead to severe associated injuries, closed head trauma and neurocranial trauma occur more often. Thus, even isolated or non-displaced fractures in children require treatment in hospital settings.

Etiology. Usually, children in the early years of life are in a protected environment. As children get older, they become more active, playing sports, climbing trees, and having more contact with older children. Although there is some variability in the studies on fracture frequency from different countries, this may reflect the nature of the referral pattern in a particular city and the socioeconomic status of the population in that region. Falls, sports-related injuries, and road traffic accidents are the most frequent causes of craniofacial trauma in children. In infants, falls in the home environment are the most common cause of craniofacial trauma. With increasing age (up to 6 years of age) and outdoor exposure, falls tend to occur outside the protected area of the home and parental supervision. The majority of sports-related traumas in the school setting occur in children aged 8 to 14. Interpersonal violence occurs more commonly in adolescents.

Motor vehicle accidents are the most common cause of mandibular fractures (80.2%), followed by accidental causes such as falls (48%; falls from heights, trees, etc.). After violence and other causes (9%), sports-related injuries are the second most common cause (42%).

General considerations. Children with bone fractures may present with the same clinical findings seen in adults with similar injuries.

It is difficult to obtain a good clinical examination while examining clinically the patient in order to diagnose a fracture. The physician should deal with children in such a way as to develop their trust so they will allow the physician to perform an examination. A normal child coming into the office at 3 years of age for an elective examination can sometimes be a challenge. A child who has experienced a tremendous amount of trauma and is suddenly sitting in a frightening emergency room is under a great deal of stress. The same is true for obtaining an X-ray. It is very difficult to get a good series of X-ray to diagnose facial fractures due to the child's behavior and the difficult collaboration between the physician and the young patient. Clinical examination can be a challenge because of the severe swelling of the soft tissue. Despite the difficulty, the physician should make certain not to accept studies that are less than optimal. It may be necessary to sedate a child, even to the point of placing the child under general anesthesia, to get a thorough physical examination and an adequate X-ray.

The radiographic images of the oro-maxillofacial area in children have peculiar features:

1. The image of the gums in newborns and infants is projected at the level of the palatal shelves.
2. Teeth buds in young children are located under the orbital floor. As the jaw develops, they move downward.
3. The sinuses of the jaws are crack-shaped, with the lower edges erased, disappearing amid the dental buds.

By the age of 8-9, the sinus floor projects to the lower edge of the piriform aperture. The density of primary teeth, compared to permanent ones, is lower. With the advent of better investigative facilities, CT scans and 3D reconstruction can be obtained, and optimal treatment plans can be formulated.

8.2. General considerations for lower jaw fractures

The pattern of craniomaxillofacial fractures seen in children and adolescents varies, involving skeletal anatomy and socio-environmental factors. The general principles of clinical signs and symptoms are the same in adults. The clinical signs include mobility of bone fragments at the line of fracture and scratch, interruption of bone continuity, deformation of the bone, and malocclusion. There are some other signs such as pain localized in the area of the fracture or irradiated, spontaneous or provoked pain, the antalgic position of the mandible, bruising, swelling, laceration, hematoma, hypersalivation, chewing, swallowing and phonation disorders, intraoral or facial lacerations.

Mandibular fracture. Mandibular injuries are classified as partial (located on the alveolar process, condyle, or inferior border of the mandible), complete or incomplete (located on the median, paramedian, and laterallines, angle, ramus, or subcondylar region), non-displaced, or significantly displaced mandibularfractures. There are single, double, triple, multiple or comminuted fractures, as well as closed or open fractures, penetrating the oral cavity. Also, they are divided into fractures of the tooth-bearing region (body and symphysis fractures) and the non-tooth-bearing region of the mandible (angle, condylar, subcondylar).

Mandibular fractures are the most common facial injuries in children. Mandibular fracture sites include the condyle (55%), the parasymphysis (27%), the body (9%), and the angle (8%). Most pediatric mandibular fractures are non-displaced („greenstick"). Those located in the tooth-bearing region are also called „subperiosteal fractures".

Mandibular body, symphysis, and ramus. Children with mandibular fractures may present with the same clinical findings seen in adults with similar injuries, such as pain, edema, malocclusion, intraoral laceration, hematoma, facial lacerations, and paresthesia. Most pediatric mandibular fractures are non-displaced injuries. Clinical-radiological findings of tooth-bearing region injuries („subperiosteal") include bruising, hematoma on the buccal floor, mucoperiosteal wound on the gum, and sometimes mobility of the fragments on the gum area along the fracture. X-ray pictures detect non-displaced fractures because embedded tooth buds and thick periosteum hold the fragments together „like glue".

Earlier, most of the pediatric cases were treated with conservative methods or closed reduction techniques. Only recently have the distinct advantages of accurate primary repair and the stable fixation of facial fractures been applied to the rehabilitation of injuries in children too. With the advent of better investigative facilities like CT scan and 3D reconstruction, as well as newer airway management techniques with reliable anesthesia techniques and specifically introduction of mini- and microplates, open reduction and fixation of pediatric facial fractures is becoming more common.

Although the management of mandibular fractures in the pediatric population differs somewhat from that of adults, mainly because of concern for possible disruption of growth. In children, the final result is determined not merely by the initial treatment but also by the effect that growth has on form and function.

Non-displaced fractures without malocclusion can be treated with a conservative approach when possible, which consists of careful observation, a blenderized diet, and avoidance of physical activity. It means seeing the child on a weekly basis. If there is any change in the occlusion and the occlusion does not look perfect, another X-ray examination should be taken to make certain that the child has not developed any distraction of the fracture, which requires a more aggressive treatment. Orthodontic devices (Mac-Lennan type cap splint, Frankel functional appliance, Andreza-Hoopla appliance, Veber appliance), mouth guards, gunning splints, or lingual splints are sometimes recommended.

If the mandible is displaced, closed reduction and immobilization are performed using maxillomandibular fixation or internal skeletal fixation, or a combination of these techniques. The exact method of immobilization depends on the child's chronologic age and state of dental development. Slight occlusal discrepancies resulting from lack of perfect reduction correct spontaneously as permanent teeth erupt and bone undergoes remodeling with function.

The first deciduous teeth (incisors) erupt in children at about 6 months of age. As the incisors continue to develop, the molars begin to erupt, followed by the canines. At this age, the roots of the primary teeth develop. Between the ages of 3 and 6 years, there is enough tooth root to be able to secure a primary tooth to an arch bar without avulsing it and obtain support for the bars for the maxillomandibular fixation. After the age of 15 years, maxillomandibular fixation with an arch bar can be established in the same way it is in adults because the adult dentition has erupted and root formation has matured enough to provide the necessary support. Between 6 and 15 years of age, a stage of mixed dentition occurs when obtaining support for an arch bar to place a patient in maxillomandibular fixation using the teeth becomes very difficult. The very early stages of resorption of the primary tooth roots are at about 5 years of age. The first adult tooth, which starts to erupt at about 6 years of age, does not have well-formed roots. In addition, the crown is not very prominent through the gum line; therefore, it is difficult to get a wire below the height of the tooth to stabilize an arch bar. As a child gets older, more of the primary tooth roots develop, while the adult dentition slowly starts to develop the root structure. From 6 to 15 years of age, there is a considerable amount of developp-ment and stability of teeth caused by the various stages of root develop-ment that may vary from child to child.

The fracture is manipulated, and the patient is put into maxilla-mandibular fixation using the technique appropriate for the child's dental development. If possible, arch bars are placed and elastic maxillomandibular immobilization is performed. Tigerstedt-type splints are widely used for maxillomandibular immobilization and are usually used in permanent or primary dentition when tooth roots are developed. They can be applied as an additional device when immobilizing the jaw with titanium plates or when closed reduction is performed. If the teeth are inadequate, the fracture site is immobilized with a gunning or lingual splint. Appliances should be fixed in place using circummandi-bular wires, one on either side of the fracture, or two wires to add stability to the splint. If intermaxillary fixation is required, then wires can be added from circummandibular wires to wires at the pyriform region or zygoma. The splint should be left in place for three weeks.

A single arch mandible splint secured with circummandibular wires or interdental wire ligatures like the Hippocratic type of ligature (a wire of 0.2-0.4 mm passed in 8 around teeth, with 3-4 teeth on both sides of the line fracture and twisted at one end) is another option for mandibular immobilization.

A closed reduction is considered the best approach for a mandi-bular fracture. However, with unstable fractures that cannot be secured with closed reduction techniques, an open reduction and internal fixation are necessary. This is a more aggressive technique to reesta-blish the normal preinjury anatomic relationships of the jaws. When open reduction and internal fixation are necessary, there is very little room for bone fixation devices. There is slightly more room at the mandibular angle and symphyseal regions for fixation. The mandibular canal, which passes through the bone of the mandible and tooth buds located in the bone, must be considered when placing hardware. For this purpose, the right miniplates are used. After finishing the internal fixation, it is critical to make sure the condylar heads are not dislocated. In comparison with titanium miniplates, the resorbable types of miniplates „Lactosorb” are best for children. Miniplates with mono-cortical screws (2-4 mm) placed between the inferior margin of the mandible and the gum (Champy's line) or at the inferior border are called „stabilization systems” and can be used for open immobilization in children too. Monocortical screws do not provide stability for fracture bonds, and children need further maxillomandibular fixation or orthodontic devices such as mouth guards, gunning splints, or lingual splints. Open reduction and internal fixation provide stable immobilization, promote primary bone healing, and make patients comfortable immediately after surgery (free breathing, nutritional intake, and oral hygiene care).

8.3. Clinical and radiological findings of „greenstick” condylar injuries

„Greenstick” condylar injuries occur when a child strikes his chin. The force of the impact is transmitted back to the condylar head, and the sponge may get crushed. Clinical findings of upper mandibular injuries include chin deviation toward the fractured side due to the pull of the lateral pterygoid muscle on the non-fractured side. Pain on opening, an open bite, and swelling in front of the ear are mostly overlooked because of the thick overlying soft tissue.

Palpation into the external auditory duct on opening does not feel movements of the condyle, because it has been pushed into the middle cranial fossa. Radiological findings depend on the type of condylar injury (*figure 12*). It can appear with subluxation (various degrees of displacement), dislocation or avulsion, and crush injuries of the head. If the condylar head has been subluxed, X-ray films show that the continuity of the periosteum and cortical plate on the lateral aspect of the subcondylar area has been disrupted, while the integrity of the periosteum and medial cortical plate on the medial aspect has been maintained. If the condyle has been pushed into the medial fossa, the loss of ramus height can be observed, the condyle being located in the middle cranial fossa.

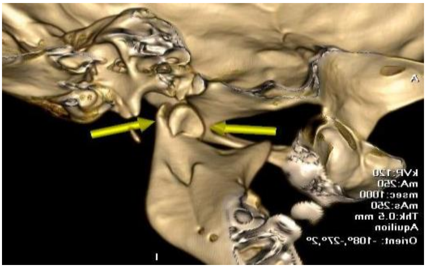

Fig. 12. **Condylar process fracture with dislocation**

Condylar head injuries are important because of the mandibular growth. A large portion of the mandibular growth is centeredat the condyle and the posterior portion of the ramus. Development takes place by bone deposition posteriorly and superiorly, involving a major remodeling of these regions as growth progresses. The condyle and ramus of the mandible play an adaptive role, responding to the growth of the cranial base, midface, and surrounding soft tissue influences. When teeth start to erupt as a child gets older, the alveolar bone develops fully. Although this is a simplistic summary of mandibular growth, which is a very complex process of deposition and remodeling, clinically, one will perceive a downward and forward growth of the mandible. The mandibular condyle in children is a major region of growth, able to change in any direction to accommodate surrounding anatomic stimuli. The head of the condyle is soft until approximately 3 years of age. Consequently, a crush injury is more likely than a routine fracture in a child less than 3 years of age. Anything that disrupts the condyle circulation, causing bleeding or scarring in the area, willcreate a risk of temporomandibular joint ankylosis. In addition, anything that disrupts the condyle head can also disrupt the growth of the mandible.

Therefore, the age at which a child sustains a condyle injury becomes significant. Before 3 years of age, one might see a severe growth deformity resulting from trauma to this region. After about 12 years of age, the same type of trauma may cause fewer problems regarding development but still lead to persistent malocclusion.

The location of condylar injuries affects the management of the patient. An intracapsular injury that involves the condylar head within the confines of the capsule should be managed with immediate mobilization. A child with this type of injury can be given chewing gum to stimulate mobility, and doctors must evaluate the child weekly and encourage continuous movements. There is a significant risk of ankylosis of the joint if these injuries are immobilized. Even if the condylar head has been subluxed, there is such active growth and adaptation in young children in this area that the condyle has the ability to remodel and reestablish a normal anatomic relationship with the glenoid fossa.

Subcondylar fractures are most common in children older than 5 years of age. After 5 years, the TMJ is more calcified and refined and the neck of the mandible becomes the weakest area. Subcondylar injuries with loss of ramus height are treated by closed reduction techniques. The child is placed in maxillomandibular fixation. The child should not remain in fixation for extended periods because ankylosis can occur even if the joint is not directly injured. Mandibular fixation is removed after 2-3 weeks, and subsequently, functional therapy of the TMJ is required. With bilateral fractures, fixation should be left longer, 3 full weeks, to enable the bone to become more rigid and maintain the ramus height. Functional appliances such as Frankel and **Andrezena-Hoiplea** appliances can be used to immobilize subcondylar fractures in children.

Birth trauma refers to damage to the tissues and organs of a newly delivered child. The causes of craniofacial injuries are physical pressure, cephalo-pelvic disproportion, quick and rapid delivery, delayed and prolonged delivery, the use of forceps, and vacuum extraction.

Craniofacial birth trauma occurs in 11.4% of cases and is usually located in the skull, condyle, and zygomatic arch regions. The facial bone fractures are usuallyoverlooked after birth and appear over time with facial deformities and dysfunction of the temporomandibular joint.

Periosteal and bone contusions often occur in young children and adolescents. They appear in the place of the blow. They begin and progress as a closed trauma and can result in aseptic bone inflammation. Clinically, they represent local thickening of the bone, which appears 3-4 weeks after the blow, imitating a tumor process. Periosteal contusions cause the stimulation of bone tissue production, which is radiologically manifested by its depositing on the bone surface. At the beginning of its formation, the bone structure is fragile with a trabecular pattern, later transforming into condensed bone on the surface of the cortex.

Partial fractures of the alveolar process are most commonly found in the anterior area of the maxilla, and are often associated with mucosal and adjacent soft-tissue lesions, dental lesions, muco-periosteal laceration, swelling and bruising, and hematoma of the adjacent soft-tissues. They are rarely accompanied by lesions of the dental buds, which become contaminated and lead to their mortification.

8.4. Midface fractures in children

The facial area is composed of 15 bones welded together and fixed at the base of the skull. Midfacial fractures show many of the symptoms seen in adults, such as a mobile or impacted maxilla, malocclusion, periorbital ecchymosis, lacerations around the midface, hematoma, edema, paresthesia, and bony step-offs. Midfacial fractures are much rarer than mandibular ones. Jaw fractures are usually uncommon in young children. The incidence of facial fractures in children under the age of 12 is 0.5%**.** In the Republic of Moldova, the incidence of maxillofacial injuries in children up to the age of 16 years accounts for 2.7%. The low frequency is accounted for by the presence of protection structures in the skull and mandible, incomplete pneumatization of the jaws and sinuses, mixed dentition, and erupting teeth. Maxillary fractures result from the action of a strong force (falls from heights, such as from a roof or construction site, or road accidents) and are usually accompanied by craniocerebral trauma. Trauma to the jaw, ranging between 25% and 88%, is associated with various injuries, ranging from severe to very severe ones.

Le Fort I fractures are rare because of the elasticity of the maxilla and the absence of a well-developed maxillary sinus with thin cortical bone walls. In 4-year-olds, the majority of the maxilla is supported by developing tooth buds that have a high concentration of soft tissue, making the maxilla more flexible and resistant to fracturing. There is no well-pneumatized sinus with thin cortical walls. In newborns, there is a small, rudimentary maxillary sinus, very small and localized along the pyriform buttresses, medial to the infraorbital nerve.

At about 5 years of age, the sinus starts to pneumatize more significantly. The sinus is centered almost entirely behind the infraorbital nerve. The sinus expands significantly, but the maxilla is still filled with developing tooth buds. Le Fort II fractures are more common because the nasal ethmoid area is delicate in children and there is no soft tissue atthe developing tooth buds in that region to support them.

Le Fort III fractures (craniofacial disjunction) are very rare. Usually, the fracture lines are directed towards the regions with low resistance. Fractures of the palatine suture are common. To manage maxillary fractures, a conservative approach is by far the best approach. Greenstick injuries most often can be managed with a soft diet and careful observation of occlusion. In complete fractures, it is necessary to reduce the fracture and stabilize it with closed techniques using splint maxillomandibular fixation and circumferential bone wires. Fixation is kept in place for 3 to 4 weeks. If the reduction using splints and suspension wires is not satisfactory or if there is still displacement or instability, an open reduction and internal fixation are necessary. Care must be taken not to plunge into the underlying tissue of the tooth buds during open reduction because they can be very superficial in the maxillary walls. Inyoung children, in the area of the maxilla between the buttresses, the developing tooth buds may be separated from the mucosa only by a thin layer of periosteum and may not have a well-developed bony cortex.

Maxillary fractures heal extremely rapidly, within a period of about 2 to 4 weeks. Due to rapid healing, these fractures need to be addressed within the first 3-4 days after injury. Because of the potential injury to tooth buds, it is more difficult to perform an elective osteotomy in a child after the maxilla has healed in an abnormal position. The best chance of a perfect reconstruction is at the time of the primary injury and early in thecourse of that injury.

Zygoma fractures in children are rare. The maxillary sinus is not well developed, but there is good support for the zygomaticomaxillary buttress. Tooth buds in the buttress region contribute to energy absorption. Because the zygoma is a dense, stable bone, displacement is usually caused by a significant force. Pediatric zygoma fractures present the same clinical findings as in adults: asymmetry, palpable infraorbital rim step-off, malposition, periorbital ecchymosis, subconjunctival ecchymosis, edema, and diplopia. Early repair and accurate treatment must be performed in a very short time, in the first 4-5 days after injury. The injury has a tremendous potential to heal rapidly and, consequently, form a strong fibrous union in a very short time. Secondary reconstruction is extremely difficult because it may damage developing tooth buds.

Orbital fractures. The etiology and clinical findings are identical to those in adults. It is important to consider children's orbital developpment. The orbits reach their full size by the time the child reaches about 7 years of age. The small maxillary sinus and flexible bone result in fewer blowout fractures in children. The orbital floor is supported by developing tooth buds, particularly in the inferior medial region. Because there is little development of the maxillary sinus, often there is no large void for the orbital floor to be extruded into, but displacement can occur. A conservative approach is best for orbital injuries. The indications for open reduction and internal fixation of orbital fractures include diplopia, enophthalmos, vertical malposition of the globe, displacement of the zygoma or orbital walls, and infraorbital nerve paresthesia.

8.5. Pediatric fractures and growth

At about 3 months of age, the child reaches approximately 50% of his adult facial size, and about 70% of his adult size at 2 years of age. By the age of five and a half, the child's facial skeleton is nearly 80% of its adult size. Facial growth is not something that starts at birth and continues at a steady rate until 16 to 18 years of age. There are several growth periods when children are very active in their growth. The most active periods are from birth to 6 months and from 4 years to 7 years of age. Fractures during rapid growth periods have a greater potential to disrupt growth. Younger children may experience more severe growth problems resulting from injuries than older children with similar injuries who have completed most of their growth. There is a much greater potential for disruption of growth in a fracture in someone who is 2 years old than in someone who is 16 years old. Some occlusal changes occur with growth. For this purpose, in children up to one year, the fixing elements must be removed at 2.5-3 weeks; at 1-3 years, they must be removed at 3-4 weeks; at 3-7 years – after 3-5 weeks; and from 7-14 years – over 6-8 weeks. The term of regeneration depends on the type of fracture and the general condition of the child.

A decreased vertical height of the mandibular body and alveolus may occur after fracture of the horizontal ramus of the mandible if teeth are lost due to injury or hardware through tooth buds. Contour defects may occur due to severely comminuted or compound fractures when bone undergoes resorption during remodeling. Fractures can have an effect on growth, although surgery with periosteal stripping and internal fixation also has an impact on growth.

Children do not necessarily grow back into a normal occlusal pattern, and a child who has a displaced mandible cannot be expected to re-establish a normal occlusion. But if surgery does not establish good intercuspation at the time of the fracture, it can result in a poor outcome.

Obviously, a conservative approach based on observation and the use of splint fixation with removable hardware would be ideal, but under some circumstances, this will not always provide a reduction that will facilitate adequate function. When opening a fracture, doing as little periosteal stripping as possible, placing the least amount of hardware possible and using autogenous bone for reconstruction of defects when needed are the main principles when dealing with the growing skeleton of a child. Although considering potential cerebral spine injuries in all facial fractures is important, this becomes more significant in children. Intensive care management of the head trauma includes the surgical approach of the injury and intensive care management during evacuation from the accident scene to definitive management.

Concomitant injuries. Children with facial trauma often have multiple traumas. In 25-75% of cases, there are associated injuries. These include closed head trauma, cervical-spinal and neurocranial injuries, temporal bone fractures, extremity fractures, abdominal and thoracic injuries, as well as dental and soft tissue lacerations. In children, craniofacial fractures are caused by high energy. Children have lower total blood and stroke volumes than adults. The risk for hypothermia, hypotension, and hypoxia after blood loss is higher in the pediatric population. Clinical signs of shock may occur with even insignificant amounts of rapid blood loss due to small blood volume. Because of the small size of the airway, laryngeal edema or retroposition of the base of the tongue may cause sudden obstruction. Even mild airway swelling or mechanical airway obstruction can quickly compromise the airway. Thus, maintenance of the airway and breathing, control of hemorrhage, and early resuscitation are even more critical and time-dependent in children than in adults. Pediatric facial fractures in concomitant trauma are sometimes not suspected or overlooked in the emergency room. The index of suspicion may be low because the facial injuries are uncommon and the swelling is extensive. It is often difficult to obtain a clinical examination in uncooperative young patients, and the accompanying caretakers may not have witnessed the accident.

For this reason, the emergency management of facial trauma in the pediatric population needs extra consideration. Craniofacial trauma should be treated as a priority, with an emphasis on initial resuscitation measures such as airway securing, hemodynamic stabilization, and evaluating and treating injuries to other vital organs. A thorough discussion of the case among the various specialists of the trauma team can help guide the decision between early and delayed surgical intervention.

8.6. Management of facial trauma in children

Initial care includes pre-hospital care and the timely transportation of these accident victims to the health centers and/or to a well-equipped health center with a well-trained trauma team.

1. Primary survey or pre-hospital care. Management of life-threa-tening emergencies takes priority, namely *asphyxia, craniocerebral trauma, shock, hemorrhage* according to the Glasgow Coma Scale /Score. Initial management of patients with craniofacial trauma follows management of airway, breathing and circulation, disability or neurological deficit, exposure, and temperature control. Upper airway obstruction due to craniomaxillofacial trauma invariably results in a threatened airway. Pre-hospital care includes anticipating and recognizing air obstruction, particularly in unconscious children. In a higher-velocity trauma that involves the mandible, the swallowing mechanism is altered due to pain and an ineffective protective reflex. Airway compromise may occur due to the displacement of the tissue and swelling caused by the injury. Many areas of the face are very sensitive, and even low-velocity trauma can cause tremendous pain relative to their size. Given that facial traumas are sudden, unexpected events often produce intense emotional distress along with the pain caused by the injury. Hence, it is important to protect the airway from blood, secretions, saliva, a full stomach, vomiting, and milk teeth that usually have low implantation in the gum to prevent aspiration and further pulmonary complications. Often, airway obstruction can be managed with patient repositioning. Emergency care includes suctioning or a finger-sweep technique to clear the airway by removing blood and debris from the oropharynx, keeping the patient in a supine position, and performing a chin lift and jaw thrust maneuver to prevent tongue fall. If the nasal and oral cavities are clear, then an artificial airway can be used if needed.

Although traumatic facial injuries frequently result in severely disfigured appearances, they are rarely life-threatening. The protocol entails history taking and physical examination to determine the possible mechanism of injury, identify source of hemorrhage and its exact location, check for contamination and the presence of foreign bodies, and determine the extent of the associated injury or fracture. Additionally, the wound needs to be washed with tap water. During assessment or exploration of the wound, it bleeds actively. The most effective way to stop bleeding is by applying direct pressure to the wound with hand-held surgical sponges or using an epinephrine-moistened sponge applied with pressure to the wound for 5 to 10 minutes and keeping the dressing until medical care is provided at the health center. During assessment or exploration of the wound, it bleeds actively. A nasal hemorrhage may require packing.

Temporary partial reduction and stabilization of the fracture segments provide symptomatic relief, help control bleeding, and minimize damage to the inferior neurovascular bundle during transportation to the nearest health center from the place of accident. This can be performed with Barton's bandage (a circumferential wrap from the chin to the skull vertex) or by placing a stainless-steel bridal wire around the necks of two teeth on either side of the line of fracture.

2. Medical care at the health center. Maintenance of the airway and timely transportation to the hospital setting are of primary importance. Medical health centers must have an available intensive care unit and trauma team so that the necessary interventions can be carried out at the earliest possible time.

Resuscitative measures must be taken over by the trauma team in the emergency department after admission. The main diagnostic interventions that should be performed during initial resuscitation are airway assessment, circulation support, fluid and electrolyte balance, adequate nutritional intake, analgesics, blood samples, a computed tomography scan, an ultrasound, and an X-ray. Care must be taken for cervical spine injuries and neurologic injuries. A spine injury should be assumed until excluded clinically and radiographically. A primary survey includes a brief but detailed history of the injury timing and mechanism, as well as any previous treatment.

A systemic and detailed physical examination is performed in order not to miss any associated injuries. Based on the presence of associated injuries after completion of a detailed physical and imaging assessment, the examiner should consult with such specialists as a neurosurgeon, anesthesiologist, ophthalmologist, and dentist.

Some of the patients have to undergo emergency surgical interventions. The most common indications for immediate surgical intervention include depressed skull fracture, extradural hematoma, brain contusion with intracranial bleed, craniospinal fracture rhinorrhea, and fractured maxillofacial bone interfering with the airway. Other patients must be transferred to the intensive care unit for observation and management. After surgery, patients who have undergone surgery must also be transferred to the intensive care unit. The majority of the patients must be mechanically ventilated, and in a few of them, tracheostomy should also be performed for various indications. In the intensive care unit, routine monitoring must be carried out, which includes heart rate, blood pressure, pulse oximetry, ECG, end-tidal carbon dioxide, etc. Day-to-day investigations should be carried out to monitor and individualize the treatment. Only those patients are discharged who have regained consciousness and made a good recovery.

Once the patient is stable, a trauma evaluation is completed, and an open airway is secured, a more focused examination of the injury can be undertaken. When caring for these injuries, operative or nonoperative treatment, as well as inpatient or outpatient management, should be determined. Some patients may benefit from 24-hour overnight observation to assess heart rate, blood pressure, pulse oximetry, pain management, and oral intake.

Although over 95% of cases require direct suturing for primary closure, all of the head injuries have a Glasgow coma scale less than 4, indicating that the injuries are more like brain contusions with a transient loss of consciousness. Primary closure in patients with multiple injuries and large facial wounds is necessary pending when the presence of concomitant injuries is ruled out. Several aspects of wound care are important in predicting the quality of healing in children: irrigation, minimal debridement of tissues, maintaining tissue viability, eliminating foreign body contamination and the resulting excessive inflammatory response, keeping sutures below the skin surface, if possible, using supportive skin dressings such as wound support tapes and temporary immobilization for jaw fractures during the first 2 weeks of wound repair, protecting the wound from subsequent injury, excessive drying, wetting, or temperature variations. Bleeding control with clamps and hand-tied ligatures is indicated for larger single bleeding vessels. The use of antibiotics depends on the mechanism of injury. The tetanus status of the patient should always be verified. Specific surgeries to the nose, the lips, the ear, or the eyelids could vary from simple wound closure to staged reconstructive surgeries. Early diagnosis and repair of nerve and duct integrity are paramount.

3. Definitive treatment must be carried out in a special cranio-facial department. The final treatment of the craniofacial area must be done in a well-equipped department. Surgical treatment of the head and neck area is performed in the operating room under nasotracheal anesthesia. The goal of definitive surgical treatment is three-dimensional reconstruction of the skeletal structures in order to restore the face to its original width, height, and length.

Before proceeding with definitive management such as suture placement, several issues have to be considered: wound exploration, cleansing, irrigation, tissue debridement, and excision.

Pre-injury skeletal and dentoalveolar anatomy and function are re-established by anatomic reduction of the fracture based on the occlusion. The surgeon's preference, the patient's age, and the location of the fracture determine the type of maxillomandibular immobilization that is used. Some maxillomandibular techniques include maxillomandibular fixation screws, acrylic splints, circummandibular wires, transnasal wires, single-arch mandibular splints, Risdon cables, and a combination of restorable screws and sutures. Severe facial trauma requires the use of a variety of methods to import tissue to the head and neck region. Local tissue flaps have limited amounts of tissue and a modest vascular supply, and thus are often saved for the final stages of reconstruction for minor contouring. Pedicled myocutaneous flaps offer large amounts of tissue with reliable vascularity for soft tissue coverage, but are often bulky and are limited by the length of the vascular pedicle. Free tissue transfer allows the early reconstruction of damaged bones and provides soft tissue coverage soon after injury. Additional reconstructive techniques and tools include implants, tissue expanders, and epidermal skin grafting.

C*omplications*. The outcomes of pediatric facial trauma depend on the location and severity of the fracture and the type of treatment.

The outcomes change as the child grows and develops. Complications after facial fracture repair are less common in children than in adults. These include infection, malunion, nonunion, malocclusion, facial asymmetry, mandibular growth restrictions, and TMJ dysfunction. A classification scheme of all adverse outcomes after facial trauma repair was developed by Rottgers et. al. In this system, type 1 outcomes are related to the fracture itself. Type 2 outcomes are adverse outcomes that are related to treatment. Type 3 outcomes result from interactions between the fracture, its treatment, and subsequent growth and development. When possible, nonoperative management is preferred to reduce type 2 and type 3 adverse outcomes.

I. Rehabilitation is a system of medical, psychological, pedagogical, and socio-economic measures aimed at helping the person return to his previous level of functioning. Maxillofacial injuries increase chronic disability, which leads to a significant decrease in the quality of life manifested in the limitation of communication and the impossibility of full participation in social life.

Inpatient management is required for those with associated severe trauma. The goal of inpatient rehabilitation is to prevent immediate post-traumatic complications of inflammatory origin. The management of rehabilitation for the acute stage is individualized to the person's particular pattern of deficits and includes: 1) adequate pain relief through local anesthesia immediately after the accident and the temporary immobilization of the fractures during the transportation to the hospital; 2) immediate reduction and immobilization of the trauma with single arch bars, splints, or interdental wire ligatures, as well as a Barton's bandage, in cases where definitive treatment in a specialized service will be delayed due to the general condition (shock, hemorrhage, neuro-cranial trauma); 3) suturing the wounds on the oral mucosa to separate the oral cavity from the bone fractures; 4) final fixation of the fragments as soon as possible, using simple methods that will not further injure the bone tissue and growth areas and will not cause innervation and vascularization disorders (mouthguards, monomaxillary or bimaxillary wires, mento-cephalic bandages, osteosynthesis with metal or resorbable stabilizing miniplates); 5) removal of primary and permanent teeth with pulp necrosis from the fracture line, teeth with chronic periapical abscesses, oral hygiene, drainage to prevent hematomas, antibiotic prophylaxis;
6) restoration of innervation and blood circulation, repair of the parotid duct and facial nerve integrity; 7) diet therapy. The doctor must not miss injuries by re-evaluating patients 24 hours after trauma by means of an anamnesis and physical examination.

II. Growth disturbances and development of the face and mandible after craniofacial trauma. In the pediatric population, injuries typically heal with significant improvement and function. The cause of actual growth disturbances remains unclear, but certain children may have lost growth stimuli or suffer from decreased regional vascularity, resulting in growth restriction. Thus, the immediate satisfactory results may be unstable. With the development of the mandible and teeth, due to eruption and delayed physiological change, partial or total disorders in the development of the mandible can occur. Given the concerns about these issues, close postoperative and long-term follow-up are recommended.

The prophylaxis of facial growth disorders and symmetric development of the mandible include:

1) Early reduction and fixation of craniofacial bone fractures, re-establishing preinjury occlusion, and the integrity and anatomical shape of the mandible and craniofacial bones by surgical or orthodontic means.

Closed postoperative follow-up includes an active period of maintaining maxillomandibular fixation or internal rigid fixation. If a patient is left in maxillomandibular fixation once discharged from the hospital, parents or caregivers should have the wire cutters on hand at all times. During all periods, the mouth should be copiously irrigated by a caregiver, parents, or dentist. The child is monitored once every 7 days after discharge from the hospital in an outpatient setting. Oral intake for patients may be accomplished with the passage of a straw through the gap between teeth if present or with a right-angle straw passed behind the maxillary tuberosity. During the period of maxilla-mandibular fixation, the child can lose weight (2,27-4,53kg). Calorie-dense foods such as milkshakes or protein shakes are recommended to allow for weight maintenance and adequate nutrition for healing at the fracture site. After a period of maxillomandibular fixation, the arch bars and wires may be removed under short-acting sedation in the operating room or general anesthesia.

A. Although the injury typically heals with significant improvement and function, the patient and their parents must be conscious of the possibility of long-term growth restriction. Abnormal growth results in facial asymmetry and deviation of the chin and may not become apparent for several years. A long-term follow-up twice a year is mandatory to detect these disorders as soon as possible. If growth or developmental disturbances of the face or the mandibular shape and occlusion are determined, orthodontic treatment should be initiated immediately. B. Malunion and nonunion often require orthodontic therapy and additional surgical treatment. Sometimes, immediately after removing arch bars and wires, additional intervention may be required, ranging from fat grafting to orthodontic therapy to combined orthodontic-orthognathic surgery. C. The duration of the orthodontic treatment depends on the character of the mandibular deformities and the occlusion disorders. After rehabilitation of the occlusion and the anatomical shape of the mandible during the primary dentition, the orthodontic treatment will be completed, but the patients will be followed up until the permanent teeth develop. The need for follow-up care and repeat orthodontic treatment is determined during the treatment stages. D. All patients who have suffered trauma to the jaws during the primary or mixed stage should be examined once or twice a year, up to the age of 15 years.

2) Patients with persistent malocclusion after unilateral or bilateral condylar fractures treated with maxillomandibular fixation can be further treated nonsurgically. Some types of functional therapy are recommended to address the abnormal occlusion relationship. This functional therapy can be as simple as using elastics in conjunction with orthodontic appliances or occlusal splints, or it may require a formal functional appliance. Functional appliances seek to mechanically reposition the jaw into proper occlusion and promote proper mandibular function. Maintaining an appropriate range of motion at the TMJ is important for maintaining proper mandibular growth, as well as avoiding ankylosis and TMJ dysfunction.

3) Preservation of dental buds if they are located in the fracture line. Dental buds are removed only 3-4 months after the trauma occurs, following a long persistent period of purulent process in the fracture area (due to dental bud necrosis) and radiological confirmation of their sequestrum.

III. Intraosseous tooth buds and erupting teeth in the fracture line can be traumatized during reduction or placement of the screw and plate. The prophylaxis of the disorders of development and eruption of the permanent dental buds includes: 1) the reduction of fragments and the restoration of the mandibular integrity; 2) infection prevention and control; 3) orthodontic treatment in the post-traumatic period of children with disorders of eruption and dental position; 4) remineralization of tooth crowns by local and general remedies; 5) vital tests; 6) X-ray examination. Patients require long-term follow-up until the permanent teeth develop.

Learning objectives

1. Age periods and the most common regions of facial bone lesions.
2. Etiology of mandibular fractures in children.
3. Anatomo-physiological peculiarities of the oro-maxillofacial area in children and clinical manifestations of the jaw lesions.
4. Clinical picture of mandibular fractures in children.
5. Peculiarities of the treatment of mandibular fractures according to the patient's age.
6. Principles of follow-up of children with mandibular fractures.
7. Clinical and radiological peculiarities of jaw fractures in children.
8. Principles of treatment of children with jaw fractures accompanied by associated trauma.

TESTS

1. MC. What types of late complicationscan occur as a result of alveolar process trauma

A. deforming osteoarthrosis;
B. pseudoarthrosis;
C. microgenia;
D. eruption anomalies;
E. dental position anomalies.

(D, E)

2. MC. What types of late complications can occur in children as a result of the trauma to the tooth-bearing portion of the jaw:

A. deforming osteoarthritis;
B. unilateral microgenia;
C. anomalies of dental positions in the fracture line;

D. eruption anomalies;
E. pseudoarthrosis.

(C, D)

3. SC. Which diagnostic method should be usedto make the diagnosis of the „greenstick” fracture of the mandible:
A. inspection of the teeth;
B. blood test;
C. X-ray of the jaw;
D. bimanual palpation;
E. inspection of the occlusion.

(C)

4. MC. Which of the answers below are characteristic of subperiosteal fractures of the lower jaw:
A. swelling of the alveolar process;
B. traumatic osteolysis;
C. interrupted continuity of the mandibular bone;
D. preserving the periosteum integrity;
E. interrupted continuity of the periosteum and cortical plate.

(C, D)

5. SC. Name the mandibular fractures in which the integrity of the periosteum is preserved:
A. „greenstick”;
B. subperiosteal;
C. mandibular fracture with displacement;
D. condylar dislocation;
E. periosteal contusion.

(B)

6. SC. „Greenstick” fracturesare located in:
A. the mental area;
B. the mandibular body;
C. the coronoid apophysis;
D. the mandibular angle;
E. the condylar apophysis.

(E)

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9. DENTAL TRAUMA IN PRIMARY AND PERMANENT DENTITION

9.1. Peculiarities of dental trauma in children. Etiology. Classification

Dental trauma is an impact injury to the teeth and other hard and soft tissue within and around the oral cavity. The prevalence of dental trauma varies between different countries, age groups, genders, socioeconomic environments, etc. One third of all preschool children suffer traumatic dental injuries involving the primary dentition, and one fourth of all schoolchildren and almost one third of adults suffer trauma to the permanent dentition. Traumatic dental injuries, or tooth trauma, have a global prevalence of 10-15%. This can occur in isolation or be associated with facial and bodily injuries. Trauma to the body, especially to the oral cavity, is a significant public health problem worldwide. In certain groups, dental trauma is almost as common as one fifth of all bodily injuries. Acute and chronic dental trauma are identified based on clinical appearance. Acute trauma is typically sudden, circumstantial, and unexpected, necessitating immediate medical attention. Chronic ones appear after a long period of force overloading on the tooth. Every third child is exposed to dental trauma. In secondary anodontia, traumatic dental injuries occupy second place after caries. In boys, the peak incidence is at 2-3.5 years and 10-12 years; in girls, it is at1-4 years. In 60% of cases, injuries occur in the autumn and winter. Traumatic dental injuries to the maxilla occur three times more often than those to the mandible.

The majority of dental trauma in permanent and primary dentition involve the anterior teeth of the upper jaw especially the maxillary central (66,7%) and lateral incisors (17,4%). Traumatic dental injuries affect a single tooth, but certain trauma events, such as sports, violence, and road traffic accidents, result in multiple injuries.

Enamel fractures and injuries involving dentine are the most common type of dental trauma in permanent dentition (42.5%). Different types of luxation injuries are most common in the primary dentition (subluxation 38.6%, lateral luxation 22.5%, and avulsion 16.6%). Boys are subjected to traumatic injuries at least twice as often as girls (1.5:1.0; 2.5:1.0) because they are more energetic and braver.

Etiology of dental trauma. Dental injuries can result from either a direct or indirect impact. The extent of the damage is related to such factors as the energy of the impact, the resilience and shape of the impacting object, the direction of the impact, and the reaction of the surrounding tissues of the tooth. The etiological factors are varied and change their appearance according to age (toddlers, children, adolescents, and young adults). The patient's home is the main location for sustaining dental trauma for both primary and permanent dentitions. This is followed by injuries at school. Falls are the most common activity causing trauma and account for the most injuries in the primary dentition. Children with a low socioeconomic background are more likely to have traumatic dental injuries, while children from higher socioeconomic backgrounds have a higher risk for dental trauma due to their greater access to leisure activities.

The child has a more inactive life for up to 12 months and sustains injuries from involuntary falls. Craniofacial traumas are quite rare at this age and are usually caused by falling from the caregivers` arms or from the cradle. For toddlers, as part of their development, it is typical to crawl, stagger, and fall before being able to walk. Typically, soft tissue concussion with facial or dental-maxillary hematomas can be detected on the first examination. After such small lesions on the skin or buccal mucosa, some consequences, such as different dental and alveolar anomalies, occur after 2-3 years or 7-8 years. Sometimes it can lead to eruption disorders in both primary and permanent teeth. Eruption of the permanent teeth in the nostrils or in the maxillary sinuscan be observed as a result of the displacement of the permanent tooth buds. The traumatic lesions of the growth center of the upper jaw in this period may be the cause of malocclusion disorders during the growth and development period.

Trauma in preschoolers. The most common age group in which primary tooth injuries occur is 1 to 3 years of age. Traumatic dental injuries occur most frequently as a result of accidents in the home. Falls and collisions are common causes of trauma and account for most injuries in the primary dentition. After 12 months, children develop walking skills with less control over motor coordination, making them more vulnerable to falls. Dental trauma in children under 6 years of age is due to the increase in independent movements and can be related to such factors as assault, fights, impact injuries, collisions, animal injuries, and being hit by something.

Domestic violence and child abuse by parents or caregivers pose a significant risk of injury. Missing teeth in the primary dentition are clinical signs of physical abuse, along with unexplained injuries, bruises, hematomas, and excoriations on the face and body. Dentists can be the first ones to detect children who have suffered physical abuse. If violence is suspected, the child's doctor or health care provider, a local child protective agency, and the police department should be contacted.

The period from 6 to 12 years of age is an active period when children want to showtheir personality. Multiple etiological factors are associated with dental trauma. Accidents during games and sports injuries are common in children aged 7 to 15 years. Falls are the main cause of dental trauma (31.7%-64.2%). Sport activities are one of the main causes of dental trauma (up to 40.2%). They are classified according to the risk groups: high-risk (football, ice hockey, martial arts sports, lacrosse, rugby, and skating) and medium-risk (basketball, team handball, diving, squash, gymnastics, parachuting, and water polo). Bicycling accidents are a common cause of dental trauma in school-children (up to 19.5%). Dental trauma following road traffic accidents is a special injury due to the predominance of multiple injuries (7.8%). Other types of facial trauma are caused by airbag explosions in cars during traffic accidents.

Piercing of the tongue and lips is quite a new group of traumatic dental injuries (19.2%). This may lead to cracked tooth syndrome.

There are certain factors important for determining the type and severity of injury, its emergency and comprehensive management, the sequelae, and the prognosis. These include the child's age, the stage of the tooth development, the direction and intensity of force, the size and shape of the impacting object, the type and timing of emergency dental treatment provided.

Some individual anatomical features serve as predisposing factors to a higher incidence of dental trauma. Children with class II division 1 malocclusion sustain injuries in 70% more cases than children with other types of occlusion. Children with an overjet with a size greater than 3.0 mm are 5.4 times more likelyto present with a dental injury than children with an overjet size lower than 3.0 mm. Inadequate lip coverage makes children 3.4 times more likely to suffer from a traumatic dental injury than children with adequate lip coverage. Factors such as proclined upper anterior teeth, an underlying bone structure that is less mineralized and has low resistance, the phenomenon of formation and resorption of the dental root, the presence of chronic apical infection, and repeated trauma episodes make children more susceptible to dental trauma.

Classification of traumatic dental-periodontal lesions

According to WHO 1994, there are two main groups of traumatic dental injuries.

I. Injuries to the hard dental tissues. II. Injuries to the periodontal tissues or tooth-supporting structures:

1. Injuries to the hard dental tissues:
   a) Crown infraction:

an incomplete fracture of the enamel without tooth structure loss;

   b) Uncomplicated crown:

crown # with unexposed pulp;

   c) Complicated crown:

crown # with exposed pulp;

   d) Uncomplicated crown-root:

crown # extending below the gum line & involving the tooth root but not exposing the pulp;

   e) Complicated crown-root:

crown # extending below the gum line & involving the tooth root but also exposing the pulp;

2. Root fracture: only the tooth root is involved:

   a) Concussion:

refers to injury to the tooth-supporting structure without abnormal loosening or displacement;

   b) Subluxation:

loose tooth with no rupture of the neurovascular bundle

   c) Intrusive luxation:

the tooth is pushed away from the tooth socket but is not avulsed.

   d) Lateral luxation:

the tooth is pushed either anteriorly-posteriorly or mesially-distally. i.e. sideways of its long axis

   e) Avulsion: the tooth has completely come out of the socket.

9.2. Diagnosis of dental trauma in primary and permanent dentition in children and adolescents

Clinical study of children with traumatic dental injuries. The history of traumatic dental lesions and the clinical examination are the basis of the diagnosis and are fundamental for all dental injuries. The diagnosis should begin with discussions, and it includes personal data such as the child`s name and surname, the place of living and activity (kindergarten, school, etc.), symptoms, life history, dental history, and neurological status.

The child's age when the trauma occurred plays a significant role in determining the treatment management and the final results. The traumas to primary and permanent teeth during the period of root formation appear as complete and incomplete dislocations. Crown fractures are caused by trauma that occurs when the apex is closed.

Anamnesis specifies the disorders of the organs and systems, as well as whether the child is under the supervision and treatment of other specialists. Medical care (specialized outpatient or inpatient care) and some treatment changes are determined based on these data. Children with affected cardiovascular, respiratory, and immune systems require antibacterial treatment, while those with allergic reactionsneed corticosteroids and desensitizers.

Traumatic craniocerebral injuries (whether or not associated with consciousness) are common in maxillofacial and dental trauma in infants. The neurological status of children with trauma will be determined immediately after the accident. Clinical signs of suspected craniocerebral injury are irritability, lethargy, instability during walking, decreased consciousness or confusion, coloration of the skin, blurred speech, nausea or vomiting, headache, bleeding or fluid leakage from the external auditory duct, asymmetrical pupil reaction, and non-uniform breathing.

Examination of the patient with dental trauma. The record of the present illness of the dental trauma is the first step of examination. Environmental details of the accident should guide the evaluation of the extent of trauma. When, where, and how are important, especially in avulsion and displacement injuries. Also, treatment delays may signal possible child abuse. To establish a correct diagnosis and rational treatment plan, it is important to realize how much time has passed from the accident to primary care. The shorter the period, the greater the chance of tooth survival. Often, acute traumatic injuries are overlooked, contaminated from the impact, and covered with blood and debris. These injuries must be cleaned with a mild detergent prior to examination, and antibiotics must be administered. Previous injuries to the teeth can affect treatment options. Some children are accident-prone, and those who participate in various sports frequently show radiographic evidence of previous trauma. A change in the bite and increased sensitivity to temperature following an injury would indicate possible tooth luxation, an alveolar fracture, or a crown lesion exposing the dentin. It is essential to establish a pertinent medical history (allergies, blood disorders, or other conditions may influence the management plan and treatment outcomes).

Extraoral and intraoral examinations are part of the clinical examination. The diagnosis is based on the local and general clinical examinations (extraoral and intraoral). The extraoral examination will detect changes in the soft tissues (asymmetry, swelling, excoriation, bruises, and wounds). It will be determined by soft tissue wounds, the location and relationship with the facial orifices, the direction, length, and depth of the wound, the presence of foreign bodies (strained teeth, root residues), injury to the blood vessels and peripheral nerves, and hemorrhages.

Intraoral inspection includes examination of soft tissues, bones, and dental tissue. The examination of the intraoral soft tissues will aim at detecting the lesions on the mucosa of the alveolar process. The mobility of the tongue will be checked by keeping the tongue up and extending it laterally.

Dental movements in the three planes (sagittal, horizontal, vertical), dental mobility, affected teeth, and the condition of apical tissues must be determined. The horizontal or vertical mobility can be identified by a slight palpation of the lingual and vestibular surfaces of the teeth, with the help of the polices or the dental mirrors. The degree of mobility should be recorded, thus: 0=no loosening or changes, 1 = horizontal loosening, ≤ 1mm, 2 = horizontal loosening > 1mm, 3 = axial (vertical) loosening. The type of luxation can be related to the degree of mobility. The mobility of two or three teeth indicates an alveolar fracture.

The percussion test can provide information about the relationship between the tooth and adjacent bone. Tenderness when touching a tooth indicates damage to the periodontal dental ligaments. A higher metallic tone indicates lateral or intrusive displacement. The follow-up examination, such as the percussion tone, indicates tooth ankylosis.

Radiological examination during the pretreatment period can determine the stage of root formation, the presence of root fractures and their location, periapical changes, the degree of intrusion and extrusion, and the ratio of damaged primary teeth to permanent dental buds. During the post-treatment period, the radiological examination must be performed every 3 months for 12 months, once every 6 months for 2 years, and once every 12 months for 3 years to obtain information on root formation, the development of the dental buds, and the periapical changes occurring during the regeneration period.

Pulp sensibility is currently the most useful test to assess the neurovascular supply to the pulp of a traumatized tooth. The pulp vitality test is usually performed during the post-treatment period, every 3 months for 12 months, in order to obtain detailed information on the evolution of traumatic dental injuries. The pulp vitality test can be carried out by three methods: thermal, electrical, and mechanical.

The pulp vitality test using the thermal method is performed by the action of ethyl chloride, ice, carbon dioxide, or a burnt gutta-percha cone on the dental crown for 2 seconds. The mechanical method includes drilling the dental crown, during which pain sensations can appear. It is a traumatic method and is rarely used because, in some cases, pain sensations occur only near the neurovascular bundle.

In some cases, however, the lack of cooperation of children makes these methods ineffective for proving pulp necrosis. Developing or mature teeth do not respond and do not give informative data on the neurovascular pulp supply. The response at the first examination of the injury does not provide a good baseline to compare it to at a subsequent follow-up examination. Therefore, the pulp vitality test in children is carried out to determine clinically the changes in the dental crown color, the diminished enamel gloss, and radiologically the cessation of the root development, comparing the teeth with the similar teeth on the opposite side.

9.3. Treatment concepts

The goal of treatment of traumatic dental injuriesis to return the teeth to acceptable function and appearance according to the type of injury and stage of development. A normal function requires teeth repositioning, while an acceptable appearance necessitates repair and restoration of the dental fracture and surrounding tissue. Restoring the function and cosmetic outcomes of the teeth cannot be achieved within the first few hours after trauma. The best outcome can be achieved if various treatment priorities are considered and the suitable treatment is selected according to the type of trauma. Thus, an acceptable guideline should be recommended for treatment priorities.

Acute treatment is indicated for cases where treatment within a few hours can significantly affect the outcome. Such cases include tooth avulsions, alveolar fractures, extrusive and lateral luxations, and possibly root fractures. Early repositioning and stabilization will promote the best dental-periodontal repair. Treatment of acute trauma will be initiated in the emergency department and includes pulp protection for continued root formation in developing teeth with complicated crown fractures. Luxation and avulsion injuries require reduction and replacement in order to stabilize the tooth in its normal position and allow re-attachment and re-organization of the periodontal ligament support.

Subacute treatment is performed within 24 hours after trauma and includes proper care for concussions, subluxations, intrusive luxations, crown fractures with pulp exposure, and dental trauma. The goal of treatment is the management of pulp trauma, and every effort must be made to protect vitality and allow pulp revascularization inimmature, developing teeth.

Delayed treatment: Crown fractures without pulp exposure appear to have the same prognosis whether treatment is performed within a few or several hours. This is caused by coagulation-type necrosis of the pulp, due to the severance of the apical blood supply and the invasion of bacteria into the necrotic pulp tissue that stimulates infection-related resorption.

9.4. Management of tooth fractures

An infraction is an incomplete fracture (crack) of the enamel without any loss of tooth structure.

Diagnosis: normal gross anatomic and radiographic appearance, apparent crack line, especially with transillumination.

Treatment objectives: to maintain structural integrity and pulp vitality

Prognosis: Complications are uncommon.

Uncomplicated crown fractures refer to enamel fractures or enamel-dentin fractures that do not involve the pulp. Crown fractures of the permanent teeth occur in 67% of cases.

Diagnosis: clinical and/or radiographic findings reveal a loss of tooth structure confined to the enamel or to both the enamel and dentin.

Treatment objectives*:* In mature teeth, maintaining pulp vitality and restoring normal esthetics and function can provide a good prognosis. In developing teeth, there must be more concern over the risk of bacterial contamination involving the exposed dentin with larger diameter tubules. Injured lips, tongue, and gingiva should be examined for tooth fragments. When looking for fragments in soft tissue lacerations, radiographs are recommended. For small fractures, rough margins and edges must be smoothed. For larger fractures, the lost tooth structure must be restored.

Prognosis: The prognosis of uncomplicated crown fractures depends primarily upon the concomitant injury to the periodontal ligament and secondarily upon the extent of exposed dentin. Optimal treatment results follow timely assessment and care.

Complicated crown fractures refer to enamel-dentin fractures with pulp exposure. Crown (6.5%) and root (0.5%) fractures are uncommon in primary teeth.

Diagnosis: Clinical and radiographic findings reveal tooth structure loss with pulp exposure.

Treatment objectives: to maintain pulp vitality and restore normal esthetics and function. Injured lips, tongue, and gingiva should be examined for tooth fragments. When looking for fragments in soft tissue lacerations, radiographs are recommended.

- Primary teeth: Decisions often are based on the life expectancy of the traumatized primary tooth and pulp vitality. Pulp therapy alternatives are pulpotomy, pulpectomy, and extraction.
- Permanent teeth: Fully developed teeth require a prosthetic crown, thus the patient may wisely choose to have root canal treatment prior to restoration. The pulp therapy alternatives recommended for developing teeth to allow continued root formation are direct pulp capping and partial pulpotomy. There is increasing evidence to suggest

that conservative vital pulp therapy for mature teeth with closed apices is as appropriate a management technique as when used for immature teeth with open apices.

Prognosis*:* The prognosis of crown fractures appears to depend primarily on a concomitant injury to the periodontal ligament. The age of pulp exposure, the extent of dentin exposure, and the stage of root development at the time of injury secondarilyaffect the tooth prognosis. Optimal treatment results follow timely assessment and care.

Crown or root fractures refer to enamel, dentin, and cementum fractures with or without pulp exposure.

Diagnosis: Clinical findings usually reveal a mobile coronal fragment attached to the gingiva with or without pulp exposure. Radiographic findings may reveal a radiolucent oblique line that comprises crown and root in a vertical direction in primary teeth and in a perpendicular direction to the central radiographic beam in permanent teeth. While radiographic demonstration is often difficult, root fractures can only be diagnosed radiographically.

Treatment objectives*:* to maintain pulp vitality and restore normal esthetics and function.

- Primary teeth: When the primary tooth cannot be restored, the entire tooth should be removed unless retrieval of apical fragments may result in damage to the succedaneous tooth.
- Permanent teeth: The emergency treatment objective is to stabilize the coronal fragment. Definitive treatment alternatives are as follows: removal of the coronal fragment followed by supragingival restoration or gingivectomy, osteotomy, or extrusion to prepare for restoration. If the pulp is exposed, pulp therapy alternativesinclude pulp capping, pulpotomy, and root canal treatment. In fully developed teeth, all of the procedures are associated with root canal therapy.

Prognosis: Although the treatment of crown-root fractures can be complex and laborious, most fractured permanent teeth can be saved. Fractures extending significantly below the gingival margin may not be restorable.

Root fractures involve the pulp, dentin, cementum and periodontal ligaments.

Diagnosis: Clinical findings reveal a mobile coronal fragment attached to the gingiva that may be displaced. Radiographic findings may reveal one or more radiolucent lines that separate the tooth fragments in horizontal fractures. A root fracture in a primary tooth may be obscured by a succedaneous tooth.

Treatment objectives*:*

- Primary teeth: Treatment alternatives include extraction of coronal fragments without insisting on removing apical fragments or observation. It is not recommended to reposition and stabilize the coronal fragment.
- Permanent teeth: The coronal fragment must be repositioned and stabilized in its correct anatomical position as soon as possible to optimize healing of the periodontal ligament and neurovascular supply while maintaining the esthetic and functional integrity.

Prognosis*:* Pulp necrosis in root-fractured teeth is attributed to displacement of the coronal fragment and mature root development. In permanent teeth, the location of the root fracture has not been shown to affect pulp survival after injury. Therefore, preservation of teeth with root fractures occurring in the cervical third of the tooth should be attempted. Young age, immature root formation, positive pulp sensitivity at the time of injury, and approximating the dislocation within 1 mm have been found to be advantageous to both pulpal healing and hard tissue repair of the fracture.

Concussion is the most common traumatic lesion of both primary teeth (2.5%) and permanent teeth (1%). It is an injury to the tooth-supporting structures that does not result in abnormal loosening or displacement of the tooth.

Diagnosis: Because the periodontal ligament absorbs the injury and is inflamed, clinical findings reveal a tooth that is tender to pressure and percussion without mobility, displacement, or sulcular bleeding. Radiographic abnormalities are not expected.

Treatment objectives: To optimize healing of the periodontal ligament and maintain pulp vitality, patients with minor involvementrequire a soft diet, occlusal adjustment, and observation for delayed pulp necrosis.

Prognosis: For primary teeth, unless an associated infection exists, no pulp therapy is indicated. Although there is a minimal risk for pulp necrosis, mature permanent teeth with closed apices may undergo pulp necrosis due to associated injuries to the blood vessels at the apex and, therefore, must be examined carefully.

Subluxation includes injury to tooth-supporting structures with abnormal loosening but without tooth displacement. Incomplete luxations are found in 58% of cases in the primary dentition and in 18% of cases in the permanent dentition.

Diagnosis: Because the periodontal ligament attempts to absorb the injury, clinical findings reveal a mobile tooth without displacement that may or may not have sulcular bleeding. Radiographic abnormalities are not expected.

Treatment objectives*:* to optimize healing of the periodontal ligament and neurovascular supply.

- Primary teeth: The tooth should be examined for pulp necrosis.
- Permanent teeth should be stabilized and relieved of any occlusal interferences. For comfort, a flexible splint can be used for no more than 2 weeks.

Prognosis: The prognosis is usually favorable. The primary tooth should return to normal within 2 weeks. Permanent teeth with closed apices may undergo pulp necrosis due to associated injuries to the blood vessels at the apex and, therefore, must be followed carefully.

Lateral luxation refers to a displacement of the tooth in a direction other than axially. The periodontal ligament is torn, and a contusion or fracture of the supporting alveolar bone occurs. The luxation of permanent teeth occurs in 18.3% of cases. Clinically, pain is caused by touching the tooth or chewing. Gum is more or less displaced.

Diagnosis: Clinical findings reveal that a tooth is displaced laterally, in the palatal or lingual direction, and may be locked firmly into the new position. The tooth is usually not mobile or tender to touch. Being painful, the child avoids touching it; sometimes it makes them change the position of the mandible. Bleeding, salivation, and disorders in mastication and phonation occur. Radiographic findings reveal an increase in periodontal ligament space and displacement of the apex toward or through the labial bone plate.

Treatment objectives:

- Primary teeth: to allow passive or spontaneous repositioning if there is no occlusal interference. When there is occlusal interference, the tooth can be gently repositioned or slightly reduced if the interference is minor. When the injury is severe or the tooth is nearing exfoliation, extraction is the treatment of choice.
- Permanent teeth: to reposition as soon as possible and then stabilize the tooth in its correct anatomical position to optimize healing of the periodontal ligament and neurovascular supply while maintaining the esthetic and functional integrity. The tooth is repositioned with digital pressure and little force. A displaced tooth may need to be extruded to free itself from the apical lock in the cortical bone plate. Splinting for an additional 2 to 4 weeks may be needed with the breakdown of the marginal bone. Developing teeth with immature roots and open apices (>0,5mm in diameter) have the potential for revascularization, which will allowfor continued root development. Pulp necrosis in young, developing teeth has been managed by the endodontic technique of apexification.
- Prognosis: Primary teeth requiring repositioning have an increased risk of developing pulp necrosis compared to teeth that are left to spontaneously reposition. In mature permanent teeth with closed apices, pulp necrosis and pulp canal obliteration are common healing complications, while progressive root resorption is less likely to occur.

Intrusion is an apical displacement of the tooth into the alveolar bone, and damage occurs to the cementum and the periodontal ligaments;

the neurovascular pulp supply is crushed. The tooth is driven into the socket, compressing the periodontal ligament, which commonly causes a crushing fracture of the alveolar socket. Intrusion occurs in 21.9% of cases in the primary dentition.

Diagnosis: Clinical findings reveal that the tooth appears to be shortened (*figure 13*) or, in severe cases, it may appear missing. The tooth apex is usually displaced labially toward or through the labial bone plate in primary teeth and driven into the alveolar process in permanent teeth. The tooth is not mobile or tender to touch. Radiographic findings reveal that the tooth appears displaced apically and the periodontal ligament space is not continuous. Determination of the relationship of an intruded primary tooth with the follicle of the succedaneous tooth is mandatory. If the apex is displaced labially, the apical tip can be seen radiographically, with the tooth appearing shorter than its contralateral one. If the apex is displaced palatally towards the permanent tooth germ, the apical tip cannot be seen on the X-ray and the tooth appears elongated. An extraoral lateral radiograph can also be used to detect the displacement of the apex toward or through the labial bone plate. An intruded young permanent tooth may mimic an erupting tooth.

Fig. 13. **Dental trauma with intrusion.**

Treatment objectives:

• Primary teeth: to allow spontaneous re-eruption except when displaced into the developing successor (soft tissue, maxillary sinus, or jaw bone). Extraction is indicated when the apex is displaced toward the permanent tooth germ. In very young, developing teeth, re-eruption can occur, and for this reason, in such young patients, it is prudent to observe for spontaneous re-eruption before initiating active treatment.

• Permanent teeth: to reposition passively (allowing re-eruption to its pre-injury position), actively (repositioning with traction), or surgically, and then to stabilize the tooth with a splint for up to 4 weeks in its correct anatomical position to optimize healing of the periodontal ligament and neurovascular supply while maintaining esthetic and functional integrity. The goal for immature teeth with higher eruptive potential (½ to ⅔ of the root formed) is to allow for spontaneous eruption. In mature teeth, the goal is to reposition the tooth with orthodontic or surgical extrusion and initiate endodontic treatment within the first 3 weeks after trauma.

Prognosis: In primary teeth, 90% of intruded teeth re-erupt spontaneously (either partially or completely) in 2 to 6 months. Ankylosis may occur if the periodontal ligament of the affected tooth has been severely damaged, thereby delaying or altering the eruption of the permanent successor. There is a high risk of pulp necrosis, pulp canal obliteration, and progressive root resorption in mature permanent teeth with closed apices. Immature permanent teeth that are allowed to reposition spontaneously demonstrate the lowest risk for healing complications. The extent of intrusion (7 mm or greater) and adjacent intruded teeth have a negative influence on healing.

Extrusion is the partial displacement of a tooth axially from its socket (*figure 14*); partial avulsion. The periodontal ligament is usually torn.

Diagnosis: Clinical findings reveal that the tooth appears elongated and is mobile. Radiographic findings reveal an increased periodontal ligament space apically.

Fig. 14. **Dental trauma with displacement of the teeth. Lip laceration**

Treatment objectives:

- Primary teeth: to allow teeth to reposition spontaneously or reposition and allow for healing for minor extrusion (<3 mm) in an immature developing tooth. Indications for an extraction include severe extrusion or mobility, the tooth is nearing exfoliation, the child's inability to cope with the emergency situation, or the tooth is fully formed.
- Permanent teeth: to reposition as soon as possible and then stabilize the tooth in its correct anatomical position, to optimize healing of the periodontal ligament and neurovascular supply while maintaining esthetic and functional integrity. Repositioning may be accomplished with slow and steady apical pressure to gradually displace the coagulum formed between the root apex and the socket floor. A splint is applied for up to 2 weeks.

Prognosis: There is a lack of clinical studies evaluating the repositioning of extruded primary teeth. There is a high risk of pulp necrosis and pulp canal obliteration in permanent, mature teeth with closed apices. These teeth must be followed up carefully.

Avulsion is the complete displacement of a tooth out of its socket. The periodontal ligament is severed, and an alveolar fracture may occur. Avulsion in the primary dentition occurs in 10.6% of cases; in the permanent dentition, it accounts for 6.9% of cases and occurs more frequently inchildren aged 7 to 12 years.

Diagnosis: Clinical and radiographic findings reveal that the tooth is not present in the socket. Radiographic assessment will verify that the tooth is not intruded when the tooth has not been found.

Treatment objectives:

- Primary teeth: to prevent further injury to the developing successor. Avulsed primary teeth should not be replanted because of the potential for subsequent damage to developing permanent tooth germs.
- Permanent teeth should be replanted as soon as possible and then stabilized in their correct anatomical location to optimize healing of the periodontal ligament and neurovascular supply while maintaining esthetic and functional integrity, unless replanting is contraindicated by:

1. the child’s stage of dental development (risk for ankylosis, where considerable alveolar growth has to take place);
2. compromising medical condition;
3. compromised integrity of the avulsed tooth or supporting tissues.

Flexible splints must be applied for 2 weeks. Tetanus prophylaxis and antibiotic coverage should be considered. Treatment strategies are directed at avoiding inflammation that may occur as a result of the tooth attachment damage and pulp infection.

Prognosis: In the permanent dentition, the prognosis is primarily dependent upon root development and extraoral dry time. The tooth has the best prognosis if it is replanted immediately. If the tooth cannot be replanted within 5 minutes, it should be stored in a medium that will help maintain the vitality of the periodontal ligament fibers. The best transportation medium for avulsed teeth is a non-physiologic medium such as saliva, physiological saline, doxycycline solution, milk, or tap water. Although water is detrimental to cell viability due to its low osmolality and long-term storage (i.e., more than 20 minutes) in water has an adverse effect on periodontal ligament healing, it is a better choice than dry storage. Limited tooth storage in a cell-compatible medium prior to replantation can produce similar healing results as compared with immediately replanted teeth. The risk of ankylosis increases significantly with an extraoral dry time of 20 minutes. An extraoral dry time of 60 minutes is considered the point where survival of the root periodontal cells is unlikely. There is a high risk of pulp necrosis, root resorption, and ankylosis of avulsed permanent teeth.

Revascularization: An immature (open apex) tooth has the potential to establish revascularization when there is a minimum of1.0 mm of apical opening. Complete pulp revascularization occurs in 18% of immature teeth. Antibiotic treatment reduces contamination of the root surface and pulp space, thereby creating a biological environment that aids revascularization. A mature tooth (closed apex or apical opening <1 mm), on the other hand, has little to no chance of revascularization.

When a tooth has been out of the oral cavity and in a dry environment for more than 60 minutes, the periodontal ligaments have no chance of survival. If such a tooth is replanted, it will undergo osseous replacement resorption and, overtime, the tooth will become ankylosed and ultimately will be lost. Given the child's growth and development, the goal for a tooth that has been avulsed for more than 60 minutes with dry storage is to delay the osseous replacement and, hence, the ankylotic process as long as possible. To slow down this process, the remaining periodontium should be removed because otherwise it becomes a cause of inflammation that accelerates infection-related resorption. The remaining periodontium can be removed by gentle scaling or by soaking the tooth in 3% citric acid for 3 minutes. This should be followed by a sodium fluoride treatment for 20 minutes. Soaking teeth in fluoride before replantation has been shown to significantly reduce the risk of resorption after a 5-year follow-up. Despite these recommendations, teeth that have been out of the oral cavity for more than 60 minutes with dry storage have a poor prognosis and will not survive long term.

Possible contraindications to replantation are immunecompromised health, severe congenital cardiac anomalies, severe uncontrolled seizure disorders, severe mental disability, severe uncontrolled diabetes, and a lack of alveolar integrity.

Orthodontic movement of traumatized teeth. Traumatized teeth must be evaluated carefully prior to beginning or continuing orthodontic movement. Even with simple crown or root fractures without pulp involvement, waiting for 3 months is recommended before tooth movement should begin. Other minor traumas to the tooth and periodontium (e.g., minor concussions, subluxations, and extrusions) also require a 3-month wait. In cases of severe trauma or damage to the periodontium, a minimum of 6 months is recommended to wait. Teeth that have sustained root fractures cannot be moved for at least a year. If there is radiographic evidence of healing, the teeth can be successfully moved. In teeth that require endodontic treatment, movement can begin once healing is evident. Because teeth that have sustained severe periodontal injury can undergo pulp necrosis when orthodontic movement is initiated even after a rest period, light intermittent forces are recommended along with avoidance of prolonged tipping forces and contact with the buccal or lingual cortical plates.

9.5. Consequences of trauma in the primary dentition

1. Loss of space on the gum, inclusion of permanent teeth, defects in the permanent tooth structure, ankylosis, early eruption of permanent teeth, persistence of the milk tooth (through fibrosis) with the permanent tooth inclusion, delayed crown formation, delayed root formation, permanent tooth position abnormalities.
2. Tooth discoloration (the yellow color of the tooth) that appears a few months after the trauma. It denotes a deposition through which the pulp chamber and root canal decrease in volume. It does not disturb the process of physiological resorption.
3. The gray-blue color of the dental crown is a sign of pulp necrosis, requiring root treatment or extraction.
4. Apical cysts can affect the crown of erupting permanent teeth. Teeth with an apical cyst should be removed with the offending primary teeth.
5. Disorder of the resorption of primary tooth roots. In some cases, the resorption of the root is accelerated, and in others, the resorption is stopped. Permanent dental buds can erupt with a single stain of the crown to extend hypoplasia of the enamel and dentin. Arrest of the root formation of permanent teeth can appear after trauma to the permanent ones.

Follow-up evaluation: The management of dental injuries includes follow-up examinations to complete or confirm the diagnosis, assess the response to treatment, determine the need for additional treatment or treatment change, and evaluate the treatment outcome or complications. 1 week - after 7-10 days, the splint placed on a replanted, avulsed tooth should be removed, and endodontic treatment, if indicated, should be initiated. 3-4 weeks - the splint applied to luxated teeth can usually be removed after 3-4 weeks. A radiographic examination should be performed to look for the possible beginning of root resorption or periradicular lesions. 6 weeks. - at this time, clinical and radiographic examinations may reveal evidence of pulp necrosis and infection-related root resorption. 3-6 months. - examination may be necessary to establish a definitive diagnosis of pulpal and periodontal healing complications. 1 year and up to 5 years - one year is a minimal follow-up period for traumatic dental injuries; some may require additional observation periods to assess the final outcome.

Learning objectives

1. Etiology of dental lesions in children.
2. Classification of acute lesions of the primary and permanent dentition.
3. Peculiarities of dental trauma in the primary dentition.
4. Consequences of trauma in the primary dentition.
5. Diagnosis of dental lesions in children.
6. Dental inclusion (tooth impaction) in primary teeth and medical care.
7. Radiological image of the dental trauma with displacement in the axis.
8. Crown trauma in permanent dentition. Incidence. Treatment stages.
9. Partial dislocations of permanent teeth and treatment according to root formation.
10. Total dental dislocation. Treatment methods. Consequences. Hospitalization indications.

TESTS

1. MC. The signs of a tooth with dislocation in the axis (slight intrusion) on a radiological image are as follows:

A. incisal margin higher than neighboring teeth;
B. narrow periradicular space;
C. missing periradicular space;
D. interrupted periradicular space;
E. incisal margin lower than neighboring teeth.

(B, C, D, E)

2. MC. The most common dental lesions in the primary dentition are:
   A. Complete dislocations;
   B. Root fractures;
   C. Crown fractures;
   D. Incomplete dislocations;
   E. All answers are correct.

(A, D)

3. MC. Specify the treatment strategy of a temporarily intruded tooth that does not show resorption:
   A. Expecting the re-eruption;
   B. Wound suturing;
   C. Tooth extraction;
   D. Immediate reimplantation;
   E. Immobilization.

(A, B)

4. SC. After a traumatic injury, the milk tooth is removed in cases when:
   A. it is located in the neighboring soft tissues;
   B. it is located in one of the facial cavities;
   C. it contuses the permanent tooth bud;
   D. it injures seriously the alveolar wall;
   E. all answers are correct.

(E)

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