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"SEMIOTICS OF CHILDREN'S DISEASES: SYMPTOMS AND SYNDROMES OF DAMAGE TO ORGANS AND SYSTEMS"

TEXTBOOK

For direction

5510100 - medical business, 5111000 - Vocational education (5510100 - medical business)

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LIST OF CONVENTIONAL ABBREVIATIONS

BP - blood pressure

ADH - antidiuretic hormone

ALT - alanine aminotransferase

ASAT - aspartate aminotransferase ongenital heart disease

HLP - left atrial hypertrophy

RVH - right ventricular hypertrophy

GER - gastroesophageal reflux

GERD - gastroesophageal reflux disease

VSD - ventricular septal defect

ASD - atrial septal defect

RF - respiratory failure

VC - vital capacity of the lungs

GIT - gastrointestinal tract

ZPR - delayed sexual development

CHF - congestive heart failure

ALV - artificial lung ventilation

BMI - body mass index

CPHK - creatinine phosphokenase

LDH - lactate dehydrogenase

NS - nephrotic syndrome

NUC - non-specific ulcerative colitis

PDA - patent ductus arteriosus

ABO - acute bronchial obstruction

AII - acute intestinal infection

ARF - acute renal failure

LSD - lack of sexual development

ARVI **-** is an acute respiratory viral infection

ARI - acute respiratory infection

OSLT - acute stenosing laryngotracheitis

OHP - osteochondropathy

VCB - volume of circulating blood

PPR - precocious puberty

PTH - parathyroid hormone

HF - heart failure

AIS - Adrenal Insufficiency Syndrome

ESR - erythrocyte sedimentation rate

TSH - thyroid stimulating hormone Ultrasound - Ultrasound

FGDS - fibrogastroduodenoscopy

CVP - central venous pressure

CGN - cerebro-pituitary nanism

CNS - central nervous system

HR - heart rate

ECG - electrocardiogram

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This textbook for 3rd year students of the Faculty of Pediatrics -5510200 in the field of education of Health Care - 510000 medical universities, details the amount of theoretical and practical knowledge necessary for mastering the theoretical basis of the subject "Pediatrics", given in block 3.02 of the curriculum, in the process of studying at department of pediatrics. The book contains a lot of illustrative material, including original drawings and photographs, diagrams and tables. The publication is intended for students of medical universities, interns, residents, pediatricians, general practitioners, doctors of other specialties. In order to determine the final level of students' knowledge, the textbook contains situational tasks, questions, tests and a glossary.

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Council Secretary, Associate **N.A. Nasritdinova**

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INTRODUCTION

The doctrine of methods for recognizing diseases is called diagnostics (from the Greek διαγνωστικός - capable of recognizing). Diagnostics is a branch of medical science that outlines research methods for establishing the disease and the patient's condition in order to prescribe the necessary treatment and preventive measures. The result of the diagnostic process is the diagnosis of the disease (from the Greek διάγνωσις - recognition, distinction).

Diagnosis is a brief medical conclusion about the nature of the disease and the patient's condition, expressed in modern medical terms.

Diagnostics as a scientific discipline consists of 3 sections:

1. Medical diagnostics - the study of methods of observation and examination of the patient.

2. Semiotics, or semiology (from the Greek Σημειωτική - sign, sign) - a science that studies the properties of signs and sign systems, in medicine - the study of the signs of a disease) - the study of the diagnostic value of the symptoms of a disease.

3. Methodology of diagnosis - the study of the peculiarities of thinking in recognizing the disease.

Symptom (from the Greek σύμπτωμα - case, coincidence, sign) is a sign of a disease that is detected by a doctor when examining a patient and is used to make a diagnosis and determine the prognosis of diseases.

According to the way they are detected, the symptoms are divided into:

- subjective - symptoms that the doctor learns about from the patient's words during questioning;

- objective - symptoms that the doctor reveals using basic and additional diagnostic methods. Syndrome (from the Greek σύνδρομον, σύνδρομο - on a par, in agreement) - a set of symptoms united by a common pathogenesis and characterizing a certain pathological state of the body.

There are two types of syndromes: anatomical and functional. Syndromic diagnosis is very important, because on the basis of individual syndromes, the

doctor makes the first conclusion about the anatomical and functional state of the internal organs.

SEMIOTICS OF NERVOUS SYSTEM DAMAGE

Damage to the central nervous system can be caused by various reasons: neuroinfections (meningitis, encephalitis, poliomyelitis), toxic effects of various etiologies (viral, bacterial, poisoning, etc.). Coma in children can develop with injuries, diabetes, renal, hepatic and adrenal insufficiency. Asphyxia during childbirth, congenital diseases with the accumulation of substances that have a toxic effect on the central nervous system (for example, phenylketonuria, galactosemia, etc.) can lead to damage to the central nervous system.

In the first year of life in children with CNS lesions, the most common are:

1. **Hyperexcitability syndrome** - the child has tremor of the hands, chin, nystagmus, restlessness, sleep disturbance, increased congenital reflexes, increased reflex excitability. This syndrome is observed in perinatal lesions of the central nervous system, metabolic disorders.

2. **Hypoexcitability syndrome** - the motor and mental activity of the child is reduced, lethargy, lack of flexor position of the limbs are observed. It is observed in premature infants, in children with intracranial trauma, as well as in patients who have undergone hypoxia.

3. **Syndrome of movement disorders** - manifested by a change in muscle tone and reflex activity.

4. **Syndrome of intracranial hypertension** - is manifested by an increase in the size of the head in a child, a divergence of the cranial sutures, an increase and bulging of a large fontanel. The child is excitable, his cry becomes sharp, piercing, his sleep becomes superficial. There is a horizontal nystagmus, a symptom of the "setting sun", converging strabismus. This syndrome is often combined with hydrocephalic syndrome (expansion of the ventricles and subarachnoid spaces).

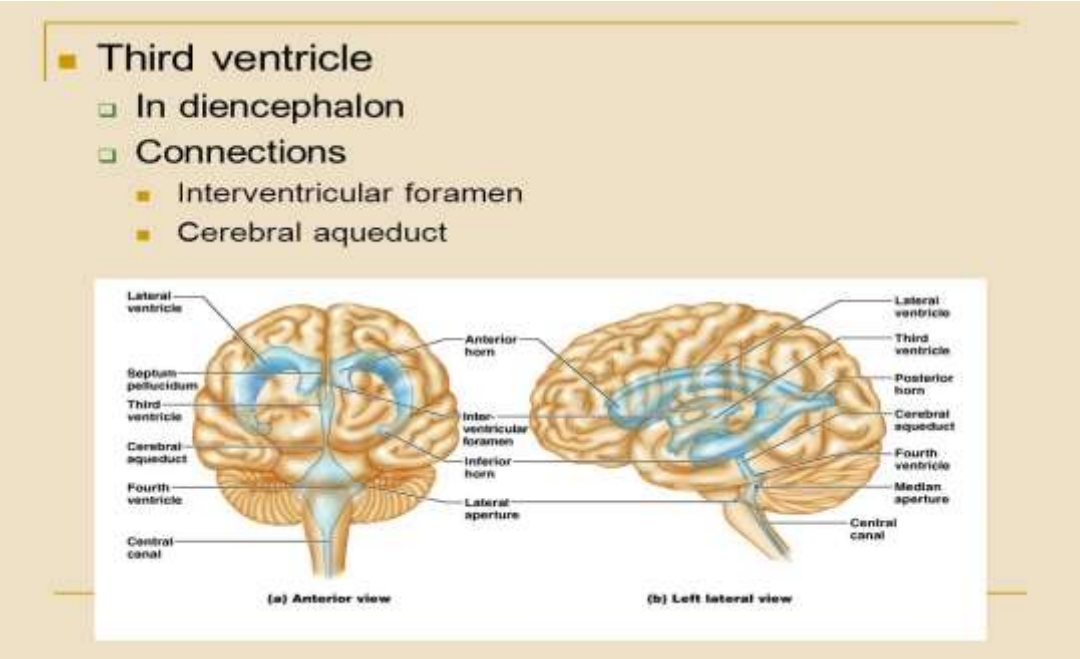

Figure No. 1 Schematic representation of the expansion of the ventricles and subarachnoid spaces in children.

5. Convulsive syndrome - in newborns, clonic convulsions, twitching of facial muscles are more common, in infants - generalized tonic-clonic convulsions, which are accompanied by autonomic disorders.

6. **Syndrome of vegetative**-visceral disorders - rarely observed in isolation, often combined with other syndromes. Manifested by various dysfunctions of the internal organs - transient cyanosis, tachycardia, tachypnea, arrhythmias, poor weight gain, regurgitation, vomiting, diarrhea, constipation, pylorospasm.

Figure No. 2. View of a sick child with tonic - clonic convulsions

ANOMALIES IN THE DEVELOPMENT OF THE NERVOUS SYSTEM

Craniocerebral hernia - a hernial protrusion in the region of a defect in the bones of the skull; There are two forms: meningocele (the dura mater and skin are part of the hernial sac, the contents are cerebrospinal fluid) and meningoencephalocele (any part of the brain is in the hernial sac).

Figure No. 3 A newborn child with a craniocerebral hernia

Spinal hernia (spina bifida) is an anomaly in the development of the spinal column resulting from a violation of the process of closing the neural tube. Exit through a defect in the membranes of the spinal cord is called a meningocele. If the hernial sac contains nerve tissue, the formation is called a meningomyelocele. More often the defect is located in the lumbar and sacral spine.

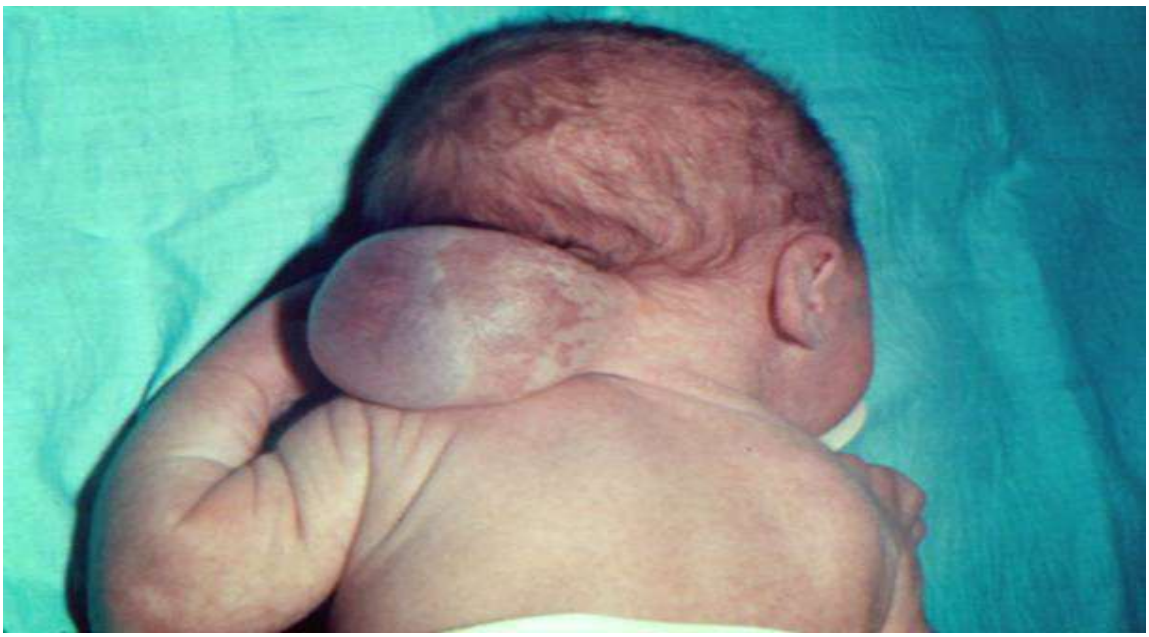

Figure #4. Newborn baby with spinal hernia

Microcephaly is the small size of the brain skull and brain of a child. Criteria for diagnosis: a decrease in the circumference of the cerebral skull by more than 5 cm in relation to the average age. Children with microcephaly have various neurological symptoms: impaired muscle tone, spastic paresis, convulsions, mental underdevelopment.

Hydrocephalus is an increase in the size of the ventricles of the brain, in most cases accompanied by an increase in the size of the head. There are internal and external hydrocephalus.

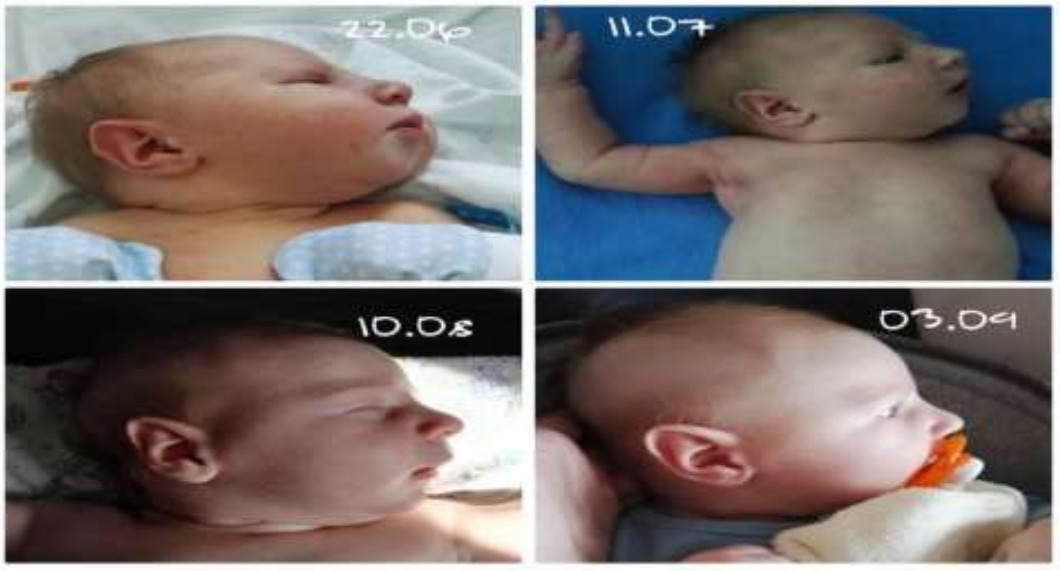

Figure 5. View of a newborn baby with microcephaly.

With internal, accumulation of cerebrospinal fluid is noted in the lateral ventricles of the brain, with external - in the subarachnoid space. As a result, there is an increase in the size of the head, a divergence and thinning of the bones of the skull, and bulging of the fontanelles. On the forehead, scalp, dilated cutaneous veins can be seen.

Figure #6. View of an infant with hydrocephalus

Ventriculomegaly is an isolated expansion of the ventricles of the brain, which is not accompanied by an increase in the size of the head. In most cases, it develops as a result of a violation of the outflow of cerebrospinal fluid.

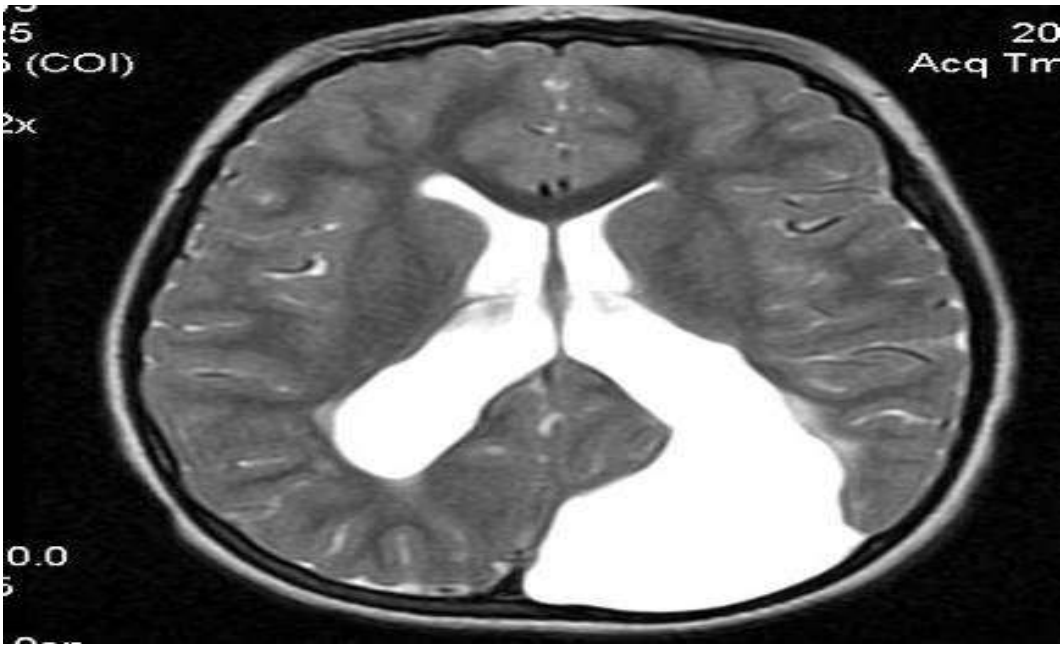

Figure #7. Schematic representation of an isolated expansion of the right ventricle in children.

Anencephaly is established by the absence of the bones of the brain skull and brain tissue.

Acrania is a condition where the fetal brain is not surrounded by a bony vault. Anencephaly and acrania are malformations incompatible with life.

Craniostenosis - premature overgrowth of the cranial sutures, leading to deformation of the skull.

Figure #8. View of an infant with craniostenosis

Scaphocephaly - the skull increases in the anteroposterior direction and decreases in the transverse due to premature overgrowth of the sagittal suture. At the same time, the child's head is narrowed, the forehead and back of the head protrude, the fontanelles are narrowed.

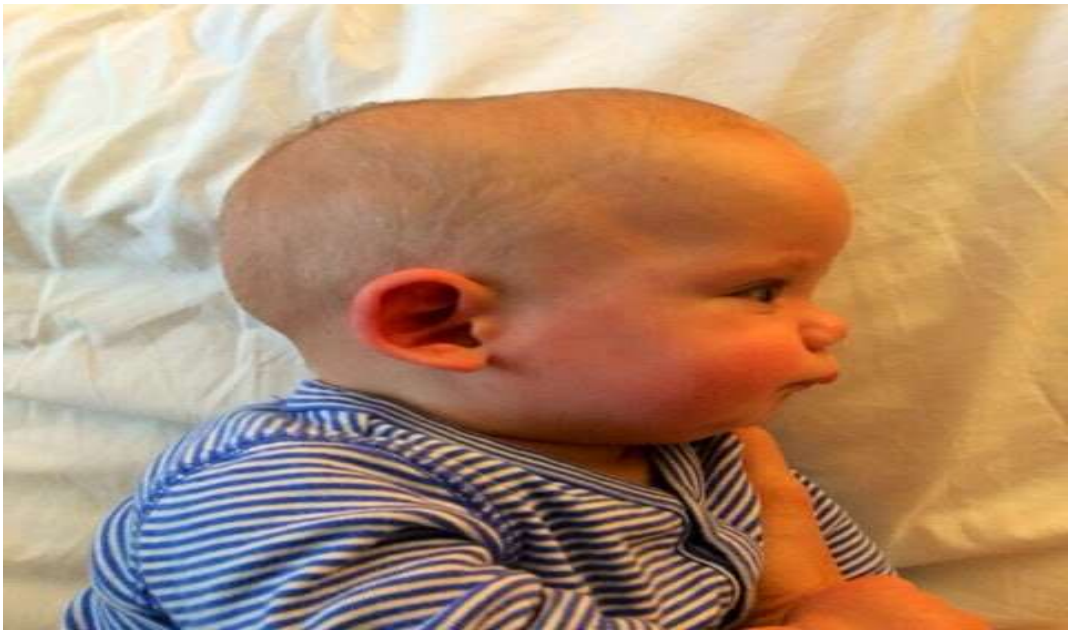

Figure #9. View of an infant with scaphocephaly

Plagiocephaly - accelerated healing of only one of the coronal sutures. The skull is asymmetrical, there is a unilateral exophthalmos.

Acrocephaly is premature overgrowth of the coronal sutures of the skull, which leads to limitation of its growth in the anteroposterior direction. The cranial vault of the child is high, wide and short, the face is flattened, exophthalmos may be noted.

Trigocephaly - early fusion of the frontal bones in the region of the frontal suture. The frontal tubercles are not developed, the skull acquires a triangular shape. Oxycephaly (tower skull) - occurs in case of premature fusion of the coronary and sagittal sutures. Accompanied by increased intracranial pressure.

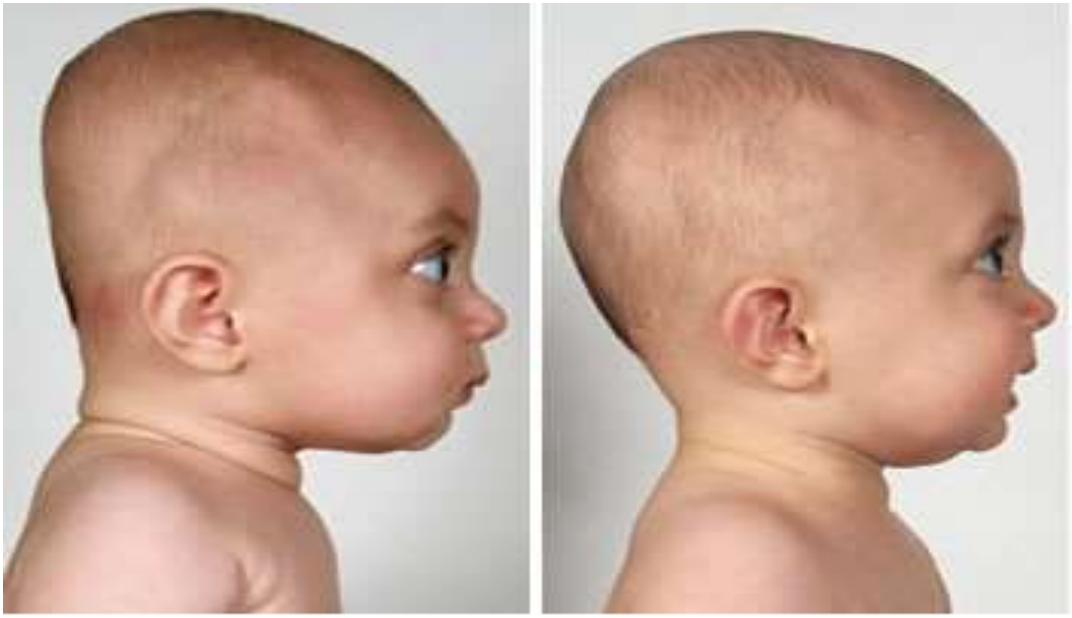

Figure #10. View of an infant with oxycephaly

GENERAL SYMPTOMS AND SYNDROMES

Disturbances of consciousness. There is currently no simple definition of the concept of "consciousness". Usually its interpretation is based on the opposition of states of sleep and wakefulness. During sleep, there is no consciousness, waking up, we begin to feel, think, act and convey our feelings with the help of words to other people. Consciousness expressed in words is called verbal. A healthy child has a clear mind. Being in a clear mind, the child is well oriented in his own personality, time and space. A disturbance of consciousness is understood as a disorder in the reflection of the environment, objects, phenomena and their connections, manifested by the complete impossibility or indistinct perception of the environment, disorientation in time, place, surrounding persons, one's own personality, incoherence of thinking. Violations of consciousness are conditionally divided into two groups - oppression and changes in consciousness. Oppression of consciousness is characterized by a deficit of mental activity, a decrease in the level of wakefulness, depression of intellectual functions and motor activity. This condition includes stupor, stupor, stupor, and coma. With severe depression of consciousness, vital functions are violated. Changes in consciousness develop against the background of wakefulness and are characterized by a disorder

of mental functions, a distorted perception of the environment and one's own personality (changes in consciousness are studied in the course of psychiatric diseases). When examining a patient, it is necessary to focus on a certain sequence of occurrence of signs of a violation of well-being and consciousness in children. A child in a stupor is in a state of stupor, from which it comes out with difficulty after vigorous braking. There are frequent periods of motor restlessness with athetosis-like movements.

Reflexes are reduced. The reaction to pain irritation is distinct, but short-lived. Sopor - deep sleep, stupor, no reaction to the environment. The child cannot be awakened. Skin sensitivity is not determined, but tendon reflexes are caused, albeit with difficulty, inconsistently. The reaction to pain (injection) is preserved, but it is indistinct. Pupillary and corneal reflexes, as well as swallowing, were preserved in the patient. With a significant degree of oppression of the cortex and damage to the centers of the brain, loss of consciousness occurs - coma.

Coma - this condition is characterized by the complete absence of reflexes and skin sensitivity in the child, there is also no reaction to injections and other manipulations, muscle hypo- and atony is observed. The pupils are wide, there is no corneal reflex, there is no reaction to light and other stimuli (pain, sound). In such patients, it is imperative to check the reflexes from the larynx and pharynx, which allow you to determine the depth of the coma. Coma can be with diabetes mellitus, cerebral hemorrhage, poisoning, chronic nephritis, severe liver damage.

By depth, it is customary to distinguish 3 degrees of coma:

I degree - mild, while there is no consciousness and voluntary movements, corneal and corneal reflexes are preserved.

Grade II is characterized by a lack of consciousness, areflexia (pupil reflexes are preserved), respiratory distress is often observed, and at grade III, the absence of all reflexes, deep respiratory rhythm disorders are observed. When excited with euphoria, the patient has an increase in motor activity and play activity, unusual talkativeness or babble (in infants). The child's contact with people close to him worsens (he does not seem to hear appeals, requests and instructions), appetite

worsens, the time to fall asleep and wake up increases. With the progression of the pathological condition, excitation with negativism develops, in which, against the background of a general excited state, positive emotions disappear in the child, he becomes capricious, screams and cries for any reason, discards toys, refuses to eat, does not know what he wants. Asks for hands, can not sleep. The sensitivity of the skin is increased - hyperesthesia - the child reacts sharply to touch, throws off the blanket. Skin and tendon reflexes are increased. Excitation in combination with somnolence is manifested by alternating periods of excitement with temporary calm, the appearance of lethargy, drowsiness. Hypersensitivity of the skin persists, reflexes may be elevated or normal.

Doubtfulness. With somnolent consciousness, the reaction to the environment is reduced (the child feels lethargy and drowsiness), but sleep is superficial, comes in short periods. Instead of crying and screaming - a quiet moaning. Poorly responsive to examination. There is a decrease in skin sensitivity and tendon reflexes. The child reacts to strong irritation by crying, answers questions sluggishly. Changes in the activity of the central nervous system are always accompanied by autonomic changes. The excitation phase is associated with the activation of the sympathetic department, so tachycardia, tachypnea occur, and blood pressure may be increased. The defeat of the central nervous system in severe diseases is never limited to the sphere of consciousness and reflex activity. In a state of stupor, stupor and coma, progressive disorders of the vital regulatory functions of the central nervous system, in particular the brain stem sections, are always detected. As a result of these disorders, irregular breathing occurs (such as Cheyne-Stokes or Biot, gasping breathing), paralysis of the respiratory or vasomotor centers. Therefore, the progression of impaired consciousness is a harbinger of possible disorders of the brain stem.

Headache. This symptom accompanies various diseases. According to pathogenesis, headache is divided into five main types. Vascular headache occurs when the vessels are excessively stretched by the pulse volume of blood or as a result of overflow of blood vessels, especially veins that are in a state of

hypotension; at a tension of vessels as a result of volumetric process. With excessive stretching of the vessels by the pulse volume of blood, patients experience rhythmic, pulse-synchronous blunt blows to the head (“knocking in the head”). This occurs in children with arterial hypotension, when blood pressure rises due to mental or physical stress, during crises in patients with signs of vegetovascular dystonia. With vascular spasm, patients complain of a feeling of squeezing, breaking, dull pain, nausea, darkening of the eyes, "black flies" before the eyes, dizziness, pallor of the skin. A similar headache occurs with sympathetic-adrenal crises and hypertension. Venous headache occurs when there is a violation of the venous outflow from the cranial cavity. Overfilling of the venous system with blood causes a feeling of heaviness in the head, dull, arching pain, which is aggravated by prolonged work with the head low, coughing, and laughter.

Tension headache is the most common form of headache. It occurs in response to mental stress and is accompanied by an increase in the tone of the soft integument of the head. Patients complain of sensations of compression, squeezing of the head (“like a hoop tightened”, “the head, as if in a vise is clamped”). Tension headache is diffuse, bilateral, and one side may hurt more intensely. At the height of the headache, accompanying symptoms may be noted: photo and sound fear, nausea. More often the pain lasts all day and increases in the evening. Tension headache can be provoked by reflex muscle tension during antiphysiological postures (prolonged work at a computer, work with small objects, etc.), which causes long-term tension in the eye and neck muscles. Prolonged muscle tension leads to narrowing of arterial vessels and ischemia. Tension headache is aggravated or provoked by changeable weather conditions. One of the manifestations of tension headache is vertebrogenic headache, manifested by the presence of pain in the head and neck, which is caused by degenerative processes of the bone and ligamentous apparatus (intervertebral osteochondrosis, arthrosis, etc.). The most common complaint in such cases is a dull, arching headache in the back of the head or fronto-temporal-orbital region that worsens in the morning, which is accompanied by a sensation of a rush of blood, pastosity and numbness of

the face, difficulty breathing, and the presence of "bags" under the eyes.

Liquorodynamic headache occurs with changes in intracranial pressure and dislocation of intracranial structures with tension on the membranes, vessels and nerves inside the skull. With intracranial hypertension, it is bursting (“from the depths of the brain”), increases with coughing, straining, in a standing position, while walking, every step “gives to the head” with sharp, shooting pain.

Neuralgic headache is associated with irritation of the nerves by an endoneural or extraneural pathological process. This type of pain is characterized by: - paroxysmal attacks (attacks last a few seconds or minutes, follow each other at short intervals); - the existence of zones, the irritation of which provokes an attack; - irradiation of pain to neighboring or distant areas. Neuralgic headache, sharp, penetrating, cutting, burning. At the moment of paroxysm, the patient freezes, avoids any head movements.

Psychogenic headache occurs against the background of mental disorders (hysteria, hypochondria, obsessive-phobic syndrome, depression, etc.). It has a chronic relapsing character, painful, provoked by stress, psychosocial factors. Dizziness (from lat. vertigo - rotation). The condition is characterized by the illusion of rotation of visible surrounding objects (objective vertigo) and/or the observer himself (subjective vertigo). It can occur in healthy children with excessive or prolonged irritation of the vestibular apparatus (movement with significant acceleration, pitching, etc.), intense rhythmic irritation of the receptors of the eyeballs (with prolonged fixation of the gaze on a moving object) or the absence of habitual points in external space that determine spatial orientation (on high). Vertigo occurs in diseases of the vestibular analyzer (labyrinth, auditory-vestibular nerve, vestibular nuclei of the brain stem, supranuclear structures, cortex, mainly in the area of the temporo-parietal-occipital junction), visual and oculomotor apparatus, in pathologies of the gastrointestinal tract, cardiovascular system etc. Causes of damage to the vestibular analyzer can be inflammatory and non-inflammatory diseases of the labyrinth, otosclerosis, Meniere's disease, infectious, toxic, traumatic effects on the auditory-vestibular nerve, circulatory

disorders of the cerebrospinal fluid, vascular, inflammatory, toxic, tumor, parasitic and other diseases of the brain brain, less often - functional diseases of the nervous system. Dizziness is usually accompanied by nausea, vomiting, blurred vision, unsteadiness, slow heart rate, blanching, changes in blood pressure, nystagmus, and other symptoms.

Generalized seizures. The development of a generalized convulsive seizure can sometimes be preceded by symptoms that are called the prodrome, or precursors (general discomfort, anxiety, aggression, headache, irritability, etc.) that occur several hours or days before its development. An epileptic seizure can be primary or secondary generalized. A secondary generalized seizure is preceded by the onset of an aura (feelings of the unreality of what is happening, discomfort in the stomach, visual, auditory, olfactory sensations). With primary generalized convulsive seizures, there is no aura. The attack begins with a loud cry, there is tension in the muscles of the whole body, the teeth are clenched, the lips are tightly compressed, a bite of the tongue is possible. There is a short-term cessation of breathing with the appearance of cyanosis. Next, seizures develop. The attack usually stops spontaneously after 1-5 minutes.

hypertensive syndrome. It is characterized by meningism (stiff neck, upper Brudzinsky reflex, etc.), vomiting, bulging of the fontanel.

Figure 11. Signs of meningism in a 4-year-old child.

In the fundus, there is varicose veins, blurring of the boundaries of the optic nerve papilla. The defeat of the cranial nerves is manifested by the symptom of the "setting sun", converging strabismus, horizontal nystagmus.

Muscle tone can vary from hypertonicity to hypotension. Often there is a tremor of the chin, hands, regurgitation, vomiting. Children become easily excitable, restless, their cry is sharp, piercing, their sleep is superficial.

Convulsive syndrome. Seizures are sudden attacks of clonic, tonic or clonic-tonic involuntary and short-term muscle contractions with or without loss of consciousness. There are general (generalized) and partial (partial) convulsions. The causes of seizures can be intoxication, infections, injuries, diseases of the central nervous system. Convulsive syndrome is a typical manifestation of epilepsy, spasmophilia, toxoplasmosis, encephalitis, meningitis and a number of other diseases. The causes of seizures can also be metabolic disorders (hypocalcemia, hypokalemia, acidosis), endocrine pathology, hypovolemia (vomiting, diarrhea), overheating. In newborns, convulsions can be caused by asphyxia, hemolytic disease of the newborn, congenital defects of the central nervous system. Seizures are also often observed with the development of neurotoxicosis, with diseases such as influenza, parainfluenza, adenovirus and other respiratory viral infections. Manifestations of convulsive syndrome are very diverse and differ in duration, time of occurrence, state of consciousness, frequency, prevalence, form of manifestation. The nature and type of seizures depend on the pathological process, which may be the direct cause of their occurrence or play a provoking role. Clonic seizures are rapid muscle contractions that follow each other after a short period of time. They are rhythmic and non-rhythmic and are characterized by excitation of the cerebral cortex. Tonic convulsions are characterized by more or less prolonged muscle tension, mainly in an extensor posture (forced position), and are caused by damage to the brainstem-subcortical structures. They can be primary or occur immediately after clonic seizures, are general or localized.

Clonic-tonic convulsions are characterized by a periodic change of clonic and tonic phases. With a convulsive syndrome, the child suddenly loses contact with the environment, his gaze becomes wandering, then the eyeballs are fixed up or to the side. The head is thrown back, the arms are bent at the hands and elbows, the legs are extended, the jaws are clenched. Possible tongue biting. Breathing and pulse slow down, possibly apnea. This is the first - the tonic phase of clonic-tonic convulsions, which lasts no more than a minute. The second phase - clonic - begins with twitching of the muscles of the face, then convulsions pass to the arms and legs and become generalized; breathing is noisy, wheezing, foam appears on the lips; the skin is pale, tachycardia is noted. These convulsions are prolonged and can be fatal. Diagnosis of a convulsive syndrome does not cause difficulties. A certain role here is played by spinal puncture, electroencephalography, echoencephalography, fundus examination and other studies of the brain and central nervous system.

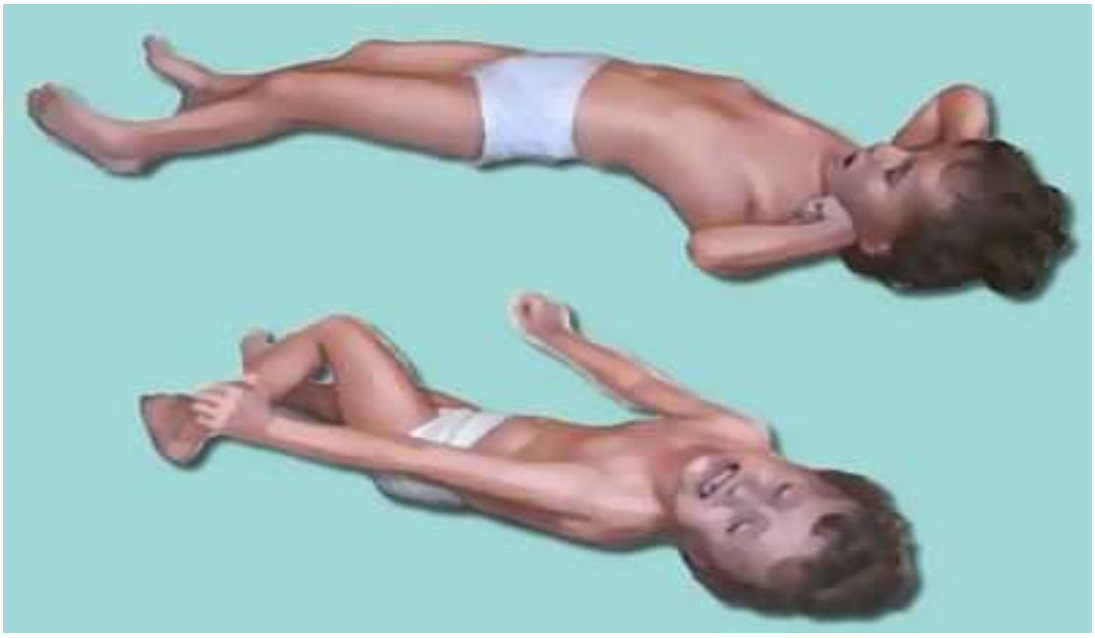

Figure 12. Seizures in a 5-year-old girl

Meningeal syndrome. It is caused by damage to the soft and arachnoid membranes of the brain, develops due to increased intracranial pressure, inflammatory or toxic damage, subarachnoid hemorrhage. It is observed when the meningeal membranes become inflamed (meningitis) or irritated (for example,

with pneumonia in young children). It is manifested by the characteristic posture of the child and positive meningeal symptoms.

Meningeal symptoms:

1. Rigidity of the neck - when trying to bend the patient's neck with the chin brought to the anterior surface of the chest, the reflex tension of the neck muscles makes flexion impossible.

2. Kernig's symptom - the inability to straighten the leg at the knee joint, previously bent at an angle of 90 ° in the knee and hip joints (both legs are examined sequentially).

3. Brudzinsky symptom (provocation of the meningeal posture):

- upper (occipital) - evaluated simultaneously with checking the stiffness of the occipital muscles - with passive flexion of the child's neck (with the chin brought to the surface of the chest), lying on his back with outstretched legs, there is a reflex flexion of the lower limbs in the hip and knee joints; often the arms are bent at the same time;

- middle (pubic) - when pressing over the pubic joint with a fist, the legs reflexively bend in the hip and knee joints;

- lower (contralateral) - is examined together with Kernig's symptom - when trying to straighten the leg bent at the knee joint, the second leg involuntarily bends at the knee and hip joints (both legs are examined sequentially).

The characteristic meningeal posture (pose of the "pointing dog") usually appears later: the patient lies on his side with his head thrown back and arms and legs bent and brought to the body, the spine is arched backwards (opisthotonus). This position of the body occurs due to involuntary reflex tonic muscle contraction. In young children, tonic muscle tension is physiological, therefore, to ascertain the presence of meningeal syndrome, the definition of the following symptoms is used:

1. Symptom of Lessage's suspension - the child raised under the armpits pulls the legs to the stomach.

2. Tension and bulging of the large fontanel (with increased intracranial pressure).

3. Ankylosing spondylitis - with percussion of the zygomatic arch, an increase in headache is noted, as evidenced by the appearance of an involuntary pain grimace on the corresponding half of the face.

4. Symptom of the "tripod" - the child sits, leaning on his hands located behind the buttocks.

5. Fanconi's symptom - the inability to stand up with straightened and fixed knee joints.

6. Symptom of "kissing the knee" - you can not touch the child's face to his knee because of the extensor posture.

7. Symptom of Meitus - with fixed knee joints, the child cannot sit up in bed (the back and legs form an obtuse angle).

Liquor changes. The pressure is usually increased, with serous meningitis it may be higher than with purulent. The liquid can be cloudy (with purulent meningitis), slightly opalescent (with tuberculous meningitis), transparent (with serous meningitis). A reflection of inflammation in the membranes is pleocytosis (an increase in the number of cells) - an increase of up to several hundred and thousands in 1 μl of neutrophils in purulent processes, lymphocytes - in serous ones. The amount of protein increases to 0.4–1 g/l or more.

Normal indicators of cerebrospinal fluid are given in table. one.

Table 1.

The main indicators of liquor

Coloring	Transparent
Quantity, ml	100–200
Ph	7.4–7.5
Pressure, MM WATER.ST	130–180
Protein, mg/l	160–330
Cytosis, cells/mm3 Of which: lymphocytes, % neutrophils, %	2-8 90-95 3–5
Sedimentary reactions (Pandi, Nonne-Apelta)	Negative
Glucose, mmol/l	1.83–3.89
Fibrin film	None

Meningism. Meningeal symptoms do not always indicate the presence of meningitis. Sometimes quite pronounced meningeal symptoms are observed with common infections in children, with intoxication. In the study of cerebrospinal fluid, except for an increase in pressure, there is no pathology. In such cases, they speak of meningism. It usually manifests itself in the acute period of infection,

lasts 3-4 days. Improvement comes after a puncture. The cause of meningism is toxic irritation of the meninges, their swelling, increased intracranial pressure. encephalitic syndrome. The development of encephalitis is accompanied by general infectious, cerebral, focal and meningeal symptoms. General infectious symptoms - fever, inflammatory changes in the blood and other signs of infection.

Cerebral symptoms (diffuse inflammatory reaction of the brain) - edema, hyperemia, hypersecretion of CSF. There are also disturbances of consciousness up to coma, sometimes excitement, epileptic seizures, muscle twitching. In severe cases - inhibition of reflexes, impaired cardiac activity and respiration. Focal symptoms of varying severity depend on the localization of the primary lesions of brain regions. There may be motor, sensory disorders, speech disorders, various hyperkinesias, cerebellar disorders, stem symptoms, as a manifestation of brain irritation - focal or general epileptic seizures.

Meningeal symptoms almost always accompany encephalitis. Even with a low severity of meningeal symptoms, there are almost always inflammatory changes in the cerebrospinal fluid (an increase in the number of cells with a slight increase in protein - the so-called cell-protein dissociation). Symptoms of increased neuro-reflex excitability.

Spasmophilia. Observed in young children with spasmophilia. The main clinical manifestations of spasmophilia - spasm and convulsions - are explained by a sharp calcium deficiency and the increased excitability of the nerves caused by it. In many children, this disease proceeds secretly, latently (hidden spasmophilia), in some patients it manifests itself with characteristic clinical symptoms (in a healthy child, all these symptoms are negative).

Chvostek's symptom (one of the most constant symptoms of latent spasmophilia) - when tapping on the fossa canina (the exit point of the trigeminal nerve), an involuntary contraction of the muscles of the lower eyelid, wing of the nose and corner of the mouth occurs on the corresponding side (checked sequentially from two sides).

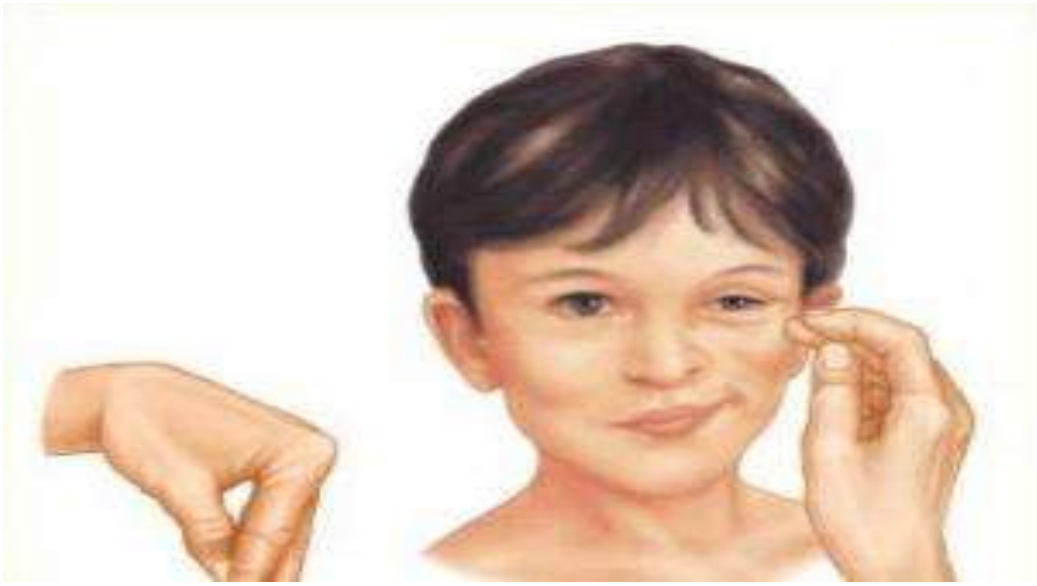

Figure 14. Manifestations of Chvostek's symptom in an 8-year-old girl.

Trousseau's symptom - if for a few minutes with a tourniquet, cuff or just fingers to squeeze the neurovascular bundle on the shoulder (in the area of s. bicipitalis, above the olecranon), the hand takes the position of the "obstetrician's hand" due to convulsive contraction of its muscles.

Lust's symptom - tapping with a neurological hammer behind the head of the fibula or squeezing the gastrocnemius muscle (in order to irritate the n. perineus passing there) causes the child's foot to flex to the back and take it slightly outward ("horse foot" - pes equinus). Instead of percussion, clamping at the level of the middle third of the lower leg can be used (similar to Trousseau's symptom). It is checked sequentially on two legs.

Maslov's phenomenon - respiratory arrest at the height of inspiration during screaming and crying in response to pain irritation (skin prick).

Hypomagnesian seizures are similar to hypocalcemic seizures, but carpopedal spasm is also commonly observed.

Syndrome of autonomic dysfunction. This syndrome includes disorders of vegetative (related to the work of internal organs) functions of various origins and manifestations, caused by a disorder in their nervous regulation.

The cause of autonomic dysfunction can be: hereditary factors (as a rule, they appear already in childhood);

- diseases of the central and peripheral nervous system (especially the hypothalamus, limbic system, brain stem);

- some chronic diseases (mainly endocrine, gastrointestinal and cardiovascular systems);

- chronic stress, overwork and overstrain. - Manifestations of autonomic dysfunction can affect a variety of organs and systems, individually or together. Below are the most common of these syndromes:

- Cardiovascular (cardiovascular) syndrome can be manifested by heart rhythm disturbances (tachycardia, bradycardia, extrasystole), volatility in blood pressure, vascular reactions (pallor or "marbling" of the skin, "hot flashes", chilliness of the hands and stop).

Often found - cardialgic syndrome - aching, stabbing or throbbing pain or discomfort in the region of the heart, which, unlike angina pectoris, is not associated with physical activity, does not go away when taking nitroglycerin. - Hyperventilation syndrome is manifested by rapid breathing, a feeling of lack of air, incompleteness or difficulty in inhaling. At the same time, the blood loses a lot of carbon dioxide, which leads to its alkalization and depression of the respiratory center. As a result, muscle spasms, sensory disturbances in the perioral region, hands and feet, dizziness develop.

Irritable bowel syndrome is manifested by spastic and aching pains in the lower abdomen, frequent urge to defecate, bloating, stool instability, appetite disturbance, nausea and vomiting, dysphagia (impaired swallowing), pain and discomfort in the pit of the stomach (all this in the absence of an organic disease). Violation of sweating, as a rule, proceeds in the form of hyperhidrosis (excessive sweating) of the palms and soles.

Cystalgia - frequent painful urination without signs of urinary system disease and changes in the anamnesis of urine. Violation of thermoregulation is expressed in a persistent slight increase in temperature, chills. Elevated temperature is easily tolerated, sometimes higher in the morning, can be raised asymmetrically in the armpits.

SEMIOTICS OF SOME DISEASES

ENDOCRINE GLANDS

Each of the endocrine glands performs a specific function, and all of them are in close interaction with each other and with the central nervous system, ensuring the unity of the body, which is reflected in the term "neuroendocrine regulation". In the development of the pathological process, an interdependent violation of the functions of various endocrine glands is revealed. By the time of birth, a single neuroendocrine system functions in a child, and after birth it continues to develop and improve intensively. In this regard, in children, unlike adults, endocrine disorders can cause irreparable changes in the growing body.

SEMIOTICS OF GROWTH DISTURBANCE

The growth of a child is determined by the interaction of many factors. Genetic factors, for example, are closely related to environmental factors. With allowance for fluctuations, the growth curve of a healthy child is quite flat, so any significant deviation in the growth line is most likely due to illness, malnutrition, or adverse psychosocial conditions.

Gigantism is a disease characterized by increased growth that does not correspond to age, sex and nationality, which develops as a result of excessive production of somatotropic hormone by the pituitary gland. It occurs predominantly in boys. Growth accelerates during puberty. In its development, craniocerebral trauma, infectious and toxic effects (encephalitis transferred in childhood, childhood infections), tumors, infectious and toxic lesions with hyperplasia of acidophilic cells of the anterior pituitary gland can play a role. The following signs of gigantism are noted: the skeleton is proportional and harmonious; expanded areas of epiphyseal cartilage; in the tubular bones, hyperostoses are noted, the formation of new bone marrow cavities with the phenomenon of osteoporosis, disorders of endochondral bone formation in the diaphysis and expansion of the periosteum; an increase in parenchymal organs;

thickening of the epidermis and atrophic disorders; an increase in growth in periods: it slows down, then accelerates; the development of internal organs is slower than the overall growth of the body; in girls, menstruation is either absent or goes through irregular cycles, the mammary glands are underdeveloped, infertility develops; nodes are detected in the thyroid gland, sometimes with symptoms of hyperthyroidism, diabetes insipidus necessarily develops, diabetes mellitus occurs; there is a decrease in intellectual abilities (up to mental retardation), irritability, infantilism, insomnia; due to the increased growth of bones and the presence of a tumor, the walls of the Turkish saddle in the skull are destroyed, a narrowing of the peripheral fields of vision occurs; bone age significantly exceeds the passport.

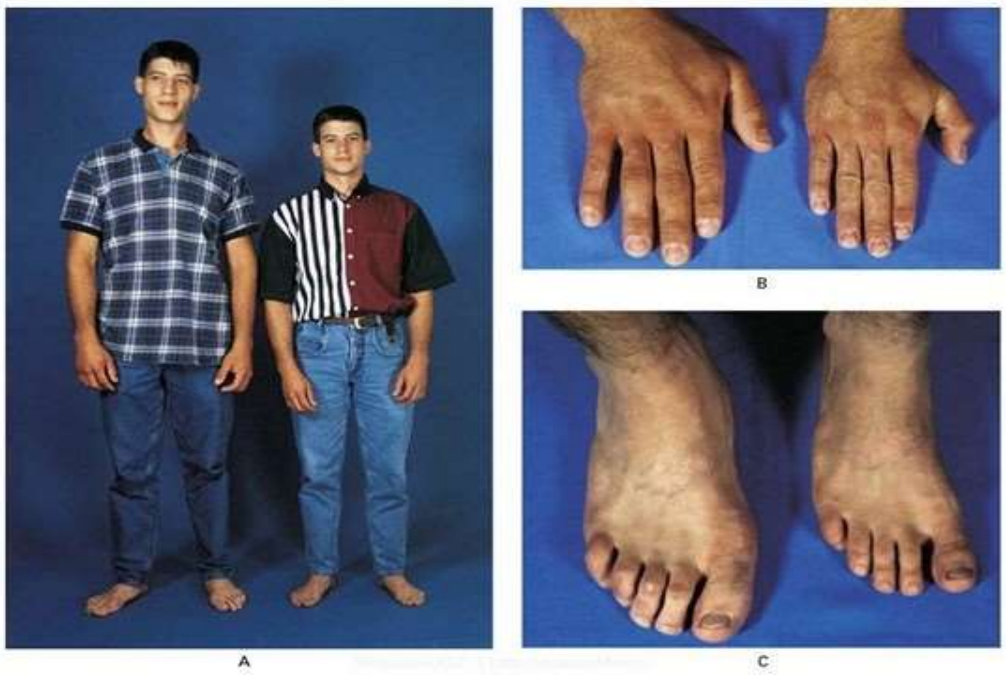

Figure 15. View of a teenager with gigantism

Acromegaly is a disease that is characterized by a pathological form of growth of bones, soft tissues and internal organs. The disease is caused by an eosinophilic adenoma of the pituitary gland or hyperplasia of its acidophilic cells, which intensively produce growth hormone. The reasons for the development of pathology are malignant tumors of the left frontal lobe; encephalitis; cysts of the large cistern after infectious diseases and traumas of the skull; congenital or acquired syphilis. Signs of acromegaly: hyperproduction of growth hormone in

adults (formation of the skeleton is complete) causes periosteal growth of bones - they deform and thicken; almost half of patients develop concomitant diabetes mellitus due to impaired glucose tolerance; the content of calcium in the blood increases (the function of the parathyroid glands increases) and inorganic phosphorus in the blood serum; a change in appearance is characteristic: enlargement of facial features, an increase in hands and feet; the bones of the skull thicken, the superciliary and zygomatic arches, the occipital protuberance protrude; jaws protrude; soft tissues of the face grow; due to hypertrophied papillae, the tongue increases; the vocal cords thicken, the voice is low, rough, speech is slurred; the skin is thick, flabby, hyperpigmented or, conversely, depigmented, with enlarged sebaceous and sweat glands, sweat with an unpleasant odor, hair growth is disturbed: baldness occurs or, conversely, hypertrichosis on the face and trunk (the latter is especially characteristic of girls); at the beginning of the disease, the muscular system hypertrophies, later on muscle atrophy occurs with progressive muscle weakness; internal organs change: the function of the cardiovascular system is disrupted, blood pressure rises, the liver and stomach increase, the intestines lengthen, the sex glands hypertrophy in girls; intracranial pressure increases, neuritis, radiculitis develop;

Figure 16. View of a patient with acromegaly

symptoms of increased intracranial pressure cause congestion, small hemorrhages in the eyeballs, peripheral visual fields fall out, sometimes with impaired color perception; vertebrae expand, intervertebral discs become loose. It is not difficult to make a diagnosis of acromegaly if changes in appearance are pronounced, associated with headaches and sexual dysfunction.

Short stature (polyetiological syndrome) is often a manifestation of morphological and functional insufficiency of the hypothalamus and / or anterior pituitary gland of a congenital or acquired nature with a decrease in the secretion of somatotropic hormone. The most common clinical variants of this condition are cerebro-pituitary dwarfism (CGN) and isolated somatotropic hormone deficiency. Another endocrine cause of short stature can be a deficiency of thyroid hormones (hypothyroidism), insulin (uncontrolled diabetes mellitus), sex hormones (hypogonadism), or an excess of sex hormones (precocious puberty),

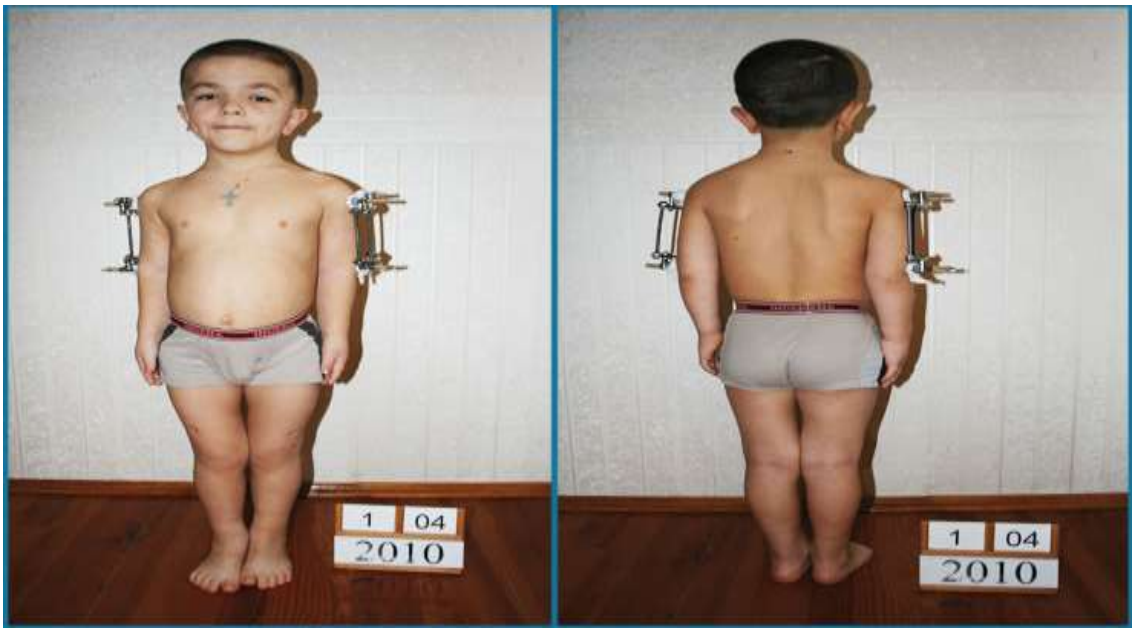

Figure 17. View of a sick child with cerebral-pituitary dwarfism

Glucocorticoids (disease and Itsenko-Cushing's syndrome). Severe somatic diseases (somatogenic dwarfism), pathology of the skeletal system, genetic and chromosomal diseases (Shereshevsky-Turner syndrome) can also lead to impaired growth. In some cases, short stature is a manifestation of constitutional features

(late puberty syndrome, family short stature). Pathology is typical for boys.

Cerebral-pituitary dwarfism is characterized by loss of functions of all tropic hormones (panhypopituitarism). There are idiopathic and organic variants of the disease. In the idiopathic variant of CGN, there are no signs of organic damage to the central nervous system, the pathological process is formed at the level of the hypothalamic structures. It occurs 2-4 times more often in boys than in girls. The relationship of the disease with the birth of a child in the breech presentation, the imposition of obstetric forceps, bleeding during childbirth was revealed. This indicates the role of birth trauma and hypoxia in the genesis of CGN. In the absence of treatment, the growth of patients (adults) does not exceed 120 cm in women and 130 cm in men. Signs of short stature: at birth and in the first months of life, the physical development of children with CGN does not differ from that of healthy children; growth retardation becomes noticeable at the 2nd year of life, gradually the growth rate decreases; bone age lags behind chronological; in children with growth hormone deficiency, there is a tendency to hypoglycemic conditions (the processes of glycogenolysis are reduced), therefore, hypoglycemia may be the first sign of the disease; deficiency of thyroid-stimulating hormone (TSH) in patients with CGN is the cause of hypothyroidism, which determines the complex of symptoms: lethargy of the child, dry skin, bradycardia, hypotension, constipation, late eruption and late change of teeth; a pronounced deficiency of TSH further worsens the processes of growth and differentiation of skeletal bones in patients with CGN.

HYPERTHYROIDISM SYNDROME - Thyrotoxicosis is a hypermetabolic syndrome in which there are clinical and biochemical manifestations of excess thyroid hormones in the blood and tissues. In most cases, thyrotoxicosis develops due to excessive production of thyroid hormones by the thyroid gland (with hyperthyroidism).

Figure 18. Thyrotoxicosis in a 9-year-old girl.

Supporting signs of thyrotoxicosis:

- damage to the central nervous system and the autonomic nervous system (ANS): irritability, irritability, tearfulness, memory loss, rapid exhaustion, sleep disturbance, tremor of the fingers and the whole body (in children of the chorea type);

- damage to the cardiovascular system (associated with sympathicotonia and the toxic effect of thyroid hormones on the heart): tachycardia that does not disappear at rest, an increase in systolic and pulse blood pressure, heart rhythm disturbances (extrasystole, atrial fibrillation), the development of heart failure;

- symptoms associated with accelerated metabolism: fever (not amenable to antipyretics), increased sweating, wet hot hands, feeling hot, increased appetite, weight loss, osteoporosis, increased basal metabolic rate;

- damage to the digestive organs: accelerated motor activity of the intestine (irritable bowel syndrome), decreased secretory activity of the stomach, functional (less often structural) changes in the liver (depletion of glycogen stores, fatty degeneration, decreased cholesterol-synthesizing function);

- "eye" symptoms associated with an excess of thyroid hormones: eye gleam (Graefe's symptom), hyperpigmentation of the eyelids (Jellinek's symptom), rare

blinking (Stelwag's symptom);

- laboratory signs: the concentration of TSH in the blood is lowered, the concentration of triiodothyronine (T3) and thyroxine (T4) is increased. During thyrotoxicosis, it is customary to distinguish three degrees of severity depending on the severity of tachycardia, weight loss, and the presence of complications (thyrotoxic heart, CNS dysfunction, myopathy, secondary adrenal insufficiency, liver damage, secondary diabetes mellitus). This pathology is also characterized by goiter transformation syndrome. Signs of goiter transformation syndrome include an increase in the size of the thyroid gland, and it does not correlate with changes in the function of the gland (Table 2)

table 2

Degrees of enlargement of the thyroid gland

Degree	Description
0	no goiter
Ia	Goiter is determined by palpation
I b	Goiter palpable a and visible with full extension of the neck: this grade also includes nodular goiter, even if there is no enlargement of the thyroid gland
II	Goiter is visible in the normal position of the neck
0 I II	Goiter visible from a considerable distance

Causes of thyrotoxicosis - diffuse toxic goiter, nodular toxic goiter, thyroid adenoma, hyperthyroid phase of autoimmune thyroiditis, iodine-induced thyrotoxicosis, TSH-producing pituitary tumor, ovarian tumor producing thyroid hormones (struma ovarii), metastases of thyroid cancer producing hormones, overdose thyroid hormone preparations. The most common cause of thyrotoxicosis is diffuse toxic goiter. Among other manifestations of this syndrome, endocrine (autoimmune) ophthalmopathy is distinguished - a pathological change in the organ of vision, which is characterized by limited mobility of the eyeballs.

Supporting signs of ophthalmopathy:

- exophthalmos;

- lag of the upper eyelid from the iris when looking down, while a white stripe of the sclera appears;

- weakness of convergence (Mobius symptom);

- lacrimation;

- photophobia;

- a feeling of pain, "sand in the eyes." Other signs may include: exophthalmos with dilated palpebral fissures (Dalrymple's symptom), rare blinking (Stelwag's symptom), increased eyelid pigmentation (Jellinek's symptom), eyelid edema (Singer's symptom), eyelid tremor (Rosenbach's symptom).

Thyrotoxic crisis is the most severe and most severe complication of thyrotoxicosis. Provoking factors are surgical operations, hyperinsolation, radioactive iodine treatment, infections, rough palpation of the thyroid gland, intoxication, discontinuation of thyreostatic drugs, physical and psycho-emotional stress.

Signs of thyrotoxic crisis:

1) a sharp increase in body temperature;

2) nausea, vomiting, diarrhea;

3) sweating;

4) increase in systolic and decrease in diastolic blood pressure, tachycardia, heart rhythm disturbance;

5) sudden excitement, hallucinations, delirium. As the crisis progresses, dehydration increases, the skin becomes dry, adrenal insufficiency develops, collapse, liver failure, parenchymal jaundice joins. Excitation is replaced by apathy, adynamia, muscle weakness, prostration. Then a coma develops.

HYPOTHYROISIS SYNDROME Hypothyroidism is caused by insufficient secretion of thyroid hormones by the thyroid gland, which leads to inhibition of all types of metabolism, oxygen utilization by tissues, a decrease in the activity of various enzyme systems, gas exchange and basal metabolism. Thyroid hormone deficiency is based on structural or functional changes in the thyroid gland (primary hypothyroidism) or a violation of the stimulating effects of pituitary TSH or hypothalamic thyroid-stimulating releasing hormone (secondary hypothyroidism).

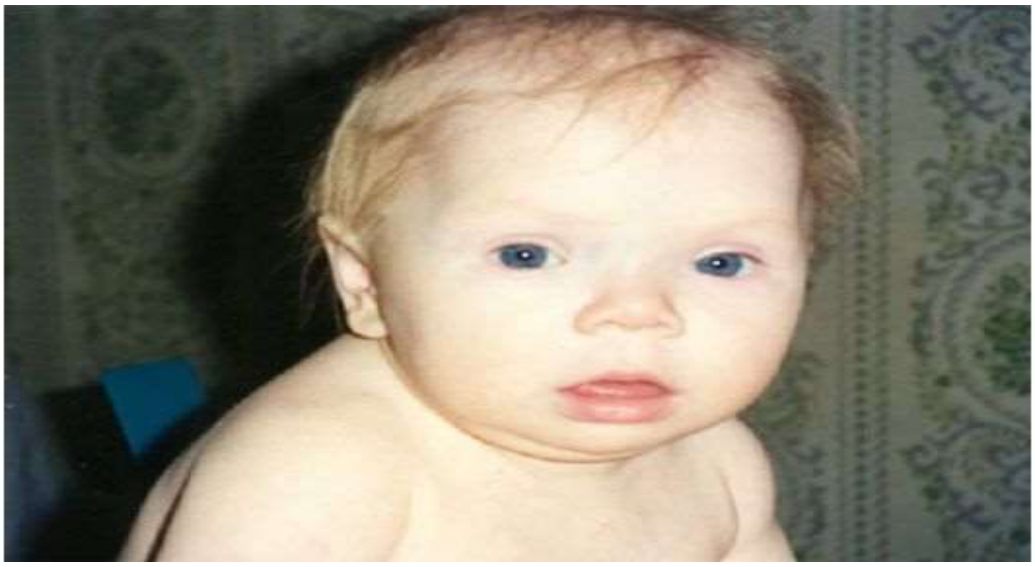

Figure No. 19. View of a sick child with hypothyroidism syndrome

Less common is hypothyroidism due to resistance of peripheral tissues to thyroid hormones (peripheral hypothyroidism). Less common is hypothyroidism due to resistance of peripheral tissues to thyroid hormones (peripheral hypothyroidism).

Supporting signs of hypothyroidism:

- damage to the central nervous system: general weakness, fatigue, decreased

performance, headache, depressed mood, drowsiness, lethargy, indifference, decreased intelligence, memory; sometimes psychoses;

-radiculitis, paresthesia and cramps in the limbs; decreased tendon reflexes;

- waxy pallor, the skin is dry, cold to the touch, dense (not taken in a fold), flaky; hyperkeratosis of the palms, soles; hair thinning, falling out, loss of the outer third of the eyebrows (Hertog's symptom); fragility of nails;

- swollen, mask-like (amimic) face, narrow palpebral fissures (edema of the eyelids); swelling of the neck, cheeks, lips, tongue, hoarseness of voice; after pressing on the edematous tissues, there is no trace left;

- damage to the cardiovascular system: bradycardia, myocardial damage (myocardial dystrophy, cardiomegaly), increased risk of atherosclerosis;

- decrease in the secretory function of the stomach and motor-evacuation function of the gastrointestinal tract (heaviness in the epigastrium, nausea, vomiting, flatulence, constipation);

- violation of the function of the urinary system: a decrease in renal blood flow and glomerular filtration, moderate proteinuria and hyperuricemia;

- anemia (normocytic, normochromic, hypochromic iron deficiency, B12- and folic deficiency);

- laboratory signs: an increase in the concentration of TSH in the blood (with primary hypothyroidism), a decrease in the concentration of T3, T4, hyperlipidemia (an increase in the concentration of total cholesterol and low-density lipoproteins, a decrease in the level of high-density lipoproteins). The causes of primary hypothyroidism are surgical treatment, treatment with radioactive iodine, radiation therapy of processes in the neck, an overdose of mercazolil, lithium, treatment with cordarone, destructive processes in the thyroid gland (thyroiditis, abscess, tuberculosis), congenital hypoplasia or aplasia of the thyroid gland, hereditary defects in the biosynthesis of thyroid glands. hormones.

The causes of secondary hypothyroidism are ischemic, tumor, inflammatory, traumatic lesions of the hypothalamic-pituitary region, treatment with large doses of reserpine, levodopa, parlodel.

The causes of peripheral hypothyroidism are diseases of the liver and kidneys, a hereditary decrease in the sensitivity of thyroid-dependent tissue receptors to thyroid hormones. Hypothyroidism must be distinguished from edematous syndrome, anemia syndrome.

hypothyroid coma. The most severe complication of hypothyroidism is hypothyroid coma. Factors provoking hypothyroid coma are inadequate treatment of hypothyroidism, infection, intoxication, hypothermia, trauma, surgery, sedatives and drugs.

Signs of hypothyroid coma:

1) hypothermia (body temperature below 35º);

2) arterial hypotension, bradycardia;

3) hypoventilation, rare breathing, respiratory acidosis;

4) atony of smooth muscles (urinary retention, dynamic intestinal obstruction);

5) progressive lethargy, apathy, drowsiness with an outcome in loss of consciousness.

HYPERPARATHYROISIS This syndrome is caused by overproduction of parathyroid hormone (PTH). Hyperproduction of PTH can develop: - due to direct damage to the parathyroid glands (adenoma, hyperplasia of the parathyroid glands, syndromes of multiple endocrine neoplasia types I and II) - primary hyperparathyroidism; - with compensatory hyperplasia of the parathyroid glands due to severe electrolyte disturbances (hypocalcemia, hyperphosphatemia) in pathology of the kidneys, gastrointestinal tract, bone diseases (Fanconi syndrome, canalic acidosis, chronic glomerulonephritis and pyelonephritis in the stage of severe chronic renal failure (CRF), gastrointestinal diseases with malabsorption syndrome, biliary cirrhosis of the liver, puerperal and idiopathic osteopathies, rickets - secondary hyperparathyroidism; - as a result of the formation of parathyroid adenoma in conditions of prolonged existence of secondary hyperpathyreosis - tertiary hyperparathyroidism; - due to the production of PTH by a tumor of non-parathyroid origin - pseudohyperparathyroidism.

Supporting signs of hyperparathyroidism:

- damage to the musculoskeletal system: muscle weakness, pain in muscles and bones, aggravated by movement and palpation, "duck" gait, skeletal deformity, osteoporosis, loosening and loss of teeth, cystic formations in the jaw, pathological bone fractures;

- damage to the urinary system: polyuria, decreased specific gravity of urine, nephrolithiasis;

- damage to the gastrointestinal tract: loss of appetite, nausea, flatulence, constipation, symptomatic gastroduodenal ulcers, bleeding, pancreatocalcinosis;

- neuropsychiatric disorders: irritability, tearfulness, depression;

- laboratory signs: increased levels of PTH in the blood, hypercalcemia, hyperphosphatemia, increased activity of alkaline phosphatase, hypercalciuria. Hyperparathyroidism is differentiated from other diseases and conditions accompanied by hypercalcemia (osteolytic metastases of malignant tumors in the bone, multiple myeloma, lymphomas, lymphogranulomatosis).

Figure #20. Key signs of hyperparathyroidism

HYPOPARATHYROISIS Pathology is caused by absolute or relative insufficiency of PTH. A decrease in calcium in the blood leads to seizures. Violation of the intake of calcium into the body or an increase in its excretion from the body may be due to insufficient absorption of calcium in the intestine or a violation of the kidneys. Absolute insufficiency of PTH is associated with a

violation of its production in case of damage to the parathyroid glands (aplasia or hypoplasia of the parathyroid glands, postoperative hypoparathyroidism, radiation therapy, hemorrhages in the parathyroid glands, infectious lesions of the parathyroid glands). Relative insufficiency of PTH is associated with tissue insensitivity with normal or even increased production (adenylate cyclase-dependent or adenylate cyclase-independent insensitivity of target organs - Albright's hereditary osteodystrophy).

Supporting signs of PTH deficiency:

- convulsive contractions of various muscle groups: skeletal (fibrillar twitching, tonic convulsions, paresthesia), respiratory (laryngospasm, bronchospasm), gastrointestinal tract (dysphagia, vomiting, diarrhea or constipation);

- increased neuromuscular excitability: positive symptoms of latent spasmophilia (Khvostek, Trousseau, Schledenger (cramps in the extensor muscles of the thigh and supination of the foot with rapid passive flexion of the leg in the hip joint with a straightened knee joint), Maslov's phenomenon, etc.);

- vegetative disorders: dizziness, headaches, fever, chills, palpitations, pain in the heart area; - trophic disorders: defects in tooth enamel, brittle nails, impaired hair growth, early graying;

- mental disorders: neurosis, depression, memory loss;

- laboratory signs: decrease in the level of PTH in the blood, hypocalcemia, hyperphosphatemia, hypocalciuria. Hypoparathyroidism must be distinguished from diseases and syndromes accompanied by convulsions and/or hypocalcemia (rickets (spasmophilia), malabsorption syndrome, chronic renal failure syndrome, vomiting, epilepsy).

DIABETES INDIABETES The syndrome is caused by a deficiency of antidiuretic hormone (ADH). ADH deficiency can be absolute (impaired secretion of ADH by the hypothalamus) or relative (insensitivity of the renal tubular epithelium to ADH).

Reference signs of ADH deficiency:

- polydipsia (the amount of fluid drunk ranges from 3 to 20 liters per day);

- polyuria (frequent, profuse urination);

- developing in connection with polyuria and polydipsia sleep disturbance;

- general dehydration (dry skin, mucous membranes, decreased salivation and sweating);

- decrease in the specific gravity of urine to 1001-1004, plasma hyperosmolality (more than 290 mosm / kg), hypernathyremia (more than 155 mmol / l), urine hypoosmolality (100-200 mosm / kg);

- ADH in the blood: with an absolute deficiency - a decrease, with a relative - the norm or increased. Causes of diabetes insipidus: congenital diabetes insipidus (familial and non-familial), neoplastic, traumatic, inflammatory, ischemic, autoimmune lesions of the hypothalamus, nephrogenic diabetes insipidus, kidney amyloidosis. Diabetes insipidus is differentiated with a syndrome of diabetic symptoms, psychogenic thirst, a polyuric phase of chronic renal failure.

SYNDROME OF DIABETIC SIGNS Symptom complex develops as a result of absolute or relative insulin deficiency, which leads to disruption of all types of metabolism, primarily carbohydrate metabolism, damage to blood vessels (angiopathies), nervous system (neuropathies), as well as other organs and tissues.

"Big" (early) diabetic signs:

- polydipsia (intense thirst);

- polyuria (frequent, profuse urination);

- polyphagia (increased appetite);

- weight loss; - hyperglycemia, ketoacidosis;

- glycosuria. "Small" diabetic signs:

- skin itching, especially in the perineum and genitals;

- furunculosis and susceptibility to infectious diseases;

- frequent lesions of the oral mucosa;

- periodontitis;

- impaired tolerance to carbohydrates;

- elevated blood glucose during an oral glucose tolerance test (mmol / l).

Hypoglycemia. The syndrome is caused by a drop in the concentration of glucose in the blood below the minimum level to which the body is adapted (below 2.8 mmol / l). Symptoms of hypoglycemia can be divided into two groups: - sympathetic (adrenergic), caused by increased secretion of catecholamines: weakness, sweating, tachycardia, tremor, nausea, vomiting, hunger, irritability, irritability, anxiety, tingling of lips and fingers; - neurological, caused by dysfunction of the central nervous system: headache, hypothermia, fog before the eyes, diplopia, decreased attention, strange behavior, lethargy, stupor, amnesia, convulsions, coma. In patients with diabetes mellitus, the clinical picture of hypoglycemia may develop with a rapid decrease in blood glucose concentration from a very high level to a lower, but significantly higher than normal (for example, from 20-23 mmol / l to 10-11 mmol / l), while in there is enough glucose in the blood, but it does not enter the cells.

Types of hypoglycemia:

- reactive - the level of glycemia on an empty stomach is within the normal range, after eating it is reduced;

- alimentary - late dumping syndrome in patients operated on the stomach;

- idiopathic - increased vagal tone, gastric emptying rate and insulin secretion rate, "early" diabetes mellitus. Characteristic is the low level of glycemia 2–4 hours after exercise during the study of the glucose tolerance test;

- fasting hypoglycemia (fasting hypoglycemia) - manifests itself in the form of more pronounced and prolonged symptoms, often neuroglycopenic. Hypoglycemia is typical 5 hours after a meal or after an overnight fast. It is caused by a decrease in the absorption of glucose and its peripheral utilization, a low level of insulin and an increase in the secretion of contrainsular hormones. It is observed in insulinoma, islet cell hypoplasia, extrapancreatic tumors (gastrointestinal tract, mesothelioma, sarcoma, adrenal cancer, hepatocellular carcinoma), adrenal, renal and hepatic insufficiency. It should be remembered that with insulinoma, adrenal insufficiency and the formation of autoantibodies to insulin, hypoglycemia can occur both on an empty stomach and after eating.

Iatrogenic (drug) hypoglycemia can be caused by the administration of insulin, sulfonamides - rare forms of hypoglycemia in leukemia, leukemoid reactions, hyperlipidemia, severe glycolysis during blood sampling. Hypoglycemic coma is an extreme manifestation of hypoglycemia, which develops with a rapid decrease in the concentration of glucose in the blood, followed by its decrease in the brain tissue. Provoking factors - an overdose of hypoglycemic drugs (often insulin), untimely meals after the administration of drugs or eating with insufficient carbohydrates; increased sensitivity to insulin (especially in children and adolescents), intense muscle load; psycho-emotional stress; acute infectious diseases; postpartum period; change in diet in diabetic patients; liver dysfunction in diabetic patients; alcoholism; diseases of the endocrine system (deficiency of contrainsular hormones, insulinoma).

Supporting signs of hypoglycemic coma:

1) acute onset and rapid development;

2) pronounced adrenergic and neurological symptoms of hypoglycemia;

3) increased tendon and periosteal reflexes, the appearance of the Babinsky symptom, clonic-tonic convulsions are possible, the tone of the eyeballs is increased, tachycardia, blood pressure is normal or increased; stupor develops, turning into a deep stupor;

4) in the stage of deep coma, areflexia, cardiac arrhythmias, arterial hypotension, abnormal breathing appear.

Cushing's syndrome. Hypercorticism is a state of excess glucocorticoids of any origin.

Supporting signs of Cushing's syndrome:

- dysplastic obesity (fat deposits are more pronounced on the trunk, moon-shaped face, supraclavicular fat pads, "buffalo scruff", relatively thin limbs);

- trophic changes in the skin (thinning of the skin with a blush on the face, purple stretch marks - striae, easy bruising);

- arterial hypertension, myocardial damage syndrome;

- muscle weakness, atrophy of the proximal muscles;

– osteoporosis;

- steroid diabetes or impaired glucose tolerance;

- dysfunction of the sex glands;

- steroid encephalopathy and emotional and mental disorders (emotional lability, excitability, depression, sleep disorder);

- secondary immunodeficiency (poor wound healing, increased frequency of infectious diseases, pustular and fungal skin diseases);

- laboratory indicators indicating an increase in daily production of cortisol: an increase in the level of cortisol in the blood, an increase in total 17-OCS in the urine.

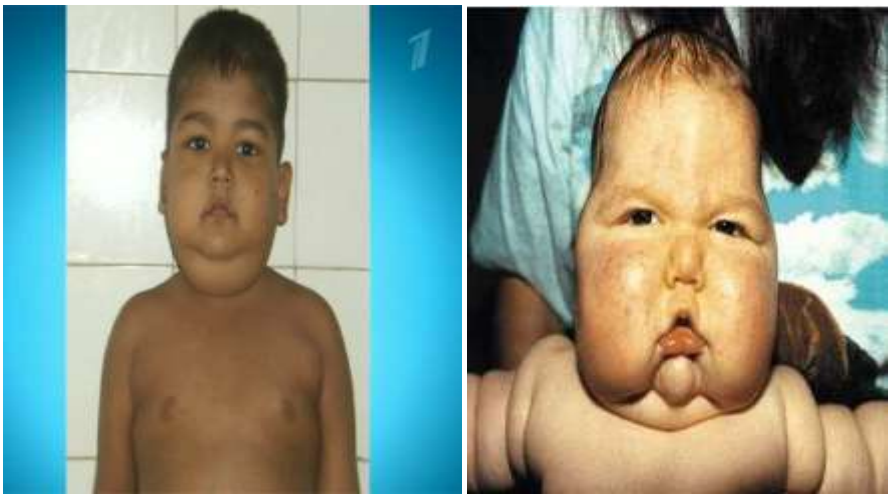

Figure #21. View of a sick child with Cushing's syndrome

In clinical practice, different variants of hypercortisolism should be differentiated: - endogenous - Itsenko-Cushing's disease (tumor of the pituitary gland), tumor of the adrenal cortex (corticosteroma-blastoma) - Itsenko-Cushing's syndrome; juvenile dysplasia of the adrenal cortex; ACTH-ectopic syndrome (tumor of the bronchi, pancreas, thymus, liver, intestines, etc., secreting corticotropin-releasing hormone or ACTH); - exogenous - long-term treatment with synthetic corticosteroids (iatrogenic Itsenko-Cushing syndrome); - functional - hypothalamic syndrome, pubertal and youthful dispituitarism, diabetes mellitus, alcoholism, obesity, pregnancy.

SYNDROME OF ADRENAL INSUFFICIENCY. HF develops in acute or chronic hypofunction of the adrenal cortex and is manifested by a deficiency of glucocorticoids or (less often) mineralocorticoids. HNS can be primary (due to a primary lesion of the adrenal glands, in which more than 90% of the cells of the cortical substance that produce corticosteroids are destroyed) and secondary (due to diseases of the hypothalamus and pituitary gland, leading to a deficiency of corticoliberin or adrenocorticotropic hormone). Iatrogenic adrenal insufficiency occurs upon discontinuation of glucocorticoid therapy or a rapid drop in the level of endogenous glucocorticosteroids against the background of prolonged inhibition of the hypothalamic-pituitary-adrenal system by exogenous or endogenous glucocorticoids.

Supporting signs of chronic adrenal insufficiency:

- weakness, fatigue, weakness, asthenia;
- weight loss;
- arterial hypotension;
- dyspeptic disorders (nausea, vomiting, anorexia, abdominal pain);
- hyperpigmentation of the skin and mucous membranes (with primary adrenal insufficiency);
- hyponatremia, hyperkalemia (with primary adrenal insufficiency), a decrease in the content of cortisol in the blood, a decrease in the excretion of 17-OKS and free cortisol in the urine. In clinical practice, it is necessary to distinguish between primary and secondary adrenal insufficiency. The causes of primary adrenal insufficiency are idiopathic autoimmune atrophy of the adrenal cortex, tuberculosis, hemochromatosis, syphilis, brucellosis, scleroderma, tumor metastases of the lungs, mammary glands, etc. to the adrenal glands, adrenalectomy for adrenal tumors, Itsenko-Cushing's disease, adrenal necrosis in patients with HIV.

The causes of secondary adrenal insufficiency are tumors, hemorrhages, injuries of the hypothalamic-pituitary region, infectious (syphilis) and non-

infectious (sarcoidosis, granulomatous hypophysitis, autoimmune hypophysitis) processes in the pituitary and hypothalamus.

Acute adrenal insufficiency occurs when a sudden sharp decrease (or cessation) of the secretion of hormones of the adrenal cortex. Provoking factors - physical or mental stress in patients with chronic adrenal insufficiency (especially with inadequate replacement therapy); meningococcal, streptococcal, pneumococcal infection; sepsis, burns; treatment with adrenocorticotropic hormone and anticoagulants; pregnancy; sudden withdrawal of corticosteroids in patients who have received these drugs for a long time.

Supporting signs of acute adrenal insufficiency:

- acute vascular insufficiency syndrome (collapse), muffled heart sounds, hypothermia;

- anorexia, nausea, indomitable vomiting, diarrhea, dehydration, abdominal pain;

- convulsions, meningeal symptoms, lethargy, stupor.

OBESITY

The syndrome develops as a result of excessive accumulation of fat (adipose tissue) in the body, leading to an increase in body weight of more than 20% compared to the average normal values. Obesity is an energy imbalance in which the intake of calories from food exceeds the energy expenditure of the body. Possible determinants of obesity can be genetic, demographic, socioeconomic, psychological, behavioral, metabolic, hormonal factors, or (most often) a combination of them. By the nature of the distribution of adipose tissue, obesity is distinguished according to the upper type (central, abdominal, android), the lower type (gynoid, gluteal-femoral) and mixed (intermediate). To diagnose obesity and determine its degree, body mass index (BMI) or Quetelet index is used. BMI is not only a diagnostic criterion for obesity, but also an indicator of the relative risk of developing diseases associated with obesity (diabetes mellitus, arterial hypertension, osteoarthritis). The index is calculated as the ratio of body weight (in kg) to height (in meters) squared. Basic signs of obesity: - an increase in body

weight by more than 20% of the norm; - body mass index - an indicator of the ratio of waist circumference to hip circumference for the abdominal type of obesity (with a BMI less than 35) is more than 0.9 in men and 0.83 in women; - waist circumference (with abdominal type of obesity) more than 94 cm in men and 80 cm in women. The causes of obesity are the wrong stereotype of nutrition (especially in combination with low physical activity), psychogenic disorders (bulimia nervosa, depression, night eating syndrome, etc.), hypothalamic lesions, Itsenko-Cushing's disease and syndrome, hypothyroidism, hypogonadism, insulinoma, medication drugs (corticosteroids, antipsychotics, tricyclic antidepressants, antiserotonin drugs, medroxyprogesterone). Obesity must be distinguished from massive edema (anasarca).

Figure #21. A child with metabolic obesity.

SEMIOTICS OF DISORDERS OF SEXUAL DEVELOPMENT

Puberty is the period during which secondary sexual characteristics appear. It is not clearly limited in duration, but usually lasts about 2-3 years. The onset of puberty has wide age limits. In 95% of healthy girls, secondary sexual characteristics begin to appear at the age of 8.5–13 years, in 95% of healthy boys at the age of 9.5–13.5 years. The initial manifestation of puberty in 85% of girls is the growth of the mammary glands (thelarche), in 15% pubic hair begins to grow. The

first menstruation (menarche) usually occurs 18–24 months after the onset of breast growth (mean age 12.8 years, ranges from 10 to 16 years). In the first 1–2 years after menarche, cycles may be anovulatory. Intensive body growth in girls (growth jump is about 25 cm), in contrast to boys, begins early, in parallel with the increase in mammary glands, and ends with the onset of menarche. Sexual development in boys occurs gradually and begins with the disappearance of subcutaneous fat in the scrotum, the appearance of its pigmentation and numerous small folds. The testicles enlarge and descend to the bottom of the scrotum, the growth of the penis begins. Pubic hair at first has a female type, from 16–17 years old it passes to a male one. Hair appears in the axillary regions, the fluff above the upper lip, on the cheeks and chin is gradually replaced by rod hair. The size of the prostate gland changes, the breast tissue begins to grow, the nipples become conical (juvenile gynecomastia), they become larger, the areola are pigmented. The larynx increases, voice mutation occurs, acne appears. The processes of spermatogenesis are intensifying, although a full-fledged reproductive function is formed much later - by the age of 16–17. The growth spurt (about 28 cm) begins in the middle of puberty and ends in its final stage. The reproductive system is represented by the sex glands (gonads) and additional (auxiliary) genital organs. In men, these include the testes, vas deferens, seminal vesicles, prostate, and penis; in women - ovaries, fallopian tubes, uterus, vagina, external genitalia. The mammary glands also belong to the female reproductive system. In case of violation of the development of the genital organs, it is possible, upon examination, to see their uncertain (intersex) state, the presence of anomalies in the structure.

Premature (early) sexual development (PPR). Puberty is considered premature if it begins in boys under the age of 8 years, in girls - up to 7 years. PPR is distinguished true (early appearance of secondary sexual characteristics corresponding to the sex of the child with an increase in the size and activity of the gonads) and false (the gonads do not mature, secondary sexual characteristics may correspond (isosexual) or not correspond (heterosexual) to the sex of the child).

True isosexual PPR is due to an increase in the secretion of GnRH,

hypersecretion of gonadotropic hormones, and dysregulation in the hypothalamic-pituitary system. Signs of this disorder in boys are an increase in the size of the testicles, growth of facial, pubic and armpit hair, the appearance of acne, breaking and coarsening of the voice, rapid (but stopping early) body growth, spermatogenesis is established early. Girls develop mammary glands, feminine fat deposition is noted, menstruation appears, an ovulatory menstrual cycle is established and the ability to conceive. The reasons may be anomalies of the organs of the reproductive system, thyroid disease, brain tumors, changes caused by head injuries, the consequences of infectious diseases (meningitis, encephalitis), a number of genetic factors.

True PPR is considered idiopathic if its cause cannot be identified. It is believed that idiopathic PPR is due to early activation of impulse secretion of GnRH. CNS diseases are the most common cause of true PPR. Other causes are traumatic brain injury, anomalies in the development of the skull and brain. Sometimes a familial nature of the PPR (constitutional PPR) is found. False PPR is due to autonomous hypersecretion of androgens. False PPR is manifested by the same symptoms as the true one, but is not accompanied by an increase in testicles in boys and ovaries in girls.

Heterosexual false PPR is characterized by the appearance of signs of the opposite sex in the child. The most common clinical form of heterosexual PPR is a pathology called "adrenogenital syndrome", which is known as congenital adrenal hyperplasia, false female hermaphroditism or heterosexual female PPR. Incomplete forms of PPR include: 1) premature adrenarche - the appearance of pubic or axillary hair growth in boys and girls 5-6 years old. There may be acne, a strong smell of sweat and a coarsening of the voice. Accelerated growth and maturation of the skeleton and increased there is no penis. Due to the early increase in the secretion of adrenal androgens; 2) premature menarche - the development of several episodes of uterine bleeding in girls; 3) premature thelarche - isolated premature development of the mammary glands. Hypogonadism. Persistent, often irreversible damage to the reproductive system with a decrease in the production of

sex hormones is called hypogonadism. Primary (hypergonadotropic) hypogonadism develops as a result of damage to the ovaries or testicles. Secondary hypogonadism is the result of damage to the pituitary gland, and tertiary hypogonadism is the result of damage to the hypothalamus. Primary hypogonadism can be congenital (with some hereditary syndromes, such as Klinefelter's syndrome, Noonan's syndrome) and acquired. In both cases, testosterone deficiency leads to an increase in the levels of follicle-stimulating and luteotropic hormones, however, this increase is usually detected only during adolescence.

Klinefelter syndrome occurs in 1 in 500 boys. Patients with the classic variant of the syndrome have a 47,XXY karyotype. Other karyotypes are also possible, and 10% of patients have 46,XY/47,XXY mosaicism. The syndrome usually manifests in adolescence as a delay in sexual development: the penis and testicles are reduced, the physique is eunuchoid, there is gynecomastia and moderate mental retardation. Patients are predisposed to diabetes mellitus, diseases of the gland and breast cancer.

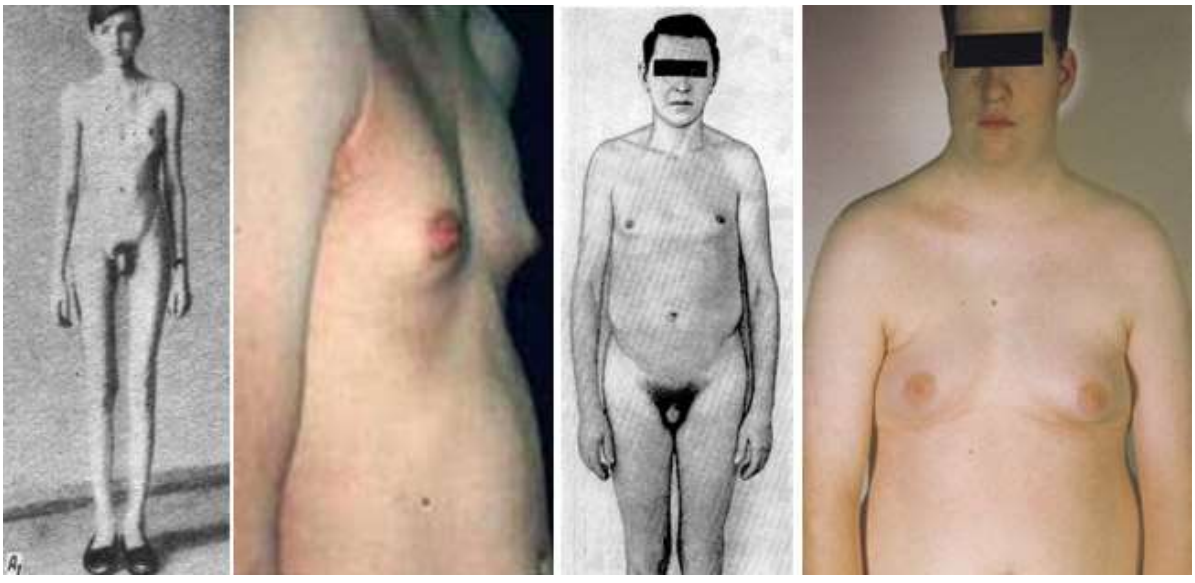

Figure #22. Type of patient with Klinefelter's syndrome

Noonan syndrome occurs in 1 in 8,000 newborns; karyotype is normal. Inheritance is autosomal dominant. According to clinical manifestations (pterygoid folds on the neck, valgus deformity of the elbow joints, short stature, lymphatic edema of the hands and feet), Noonan's syndrome is similar to Turner's syndrome.

Other signs of Noonan syndrome are ptosis, a sunken chest, right heart defects (pulmonary stenosis), a triangular face, and mental retardation. Boys have cryptorchidism or micropenia. Acquired primary hypogonadism (acquired testicular insufficiency) may be the result of viral orchitis (mumps virus, Coxsackie B virus and ECHO viruses), the use of antitumor agents, targeted radiation, etc.

Figure #23. Type of patient with Noonan syndrome.

With secondary hypogonadism (secondary testicular insufficiency), sexual development begins with a great delay and proceeds slowly, or begins but does not end. Patients are at high risk of infertility. The child grows normally until adolescence, then growth slows down, and signs of sexual development do not appear. Adolescents with this pathology have a eunuchoid physique. Isolated deficiency of gonadotropic hormones as a separate disease is rare and is inherited in an autosomal recessive manner in about half of the cases.

Much more often, an isolated deficiency of gonadotropic hormones is combined with other developmental anomalies: loss or decrease in the sense of smell (with Kalman syndrome), defects in the median structures of the brain and skull, micropenia, cryptorchidism, color blindness, anomalies of the kidneys and metacarpal bones.

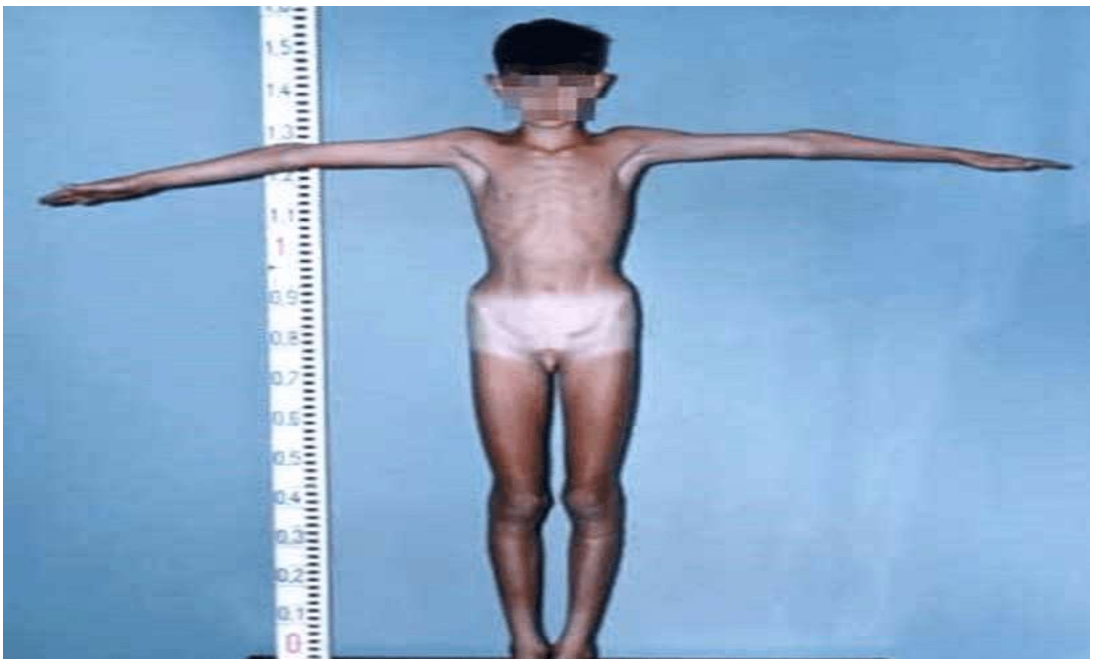

Figure #24. View of a sick child with hypogonadism.

Idiopathic hypopituitarism. Under this name, several sporadic and hereditary diseases are combined. Congenital idiopathic hypopituitarism presents in neonates with severe hypoglycemia, hyponatremia, and hepatitis-like liver lesions. Micropenia is almost always observed. Idiopathic hypopituitarism in older boys in 50–60% of cases is the result of birth trauma and hypoxia.

Causes of dysfunction of the hypothalamus and pituitary gland:

- Tumors (craniopharyngioma, astrocytoma, optic nerve glioma, dysgerminoma, teratoma, etc.);

- developmental anomalies (defects in the median structures of the brain and skull, hydrocephalus);

- infections (meningitis, encephalitis);

- traumatic brain injury;

- Radiation therapy for leukemia and brain tumors.

Delayed sexual development (DSD). They say about ZPR if there is a delay in the appearance of signs of puberty by more than 2 years compared to the average terms. Constitutional retardation of growth and sexual development is a variant of the norm. It is caused by a delay in the activation of the hypothalamic-pituitary-gonadal system. Due to low levels of LH, FSH, and testosterone, puberty

can begin at age 15 or later. Chronic systemic diseases can cause stunted growth, skeletal maturation, and sexual development. These diseases include chronic renal failure, cystic fibrosis, celiac disease, bronchial asthma, chronic inflammatory bowel disease, severe hypothyroidism. CNS diseases (cerebral forms) can also cause ZPR.

Cerebral disorders of an organic nature: traumatic, toxic, infectious lesions (encephalitis, epilepsy, tumors of the hypothalamic region) - cause ZPR if the hypothalamus is involved in the process. Cerebral forms include ZPR in psychoses, neuroses. One of the cerebral forms of mental retardation - anorexia nervosa (anorexia nevrosa) - is regarded as a neurotic reaction in response to pubertal changes in the body. ZPR closely adjoins this form against the background of weight loss. Suspicion of ZPR can be caused by the presence of the following signs: a “frail” physique, relatively long limbs, a high waist, often hips wider than shoulders. Typical deposition of subcutaneous fat on the chest, waist, lower abdomen. The genitals are not developed, the boys do not have folding and sagging of the scrotum, there are no pollutions; girls have amenorrhea. Patients do not grow hair on the pubis and under the arms. ZPR should not be identified with primary amenorrhea (absence of menarche at the age of 16 years and older). ZPR is a broader concept and primary amenorrhea is often a symptom of it.

Lack of sexual development (LSD). Absence of menarche, development of the mammary glands, genital and axillary hair growth after the age of 16 are symptoms of OPD. The reason is aplasia of the gonads or a malformation of their development, in which there is no functionally active hormone-producing ovarian tissue. Sexual development does not occur in girls who, according to indications, underwent ovarian removal before the age of 8–10 years. Another cause of OPD is a genetically determined malformation of the sex glands called gonadal dysgenesis. Gonadal dysgenesis is a rare pathology, its frequency is 1 case 36 per 10-12 thousand newborns. The reason for the development of gonadal dysgenesis are chromosomal disorders in the form of the loss of one X chromosome or part of it. Most often there are 4 clinical forms of gonadal dysgenesis.

Shereshevsky-Turner syndrome. This form is characterized by a 45,X karyotype. Patients differ in specific appearance. Mandatory symptoms are short stature (up to 150 cm), a short wide neck with a low hairline, skin neck folds, high palate, low-lying auricles, deformity of the elbow joint, widely spaced nipples of the mammary glands, multiple pigment spots on the skin. Variable signs are malocclusion, strabismus, third eyelid, thyroid chest, pterygoid scapulae. There are anomalies in the development of the kidneys and ureters, malformations of the cardiovascular system. The external and internal genital organs are hypoplastic, there is a pronounced sexual infantilism. According to ultrasound, the gonads are thin (about 0.3–0.5 cm wide) connective tissue strands.

Figure #25. Newborn baby with Shereshevsky-Turner syndrome

Erased form of gonadal dysgenesis. Most often, with this type of gonadal dysgenesis, it has a mosaic character 45,X/46,XX. The severity of clinical manifestations depends on the percentage of normal and aberrant cell clones. The higher the percentage of clone 45,X, the closer the patients in appearance and clinical picture of the disease to patients with Shereshevsky-Turner syndrome. They have low growth, there may be insufficient, but spontaneous development of secondary sexual characteristics in the presence of primary amenorrhea; in 20% of patients, menarche occurs in normal terms, and in 10% of patients, relatively

regular menstruation is noted for about 10 years after menarche, which then turn into oligomenarea and secondary amenorrhea. The external and internal genital organs are hypoplastic. Ultrasound and laparoscopy revealed sharply hypoplastic gonads.

A pure form of gonadal dysgenesis. This clinical variant is characterized by normal growth, the absence of somatic malformations, underdeveloped secondary sexual characteristics, and pronounced sexual infantilism. The physique varies from eunuchoid to intersex. The karyotype of patients is most often 46,XX or 46,XY (Swyer's syndrome). Gonads are fibrous strands, which sometimes contain elements of stroma. Mixed form of gonadal dysgenesis. Patients with this form are characterized by normal body length, intersex physique and elements of virilization of the external genital organs: a slight increase in the clitoris, the fusion of the lower third of the vagina and the urethra. They also have developmental anomalies characteristic of the Shereshevsky-Turner syndrome. In the karyotype of these patients, with a wide variety of mosaic sets, the Y chromosome or its fragment is necessarily present. The most common karyotype is 45,X/46,XY. The gonads in these patients have a mixed structure. In girls, during laparotomy, a fibrous cord is found on one side, and an underdeveloped testicle on the other. During puberty, these patients often develop gonadal tumors. By the age of 20–25 years, tumors develop in most patients with a mixed form of gonadal dysgenesis.

SEMIOTICS OF DAMAGE TO THE SKIN AND SUBCUTANEOUS FIBRE Numerous functions, the closest physiological connection with various organs and systems make the skin a kind of screen that reflects numerous pathological processes in the body. Therefore, the correct assessment of her condition is of great practical importance in diagnosis.

When pathological changes in the skin are detected (discoloration, rash, violation of integrity, scars, peeling, etc.), it is necessary to find out:

1) when certain changes appeared;

2) how quickly the change in skin color appeared;

3) where the first elements of the rash appeared, how they looked, whether

they were single or multiple;

4) what is the rate of spread of the rash, its localization, symmetry;

5) how the rash changed over time (changes in color, shape, size of elements, the appearance of peeling);

6) whether skin changes were accompanied by a temperature reaction;

7) whether the child was in contact with an infectious patient, whether similar rashes were noted earlier.

Semiotics of skin color.

The color of a child's skin is determined by the relative content of melanin, oxyhemoglobin, reduced hemoglobin and carotene, the thickness of the stratum corneum, and the degree of blood supply. Melanin is the main pigment that determines the color of the skin, hair and eyes. Normally, skin color is determined by genetic or constitutional factors and persists in some areas of the body (buttock area), since the skin is not exposed to external influences, or its color changes under the influence of sunlight (sunburn), as a result of increased pigmentation under the influence of hormones that stimulate melanocytes.

Semiotics of pigmentation Disturbances in the melanocyte system are divided into hypermelanosis (increase in melanin in the epidermis or dermis) and hypomelanosis (reduction or absence of melanin in the dermis, leukoderma), which in turn can be generalized or localized. Some of these disorders are due to hormonal changes (Addison's disease), others are local developmental defects (white spots in tuberous sclerosis) or the result of skin inflammation (post-inflammatory hypo- or hyperpigmentation).

Hypermelanoses. They are divided into epidermal (brown color) and dermal (blue, bluish-gray, gray color). Brown hypermelanoses (melanoderma) are associated with an increase in melanin content in the epidermis as a result of increased activity of melanocytes, an increase in the number of secretory melanocytes, the number of melanosomes, or their size.

Figure #26. View of a sick child with symptoms of hypermelanosis

Bluish-gray hypermelanoses (ceruloderm, blue skin) are similar to false tattoo melanin and are due to the presence of melanin in the dermis, in ectopic dermal melanocytes or dermal macrophages, which, as a result of the Tyndall effect, give the skin a characteristic gray, grayish blue or blue color. Hyperpigmentation of the skin can be generalized uniform, generalized patchy or limited, in separate areas of the skin.

Diffuse congenital melanosis is characterized by hyperpigmentation already at birth (other organs are unchanged), later hyperkeratosis of the hands joins. Constitutional hyperpigmentation is observed in dark blond children, mainly of the peoples of the Mediterranean.

Diffuse brown hypermelanosis is characteristic of adrenal insufficiency (Addison's disease), in which hyperpigmentation of the skin is expressed in places of pressure on it (vertebrae, interphalangeal, elbow and knee joints), in the folds of the body, on the palmar surfaces, the mucous membrane of the gums. Adrenalectomy, tumors of the pancreas and lungs lead to hypermelanosis. Under these conditions, hyperpigmentation is due to overproduction of melanocyte-stimulating hormone and ACTH, in which the amino acid sequence is the same.

Generalized hypermelanosis is a typical sign of hemochromatosis, late cutaneous hematoporphyria, and variegated porphyria. In hemochromatosis, the

hyperpigmentation is greyish-brown or brown and indistinguishable from that of Addison's disease, and the diagnosis can be made by skin biopsy showing hemosiderin deposits in the sweat glands and melanin deposits. With late cutaneous porphyria, the diagnosis is established by vesicles, blisters, atrophic spots, sclerodermoid changes and a millet-like rash on the skin of exposed parts of the body, and is confirmed by the presence of an increased content of uroporphyrin in the urine (the ratio of uroporphyrin and coproporphyrin is normally more than 3:1) or red fluorescence of acidified urine. Skin changes in variegated porphyria are identical to those in cutaneous porphyria tarda, differentiate them by a different response to treatment.

Figure #27. View of a sick child with nevoid hyperpigmentation

Hyperpigmentation of healthy skin is observed in chronic renal failure, primary biliary cirrhosis. With chronic nutritional deficiency (kwashiorkor, nephrotic syndrome, malabsorption syndrome, etc.), hyperpigmented spots appear on the skin of the trunk. With pellagra, the pigmentation zone is limited to areas of the skin exposed to light or trauma; vitamin B12 deficiency is accompanied by premature graying of the hair and hypermelanosis, especially pronounced around

the small joints of the hands. Hypermelanosis may be a consequence of treatment with myelosan, cyclophosphamide, methylurea, chlorpromazine, etc.

Hyperpigmented skin elements.

Freckles (ephelids) are small pigment spots located at the level of the skin on the face on both sides of the nose, on the shoulders. Larger coffee-au-lait spots can be a manifestation of Recklinghausen's neurofibromatosis, in which neurofibromatosis of the skin and peripheral nervous system, hypertension, and precocious puberty are found.

Leopard syndrome is an autosomal dominant condition with a generalized distribution of dark brown spots in combination with neurosecretory deafness, growth retardation, heart defects, and genital anomalies.

A blue nevus is a group of pigment cells that have accumulated in the dermis. The epidermis translucent above them looks like bluish spots. When localized in the region of the sacrum, they are called Mongolian spots, which disappear after the age of 3 years. In children, there are also benign and malignant variants of melanoma (tumor-like, growing pigmented nevus).

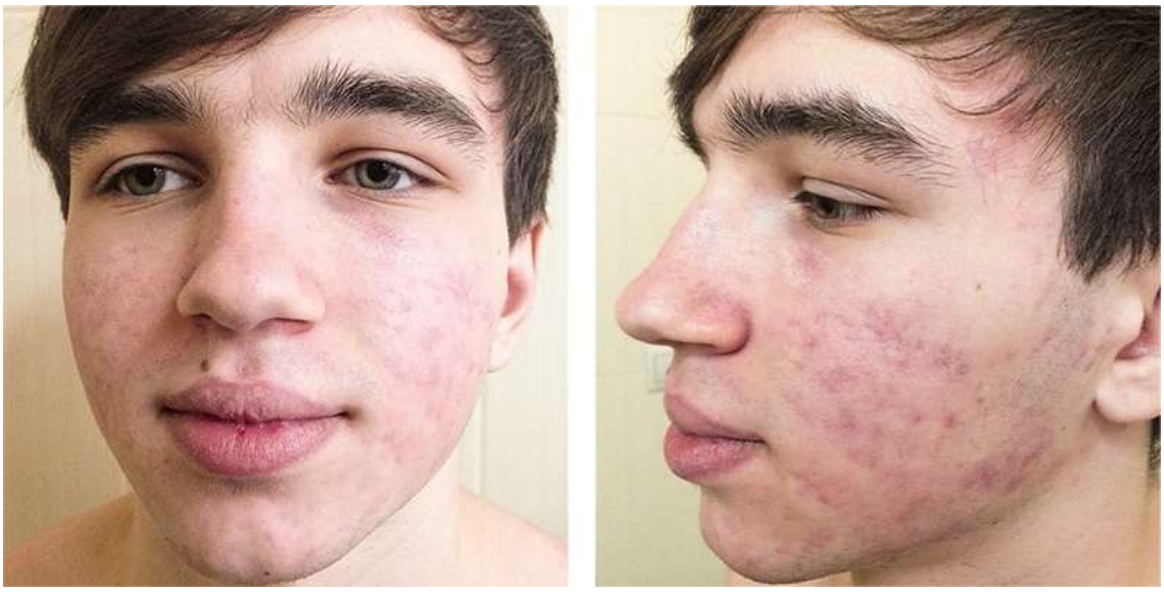

Figure #28. View of a sick child with a blue nevus

Mastocytosis, or urticaria pigmentosa, is a disease characterized by paroxysmal rashes of spots, papules, blisters of pink-red color of a round or oval shape, localized on the trunk, limbs, scalp, face and (rarely) on the palms and soles. Mastocytosis usually begins before the age of 2 years. The child is restless due to

severe itching, the mucous membranes are not affected. The symptom of Unna-Darye is considered pathognomonic for mastocytosis, when, when a spot or papule is rubbed with a spatula, or after touching a warm object, redness and swelling of the element soon appear - it takes on a blister-like appearance. Systemic signs of histamine release during mastocytosis (episodic "hot flashes", tachycardia, respiratory failure, headache, intestinal colic, diarrhea, hypotension) are almost constant.

Hypomelanosis. Observed in albinism, Hermansky-Goodluck syndrome (tyrosinase-positive albinism with platelet defects and hemorrhagic diathesis), Cross-McKusick-Brin syndrome (tyrosinase-positive albinism with microphthalmia, developmental delay, muscle hypertonicity and athetosis).

Partial albinism (Piebaldism), inherited by an autosomal dominant type, is characterized by amelanotic plaques in the frontal region, the front of the head (resulting in a white strand of hair), on the chest, in the knee and elbow joints. Plaques are caused by a local absence or decrease in the number of melanocytes and do not disappear.

Wardenburg's syndrome is inherited in an autosomal dominant manner, characterized by white strands of hair, pigment defects and hypopigmentation of the skin, heterochromic irises, a wide bridge of the nose, dystopic corners of the eyes, and congenital deafness.

Tuberous sclerosis is inherited in an autosomal dominant manner, characterized by small white leaf spots (1-3 cm), localized mainly on the trunk, fibromatous nodules on the skin of the forehead, trunk, arms and legs, mental retardation, epilepsy, tuberous nodules in the cortex and subependymal areas, retinal phakomatosis, heart rhabdomyomas, cysts of the kidneys, lungs and bones. Hypomelanosis of Ito (achromatic pigment incontinence) is a congenital disease characterized by bizarrely shaped hypopigmented macules that form well-demarcated patterns, stripes and plaques over the entire surface of the body, which disappear in adulthood. Vitiligo (acquired pigment defect) occurs at any age and is characterized by depigmented spots of various shapes and sizes with clear

boundaries, localized on the skin of the face (around the eyes and in the mouth), in the genital area, hands and feet, elbow and knee joints, upper half of the chest. Skin elements may spontaneously disappear, new spots may appear, permanent depigmentation is possible.

Figure #29. Albinism in a child

PALE SKIN. Paleness may depend on individual anatomical and histological features of the skin, low blood pressure. Paleness of the skin due to anemia, insufficiency of blood filling of peripheral vessels, and edema is most often observed. So, insufficient blood circulation in peripheral vessels may be due to a decrease in blood circulation in the periphery (centralization of blood circulation) or a decrease in cardiac output (acute left ventricular failure in diphtheria, pneumonia, endomyocarditis, pericarditis, aortic stenosis, etc.). The immediate causes of pallor of the skin can be glomerulonephritis, collapse, shock, fear, cold, pain, etc.

Diffuse pallor of the skin indicates a decrease in hemoglobin in the blood (impaired formation of red blood cells: iron deficiency, hypo-, aplastic and other anemia; acute or chronic bleeding: massive or microbleeding; increased destruction of red blood cells: hemolytic anemia, hemoglobinopathies). Pallor of the skin may be associated with thickening of the layers of tissues lying above the capillaries, with edema with severe hypoproteinemia (glomerulonephritis, nephrotic syndrome,

exudative enteropathy, malabsorption syndrome, burn disease), hypothyroidism, hypovitaminosis A, constitutionally caused thickening of the skin. It is important to distinguish pallor associated with a change in the qualitative or quantitative composition of the blood from pallor due to vasospasm - pseudoanemia: with true anemia, the mucous membranes become pale, with pseudoanemia they remain pink. In some conditions, pallor acquires a characteristic shade: yellow - with hemolytic anemia; waxy - with hypo- and aplastic anemia; the color of coffee with milk - with infective endocarditis; earthy gray - with purulent-septic diseases; greenish - with chlorosis. Pallor of the skin may be due to a deficiency of melanin in albinism, phenylketonuria (Felling's disease).

Icteric staining. Icteric staining of the skin and sclera is observed with hemolytic anemia (lemon-yellow hue), obstructive jaundice (greenish). In the initial stages of the disease, when bilirubin begins to accumulate in the skin, it acquires an orange tint. First of all, yellowness with true jaundice appears on the sclera, the lower surface of the tongue and soft palate. With false jaundice (due to the use of carrots, tangerines, tomatoes, quinacrine, etc.), only the skin is stained - carotene jaundice occurs, while the level of bilirubin in the blood is normal. The most intense yellow coloration with an overdose of carotene (deposited in the epithelium of the skin and mucous membranes) is observed in areas of the skin with a thicker epidermis (on the palms and soles); the sclera of the eyes, where the epithelium layer is very thin, remain white.

Skin cyanosis. Cyanosis is a bluish coloration of the skin and mucous membranes, which is determined by the state of the underlying network of capillaries and is detected during physical examination. The clinical severity of cyanosis correlates with the presence of more than 50 g/l of reduced (oxygenated) hemoglobin in capillary blood. Cyanosis appears when the oxyhemoglobin content falls below 85%. Cyanosis is difficult to distinguish in patients with severe anemia (hemoglobin < 70 g/l) even with a significant percentage of reduced hemoglobin and is unlikely in blood hemoglobin concentrations below 50 g/l. Detection of cyanosis can be difficult in patients with polycythemia who have elevated levels of

both saturated and reduced hemoglobin (red cyanosis). The severity of cyanosis is influenced by the density of the subcutaneous capillary network, skin thickness and extravascular skin pigments. There are total and regional cyanosis (perioral - around the mouth, cyanosis of the nasolabial triangle, distal parts of the body (acrocyanosis) - the tip of the nose, earlobes, lips, tip of the tongue, hands, feet). More often cyanosis is observed in diseases of the respiratory and cardiovascular systems. In lung diseases, cyanosis occurs as a result of the passage of blood through poorly ventilated areas of the lungs, while the amount of unsaturated hemoglobin increases as a result of a mismatch between ventilation and perfusion. In congenital heart defects, cyanosis is caused by intracardiac mixing of venous and arterial blood (right-to-left shunt). Peripheral cyanosis may occur as a result of a decrease in peripheral blood flow, the amount of unsaturated hemoglobin in the capillary bed increases due to an increase in oxygen extraction by tissues. Cyanosis in a healthy person can develop at high altitudes, where the partial pressure of oxygen in the inhaled air is reduced.

Central (warm, arterial) cyanosis is a condition in which the oxygen content in the circulating blood is less than 85%, warming the limbs and other parts of the body does not lead to their redness, and a bluish spot appears when pressure is applied to the skin. Central cyanosis divided into hemoglobin and methemoglobin. With hemoglobin cyanosis in the peripheral blood, the amount of reduced hemoglobin is increased.

This form of cyanosis is more common:

- with a shunt of blood from right to left (tetrad, pentad or triad of Fallot);

- reverse shunt (defects of the interventricular and interatrial septa, Ebstein's syndrome, patent ductus arteriosus, severe right ventricular failure). Cyanosis, more pronounced in the arms than in the legs, indicates transposition of large arteries with the presence of high coarctation or aortic stenosis, and the resulting pulmonary hypertension reduces the degree of shunt through the patent ductus arteriosus, resulting in more oxygenated blood to the extremities. Drumstick toes and cyanosis, more pronounced on the legs than on the left hand, while the right

hand is relatively normal in color, confirms the diagnosis of pulmonary hypertension with backflow of blood through the patent ductus arteriosus, resulting in less delivery to the lower extremities. oxygenated blood.

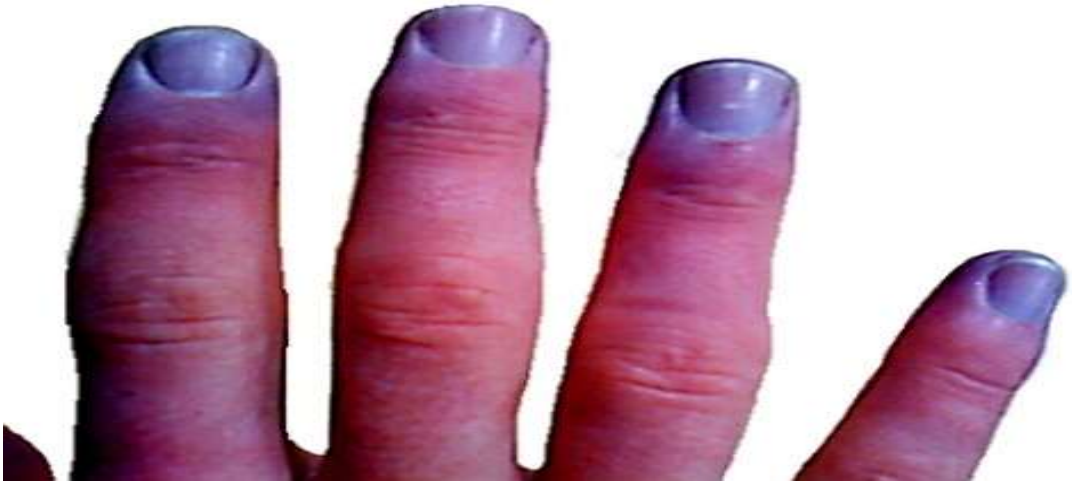

Figure 30. Severe cyanosis of the fingers.

Cyanosis appears with damage to the respiratory system (dyspnea with narrowing of the airways, restriction of the respiratory surface of the lungs, impaired respiratory movements, damage to the respiratory center) and depends on the severity of respiratory failure. More often it is observed in the syndrome of respiratory disorders in newborns, with pneumonia, atelectasis, pneumothorax, croup, etc.

Peripheral (cold, venous) cyanosis is a condition in which the blood in the arterioles of the cutaneous vascular plexuses has a normal oxygen content, warming the skin is accompanied by its reddening, and after pressure, a pink spot first appears, which later acquires a bluish tint. It is observed with a decrease in the minute volume of blood (heart failure, pericarditis, shelled heart), local stasis in the final sections of the bloodstream (cooling, collapse and shock of various origins, arterial embolism, polycythemia, etc.). In case of carbon monoxide poisoning, carbon monoxide, entering the blood, combines with hemoglobin iron, turning it into carboxyhemoglobin, at a blood concentration of more than 35 g / l, hemic hypoxia, respiratory failure and neurological disorders occur. Typically, patients have a bright red (cherry red) face, acrocyanosis, and severe cyanosis at a later stage. A large number of drugs and chemical compounds, including nitrates, cause

methemoglobinemia, while hemoglobin iron is oxidized to trivalent iron, which is not able to firmly bind oxygen. The cyanosis that develops at the same time gives the skin not so much a bluish, but a brownish tint. The blood is dark and chocolate-colored. In children, regardless of age (especially with a deficiency of glucose-6-phosphate dehydrogenase), sulfonamides in high doses can cause sulfhemoglobinemia, and 5 g / l of sulfhemoglobin in the blood is sufficient for the appearance of clinical signs of cyanosis.

Figure #31. Severe cyanosis of the nasolabial triangle

The light blue color of the skin in argyria may resemble cyanosis, but skin pigmentation disorders are also found in Addison's disease or hemochromatosis.

Figure #32. View of a sick child with Addison's disease

Skin hyperemia. Redness of the skin as a physiological phenomenon can occur under the influence of temperature (high or low), with mental arousal, increased physical activity, mechanical irritation of the skin, is temporary and is usually limited to one or more areas of the body.

Pathological hyperemia appears with erythrocytosis, diseases accompanied by fever, with exposure to atropine, alcohol poisoning, scopolamine or hallucinogens, with severe acetonemia, Kawasaki syndrome (mucocutaneous lymphatic syndrome with the periodic appearance of polymorphic erythema along with erythema on the palms and soles), harlequin syndrome in newborns, with fetofetal transfusion in identical twins. Limited hyperemia with localization on the cheeks, nose and around the eyes ("lupus butterfly") is typical for systemic lupus erythematosus, diffuse reddening of the face with characteristic pallor around the mouth is observed with scarlet fever, open areas of the skin turn red when exposed to solar radiation, with first degree burns. Local hyperemia accompanies foci of inflammation - inflamed joints, infiltrates, wounds. Vascular formations of the skin. The most common vascular formations of the skin are hemangiomas, among which there are superficial and deep located, undergoing regression and progressing.

Flat hemangiomas are flat, superficial, spots of various shapes and sizes, formed during the fusion of telangectasias, red in color, sometimes with a bluish tinge. They are located at the level of the surrounding healthy skin or slightly rise

above it. Vascular nevi are superficial, well-vascularized lesions that can be palpated.

Capillary hemangiomas (“flaming” nevi, “salmon” spots) are flat vascular malformations that change over time. A "flaming" nevus (a port-wine stain, a "port wine" stain) is a variant of a flat hemangioma with more pronounced vasodilation. Port-wine spots consist of mature dilated capillaries and are a permanent malformation, located asymmetrically on one half of the face or chest, sometimes on the mucous membranes of the oral cavity. A favorite localization is the back surface of the neck (nevus of Unna). The spots are bluish-red or crimson in color, have clear boundaries, vary widely in size. As the spots mature, they may rise and acquire a dense texture. Lighter elements may fade significantly over time. A stellate angioma has the appearance of nodules the size of a pinhead, dark red in color, from which dilated blood vessels spread in the form of rays. They are found in 15% of healthy preschool children and in about 45% of school-age children. Their favorite localization is on the forearms, back surfaces of the hands, face and auricles. They are observed immediately after birth and spontaneously disappear after a year, or they are removed using the application of liquid nitrogen or electrocoagulation.

Tuberous-cavernous hemangioma has the appearance of tumor-like vascular formations rising above the surface of the skin. It is localized more often on the face, scalp, less often on the limbs and buttocks, sometimes on the oral mucosa. The surface of the hemangioma is bumpy due to the bulging of the walls of varicose cavities, soft consistency, bluish-red color with a brownish tinge. Hemangiomas of various sizes, single or multiple, sometimes combined with lymphangiomas, flat hemangiomas.

Cavernous angiomas go through phases of growth, a stationary period, and a period of involution. The outcome cannot be predicted by the size and location of the mass.

Kasabach-Merritt syndrome occurs after birth or in the first weeks of life and presents with hemangioma, thrombocytopenia, and anemia. In typical cases,

hemangiomas are solitary and large. Vascular lesions are usually located on the skin and rarely - in the internal organs. Thrombocytopenia is due to sequestration or increased destruction of platelets in the hemangioma. Associated thrombocytopenia can lead to profuse bleeding associated with ecchymosis, petechiae, and rapidly enlarging hemangioma. Anemia may develop. Relatively constant signs are hypofibrinogenemia and a decrease in the level of blood coagulation factors.

Klippel-Trenaunay-Weber syndrome presents as a patchy vascular nevus (port wine-colored patches) in combination with soft tissue and bone hypertrophy and varicose veins, making up the triad of symptoms of this inherited autosomal dominant condition. The anomaly may be limited to an increase (macromelia) of one arm or leg, or both limbs, as well as part of the torso, are involved in the process. Macromelia is manifested by hypertrophy in length and less in width of the bones and soft tissues of the limb on the side affected by angiomatosis. The syndrome can be combined with angiomatosis of the intestines, bladder, kidneys. The complications of the syndrome include thrombophlebitis, congestive heart failure, gangrene of the affected limb, lung disease, hematuria.

Osler-Randu disease (hereditary hemorrhagic telangiectasia) is an inherited autosomal dominant disease characterized by a triad of symptoms: telangiectasia of the skin and mucous membranes, a tendency to bleeding, and the transmission of these properties by inheritance. Before the appearance of typical changes in the skin and mucous membranes, nosebleeds recur in the child. Telangiectasias in the form of dots, linear and star-shaped spots are localized on the face (cheeks, forehead, chin, wings of the nose, auricles) and in certain parts of the body. They occur on the mucous membranes of the mouth, nose, bronchi, brain, gastrointestinal tract and urinary tract. Massive bleeding is the most serious complication and can lead to severe anemia, splenomegaly.

Sturge-Weber-Crabbe syndrome (encephalotrigeminal angiomatosis) is characterized by a combination of unilateral cutaneous, ocular, and cerebral angiomatosis. It is found from birth in the form of a "flaming" nevus located along

the branches of the trigeminal nerve. Sometimes angiomatosis spreads to the neck, chest and abdomen, less often the mucous membranes of the gums, nose, lips are affected. Unilateral angiomatosis of the brain is manifested by epileptiform seizures, hemiparesis, oligophrenia. Eye damage can be in the form of congenital glaucoma, angioma of the conjunctiva, angioma of the choroid. The prognosis for this syndrome is poor.

Hippel-Lindau disease (cerebroretinal angiomatosis) is characterized by a combination of hemangiomas of the skin, retina, cerebellum, medulla oblongata and spinal cord, kidneys, and pancreas. Numerous tuberous-cavernous hemangiomas of the skin are observed, located on the face, sometimes on the limbs, abdomen, chest. The syndrome is detected in infancy, more often in boys, and is inherited in an autosomal dominant manner. The prognosis is unfavorable: after a few months or years, symptoms characteristic of a brain tumor appear.

Louis-Bar syndrome (ataxia-telangiectasia) is characterized by telangiectasia, cerebellar ataxia and impaired immunity, and is inherited in an autosomal recessive manner. Telangiectasias appear on the conjunctiva, then on the nasal septum, auricles, hard palate, upper chest, and limbs in children aged 3 years. Fabry's disease is a nephrocutaneous syndrome (diffuse angiokeratoma), characterized by a combined lesion of the skin, kidneys, lungs and heart. The type of inheritance is recessive. Persons of both sexes are ill. The first signs of the disease appear at the age of 6-7 years. At the onset of the disease, characteristic skin changes are noted in the form of angiokeratomas located in the elbows, knees, hips, abdomen, lower back, and scrotum. Diffuse maculo-papular rash of red color can also be on the mucous membranes of the oral cavity, gastrointestinal tract. Semiotics of rashes (exanthema). Rashes on the skin (exanthema) and mucous membranes (enanthema) can be not only with viral and bacterial infections, but also with diseases of a non-infectious nature. The pediatrician faces the differential diagnosis of skin rashes every day, so only the main differential diagnostic signs are given here without a detailed description of the diseases. For the differential diagnosis of rashes, anamnesis data is always necessary (diseases among others,

taking into account the duration of the incubation period, previously suffered diseases, a tendency to allergies, medications taken, vaccinations), examination (morphology and nature of the location of the rash: the timing of the appearance of the rash from the onset of the disease, color, the presence of a thickening of the rash and its size), data on the blood picture and the dynamics of the disease, the isolation of a specific pathogen in a microbiological study or the detection of antibodies in the dynamics of the disease to various pathogens. It is important to decide whether these changes represent a primary skin injury or whether the clinical signs have been altered by secondary factors (infection, trauma, or treatment). be in the form of congenital glaucoma, angioma of the conjunctiva, angioma of the choroid. The prognosis for this syndrome is poor.

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Rash elements. Primary and secondary elements of rashes differ. The primary elements are classified as roseola, macula, papule, nodule, blister, vesicle, blister, hemorrhage. Secondary morphological elements include pigmentation and depigmentation, scale, crust, erosion, crack, abrasion, ulcer, scar, cicatricial atrophy, lichenification, vegetation.

Roseola (roseola) - a speck of pale pink, red color ranging in size from 1 to 5 mm. The shape is rounded or irregular, the edges are clear or blurry, it does not protrude above the level of the skin, it disappears when the skin is pressed and stretched. Roseola is found in many infectious diseases, especially typical of typhoid fever. (M.P. Konchalovsky said: “One star is not yet a starry sky, but one roseola makes you think about typhoid fever.”) Multiple roseolas 1–2 mm in size are usually described as a small punctate rash (with scarlet fever), in the process of resolution they are covered with scales or disappear without a trace.

The spot (makula) has the same color as roseola, does not protrude above the level of the skin, the size varies from 5 to 20 mm. The form is often incorrect. The spot disappears with pressure on the skin and reappears after the pressure

stops. Multiple spots ranging in size from 5 to 10 mm are described as a small-spotted rash (for example, with rubella). Spots 10–20 mm in size form a large-spotted rash (for example, with measles, allergies).

Erythema (erythema) - extensive areas of hyperemic skin of red, purple-red or purple color. It occurs as a result of the fusion of large spots (more than 20 mm in size), formed by the expansion of the vessels not only of the papillary layer of the skin, but also of the subpapillary choroid plexus. Erythema is most typical for erysipelas, thermal, ultraviolet burns.

Hemorrhagia (haemorrhagia) - hemorrhage into the skin as a result of destruction of skin vessels. It looks like dots or spots of various sizes and shapes, does not disappear when the skin is stretched. The color is initially red, purple or violet, then, as the hemorrhage resolves, it becomes yellow-green and, finally, yellow (the formation of hemosiderin during the breakdown of red blood cells). Color changes are clearly visible in larger hemorrhages. Pinpoint hemorrhages are called petechiae (petechia). Multiple hemorrhages of a rounded shape ranging in size from 2 to 5 mm are described as purpura (purpura haemoghagioe). Irregular hemorrhages larger than 5 mm are called ecchymoses (echumoses). Hemorrhages can be superimposed on other elements of the rash. In such cases, they talk about the petechial transformation of roseola, spots, papules. As a rule, this is observed in the severe course of the disease.

Papula (papula) - an element of a rash that rises above the level of the skin, which is often determined by touch. It has a flat or dome-shaped surface, the size is from 1 to 20 mm. The shape and color are the same as those of roseola and spots. Papules often leave behind pigmentation and flaking of the skin. Depending on the shape and size of the papules, they are miliary, the size of a millet grain, more often conical in shape and located around the hair follicle (a horny plug or hair is visible in the center of such an element).

A tubercle (tuberkulum) is a limited, dense, cavityless formation protruding above the skin surface with a diameter of 1–2 to 5–10 mm. Tubercles are formed as a result of the accumulation of a specific inflammatory infiltrate in

the dermis. Clinically, the tubercle is similar to a papule, but differs from it in that when the tubercle is felt, a dense infiltrate in the skin is always clearly defined. In addition, tubercles, unlike papules, undergo necrosis during reverse development, often form ulcers and leave behind a scar or cicatricial atrophy of the skin. The tubercles are most typical of cutaneous leishmaniasis, leprosy and tuberculous skin lesions, tertiary and late congenital syphilis.

Knot (nodus) - a cavityless, limited, deep seal deep into the skin, often standing above the level of the skin. The size of nodes from a hazelnut to a chicken egg and more. They are formed as a result of the accumulation of cellular infiltrate in the subcutaneous tissue and in the dermis itself. Nodules of an inflammatory nature have a soft or testy consistency, fuzzy borders, the skin above them is red, they are prone to rapid resolution. The nodes that appeared as a result of a specific inflammation (colliquiative tuberculosis, syphilitic gum) have a dense texture, are sharply delimited from the surrounding tissues, and are prone to disintegration and ulceration, followed by scarring.

A blister (urtica) is an acutely inflammatory, non-cavitary element that rises slightly above the level of the skin, ranging in size from 2-3 to 10-15 cm or more, has a round or oval shape, often accompanied by itching. Color - from white to pale pink or light red. A blister usually forms quickly and disappears quickly without leaving any trace behind. It occurs as a result of limited acute inflammatory edema of the papillary layer of the skin and the simultaneous expansion of capillaries. The appearance of urticarial elements is characteristic of allergic reactions of various origins (drug, food, cold allergies), including those of an infectious nature. Sometimes it occurs in the preicteric period of hepatitis B.

Bubble (vesicula) - a cavity element ranging in size from 1 to 5 mm, is a detachment of the epidermis. Usually the vesicles are filled with clear, cloudy or bloody contents, they can shrink and give a transparent or brown crust. If the cover of the bubble is opened, then erosion is formed - a wetting surface of pink or red color limited by the size of the bubble. Bubbles do not leave scars on the skin. In the case of accumulation in the vesicle of a large number of leukocytes, it turns

into an abscess - a pustule (pustula). Inflammatory changes are noted at the base and around the bubble. Pustules are divided into single-chamber (chickenpox) and multi-chamber (natural smallpox). A group of bubbles located on the burnt skin is called herpes. Vesicles are characteristic of herpetic and enterovirus infections, chickenpox and smallpox, erysipeloid and foot and mouth disease.

Bubble (bulla) - cavity element up to 3-5 cm in diameter, located in the upper layers of the epidermis and under it. The contents of the blisters can be serous, bloody, purulent. They can fall off, forming a crust, open, forming an erosive surface, turning into unstable pigmentation. The bubble occurs more often against the background of an erythematous spot, less often against the background of unchanged skin (pemphigoid of newborns). Elements can be located both inside the epidermis, in the styloid layer (pemphigus vulgaris), and under the epidermis (polymorphic exudative erythema, dermatosis herpetiformis). It is observed with a bullous form of erysipelas, sometimes with chicken pox, thermal burns. Secondary morphological elements are formed as a result of the evolution of the primary elements of the rash.

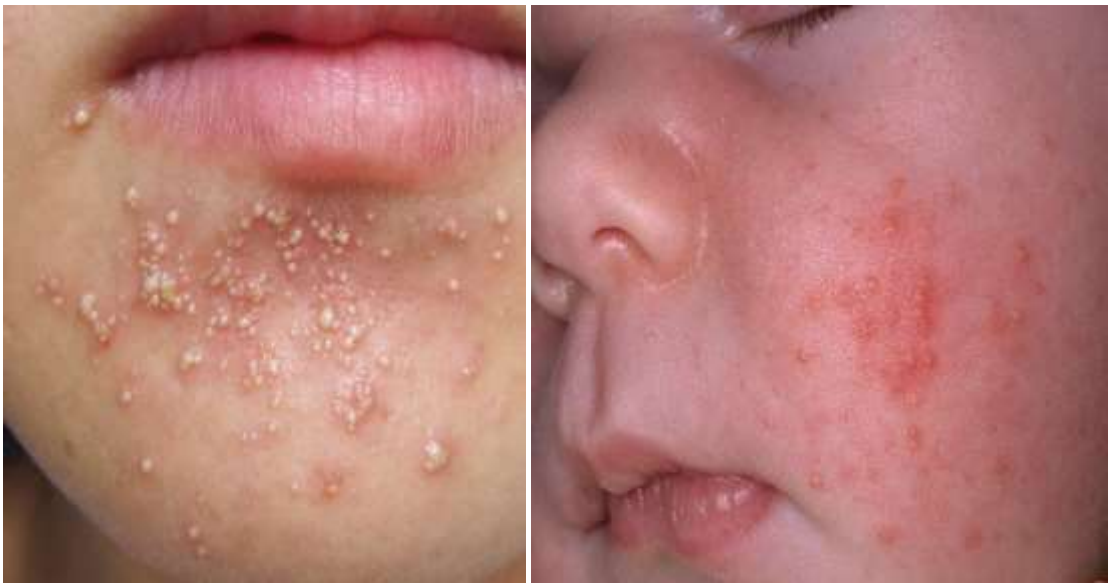


Figure #34. Manifestations of blisters in children

Hyperpigmentation (hyperpigmentatio) - a change in skin color as a result of an increase in melanin in it or the deposition of hemosiderin of primary elements. Depigmentation (depigmentatio) occurs as a result of a decrease in the content of melanin in the skin, observed after the disappearance of the nodule,

tubercle, resolution of patchy-scaly (pityriasis versicolor, eczematoids) and papular (psoriasis) elements.

Scale (sguama) - an accumulation of sloughing cells of the stratum corneum, sometimes layers of the epidermis that are subject to it. Scales are on the primary morphological elements - papules (psoriasis, syphilis), tubercles, after resolution of the vesicles (eczema), etc. Depending on the shape and size of the scales, flour-like (pityriasis scales) are distinguished, when the skin surface looks like sprinkled with flour (xeroderma), and lamellar peeling, in which horny plates of various sizes, up to parchment-like masses, are separated from significant areas of the skin (Leiner's desquamative erythroderma).

Erosion (erosio) is a defect in the skin within the epidermis as a result of the opening of a vesicle, bladder, abscess, repeating their shape and size. When vesicles and pustules merge, erosions have scalloped edges. Erosions can also occur as a result of maceration of the skin in the area of \u200b\u200bfolds or maceration of other elements of the rash, most often papules. When healing, erosion does not leave a scar, usually there is only temporary pigmentation.

An abrasion (excoriatio) is a violation of the integrity of the skin resulting from scratches, scratches, and other injuries. Abrasions can be superficial - within the epidermis, sometimes involving the papillary dermis, and heal without a scar. Deeper abrasions, involving the deep layers of the dermis, leave a scar behind. Abrasions are characterized by a tendency to infection. An ulcer (ulcus) is a deep skin defect that reaches the dermis, subcutaneous fat, fascia, muscles, and bones. It arises as a result of the decay of the tissue of the primary element (tubercle, node, ecthyma). Its size can be from 1 mm to the size of a coin or palm and more, the shape is round, oval, linear, oblong, irregular. The surrounding tissue is either inflamed (edema, hyperemia) or infiltrated. Ulcers always heal with scarring.

Cracks, tears (fissura, rhagades) - linear damage to the skin in the form of its rupture, resulting from excessive dryness due to loss of elasticity due to inflammatory infiltration or overstretching of the skin. Cracks can be located within the epidermis and dermis. Usually they are localized in the corners of the

mouth, interdigital folds, on the palms, soles, over the joints, in the anus. Superficial crack after healing leaves no traces. After healing of deep cracks, linear scars remain.

Scar (cicatrix) - the formation of connective tissue at the site of a deep defect. Occurs after healing of deep skin defects at the site of ulcerated tubercles, deep pustules, knots, deep burns, wounds. Scar formation is accompanied by the death of the sebaceous and sweat glands, hair follicles, blood vessels and elastic fibers, the disappearance of the skin pattern. Usually scars are located below the level of the skin or are at its level, less often rise above the level of the skin - hypertrophic scars (their variety is keloid). Lichenification (lichenificatio) - a focus of increased skin pattern, accompanied by its thickening and compaction, hyperpigmentation, dryness. Lichenification foci are most often localized in the neck, elbow and popliteal folds, wrist and ankle joints, inguinal folds, scrotum and occur in chronic dermatosis accompanied by itching (eczema, neurodermatitis).

Vegetation (vegetatio) is a papillary thickening of the skin that occurs as a result of the growth of the styloid layer of the epidermis and papillomatosis of the dermis during a long-term inflammatory process. More often it is formed in the area of papular elements and ulcers. Vegetations can erode, bleed, and are prone to the addition of a secondary pyococcal infection.

Striae (striae) - scar strips resulting from stretching of the skin and tearing of its corium with intact epidermis. They often form in obese older children and are more often localized on the upper thighs or on the abdomen. At first, the striae are light red, turning blue-gray, and later white. Often, from the first month of life, exudative diathesis appears on the skin in the form of persistent diaper rash in the skin folds against the background of dry skin and itching. In this case, there may be gneiss, or seborrhea, on the scalp (fatty yellowish scales around the large fontanel and on the crown), milky scab (temperature-dependent redness, and then peeling of the skin of the cheeks, which decreases outdoors in cool weather). Exudative diathesis is characterized by erythematous-papular or erythematous-vesicular rashes on the skin of the limbs and trunk, as well as or strophulus (itchy vesicles

filled with serous contents).

SEMIOTICS OF RASHES OF NON-INFECTIOUS ORIGIN.

An allergic rash is observed with serum sickness, food and drug allergies. With serum sickness against the background of the underlying disease (diphtheria, botulism, tetanus, etc.), a rash appears in a week after the introduction of heterologous serum. The nature of the rash is varied - spotted, maculopapular, medium and large sizes. Urticarial rash is very characteristic. The rash is necessarily accompanied by itching, it is located everywhere - on the face, trunk, limbs, but most of all around the joints and at the site of serum injection. Erythema multiforme exudative, like erythema nodosum, has an infectious-allergic nature. It is characterized by a spotted or papular rash, round in shape, 3–15 mm in diameter, with sharp borders, pink or bright red, characterized by centrifugal growth with retraction and a lighter color of the central part. Erythema multiforme is characterized by a symmetrical, fairly common skin lesion, sometimes the spots merge, forming figures in the form of garlands, arcs, with localization mainly on the extensor surfaces of the limbs, more often the forearms, less often the legs, rear of the foot, on the face, neck. Often, erythema is preceded by subfebrile condition, pain in the throat, joints, etc.

Stevenian-Johnson syndrome refers to the variants of the course of erythema multiforme exudative. The mechanism of development is associated with allergic reactions of an immediate type, proceeding according to the type of the Arthus phenomenon, to taking medications: sulfa drugs, pyrazolone derivatives, antibiotics, etc. The onset is acute, stormy, with fever lasting from several days to 2-3 weeks, there are pains in throat, hyperemia of the mucous membranes, runny nose, conjunctivitis, hypersalivation, joint pain. From the first hours, there is a progressive lesion of the skin and mucous membranes: painless dark red spots on the neck, face, chest, limbs (even the palms and soles are affected); along with this, papules, vesicles, blisters appear.

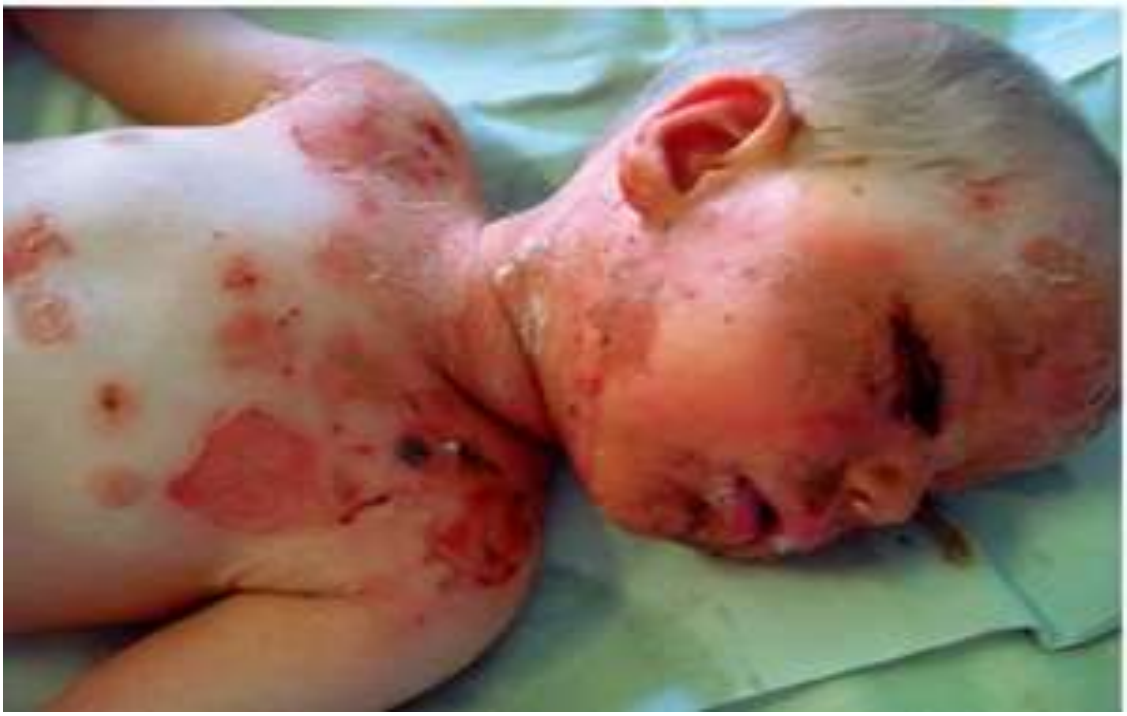


Figure #34. View of a sick child with Steven-Jones syndrome

Lyell's syndrome (toxic epidermal necrolysis) is an allergic reaction to an infectious, mainly staphylococcal, process and to medication (antibiotics, sulfonamides, analgesics) or to blood transfusion and its components. In the pathogenesis of the disease, the "explosive" release of lysosomal enzymes in the skin (not always of immune origin) is of primary importance. The disease begins acutely with chills, fever, sore throat, lower back, joints, burning and soreness of the skin. Large erythematous spots of various sizes appear, often merging and spreading throughout the body in a few hours. On some areas of the skin, vesicles, papules, blisters and then large, flat, flabby blisters appear at the site of the spots, on others - hemorrhages. As a result of severe epidermolysis, the child looks like a second-degree burn. In areas subjected to friction with clothing, the surface layers of the skin peel off, regardless of the presence or absence of blisters.

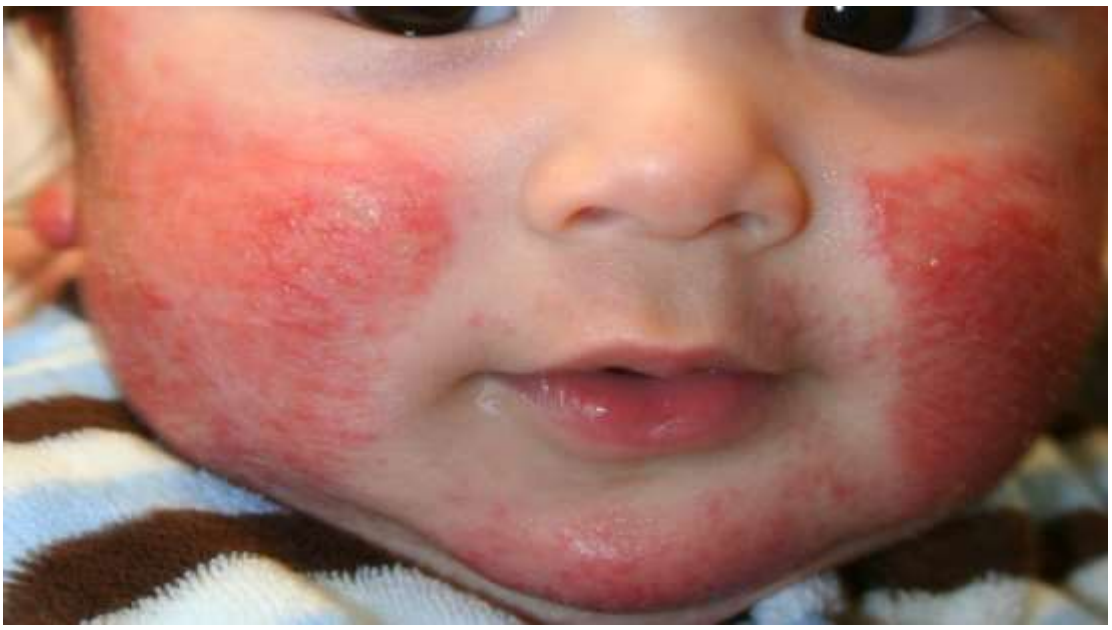


Figure #35. View of a sick child with Lyell's syndrome

Nikolsky's symptom is positive. With this syndrome, mucous membranes can also be affected. In contrast to the Stevens-Johnson syndrome, toxicosis is pronounced, the mucous membranes of the mouth and eyes are affected, myocarditis, nephritis, and hepatitis often develop.

Urticaria, angioedema are the most common allergic skin lesions. There are immune and non-immune forms of urticaria. In children, allergens are more often food substances. Allergic reactions are mediated by IgE. A few minutes or hours after eating allergens, the patient feels tingling of the tongue, lips, palate, swelling in these places, often sharp pains in the abdomen. Erythema appears on the skin of the face, which later spreads to other parts of the body, urticaria, severely itchy elements appear in place of the erythema. Rashes on the skin have a diverse character: nodules, blisters of various sizes and bizarre shapes. Conjunctivitis phenomena are often observed simultaneously, less often - difficulty breathing due to laryngeal edema, etc. In angioedema (giant urticaria, Quincke's edema), significant, clearly limited edema is detected that can occur in any part of the body, but more often in the lips, tongue , eyes, arms, legs, genitals. Edema can migrate. Common symptoms are possible: fever, agitation, arthralgia, collapse.

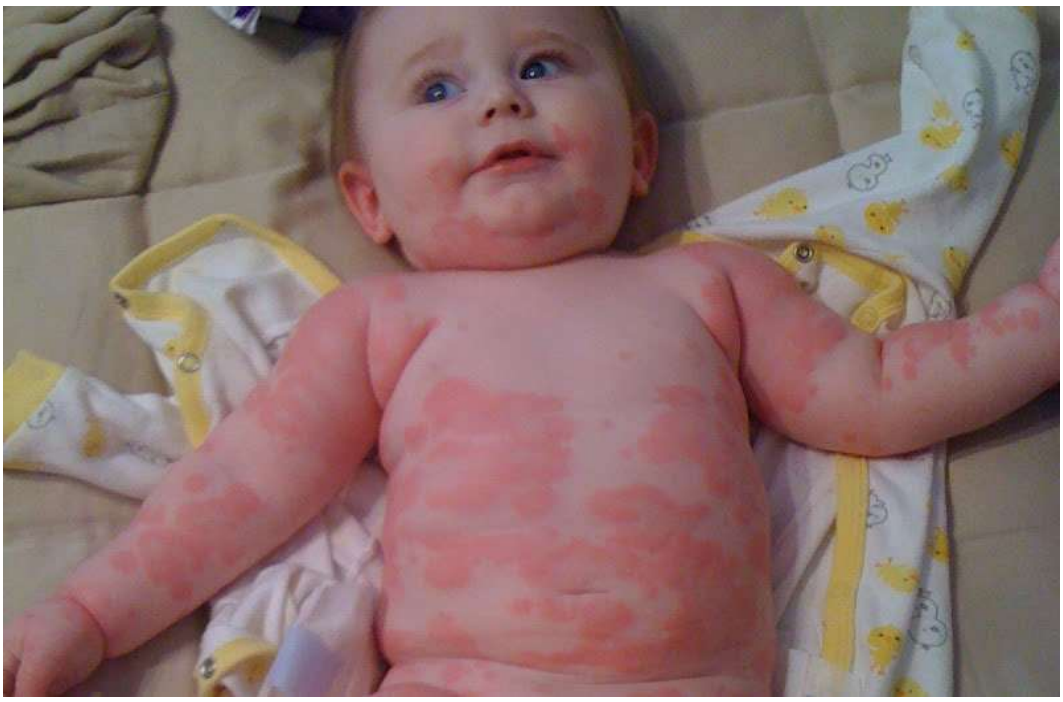


Figure #36. View of a sick child with urticaria syndrome

Desquamative erythroderma Leiner-Myssy occurs in children of the first 3 months of life. It is characterized by bright hyperemia, infiltration and peeling of the entire skin. In most children, the initial localization of the lesion is the buttocks and inguinal folds, less often the upper body, scalp, face, and armpits. Peeling scales of a dirty yellow color merge on the face and form, as it were, a shell. After peeling of the scales on the body, maceration, cracks appear in the folds, and a secondary infection is superimposed. In addition to changes in the skin, dyspeptic disorders are typical, leading to hypovitaminosis, malnutrition, iron deficiency anemia, and septic complications.

SEMIOTICS OF DAMAGE TO THE BONE AND ARTICULAR SYSTEM

Knowledge of the physiological parameters of the musculoskeletal system in children of different ages, a clear interpretation of the clinical symptoms of the pathology of individual elements and the entire organ system of the osteoarticular system as a whole is necessary for the correct diagnosis of its diseases and successful therapy. Violations of the regulation of phosphorus-calcium

metabolism. Violations of the regulation of phosphorus-calcium metabolism cause the development of many diseases in childhood. More often than other causes of bone deformities, osteopenia and early osteoporosis in children are rickets and rickets-like diseases. Hypocalcemia syndrome - can be genetically determined or acquired. It develops as a result of low intake of calcium from the gastrointestinal tract, bones and kidneys into the extracellular fluid and bloodstream or due to excessive loss of calcium. Clinical manifestations of hypocalcemia are mainly due to disorders of neuromuscular excitability. For hypocalcemia, the most characteristic are paresthesia and tetany.

Osteoporosis is a common dysmetabolic disease of the human skeleton, which is characterized by low bone mass and microstructural restructuring of bone tissue, which leads to increased bone fragility and, as a result, radiological damage.

Rickets (from Greek - spine) is a polyetiological disease caused by an increased rate of remodeling and growth of the skeleton, a large need of the body for salts of Ca, P and other nutrients, with relative weakness and imperfection of the systems that ensure their delivery and metabolism. Changes in the skeletal system, similar to D-deficient rickets, can develop in primary (genetically determined) and secondary diseases of the organs involved in the metabolism of vitamin D: in the pathology of the parathyroid gland, gastrointestinal tract, kidneys, liver, lungs, skeletal system.

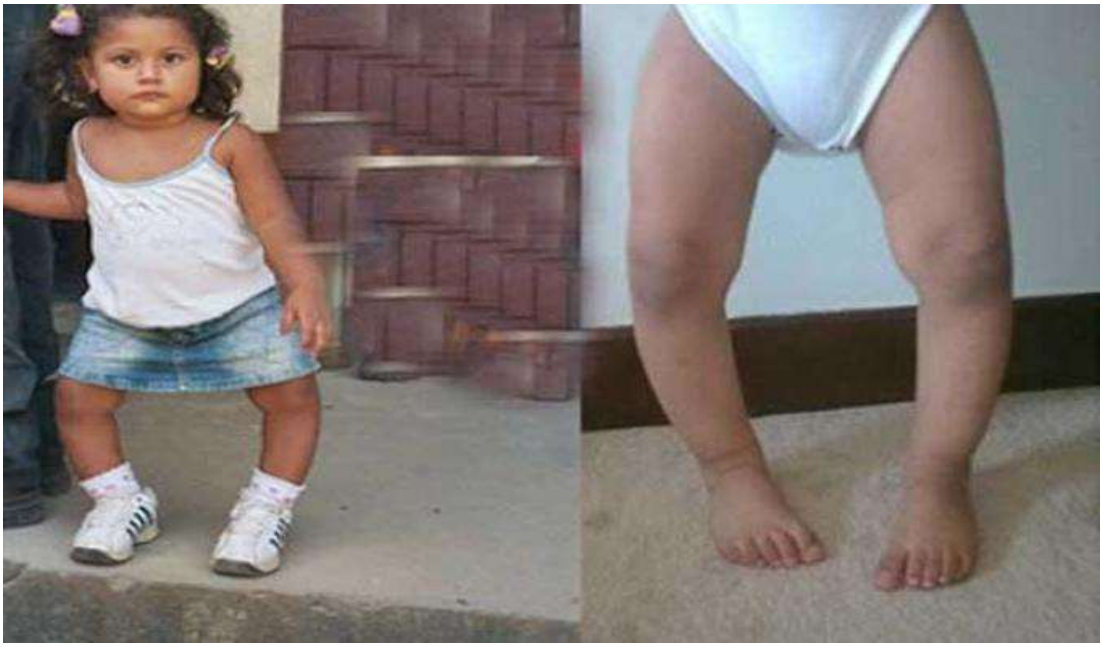


Figure 37. Curvature of the lower extremities with rickets

In such cases, a violation of phosphorus-calcium metabolism is interpreted as a rickets-like syndrome in the underlying disease (hypoparathyroidism, renal tubular acidosis, de Toni-Debre-Fanconi syndrome, etc.). Deficiency of mineralization and softening of tubular bones in children of preschool and school age, adults, in particular with rickets, is called osteomalacia.

Bone deformities in rickets. The most striking symptoms of rickets are changes in the skeletal system. Bone changes occur non-simultaneously and unevenly throughout the skeleton. First of all, and most intensively, disturbances develop in rapidly growing bones - the skull, the anterior ends of the ribs, the metaphyses of tubular bones, and later - in the vertebrae, pelvic bones, and jaws. In this regard, bone symptoms in rickets are different depending on the age of the patient: in a child of the first six months of life, lesions of the skull and chest are found, in older children - limbs and spine.

Changes in the skull with rickets:

1) along the lambdoid suture and the posterior parts of the parietal bones (less often in the region of the scales of the occipital bone), areas of softening with a diameter of 1.5–2 cm (craniotabes) appear, which, when pressed, give the feeling of soft parchment or a thin celluloid plate (symptom " ping pong ball" or "felt hat");

2) the cranial sutures close late, the closure of the large fontanelle is also late (up to 16–24 months); due to general changes in the configuration of the skull, the large fontanel changes its shape, turning from rhombic to square, triangular, or elongated along and across;

3) due to the softness of the bones and depending on the position of the child in bed, the skull may be irregular in shape - either the back of the head is flattened, or asymmetry of the head occurs, brachycephaly, sometimes the bridge of the nose (saddle nose) sinks down;

4) the head of the child is disproportionately large due to the more developed

brain part of the skull and the growth retardation of the facial bones and the base of the skull;

5) bite is disturbed due to deformation of the jaws - the upper jaw lengthens in the anteroposterior direction and protrudes forward above the lower one; the lower jaw becomes angular and flattened in front; the vault of the sky becomes high and narrow;

6) narrowing of the nasal passages;

7) due to the growth of the parietal and frontal tubercles (growth of the osteoid), the skull can be of various configurations (square (caput guadratum), saddle-shaped, rachitic brachycephaly);

8) high (“Olympic”) forehead.

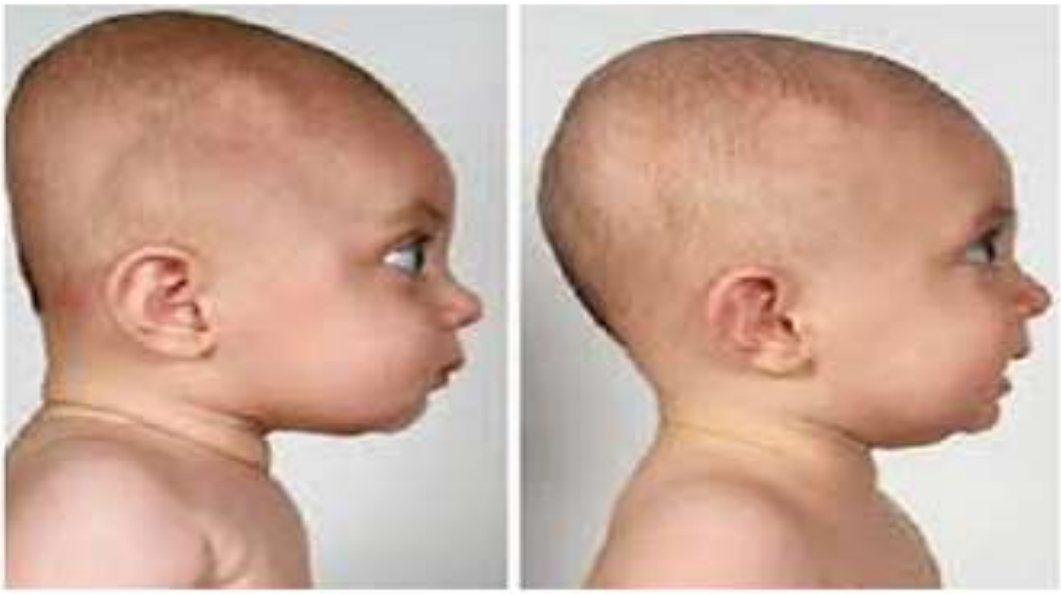


Figure No. 38. Changes in the skull with rickets

Changes in teeth:

1) milk teeth in children with rickets often erupt in the wrong order and at a later date;

2) due to a violation of the structure of dentin and enamel, teeth are easily subjected to carious processes.

Deformation of the chest:

1) the curvature of the clavicles increases;

2) as a result of the softness of the ribs and their greater mobility, a horizontal depression appears in the form of a belt (Harrison's groove), approximately corresponding to the attachment of the diaphragm, which is especially noticeable when the child takes a deep breath and cries;

3) with a significant compression of the chest from the sides (tight swaddling), the sternum protrudes forward - the so-called keeled ("chicken") chest is formed;

4) sometimes the lower part of the sternum is pressed in - a funnel-shaped chest is formed ("shoemaker's chest"); especially severe deformities of the chest are observed with curvature of the spine (posteriorly - kyphosis, anteriorly - lordosis, to the side - scoliosis);

5) the lower aperture of the chest turns outward - the so-called "hat brim" is formed; 6) as a result of increased formation of osteoid tissue, thickenings appear on the border of the bone and cartilaginous parts of the ribs - the so-called "rachitic rosary" or "rachitic rosary".

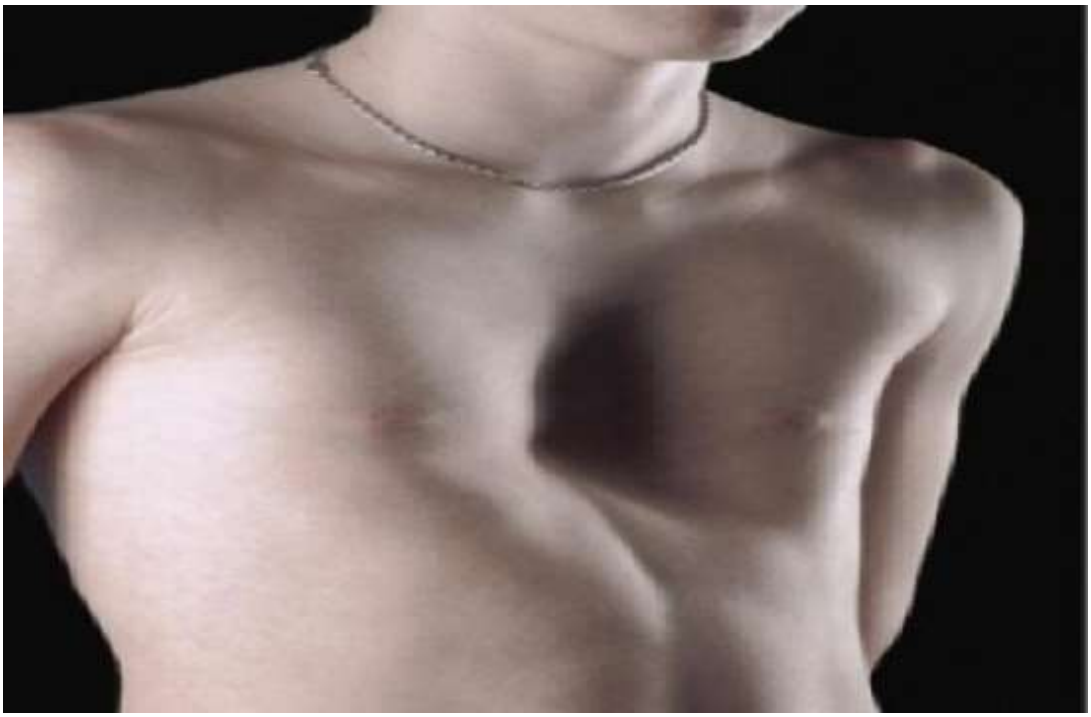


Figure #39. Rachitic "funnel" of the chest

Deformities of the spine and pelvis:

1) the curvature of the back increases (rachitic kyphosis), the formation of a

rachitic hump occurs; when the child begins to walk, sometimes lordosis joins, and in some cases scoliosis;

2) sick children in the supine position easily pull the leg to the head, even put the foot on the shoulder (symptom of the "penknife");

3) a flat rachitic pelvis is formed - the pubic arches expand, the conjugates shorten.

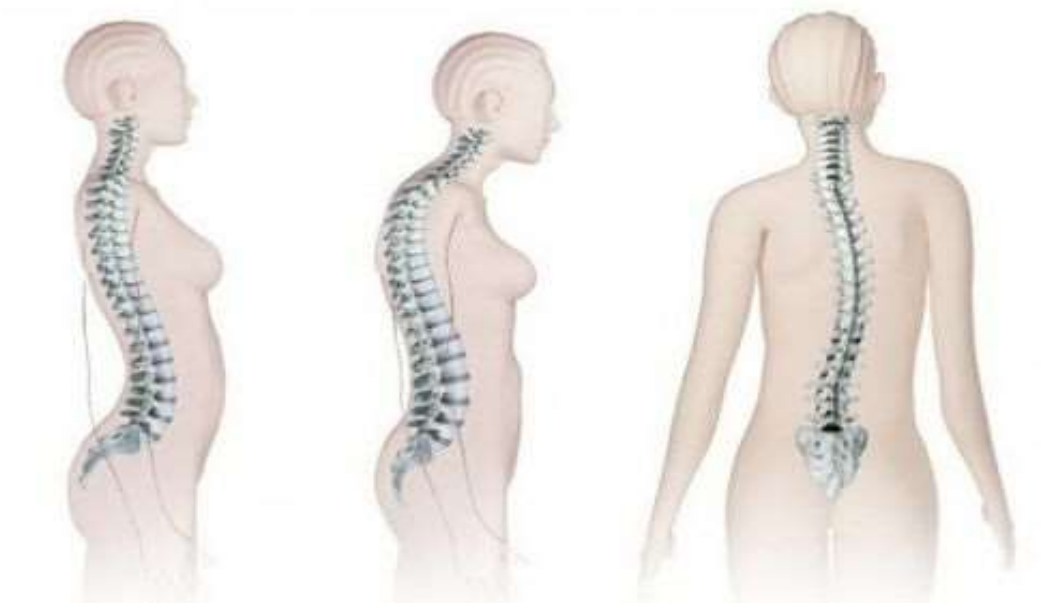


Figure No. 40. Rachitic kyphosis, lordosis and scoliosis of the spine

Deformities of the extremities:

1) as a result of deformation of the epiphyses and expansion of the metaphyses, thickenings are formed - rachitic "bracelets" - at the lower ends of the radius, ulna, tibia and fibula and "pearl threads" on the phalanges of the fingers, supracondylar thickenings are sometimes visible on the legs;

2) various curvature of the thigh forward and outward, lower leg in the lower third forward - these deformations cause the formation of 0-shaped (genu varum) and X-shaped (genu valgum), saber legs, flat feet;

3) the processes of impaired bone formation and osteomalacia often lead to fractures and fractures;

4) there is a slowdown in the growth of bones in length - this is one of the reasons for the growth retardation of children with rickets, and the delay in overall

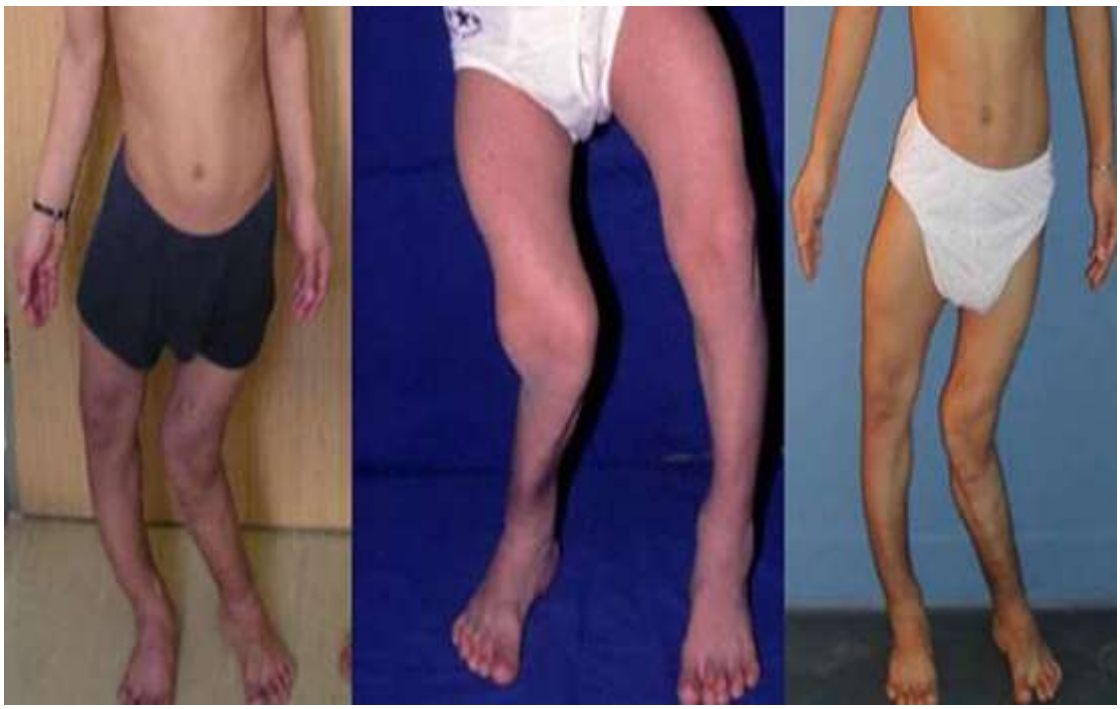


Figure #41. Curvature of the lower extremities with rickets

growth is manifested especially in the presence of changes in the vertebrae along with deformations of other parts of the skeleton.

Thus, lesions of the skeletal system in rickets can be summarized in four groups:

1) symptoms of osteomalacia (softening of the bone) prevail in acute course - compliance of the bones of the skull, the edges of the fontanel, craniotabes, brachycephaly, deformities of the skull, limbs, collarbones, flat pelvis, erosion and dental caries;

2) symptoms of osteoid hyperplasia predominate in subacute rickets - frontal, parietal tubercles, costal "rosary", supracondylar thickening of the legs, "bracelets" on the forearms, "strings of pearls" on the fingers;

3) symptoms of bone tissue hypoplasia - growth retardation with a characteristic "short-legged" due to a lag in the growth of tubular bones in length, late eruption of milk and permanent teeth, late closure of fontanelles;

4) symptoms of muscular hypotension - curvature of the spine with spinal lumbar kyphosis, scoliosis, deformity of the chest with a deployed lower aperture ("bell-shaped" chest).

Joint syndrome. Articular syndrome is a symptom complex that develops when one or more joints are involved in the pathological process. Anamnesis is one of the central and defining links in the differential diagnosis of articular syndrome in children. An important role among the anamnestic information in the diagnosis is assigned to the analysis of pain. Arthralgia (pain in the affected joint) occurs as a result of irritation of nerve endings in its various structures, excluding articular cartilage, which does not have nerve endings and blood vessels.

When characterizing pain, the following are taken into account:

- localization (asymmetric, symmetrical);
- irradiation of pain;
- the duration of the pain syndrome;
– intensity of pain (weak, moderate, strong);
- suddenness or gradual appearance;
- chronology of pain: at night, in the morning, in the evening;
- connection with movement or a certain type of movement;
- Is the sensation of numbness accompanied by pain?
- factors that reduce pain. The nature of the pain:
- inflammatory - the pain is more pronounced at rest or it is stronger at the beginning of the movement than at the end; joints ache in the morning or most of the night (a characteristic feature of rheumatoid arthritis, Reiter's disease, reactive arthritis); - mechanical - the pain is associated with movements in the joint, the more the sick child walks, the stronger the pain (a characteristic sign for osteoarthritis); - constant - severe pain, exhausting, aggravated at night (associated with osteodestruction and bone necrosis, accompanied by intraosseous hypertension). Constant "bone pain" (day and night) occurs when tumors metastasize to the bone.

Also pay attention to the following points:

1. Acute onset - the main symptoms develop within a few hours - a few days. Acute gouty and infectious arthritis begin within a few hours. Reiter's arthritis and reactive arthritis occur within a few days.

2. In subacute course, the main symptoms of arthritis develop within a month. This course is most common in rheumatoid and tuberculous arthritis, as well as diffuse connective tissue diseases.

3. Chronic course is observed in most cases of rheumatoid arthritis, osteoarthritis and ankylosing spondylitis.

4. Localization of pain - more often the pain corresponds to the affected joint, but may be in the nature of "reflected" pain, for example, if the hip joint is affected, there may be pain in the knee joint, lumbar, inguinal and gluteal regions; with flat feet - in the ankle, knee and even hip joints, etc. Sometimes joint pains can be associated with diseases of internal organs, for example, with a lung tumor, they are localized in the shoulder joint, with pathology of the pelvic organs - in the sacrum, etc.

5 Variants of the course of the pain syndrome: slow, but steadily progressing; rapidly progressing; without progression; undulating without progression; undulating with steady progression; relapsing progressive; relapsing regressive. In the occurrence of pain, mechanical factors play a role - overload of the joint, stretching of the tendon-ligamentous apparatus, irritation of the synovial membrane, as well as microcirculatory disorders, metabolic disorders in the bone skeleton, development of inflammatory and degenerative changes in the joint. The most common causes of joint pain in newborns and infants include fractures of the femur and humerus, fracture of the clavicle during childbirth, paralysis of the brachial plexus, epiphysiolysis of the humerus, metastatic arthritis in sepsis, osteomyelitis. At the age of 1–3 years, among the causes of pain in the joints are injuries, juvenile rheumatoid arthritis, tuberculous arthritis, arthritis in sepsis, arthritis and arthralgia in tubulopathies, allergic diseases, and toxic and septic diseases. In older and adolescent children, the involvement of the joints in the pathological process is noted in infectious diseases, celiac disease, serum sickness, collagenosis, rheumatism, psoriasis, diseases of the nervous system, oncological diseases, alkaptonuria, etc. The main complaints in children with articular syndrome can also include limitation movements in the affected joint (joints),

morning stiffness, swelling and change in the configuration of the joint, the presence of a crunch, clicking in it during movement, a change in the child's gait. The duration of morning stiffness can be different - from a few minutes (then they talk about stiffness in the joints) to several hours. In addition to the symptom of morning stiffness, there is also the so-called general stiffness - a symptom that reflects the condition of the spine. General stiffness can be observed in the entire spine or in its sections - cervical, thoracic, lumbar. Complaints of a general nature include an increase in body temperature (fever), as well as other complaints that reflect the presence and severity of the toxicosis syndrome, such as weakness, lethargy, unmotivated capriciousness, malaise, and a change in the child's behavior. When examining the joints, attention is paid to their position and symmetry, volume (size), contours and shape (configuration), the absence or presence of soft tissue changes over the affected joint (pallor or hyperemia of the skin, its pigmentation, fistulas) is stated. A change in temperature is carried out over the joint (several areas located nearby are compared with the back of the hand). The asymmetry of the joints often occurs when one of the limbs is shortened - hemiatrophy, or underdevelopment of the limb, or its unilateral increase - hemihypertrophy.

Deformation of the joints is a persistent change in their shape due to bone changes, the development of ankylosis, subluxations. Swelling - an increase in the volume of the joint with the smoothing of its contours. The swelling can be uniform and uneven. The uniform swelling of the joint is usually combined with the smoothness of its contours, in particular, the bony protrusions and the soft tissue retractions existing in the norm. With a pronounced exudative process, characteristic of acute inflammation, the joint increases significantly in volume and loses its usual shape, acquiring a spherical (spherical) shape. The skin over the joint is tense and shiny.

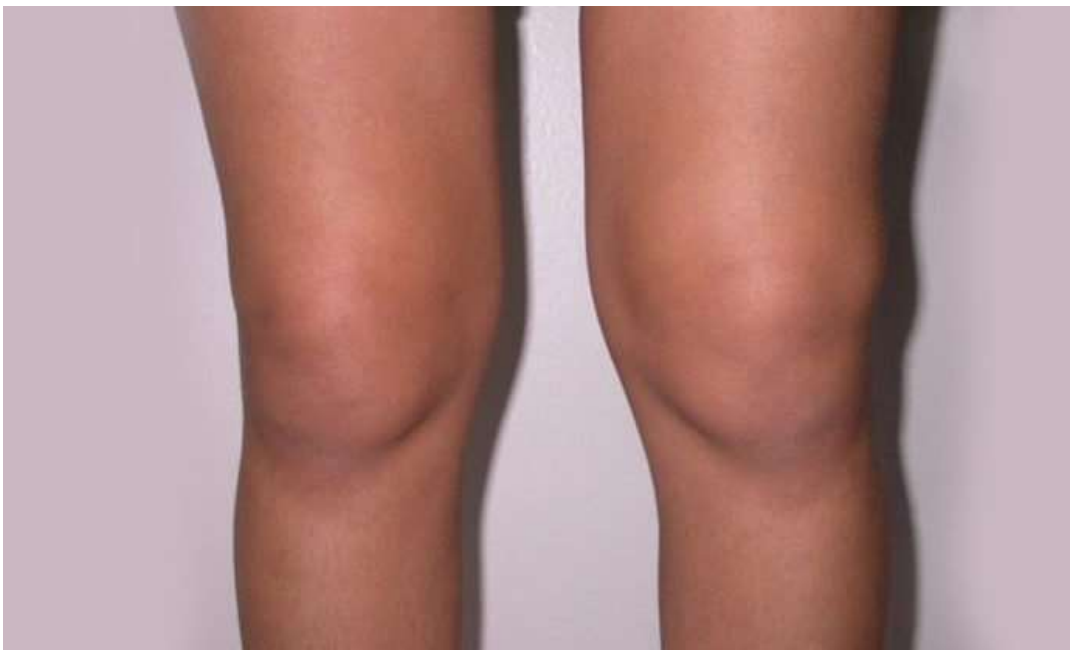


Figure #42. Deformity of the left knee joint

The cause of uniform swelling is most often a significant edema of the synovial membrane and periarticular soft tissues, and sometimes the presence of effusion (exudate) in the articular cavity. With a significant accumulation of exudate in the cavity of the joint, such as the knee or elbow, there is a bulging of the torsion of the synovial membrane. Uneven swelling of the joint is called defiguration and is most often seen in chronic arthritis. This is a change in the shape of the joint due to inflammatory edema of the synovial membrane and periarticular tissues, effusion into the joint cavity, hypertrophy of the synovial membrane and sclerotic changes in the periarticular tissues. Limited swelling in the joint area, which does not correspond to the anatomical location of its cavity, can be caused by a cyst, hygroma, tumor, but more often - an inflammatory lesion of the periarticular soft tissues, in particular tendons (tenodinitis), tendon sheaths (tenosynovitis, or tenosynovitis) or synovial bags (bursitis). When the musculoskeletal system is affected, the child’s gait suffers (“duck gait” with osteomalacia and congenital hip dislocations, “stilted gait” with ankylosis of the joints, etc.). Complaints about the feeling of a "foreign, foreign body" in the joint ("articular mouse") are much less common. Complaints of muscle pain, pain in the area of ligaments and tendons are important. Complaints of reddening of the affected joints most often suggest septic arthritis, acute rheumatic fever

(rheumatism), but sometimes are a sign of a malignant tumor. When examining and palpating the affected joints, the restriction of the range of motion characteristic of this joint is approximately established. The limitation of active and passive movements in the joints is assessed. Articular syndrome in children can be represented by arthritis, arthrosis and arthropathy. Arthritis is an inflammation of the joints that begins in the synovium. This symptom complex is characterized by: - acute onset; - course with periods of exacerbation and remission; - concomitant symptoms of intoxication: fever, feeling of "ache" in the muscles and joints, headache; - the presence of pain of an inflammatory nature (they occur and intensify at night or in the morning, are noted at rest and during movement); - complaints of "morning stiffness"; - the presence of swelling of the joint after its prolonged immobility - a symptom of "jelly"; - visible changes in the joint - its swelling, hyperemia of soft tissues; palpation - soreness, increased temperature of the skin over the affected joint; - signs of inflammation during laboratory examination: in the general blood test in the form of leukocytosis, increased ESR, anemia; in biochemical - dis-γ- and α-globulinemia, fibrinogenemia, the appearance of C-reactive protein, etc.; - changes in the synovial fluid in the form of a decrease in its viscosity, an increase in cytosis, the formation of a bad mucin clot, and also, according to the results of a biopsy, the presence of signs of synovitis with the development of granulation tissue (pannus), plasma cell reaction and lymphoid infiltration.

X-ray examination reveals signs of erosion of the articular surfaces, osteoporosis, narrowing of the joint space up to ankylosis. If one joint is affected, they talk about monoarthritis, from two to four joints - oligoarthritis, more than four - polyarthritis. Bilateral arthritis is equally localized on both sides. Symmetric arthritis has the same localization and the same degree of severity on both sides.

Arthrosis is a disease of the joints of a degenerative-dystrophic nature (primary lesion of the articular cartilage). In childhood, arthrosis as a primary process practically does not occur. For example, juvenile spondylarthrosis or discitis often manifests itself after a spinal injury. Discitis occurs due to partial

displacement of the epiphysis, more often III–V lumbar vertebrae, as they experience maximum load during anterior and maximum lateral flexion. The general condition of the child practically does not suffer, deviations of laboratory parameters from the norm are not detected. The radiograph shows narrowing of the intervertebral spaces.

In table. 3 presents the differential diagnostic criteria for arthritis and arthrosis.

Table 3

Differences between arthritis and arthrosis

Onset of the disease	**Acute**	**Long-term course with no symptoms**
Prevalence in childhood	More often	Less often
Course	With exacerbations and remissions	Sluggish, without noticeable exacerbations
The nature of the pain	Inflammatory type: sharp and strong, occur and intensify at night, in the morning, often accompanied by morning stiffness in the affected joints, are noted at rest and in motion	Mechanical type, i.e. associated with mechanical (physical) stress: dull, aching, moderate forces, constant, arise at the end of the day, the first half of the night, decrease in the morning, after physical exertion and with prolonged standing increase, subside at rest, a variety of them are “starting pains” that occur immediately at the beginning of the movement and subside as it continues
Visible changes	Swelling, hyperemia of soft tissues, pain on palpation of the joint	Pain on palpation and percussion of the joint in the absence of hyperemia and swelling
Limited mobility	Progressive deterioration of joint function with development of	There is no pronounced limitation of mobility (an exception is coxarthrosis), stiffness at any time of the

	ankylosis, morning stiffness	day, after a state of rest (for example, after prolonged standing)
Contractures, ankylosis	Persistent contractures and ankylosis are characteristic	Persistent contractures are not characteristic, flexion contractures are characteristic
Signs of inflammation	Fever present, symptoms of intoxication	None
Changes in synovial fluid	Increased cytosis, reduced viscosity, poor mucin clot	Small cytosis, good viscosity, dense mucin clot
Biopsy	Signs of synovitis with granulations (pannus), plasma cell reaction and lymphoid-histiocytic infiltration	Signs of degeneration of articular cartilage
X-ray data	In long-term arthritis: osteoporosis, erosion of the articular surface, narrowing of the joint space, ankylosis	Subchondral osteoporosis, osteophytes, narrowing of the joint space, change in the shape of the articular end of the bone
Laboratory indicators	Increased ESR, leukocytosis, anemia, dysproteinemia (increased α2-globulin fraction, γ-globulin fraction), increased fibrinogen, increased seromucoid, appearance of C-reactive protein, etc.	Increased ESR (20–25 mm/h), moderate leukocytosis , dysproteinemia (an increase in α2-globulins, the appearance of C-reactive protein, etc.)

ARTROPATHY is a secondary lesion of the joint (joints) against the background of various pathological processes. It can be both inflammatory and degenerative-dystrophic.

Common distinctive features of arthropathies are:

1) asymmetry of the lesion;

2) the presence of parallelism in the clinic of the articular syndrome with the clinic of the underlying disease;

3) positive dynamics against the background of treatment of the main process;

4) the scarcity of the x-ray picture (lack of usuration, narrowing of the joint space, signs of ankylosis). Some other features of the articular syndrome in the main nosological forms of joint pathology are presented in Table. 4.

Table 4

Features of the articular syndrome in the main nosological forms of articular pathology

Morning stiffness in the joints for more than 30 minutes. Polyarthritis. Less often oligo- and monoarthritis. The small joints of the hands and feet are affected - metacarpophalangeal, proximal interphalangeal. During the period of exacerbation and as the disease progresses, there is a pronounced deformity of the joints, a violation of their function. Usually not associated with infection	Rheumatoid arthritis
Articular manifestations appear 2.5-3 weeks after tonsillitis, pharyngitis. Large joints are affected, volatility, symmetry of the lesion, amazingly fast effect of aspirin and other non-steroidal anti-inflammatory drugs are characteristic. No articular disability	Rheumatic arthritis
There is a clear connection with the	Reactive arthritis

infection - urogenic, enterogenic, tonsilogenic. Damage to the joints by the type of mono- or oligoarthritis, often signs of sacroiliitis. There is no pronounced deformity of the joints. The effect of antibiotics and non-steroidal anti-inflammatory drugs is noted	
The triad of signs - poly- or oligoarthritis, conjunctivitis, urethritis	Reiter's syndrome
Progressive damage to the spine, large joints may be affected without their articular disability	Ankylosing spondylitis
Recurrent arthritis involving the first metatarsophalangeal	joint Gout
n the anamnesis indications of tuberculosis, gonorrhea. Predominantly asymmetric mono- and oligoarthritis	Infectious specific arthritis
The defeat of the interphalangeal, mainly distal, joints of the hands (fingers in the form of sausage or radish). There are signs of sacroiliitis	Psoriasis
Mainly large joints are affected, the pain is aggravated by exertion. There may be a pronounced deformity of the joints, secondarily - signs of synovitis	Deforming osteoarthritis

Among other pathologies of the musculoskeletal system in children of different ages that fall into the scheme of differential diagnostic search, one can single out OHP, chondromalacia of the patella, recurrent subluxation of the patella, generalized joint hypermobility syndrome, fibromyalgia.

Osteochondropathy. These are diseases manifested by aseptic necrosis of subchondral and, as a rule, parts of the skeleton subject to increased stress (more often these are the epiphyses of short and long bones). On the radiograph, one can detect a half-moon symptom pathognomonic for this process - a site of enlightenment located in the subchondral zone is determined. These diseases occur cyclically and for a long time.

Osgood-Schlatter disease is characterized by pain and swelling at the insertion of the patellar tendon to the tibial tubercle. There are no general symptoms. X-ray (lateral view) shows signs of fragmentation of the tibial tubercle. Boys aged 10–15 years are most often affected. In Legg-Calve-Perthes disease, primary subchondral aseptic necrosis of the epiphysis of the femoral head is detected, often unilateral. Children 5-10 years old, usually boys, suffer. They complain of gradually increasing pain in one of the hip joints with possible irradiation to the knee joint, gait disturbance. Change is progressing slowly. Over time, muscle atrophy on the side of the lesion may join. X-ray shows flattening and fragmentation of the femoral head. There are also chondropathy of other localization: juvenile kyphosis - Scheuermann-Mau disease, Hass disease (OHP in the humerus), Larsen-Johansson (OHP in the patella), Kienbeck (OHP in the lunate bone of the wrist). There are secondary OHP that occur during long-term treatment of a child with certain medications (for example, corticosteroids), as well as with mucopolysaccharidoses, hemoglobinopathies. In this case, the head of the femur is most often affected.

Chondromalacia of the patella. Pain in one or both knee joints in adolescent girls can also occur with other pathologies, such as patella hodromalacia. At the same time, creaking and grinding appear in the joints. The pain is aggravated when climbing stairs. Crepitus is found over the patella. A small joint effusion may be present. An x-ray may reveal articular erosions. Recurrent subluxation of the patella is accompanied by weakness of the ligamentous apparatus, a feeling of insecurity of the joint, pain in the medial part of the patella. In the anamnesis, the child may indicate a block of the knee joint. Syndrome of generalized hypermobility of the joints. Approximately 10% of people have joint mobility that is outside the normal range towards hypermobility. On average, in 10-15% of these patients, hypermobility is pathological (for example, Marfan syndrome, Ehlers-Danlos syndrome, acromegaly, etc.).

Screening control for generalized hypermobility can be a modified Bayton score that facilitates its diagnosis:

1) extension of the little finger by 90° or more (one point on each side);

2) ghosting the thumb through the side and back until it touches the forearm (one point on each side);

3) hyperextension of the elbow joint by 10° (one point on each side);

4) hyperextension of the knee joint by 10° or more (one point on each side). The diagnosis of hypermobility is made at 6 or more points.

Fibromyalgia. This often occurring syndrome is characterized by the following features:

1) poor sleep, feeling tired when waking up;

2) malaise, drowsiness;

3) irritability;

4) multiple regional pains, including pains in the spine, which do not decrease when taking analgesics;

5) hypersensitivity of certain points during palpation (the lower part of the cervical spine, the middle of the supraspinatus muscle);

6) soreness of the lateral epicondyle, lower lumbar spine, upper gluteal region.

SEMIOTICS OF THE DAMAGE OF THE MUSCLE SYSTEM THE STATE OF THE SKELETAL MUSCLES.

In the study of the musculoskeletal system, attention is paid to the condition of the skeletal muscles. When examining the muscles, the right and left sides are compared, the symmetry of the muscle groups, the presence of atrophy (amyotrophy) and the symptom of a “hollow” hand, indicating the participation of muscles in the pathological process, are ascertained. There are mild, moderate and severe degree of amyotrophy. Multiple joint involvement causes commonly widespread atrophy. Local atrophy is more characteristic of mechanical damage to the tendons, muscles or nerve. Palpation of the muscles is carried out with their complete relaxation. It gives an idea of muscle tone, the presence of myogenic contractures, rigidity. Muscle percussion allows you to detect pain points,

myogilosis - painful muscle compaction. Complaints of muscle pain - myalgia - are characteristic of both joint diseases and a number of rheumatic diseases with damage to muscle tissue. It should be noted that children with rheumatic diseases often have pain in the area of ligaments, tendons, in places of attachment of tendons to bones (enthesopathy). Muscle malformations are more often manifested in underdevelopment (for example, underdevelopment of the sternocleidomastoid muscle - torticollis, underdevelopment or absence of the pectoralis major, deltoid muscle, which causes deformation of the shoulder, disrupts its function), developmental anomalies (for example, anomalies in the development of the diaphragm with the formation of diaphragmatic hernia). Muscle injuries are divided into open (wounds) and closed (bruises, hemorrhages, ruptures).

Muscle atrophy is a reversible or irreversible violation of muscle trophism with the phenomena of thinning and degeneration of muscle fibers, a significant decrease in muscle mass, weakening or loss of their contractility. It can be congenital and acquired, primary and secondary. It is one of the main symptoms of many hereditary diseases of the neuromuscular system of a child (Werdnig-Hoffmann amyotrophy, Aran-Duchen, Charcot-Marie-Tooth, etc.). In early childhood, it occurs with malnutrition of the 2nd or 3rd degree, prolonged immobility, somatic diseases, prolonged immobilization, etc. Local muscle atrophy can develop with parasitic diseases (toxaplasmosis, trichinosis).

Muscle tone. Changes in muscle tone include a number of pathologies: Atony - loss of muscle tone (hypotension), as a result of which the affected limb is relaxed and joints are loose (Guillain-Barré syndrome, acute phase of spinal cord injury (spinal shock) or stroke).

Muscular hypotension - reduced resistance to passive movements, muscle flabbiness on palpation. Muscular hypotension is accompanied by an increase in the range of motion in the joint (hyperextension).

Muscular hypertension is an increase in resistance to passive movements. On palpation, increased muscle density is noted, spontaneous and voluntary motor activity is limited or impossible.

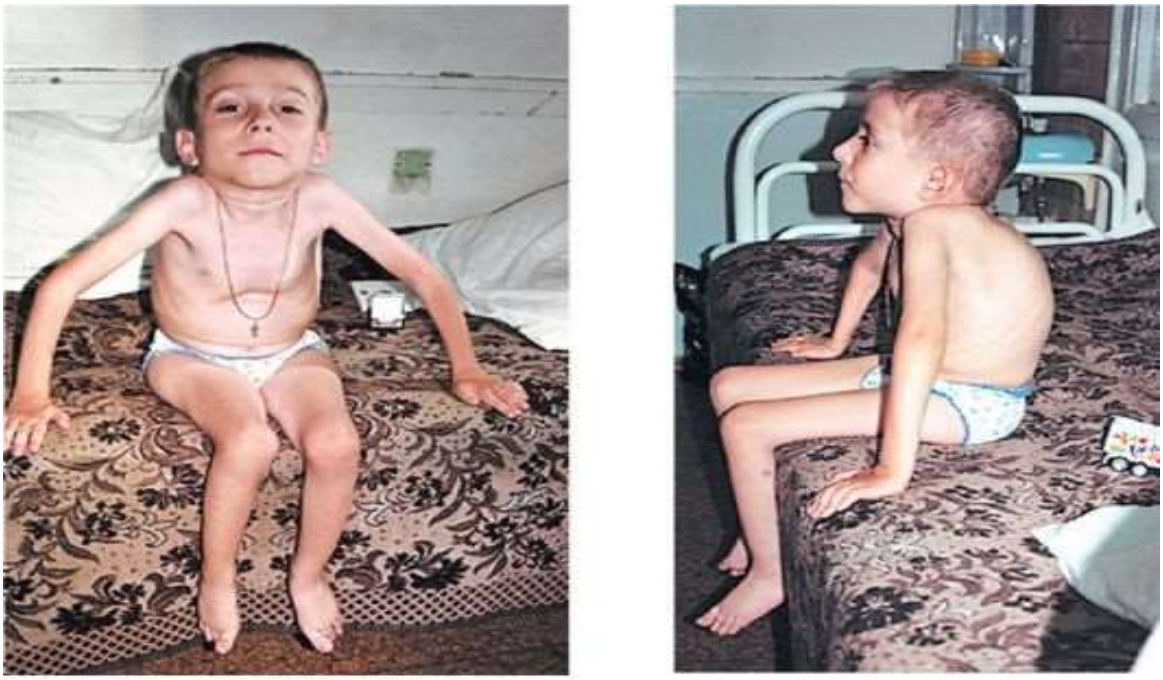


Figure No. 44. View of a sick child with muscle hypotension

Muscular dystonia - hypotension alternates with hypertension: at rest, with passive movements, muscle hypotonia is expressed, when you try to actively perform any movement, muscle tone increases.

Myotonia is a condition in which it is sharply difficult to relax the muscles after a strong contraction. Myotonia congenita is called Thomsen's myotonia. Myatonia is congenital, pronounced general hypotension or complete atony of skeletal muscles (Oppenheim's disease).

Myasthenia gravis is a neuromuscular disease with symptoms of muscle weakness and increased fatigue, related to autoimmune disorders. Occurs with a block of neuromuscular transmission (70% of patients have a thymus tumor).

Spasticity is an increase in muscle tone, depending on the speed of movement (hypertonicity). The increase in tone is more pronounced with fast passive movements than with slow ones, at the beginning of the movement and at its end. The resistance experienced at the start of rapid passive flexion and extension may suddenly disappear as the limb relaxes. This violation of tone is called the symptom of the "jackknife". It can occur with a stroke, especially in the long term.

Rigidity is a steady increase in muscle tone, a uniform muscle resistance to passive movement, for example, neck stiffness (limiting the patient's ability to bring the chin closer to the chest due to a reflex increase in the tone of the cervical muscles) with meningitis. Muscle resistance that persists throughout the duration of a passive movement, regardless of its speed, is called lead-wire rigidity. Such a violation of muscle tone is also referred to as "wax flexibility". With passive flexion of the hand or forearm, intermittent, stepped muscle resistance may occur, which is called the “cog wheel” symptom.

Occurs in parkinsonism. Paratonia is a sudden change in tone during passive movements. A sudden decrease in tone that makes movement easier is called (in German terminology) the phenomenon of mitgehen (joint movement), and a sudden increase in tone that makes movement difficult is known as gegenhalten (holding).

CHANGES IN MOTOR ACTIVITY.

Hypokinesia - limitation of range of motion due to various nervous, muscular, bone, articular pathologies.

Hyperkinesia - violent involuntary movements of the muscles of the face, trunk, limbs. Hyperkinesia can be observed in separate muscle groups of the distal (more often) or proximal sections, or acquire a generalized character. To identify hidden hyperkinesia, the child is asked to copy the movements made by the doctor, to collect scattered small balls or matches. At an older age, you can ask the child to write any text with his eyes closed.

There are the following variants of involuntary hyperkinesis:

1. Athetosis - continuous, slow, worm-like movements, tonic forced movements in the distal parts of the upper limbs, less often in the muscles of the face (distortion of the mouth, protrusion of the lips) or lower limbs. With athetous damage to the muscles of the body, they speak of torsion spasm.

2. Myoclonus - muscle spasms of different amplitude and duration, single or occurring in series in different muscles, both at rest and during movement.

3. Tremor (trembling) - rapidly intermittent involuntary contractions and

relaxation of muscle groups, causing rhythmic movements of insignificant amplitude of various parts of the body (head, eyelids, lower jaw, fingers, hands, and so on). It can be small-sweeping (10–20 twitches per 1 s), medium-sweeping (3–9 twitches per 1 s). There are pallidar (only at rest, disappears during movements) and intentional (occurs during voluntary movements, has the character of large amplitude oscillations and becomes especially sharp at the end of the motor act at the moment the goal of the movement is reached) tremor.

4. Tic - involuntary movements in the form of blinking eyelids, twitching of the shoulder.

5. Hemiballismus - sweeping, large movements, mainly in the proximal limbs of one half of the body, imitating the movements of throwing, waving a hand, throwing a sling.

6. Dystonia - movements are somewhat athetoid, but often involve more muscles, including those of the trunk. As a result of these movements, the body takes on bizarre, artsy positions. It is observed during treatment with drugs of the phenothiazine series, primary torsion dystonia and spastic torticollis.

7. Chorea - characterized by rapid non-rhythmic unpredictable twitches. They may occur at rest or interrupt normal coordinated movements. Unlike tics, movements in chorea are not stereotyped. The movements involve the face, head, upper and lower limbs. It is observed in rheumatism (Sydenham's chorea) and Huntington's disease.

8. Orofacial dyskinesia - rhythmically repetitive movements due to selective contraction mainly of the muscles of the face, tongue, which manifest themselves in the form of bizarre grimaces, movements of the lower jaw, lips, protrusion of the tongue. May be a manifestation of the side effects of psychotropic drugs with prolonged use (in particular, drugs of the phenothiazine series). It is called tardive dyskinesia. It is observed in long-term psychoses, in patients who have lost their teeth.

Coordination disorders include:

1. Ataxia - manifested by impaired coordination of movements (unsteady,

staggering gait, impaired actions with objects) or impaired coordination of balance (when sitting, standing).

2. Changes in neuromuscular excitability. Increased neuromuscular excitability is manifested by convulsions - involuntary muscle contractions that occur suddenly in the form of seizures and have a different duration. Paralysis (plegia) is the complete absence of voluntary movements due to damage to the cortico-muscular pathway.

Paresis - limited range of motion and reduced strength. Paralysis is distinguished:

- central - damage to the central motor neuron in any of its parts - muscle hypertension, hyperreflexia, clonuses of the feet, kneecaps, hands, pathological reflexes, protective reflexes, pathological synkenesias (friendly involuntary active movements);

- peripheral - damage to the peripheral motor neuron in any of its areas - areflexia, muscle atony and atrophy, sometimes fibrillar or fascicular muscle twitches. Paralysis of one limb is called monoplegia, paresis - monoparesis. Paralysis of two limbs - diplegia (paraplegia and hemiplegia). Paralysis of all four limbs is called tetraplegia.

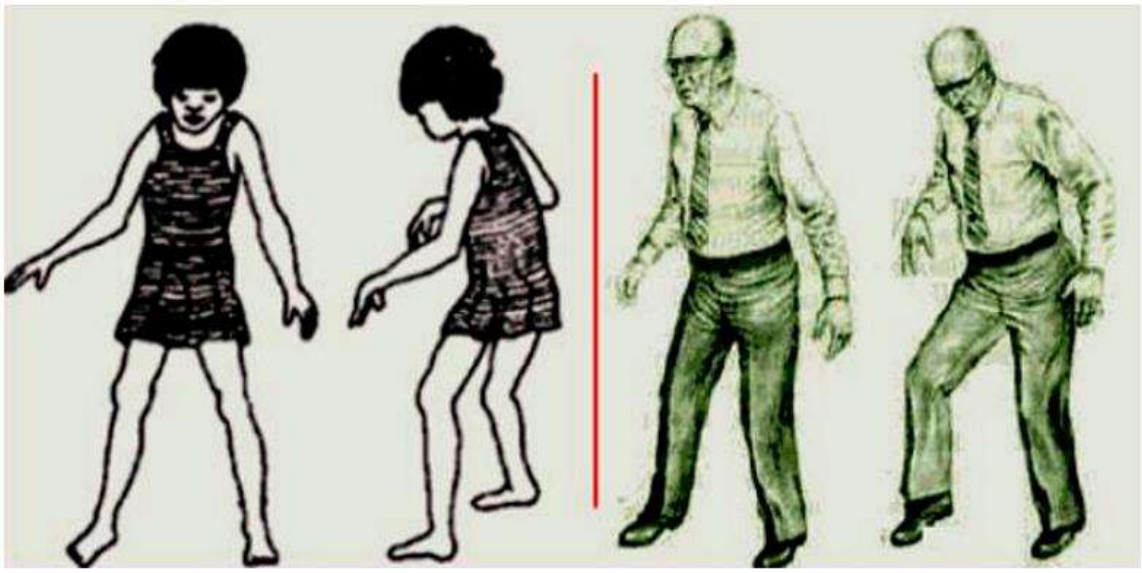


Figure 45. View of a patient with paralysis

SEMIOTICS OF CARDIOVASCULAR DAMAGE

Visual inspection. In assessing the state of the cardiovascular system, external examination is of great importance. Some forms of pathology of the cardiovascular system are accompanied by a forced position of the child. So, with circulatory failure II-III degree, patients take a sitting or reclining position, which greatly facilitates their condition. With pericarditis, patients take a bent position, leaning on something with their hands or putting a pillow to their chest. An external examination may reveal a sharp pallor or cyanosis of the skin, bulging of the chest in the region of the heart (heart hump). The heart hump is formed with congenital heart defects (CHD), cardiomegaly (which developed in early childhood, when the chest was pliable), hypertrophy of the heart muscle (mainly the left ventricle), a slight protrusion may appear with prolonged pericarditis with a large amount of effusion in the pericardium.

An external examination of the chest in some children can reveal an increase in the subcutaneous venous pattern, indicating a difficult flow of venous blood to the right heart (triad and tetrad of Fallot, Eisenmenger's syndrome,

adhesive pericarditis). In patients with cardiovascular pathology, edema may be detected, which indicates circulatory failure associated with weakness of the right ventricle. Edema of cardiac origin is distinguished by the following features: they appear and are expressed on the periphery and low-lying areas of the body: feet, lower third of the lower leg, if the patient walks; on the back, in the region of the sacrum, if the patient is lying. As heart failure progresses, edema increases, sometimes to the extent of anasarca. With a long change in body position, cardiac edema changes its location. A constant blush on the cheeks with a cyanotic tinge is characteristic of mitral valve defects. Increased pulsation of the carotid arteries is observed with an open ductus arteriosus (ODA), insufficiency of the aortic semilunar valves. Increased pulsation of the carotid arteries can be observed in febrile patients, with thyrotoxicosis, strong nervous excitement. Pronounced pulsation of the cervical veins and their increased blood supply occur with stagnation of blood and incomplete or difficult evacuation of it from the right atrium. In this case, the pulsation does not coincide with the pulse on the carotid artery, and its amplitude is small. Severe swelling of the cervical veins and their large amplitude of fluctuations accompany tricuspid valve insufficiency. With this heart disease, the pulsation coincides with the pulse on the carotid artery (a symptom of a positive venous pulse). Increased pulsation in the region of the jugular notch of the sternum may be with an atrial septal defect (ASD) due to expansion and increase in the volume of the atrium.

May occur in mitral valve disease. Pronounced epigastric (epigastric) pulsation occurs with an increase in the mass and volume of the right ventricle. Epigastric pulsation is found in mitral heart disease, aortic valve insufficiency, severe relative tricuspid valve insufficiency.

Palpation. The apex beat is determined visually and by palpation. Appears as a result of the transmission of a systolic heart beat to the anterior chest wall. The localization of the apex beat is influenced by the phase of breathing: with a deep breath, the push moves down, with a deep exhalation, it moves up. These displacements are classified as physiological. Pathological upward displacement of

the apex beat is noted with a high standing of the dome of the diaphragm, caused by ascites, flatulence. A downward shift is noted with emphysema, cor pulmonale. The apex beat shifts to the left when the entire heart is displaced to the left (displacement of the heart by pleural effusion, hydrotorox, pneumothorax on the right, with atelectasis of the left lung). To the right, the apex beat shifts with similar pathological processes of left-sided localization. Left ventricular hypertrophy shifts the apex beat to the left and down. The displacement is more pronounced with a combination of hypertrophy and dilatation of the left ventricle. The strength of the apex beat depends on changes in the heart itself and on extracardiac factors. An increased apex beat is observed with left ventricular hypertrophy, aortic valve insufficiency. The apical impulse is increased in thyrotoxicosis, significant weight loss, when the chest wall becomes very thin, with tumors in the posterior mediastinum, which tightly push the heart to the anterior chest wall. The weakening of the apex beat is observed in myocarditis, acute cardiovascular failure, pericarditis. A significant accumulation of fluid in the pericardial cavity leads to the disappearance of the apex beat. Some non-cardiac factors: obesity, subcutaneous fat edema, subcutaneous emphysema - also cause a weakening of the strength of the apical impulse. Apex beat for hypertrophy and dilatation and the heart becomes spilled, its area increases. On palpation of the chest, the phenomenon of trembling of the chest wall is determined. If the trembling coincides with the systole phase, it is called systolic, with the diastolic phase - diastolic ("cat's purr"). Systolic trembling is most often determined with congenital heart disease. With a ventricular septal defect (VSD), trembling is localized in the IV-V intercostal space at the left edge of the sternum, with ASD - at the base of the heart, with stenosis of the aortic opening - above the place of its exit, in the II-III intercostal space to the right of the sternum. With mitral valve insufficiency in children, systolic tremor can be determined at the apex of the heart. Systolic-diastolic trembling is typical for PDA and is determined in the second intercostal space to the left of the sternum or in the left subclavian fossa. However, as pulmonary hypertension develops in PDA, the diastolic component

gradually decreases and may disappear completely. The study of the pulse is performed by palpation on the radial, femoral arteries, dorsal artery of the foot. The characteristic of the pulse includes its frequency, rhythm, tension, filling, size, shape, symmetry of the listed indicators on the right and left. The pulse rate in childhood has age-related features. In young children, a more frequent pulse due to the predominant effect on the function of the cardiovascular system of the sympathetic link of the autonomic nervous system, increased metabolism at this age. In a healthy child, an increase in heart rate (tachycardia) occurs with fright, fear, joy, strong excitement, as well as after eating, physical activity, in hot weather. With cardiovascular pathology, circulatory failure, thyrotoxicosis, anemia, viral and bacterial diseases, pathological tachycardia occurs. It is believed that with an increase in temperature by 1 ° C, the pulse in children quickens by 10-12 beats / min. In diseases such as typhoid fever, tuberculous meningitis, occurring with fever, the pulse rate lags behind the level of temperature rise. With endomyocarditis, peritonitis, the pulse rate is significantly ahead of the temperature increase. Decreased heart rate (bradycardia) in healthy children is observed during sleep, may be an individual feature, occurs in young athletes. Pathological bradycardia occurs with viral hepatitis, typhoid fever, various heart blocks, myocarditis, myxedema, and an overdose of digitalis preparations. Under the rhythm of the pulse understand the alternation of pulse waves of the same amplitude at regular intervals. A change in this alternation is referred to as an arrhythmic, uneven pulse and is associated with a violation of the basic functions of the heart: excitability, contractility, conductivity and automatism. In children, the most common respiratory arrhythmia, which is a change in the rhythm of the pulse under the influence of the phases of respiration. Respiratory arrhythmia increases with the excitement of the child, crying, in children with autonomic dysfunction, especially in prepubertal and pubertal. Extrasystolic arrhythmia is characterized by the appearance behind the normal pulse wave of another, weaker wave and is caused by extraordinary contractions of the heart due to additional myocardial excitation impulses. A more serious prognosis is atrial fibrillation,

characterized by chaotic pulse waves and often a pulse deficit, i.e., a reduced number of pulse waves on peripheral vessels compared to the number of heartbeats. The pulse deficit becomes more distinct after exercise, which is used for the differential diagnosis of atrial fibrillation and extrasystole: with atrial fibrillation, the deficit increases, and with extrasystole, it decreases or disappears.

Auscultation. **Heart tones**. Auscultation of the heart reveals sound phenomena called heart sounds. I tone is formed from several components: valvular (slamming and vibration of the cusps of the bicuspid and tricuspid valves), muscle (from the contraction of the atria and ventricles), vascular (sound formed by the vibration of the walls of the aorta and pulmonary artery when stretching the blood pouring into them from the ventricles). Two components take part in the formation of the II tone: the slamming of the valves of the aorta and pulmonary artery and the oscillation of the valves of these valves. The slamming of the valves occurs in the diastolic phase, so the II tone is also called diastolic. III tone is formed due to the rapid tension and expansion of the walls of the ventricles by a stream of blood entering from the atria during the onset of diastole. It follows a short period of time after the II tone. Of great importance is the place of the best listening to the valves. The bicuspid (mitral) valve is best heard at the apex of the heart and at the site of attachment to the sternum of the IV rib on the left; tricuspid - at the point of attachment to the sternum of the 5th rib on the right, the valves of the pulmonary artery - in the second intercostal space at the left edge of the sternum, the aortic valves - in the second intercostal space on the right at the edge of the sternum. The aortic valves can be heard in the third intercostal space on the left side of the sternum (Botkin's point). Heart tones are characterized by strength, timbre, shape (configuration). Under the power of heart tones understand their loudness. By strength, heart tones can be weakened or amplified (accented). The weakening of the I tone at the apex of the heart is observed with insufficiency of the bicuspid valve (due to deformation of the valve, its slamming is incomplete, and the slamming sound is weak). Weak I tone at the apex of the heart with narrowing of the aortic orifice or aortic valves due to increased blood filling of the

left ventricle and slow expulsion of blood from its cavity. I tone can be weakened at the top of the heart with myocarditis, myocardial dystrophy. The weakening of the II tone on the aorta is observed with insufficiency of the aortic valves, as well as with narrowing of the aorta at the place of its exit from the heart, due to which the pressure in the aorta decreases and the force of the valves slamming decreases. The strengthened I tone on a top is characteristic of a mitral stenosis. With this defect, the left ventricle is not completely filled during diastole, as a result of which it contracts quickly, and the valve closes quickly with a characteristic popping sound (“clapping” tone, “cannon” tone). For the same reason, the I tone at the apex of the heart is enhanced in the case of ventricular extrasystole and atrial fibrillation. Strengthening (emphasis) of the II tone on the aorta occurs with an increase in blood pressure in the systemic circulation, in the early stages of the development of left ventricular hypertrophy. A short-term variant of the accent of the II tone on the aorta can appear in children and adolescents with excessive excitement, during sports competitions, and enhanced training. Emphasis of tone II on the pulmonary artery always appears with hypertension in the pulmonary circulation, which occurs with all heart defects with overflow of the pulmonary circulation (mitral stenosis, Eisenmenger's syndrome, ASD, VSD). The emphasis of the II tone is accompanied by such lung diseases as emphysema, pneumonia with extensive damage to the lung tissue, pneumosclerosis, effusion pleurisy, and tuberculous lesions of the lungs. Chest deformities (kyphosis, scoliosis, funnel-shaped chest) may be accompanied by an accent of II tone on the pulmonary artery due to limited lung mobility. An increase in both heart sounds can be detected in healthy children with physical exertion, nervous excitement, but most often an increase in both tones occurs with thyrotoxic goiter, high fever, left ventricular hypertrophy, a tumor process in the posterior mediastinum (the heart is pressed against the anterior chest wall), sometimes with anemia, accumulation of air in the pericardial cavity. Speaking about the third tone, it should be noted that the strengthening of the tone, which is noted during the "gallop" rhythm, is of practical

importance. The weakening or absence of the III tone does not play a diagnostic role.

Figure 46. Scheme of auscultation points of the heart

Under the timbre of heart tones is meant a sound characteristic (voiced, deaf, clapping). A deaf I tone at the apex of the heart is heard with myocarditis, intoxication against the background of acute infectious diseases (typhoid or typhus, scarlet fever). Flapping I tone at the apex of the heart, resembling the sound of a towel flapping in the wind, is heard with mitral stenosis due to sclerotic changes in the cusps of the bicuspid valve. Violation of the form, or configuration, of heart tones manifests itself in the form of their bifurcation or splitting. The basis of this sound phenomenon is the perception of the tone being heard not as a single whole, but as two short sounds following one after another. If both sounds that make up the tone are distinguishable when listening, they speak of a bifurcation of the tone, but if both components of the tone differ indistinctly, they speak of a splitting of the tone. Bifurcation and splitting of the I tone can be heard in healthy children as a result of non-simultaneous slamming of the bicuspid and tricuspid valves. Pathological bifurcation of the I tone is best heard at the left edge of the sternum at the level of the third or fourth intercostal space. Allocate systolic and presystolic bifurcation of the first tone. Systolic bifurcation of the I tone can be observed when there is a violation of conduction along the right or left leg of the atrioventricular

bundle, which causes a violation of the synchrony of the contraction of both ventricles (longitudinal dissociation of the heart). This effect occurs in rheumatic myocarditis, aortic valve insufficiency and is heard in the third intercostal space along the left midclavicular line. Presystolic bifurcation and splitting of the I tone occurs as a result of a slowdown in the conduction of an impulse between the atria and ventricles (transverse dissociation of the heart) in rheumatic carditis, myocardiosclerosis, myocardial dystrophy, during the isolation of components of the hypertrophied atrial contraction from the components of the I tone. Physiological splitting of the II tone is usually heard in the second and third intercostal space on the left. The pulmonary component of the II tone is too weak to be heard at the apex of the heart or in the aortic region. Normally, splitting increases during inhalation and usually disappears during exhalation. In some children, an unexpressed split may be heard during exhalation when the patient is sitting. Bifurcation of the II tone appears with an increase in physiological splitting, which continues throughout the entire respiratory cycle. Bifurcation may result from delayed closure of the pulmonary valves (eg, in pulmonary stenosis or right bundle branch block). The blockade of the right leg of the bundle of His also causes splitting of the I tone into its mitral and tricuspid components. A bifurcation of the II tone can occur with early closure of the aortic valves (for example, with mitral insufficiency). In contrast to the clear bifurcation and splitting of the II tone at the base of the heart, the "melody" of the heart can be heard at the top in the form of a quail rhythm or a gallop rhythm. The quail rhythm is a three-part rhythm resembling the singing of a quail and is heard with mitral stenosis. It is permanent and disappears after surgical treatment of pathology. The gallop rhythm resembles the sound of a galloping horse. With this rhythm, two normal tones and one additional tone are heard. It may be with mitral stenosis and indicates the depletion of the reserve capacity of the heart muscle. The equality of the intervals between systole and diastole is perceived auscultatively as a pendulum-like rhythm (reminiscent of the movement of a clock pendulum). Such a rhythm occurs in patients with myocarditis and also indicates a significant lesion of the heart muscle.

Heart murmurs. Description of the noise should include the following components: - period in relation to the phases of the heart - systolic, diastolic; - intensity - from quiet to loud; - character - soft (blowing), rough, rumbling, etc .; - localization in accordance with the accepted areas of auscultation of the heart; - carrying out in other areas. According to their origin, noises are divided into intracardiac, i.e., caused by damage to the heart and formed inside it, and extracardiac, formed outside the organ. Intracardiac murmurs occur as a result of damage to the valves, septa of the heart, improper discharge of large main vessels from it, with significant damage to the myocardium. In relation to the phases of cardiac activity, noises are divided into systolic and diastolic. Systolic murmurs are heard after a long interval after the I heart sound, diastolic - after the II tone (between the II and I tone). There are several types of diastolic murmurs: proto-diastolic (noise is superimposed on the II tone and replaces it), meso-diastolic (the noise is heard in the middle of the diastole), presystolic (the noise precedes the I tone, sometimes it is heard immediately before it). Intracardiac (organic) murmurs are a sign of anatomical changes in the valvular apparatus of the heart, myocardium, vessels from the heart. When the heart valves are damaged, murmurs appear due to the flow of blood through the narrowed holes between the valve leaflets or when the blood flows back through the altered leaflets that have not closed. Noises during narrowing of the holes occur during the movement of blood in physiological directions, with valve insufficiency - as a result of the movement of blood in the opposite direction. Based on this, noises during diastole appear with narrowing of the atrioventricular orifices (left and right) and aortic and pulmonary valve insufficiency, and during systole - with narrowing of the outlet openings of the aorta and pulmonary artery and insufficiency of the bicuspid and tricuspid valves. The main cause of heart murmurs in children are congenital, acquired heart defects, myocardial damage. Systolic murmur is heard in such CHD as VSD, ASD, aortic orifice stenosis, aortic coarctation, pulmonary artery stenosis, Eisenmenger's syndrome, PDA with backflow of blood from the pulmonary artery into the aorta with a high degree of hypertension in the pulmonary circulation. Of the acquired

heart defects, systolic murmurs are accompanied by insufficiency of the bicuspid and tricuspid valves, narrowing of the aortic valves and pulmonary artery valves. Diastolic murmur is heard in PDA, Ebstein's syndrome, Eisenmenger's syndrome with a sharp expansion of the pulmonary artery, with acquired heart defects - narrowing of the left or right atrioventricular orifices, insufficiency of aortic valves, pulmonary artery valves. Systolic-diastolic murmur is heard in PDA without pulmonary hypertension, as well as in combination of PDA with other malformations. The most complete information about the nature of the noise is given by listening to the patient in the supine position, standing, with little physical exertion. Organic noise is characterized by the invariability of the acoustic pattern or their amplification with a change in position or physical activity. The conduction of noises from the place of their maximum sound in certain directions is of diagnostic importance. Systolic murmur during narrowing of the aorta is carried out along the blood flow to large vessels (carotid and subclavian arteries), with narrowing of the pulmonary artery - towards the right ventricle and behind the back to the interscapular region. Conduction of noises at CHD is specific. So, with VSD, the noise is carried out in all directions - to the left, to the right, up, on the back. It can be heard in the interscapular region, on the shoulder blades and even on the mastoid processes. With an ASD, the noise is conducted up the large vessels of the neck. Noise (diastolic) with narrowing of the left atrioventricular orifice is limited towards the left atrium, with insufficiency of the aortic valves - towards the left ventricle and large vessels of the neck. Functional noises in children are due to age-related anatomical and physiological features of the structure of large vessels, changes in blood flow velocity, blood composition, and features of the jet movement of blood in the chambers of the heart and large vessels.

Features of functional noise:

1) softer timbre, not loud compared to the noise of organic origin, sometimes reminiscent of the sound of a light rustle;

2) do not have certain specific points of maximum auscultation, diffuse, distributed over the entire region of the heart;

3) are not carried out anywhere and are not accompanied by a pathological increase in heart tones;

4) change their intensity depending on the position of the patient's body: they are heard more clearly in the supine position, disappear or significantly reduce their intensity when the phonendoscope is pressed on the region of the heart, which never happens with organic noises;

5) are not accompanied by pronounced changes in the electrocardiogram;

6) occur in febrile patients, excitable, irritable children, with thyrotoxicosis, compression of blood vessels by enlarged lymph nodes, thymus gland, chest deformity;

7) can occur when blood moves from the chambers of the heart to the pulmonary artery or aorta, if the latter have a larger diameter than the average values.

Extracardiac (extracardiac) murmurs occur over the region of the heart or in areas adjacent to the heart, but can be synchronously associated with the phases of the activity of the heart. These murmurs include: pericardial friction murmur, pleuropericardial murmur, cardiopulmonary murmur. Pericardial friction noise occurs with dry pericarditis during friction of the pericardial sheets. The friction noise is superimposed on the heart tones and is heard as a four-term rhythm, two elements of which are heart tones, and two are pericardial friction noises.

The friction noise of the pericardium resembles the crunch of snow underfoot. With dehydration, pyloric stenosis, pericardial friction noise may occur due to a violation of the smoothness of the surface of the pericardial sheets. This noise is heard in uremic coma, when nitrogenous slag products sweat through the serous membranes and make them rough. Pleurocardial murmur often appears with left-sided pleurisy. With this localization of inflammation, the pleura lining the costal mediastinal sinus comes into contact with the pericardium, since with a contraction of the heart and a decrease in its volume, the lung expands and brings the inflamed area of the pleura closer to the pericardium. Noise coincides with the activity of the heart, its contraction phases. Often this murmur is mistaken for

pericardial, but unlike the latter, pleuropericardial murmur is heard along the left border of the heart, and pericardial murmur is heard along the entire perimeter of cardiac dullness. Pleurocardial murmur increases with deep inspiration, with holding the breath, either weakens or disappears altogether. Cardiopulmonary noise is heard over the projection of the left ventricle during the systole of this part of the heart and is explained by the penetration of air into the marginal alveoli of the lung tissue. Increases during inhalation.

CONGENITAL HEART DEFECTS.

Common clinical manifestations of CHD:

1) cyanosis or pallor of the skin;

2) protein-energy deficiency;

3) frequent colds, including those occurring with bronchitis (more than 3-4 times a year);

4) insufficient tolerance of adequate loads, shortness of breath;

5) an increase in the size of the heart (percussion, ECG, radiograph);

6) change in heart sounds;

7) appearance of murmurs in the heart;

8) change in the pulse on the hands, a weak pulse on the femoral artery or its absence;

9) change in blood pressure;

10) deformation of the chest;

11) violation of the heart rhythm;

12) unusual changes revealed on the radiograph. The presence of a “triad” of symptoms (cyanosis or pallor of the skin, the appearance of heart murmurs, as well as insufficient tolerance for adequate exercise and shortness of breath) with a sufficient degree of probability allows the doctor to suspect congenital heart disease in a small patient.

General characteristics of noise in CHD:

- more often it is systolic murmur;

- maximum auscultation - to the left of the sternum (second-fourth

intercostal space);

– pronounced low-frequency component; – there is no direct dependence of the noise intensity on the size of the defect. Changes in hemodynamics inside the heart:

- gateway syndrome - a narrowing or stenosis that prevents blood flow;

- reset syndrome - abnormal communication between the chambers of the left and right heart or vessels flowing into them.

Tetralogy of Fallot - CHD, accompanied by cyanosis, occurs in 10% of all children with this pathology.

Defects of the heart and blood vessels are composed of the following 4 components:

1. Patients have an intermediate position of the aorta, in which it is able to collect blood from both the left and right ventricles of the heart.

2. Narrowing (stenosis) of the pulmonary artery.

3. Defect (hole) in the interventricular septum.

4. Hypertrophy of the right ventricle. The main defect is the narrowing of the pulmonary artery. There is insufficient blood flow to the lungs. They are small-blooded. On x-ray, such lungs look transparent, with vessels depleted of blood. In the lungs, the blood is not sufficiently enriched with oxygen, so the composition of the arterial blood of patients resembles venous blood. Other defects are more adaptive in nature and allow patients to survive, although, of course, their quality of life is very low. The special, intermediate location of the aorta, the opening between the right and left ventricle and hypertrophy of the right ventricle make it possible to remove excess blood from the right parts of the heart, bypassing the stenotic area of the pulmonary artery into the left ventricle of the heart, and to let venous blood into the large (arterial) circulation. cyanotic seizures. In response to physical activity, colds, in conditions of erythrocyte polycythemia, which causes great difficulty in the movement of blood through the vessels of the lungs, blood circulation in the lungs can suddenly deteriorate sharply. There is an acute debt of the body for oxygen, which causes the brain to suffer. The child acquires a cast-

iron gray color, breathes rapidly (“panic breathing”), becomes restless, which further aggravates the severity of the condition. Against this background, hypoxemic convulsions may develop. Characteristically, children 2 years and older learn to fight the onset of an attack by squatting down. By squeezing the large arteries of the arms and legs, they reduce the movement of blood in the aorta, directing it to the pulmonary artery. Because of this, blood flow in the lungs and blood oxygenation improve. From the age of 1–2 years, the formation of the symptom of "drum sticks" and "watch glasses" is noted. When examining the heart, a visible pulsation of the chest wall to the left of the sternum is determined, which is a manifestation of right ventricular hypertrophy. Trembling of the chest wall is also found there (in about half of the children). During auscultation, rather loud tones are heard, the second heart sound is always the same (S1 + S2). Systolic murmur (ejection murmur) occupies the entire systole, is heard over the region of the pulmonary artery, complements the characteristics of the symptoms determined by cardiac examination. The heart is usually not enlarged and congestive heart failure does not develop. Ventricular septal defect (VSD) is the most common heart defect, accounting for 20–30% of all CHD cases in children.

Hemodynamic disturbances are determined by:

- discharge of blood from left to right;

– the magnitude of the reset (d-defect);

- the presence of pulmonary hypertension;

- hypertrophy of the heart.

There are two forms of defect:

- low small defects (of the muscular part of the septum);

- high defects in the membranous part. A VSD causes an abnormal movement of blood inside the heart from the left ventricle, which is more powerful, to the right. An excess of blood flow into the right ventricle of the heart causes an increase in the blood supply to the lungs, their "swamping". With a significant size of the defect, the development of congestive heart failure is possible. With a small defect, or the so-called Tolochinov-Roger disease, children

do not show any signs of trouble and develop satisfactorily. Small defects can spontaneously close. A constant and typical symptom of VSD is a rough and loud systolic murmur. Sometimes it occupies the entire systole and is therefore called pansystolic. Noise is heard above the anatomical defect, that is, in the third or fourth intercostal space to the left of the sternum. Having found such a noise, one should compare its intensity at the apex and in the epigastric region (under the xiphoid process). If its intensity at these points is more pronounced, then it may come from the mitral or tricuspid valve with their insufficiency, although congenital malformations of these valves are many times less common than VSD.

Cardiomegaly and congestive heart failure (CHF) occur with moderate to large septal defects. Hypertrophy of the heart occurs in all departments and is manifested by a “heart hump” and a pulsation of the chest wall in the parasternal region to the left of the group.

General characteristics of noise in CHD:

- more often it is systolic murmur;

- maximum auscultation - to the left of the sternum (second-fourth intercostal space);

– pronounced low-frequency component; – there is no direct dependence of the noise intensity on the size of the defect. Changes in hemodynamics inside the heart:

- gateway syndrome - a narrowing or stenosis that prevents blood flow; - reset syndrome - abnormal communication between the chambers of the left and right heart or vessels flowing into them. Tetralogy of Fallot - CHD, accompanied by cyanosis, occurs in 10% of all children with this pathology. Defects of the heart and blood vessels are composed of the following 4 components: 1. Patients have an intermediate position of the aorta, in which it is able to collect blood from both the left and right ventricles of the heart. 2. Narrowing (stenosis) of the pulmonary artery. 3. Defect (hole) in the interventricular septum. 4. Hypertrophy of the right ventricle. The main defect is the narrowing of the pulmonary artery. There is insufficient blood flow to the lungs. They are small-blooded. On x-ray, such lungs

look transparent, with vessels depleted of blood. In the lungs, the blood is not sufficiently enriched with oxygen, so the composition of the arterial blood of patients resembles venous blood. Other defects are more adaptive in nature and allow patients to survive, although, of course, their quality of life is very low. The special, intermediate location of the aorta, the opening between the right and left ventricle and hypertrophy of the right ventricle make it possible to remove excess blood from the right parts of the heart, bypassing the stenotic area of the pulmonary artery into the left ventricle of the heart, and to let venous blood into the large (arterial) circulation. cyanotic seizures. In response to physical activity, colds, in conditions of erythrocyte polycythemia, which causes great difficulty in the movement of blood through the vessels of the lungs, blood circulation in the lungs can suddenly deteriorate sharply. There is an acute debt of the body for oxygen, which causes the brain to suffer. The child acquires a cast-iron gray color, breathes rapidly (“panic breathing”), becomes restless, which further aggravates the severity of the condition. Against this background, hypoxemic convulsions may develop. Characteristically, children 2 years and older learn to fight the onset of an attack by squatting down. By squeezing the large arteries of the arms and legs, they reduce the movement of blood in the aorta, directing it to the pulmonary artery. Because of this, blood flow in the lungs and blood oxygenation improve. From the age of 1–2 years, the formation of the symptom of "drum sticks" and "watch glasses" is noted. When examining the heart, a visible pulsation of the chest wall to the left of the sternum is determined, which is a manifestation of right ventricular hypertrophy. Trembling of the chest wall is also found there (in about half of the children). During auscultation, rather loud tones are heard, the second heart sound is always the same (S1 + S2). Systolic murmur (ejection murmur) occupies the entire systole, is heard over the region of the pulmonary artery, complements the characteristics of the symptoms determined by cardiac examination. The heart is usually not enlarged and congestive heart failure does not develop. Ventricular septal defect (VSD) is the most common heart defect, accounting for 20–30% of all CHD cases in children. Hemodynamic disturbances

are determined by: - discharge of blood from left to right; – the magnitude of the reset (d-defect); - the presence of pulmonary hypertension; - hypertrophy of the heart. There are two forms of defect: - low small defects (of the muscular part of the septum); - high defects in the membranous part. A VSD causes an abnormal movement of blood inside the heart from the left ventricle, which is more powerful, to the right. An excess of blood flow into the right ventricle of the heart causes an increase in the blood supply to the lungs, their "swamping". With a significant size of the defect, the development of congestive heart failure is possible. With a small defect, or the so-called Tolochinov-Roger disease, children do not show any signs of trouble and develop satisfactorily. Small defects can spontaneously close. A constant and typical symptom of VSD is a rough and loud systolic murmur. Sometimes it occupies the entire systole and is therefore called pansystolic. Noise is heard above the anatomical defect, that is, in the third or fourth intercostal space to the left of the sternum. Having found such a noise, one should compare its intensity at the apex and in the epigastric region (under the xiphoid process). If its intensity at these points is more pronounced, then it may come from the mitral or tricuspid valve with their insufficiency, although congenital malformations of these valves are many times less common than VSD. Cardiomegaly and congestive heart failure (CHF) occur with moderate to large septal defects. Hypertrophy of the heart occurs in all departments and is manifested by a “heart hump” and a pulsation of the chest wall in the parasternal region to the left of the group.

Chronic CHF is characterized by dyspnea at rest, enlarged and pulsating jugular veins, enlarged liver, and edema of the lower extremities.

Acute CHF in children with VSD occurs during an acute respiratory tract infection. The diagnosis is based on the triad of symptoms - tachycardia, tachypnea, liver enlargement. In severe cases, interstitial-alveolar pulmonary edema may develop, which is manifested by severe functional respiratory disorders (rapid breathing, retraction of compliant chest areas) and bilateral symmetrical crepitus. Ultrasound reveals a ventricular septal defect, its size, an increase in the

size of the heart chambers and hypertrophy of their walls. X-ray examination of the chest organs shows the degree of enlargement of the heart and the degree of plethora of the lungs (enhanced pulmonary pattern).

Atrial septal defect (ASD) is one of the most common congenital heart defects, accounting for up to 30% of all heart defects. An ASD causes an abnormal movement of blood from left to right at the level of the atria, which leads to an increase in the size of the right atrium, and then the right ventricle, and a quantitative increase in pulmonary blood flow.

Although children with ASD in most cases do not have complaints, the diagnosis is possible on the basis of objective data:

1. Blood expulsion noise over the area of the pulmonary artery. He is always heard. The murmur is not loud, rarely accompanied by vibration felt on palpation of the heart area.

2. Bifurcated tone II (S1 + S2 + S2) due to different response times (clicks) of the aortic and pulmonary valves.

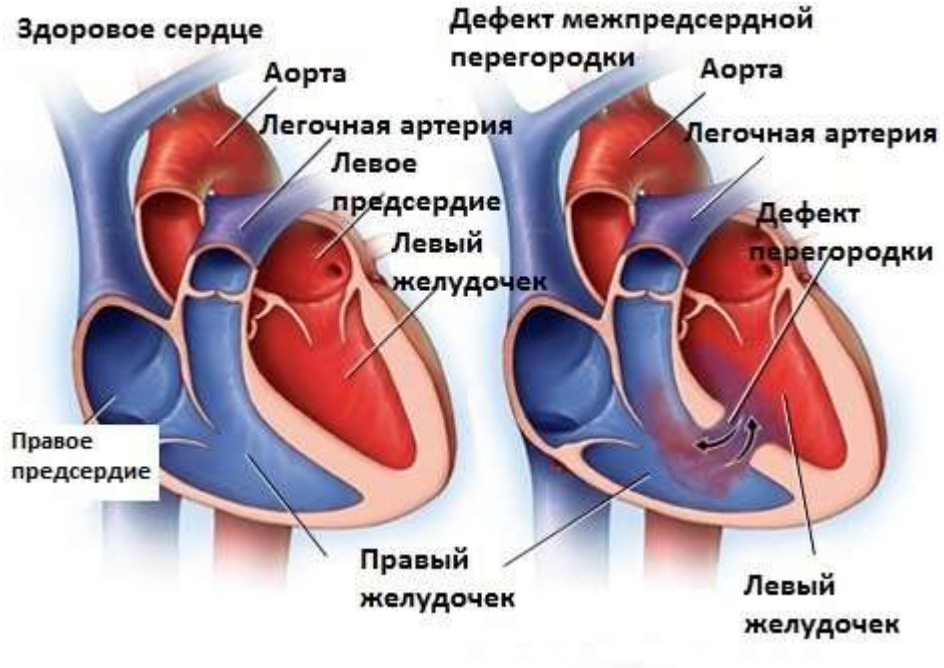


Figure 47. Scheme of hemodynamic disturbances in atrial septal defect.

3. Left-sided parasternal pulsation is determined quite often and indicates the presence of right ventricular hypertrophy.

4. Chest x-ray reveals a typical heart configuration with an enlarged right atrium and right ventricle and signs of increased pulmonary blood flow. Ultrasound reveals the location of the defect in the atrial septum and the expansion of the right atrium and ventricle. The murmur of congenital isolated pulmonary stenosis is very similar in characteristics to that of an ASD, but is associated with vibration of the anterior chest wall; The 2nd tone, although acoustically split, but its second component, due to the click of the closing of the pulmonary artery valve, is very weak, barely audible (S1 + S2 + s2). Hypertrophy of the right ventricle in this type of congenital heart disease is always strongly pronounced (ultrasound shows post-stenotic expansion of the pulmonary artery). The systolic murmur of aortic stenosis is distinguished by the point of auscultation - the area of the aorta. The intensity of the noise is high, it is rough and often accompanied by a feeling of vibration of the chest wall, and is carried to the neck. Severe left ventricular hypertrophy is always present.

Patent ductus arteriosus (PDA) is a common CHD, it can be both independent and in combination with other malformations of the heart and large vessels. The essence of the defect is the abnormal left-right movement of blood from the aorta through the duct into the pulmonary artery, resulting in the development of overflow of blood flow in the lungs. The degree of hemodynamic disturbances mainly depends on the size of the duct.

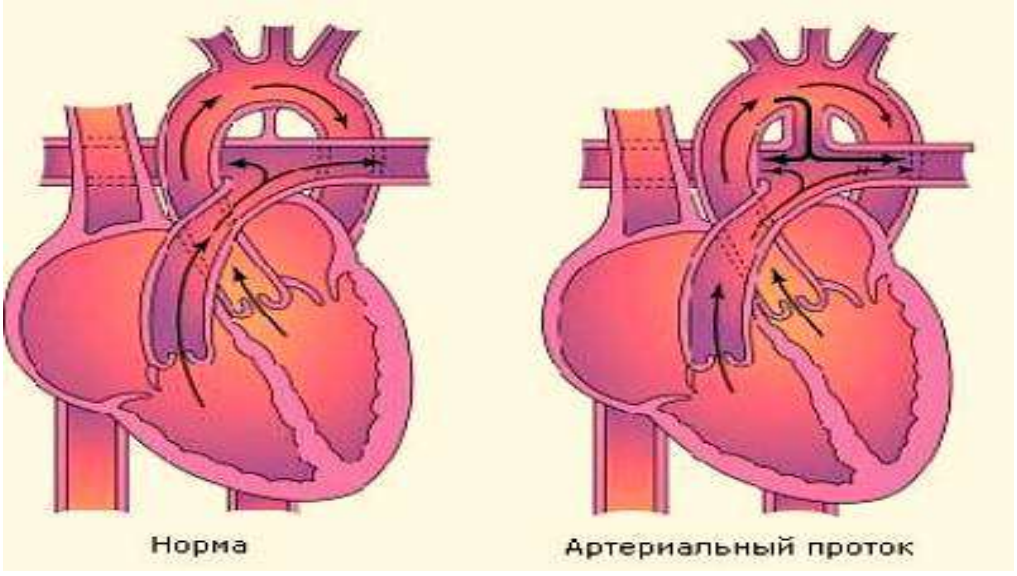


Figure 48. Scheme of hemodynamic disorders in PDA.

A small functioning duct does not provoke complaints and is a clinical finding.

Symptoms of PDA:

1. Jumping, or jumping, pulse is a high pulse that is easily detected.

2. Continuous "machine" noise over the projection of the pulmonary artery and in a wide area from the sternum along the clavicle on the left. You can always feel the vibration of the chest above this area.

3. Enlargement of the heart in size due to total ventricular hypertrophy (right and left).

4. CHF always happens if the size of the arterial duct is large enough (from medium to large).

5. Chest x-ray reveals pulmonary plethora, prominent, enlarged pulmonary artery cone, and varying degrees of cardiomegaly.

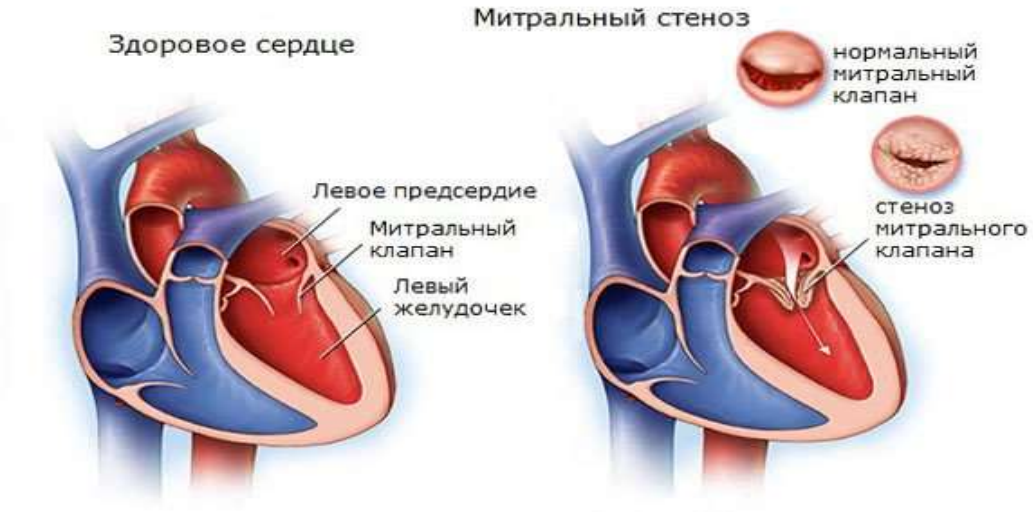


Figure 49. Scheme of hemodynamic disorders in mitral stenosis.

The narrowing of the mitral orifice prevents the filling of the left ventricle, while increasing pressure in the left atrium. As a result, it hypertrophies, blood stagnation occurs in the lungs. Then the right ventricle also hypertrophies. Therefore, left ventricular failure occurs first, and then right ventricular failure.

Symptoms:

1. Left ventricular failure - shortness of breath, orthopnea, cardiac asthma, pulmonary edema, hemoptysis.

2. Right ventricular failure - edema, liver enlargement, ascites.

3. Systemic thromboembolism - due to the formation of blood clots in the left atrium (since there is stagnation of blood).

4. Hoarseness of voice - an enlarged left atrium compresses the nerves of the larynx.

5. Chest pain due to pulmonary hypertension or myocardial ischemia.

6. "Mitral" blush on the cheeks, acrocyanosis, especially of the nasolabial triangle.

7. Atrial fibrillation, pulse deficit, carotid pulse - weak filling (reduced stroke volume).

8. Pronounced cardiac impulse.

9. Diastolic trembling at the top ("cat's purr").

10. With percussion - expansion of relative cardiac dullness up and to the right, an increase in the boundaries of absolute dullness.

11. A clapping I tone, a mitral valve opening tone, an accent of the II tone or its bifurcation above the pulmonary artery (quail rhythm) are determined auscultatory. Diastolic murmur at the apex is rough, heard after the opening of the mitral valve. Sometimes a soft functional Graham-Still murmur is detected over the pulmonary artery (due to increased blood pressure in the pulmonary circulation).

12. Wheezing in the lungs (stagnation in the pulmonary circulation).

13.Symptom Saveliev-Popov - on the left hand, the filling of the pulse is less (the subclavian artery is squeezed by the left atrium).

14. ECG shows left atrial hypertrophy (P-mitrale), right ventricular hypertrophy, deviation of the electrical axis of the heart to the right.

15. X-ray: the waist of the heart is smoothed, the pulmonary pattern is strengthened, the retrosternal space is reduced.

16. Ultrasound: enlargement of the left atrium, thickening of the mitral valve cusps.

Mitral insufficiency. Part of the stroke volume from the left ventricle goes not to the aorta, but to the left atrium, as a result, the pressure in it rises, and the ejection of blood into the aorta decreases. With the next contraction, a larger volume of blood enters the left ventricle, so both the left atrium and the left ventricle hypertrophy. There is left ventricular failure, which is accompanied by an increase in blood pressure and stagnation in the pulmonary circulation.

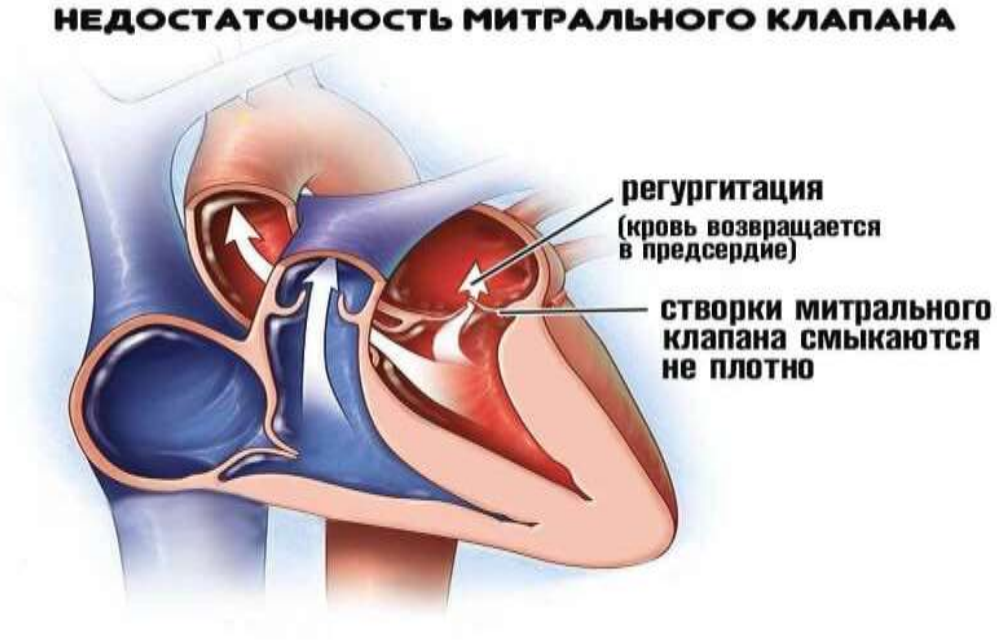


Figure 50. Scheme of hemodynamic disorders in mitral valve insufficiency.

Symptoms:

1. Left ventricular failure - shortness of breath, orthopnea, cardiac asthma.

2. Thromboembolism.

3. In severe cases, right ventricular failure may also develop.

4. Palpitations, arrhythmias.

5. Mitral flush, acrocyanosis.

6. The apex beat is strengthened and shifted up and to the left.

7. The pulsation of the carotid arteries is reduced (the stroke volume is reduced).

8. Systolic tremor at the apex.

9. During auscultation, the third tone is often heard (a large volume of blood enters the left ventricle), the accent of the second tone over the pulmonary artery, the first tone is weakened (up to complete disappearance). Systolic murmur at the apex (conducted to the axillary region).

10.ECG: signs of hypertrophy of the left atrium (P-mitrale) and left ventricle. Displacement of the electrical axis of the heart to the left.

11. X-ray: congestion in the lungs, mitral configuration of the heart.

12. Ultrasound: valve defects are noticeable, on Doppler - blood regurgitation into the left atrium.

aortic stenosis. Aortic stenosis causes left ventricular hypertrophy, which leads to an increase in myocardial oxygen demand, i.e., ischemia.

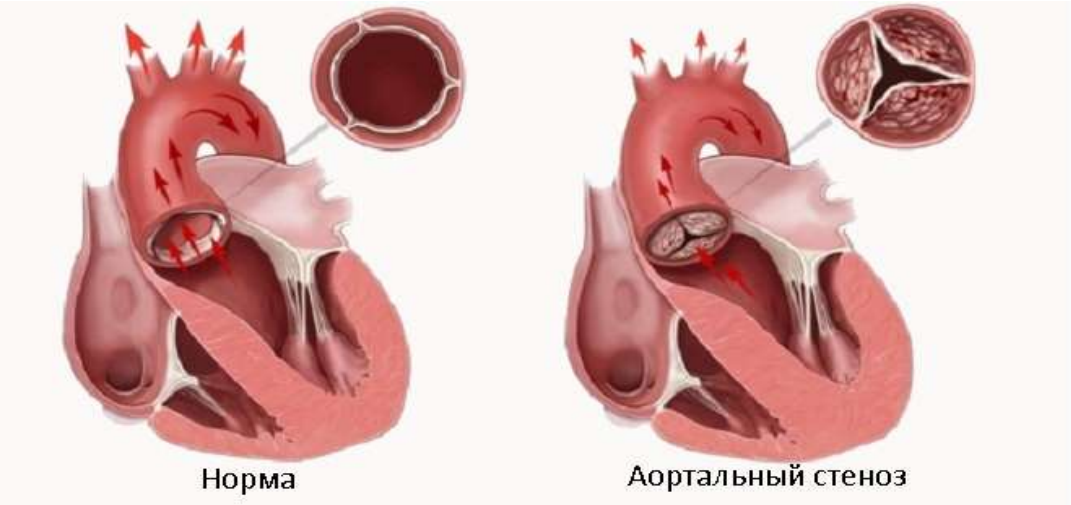


Figure 51. Scheme of hemodynamic disorders in aortic stenosis.

Symptoms:

1. Angina pectoris - coronary blood flow is reduced (due to left ventricular hypertrophy).

2. Fainting during physical exertion as a result of reduced blood flow in the brain.

3. Heart failure, especially left ventricular (shortness of breath).

4. Pale skin.

5. Delayed pulsation of the carotid arteries, and the pulse is small, slow, rare.

6. Systolic pressure is reduced, and diastolic pressure is increased.

7. Reinforced domed apex beat is often shifted to the left and down.

8. Auscultatory: muting of the second tone due to limited mobility of the aortic valve cusps. A rough systolic murmur over the aorta and at the Botkin point is carried along the course of the carotid arteries.

9. ECG: left ventricular hypertrophy, displacement of the electrical axis of the heart to the left, the T wave is sometimes negative, often a block of the left leg of the His bundle.

10. Radiography reveals calcification of the aortic valves.

Aortic insufficiency. In diastole, part of the stroke volume returns to the left ventricle, which reduces cardiac output.

The left ventricle gradually hypertrophies, which leads to an increase in stroke volume. Gradually, left ventricular failure occurs

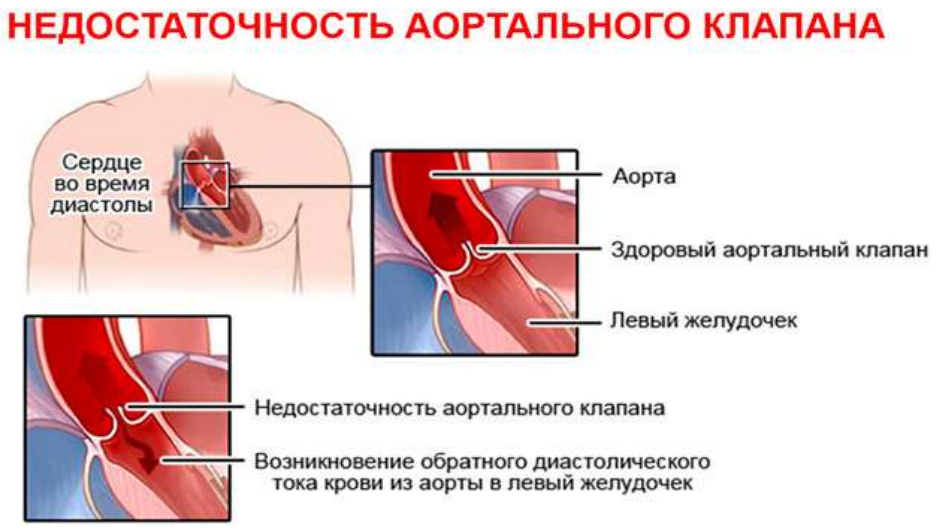


Figure 52. Scheme of hemodynamic disorders in aortic valve insufficiency.

Symptoms:

1. Palpitations, especially when lying down.

2. Left ventricular failure - shortness of breath, orthopnea, cardiac asthma.

3. Fainting due to reduced cerebral blood flow.

4. Attacks of angina pectoris, but they are less common than with aortic stenosis.

5. The apex beat is diffuse, dome-shaped and shifted down and to the left.

Shift of the left border of cardiac dullness to the left.

6. Diastolic murmur is soft, better heard in a sitting position with an inclination forward. Flint noise. Traube double tone on auscultation of the femoral arteries. Double Vinogradov-Durozier murmur during stethoscope pressure on the femoral artery.

7. Stroke volume and pulse pressure are increased.

8. Fast, racing pulse. Capillary Quincke pulse.

9. Symptom Musset - rhythmic shaking of the head.

10. Symptom of "carotid dancing".

11.ECG: left ventricular hypertrophy, displacement of the electrical axis of the heart to the left.

12. Radiography: enlargement of the heart, expansion of the proximal aorta.

13. Ultrasound: Doppler sonography reveals reverse blood flow into the left ventricle.

myocardial syndrome. Changes in the heart with myocarditis are determined by the result of the inflammatory process. There are changes characteristic of the syndrome of acute inflammation: C-reactive protein, dysproteinemia, increased mucoproteins, mild increase in the level of sialic acids. The most significant are the increase in the level of lactate dehydrogenase (LDH), creatinine phosphokenase (CPK). The results of a general blood test depend on the etiology of myocarditis. Viral myocarditis is characterized by leukocytopenia, lymphocytosis, bacterial myocarditis - leukocytosis, neutrophilic shift, accelerated ESR. An increase in temperature is not characteristic of non-rheumatic myocarditis, but it can be high in bacterial and allergic myocarditis. Complaints are inconsistent, depending on the degree of damage and on extracardiac factors. Patients note fatigue, palpitations, discomfort or pain in the region of the heart, a feeling of “incompleteness” of inspiration, poor health, headache, sleep and appetite disorders. Young children become lethargic, gain weight poorly, they develop bouts of cyanosis, fainting. In the early stage of development of the process, the pulse quickens, its frequency does not correspond to the general

condition and temperature. Sleep does not affect tachycardia with myocarditis, it is very persistent, despite the ongoing treatment. The pulse is labile, which manifests itself even with minimal exertion. Bradycardia in myocarditis indicates involvement in the inflammatory process of the conduction system of the heart. When examining the region of the heart, a weakening of the apex beat is noted. Percussion data reveal a uniform expansion of the heart, its right and left sections. The degree of muffled heart sounds depends on the intensity of inflammation and is greatest in diffuse myocarditis. Systolic (myocardial) murmur varies with inhalation and changes in body position. Its intensity depends on the degree of metabolic disorders and inflammation in the myocardium.

Syndrome of endocarditis. The clinical picture develops gradually, with the manifestation of symptoms of general intoxication. There is a special pallor ("coffee with milk"), sweating, chills, pain in the muscles, bones, persistent fever of an intermittent or undulating nature. On the part of the heart, there are signs of valvular lesions (usually aortic). Characterized by greater dynamism of physical data with the appearance or disappearance of noise, depending on the processes of thrombosis on the surface of the valves. Endocardial lesions are not reflected on the ECG, since the endocardium is devoid of muscle elements. ECG changes occur at the final stage of the process and are manifested by a violation of atrioventricular or intraventricular conduction.

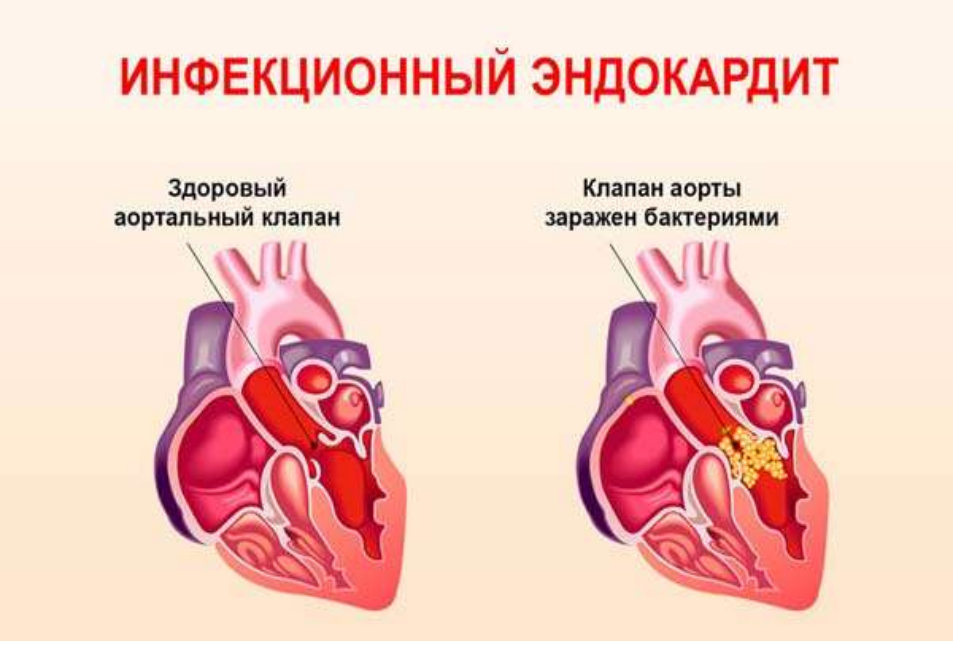
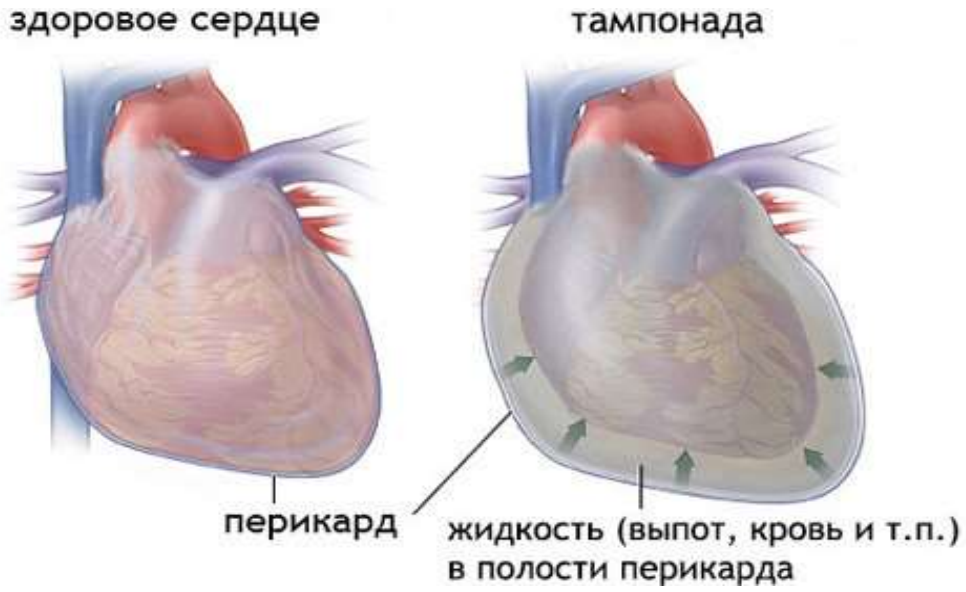


Figure 53. Scheme of hemodynamic disorders in infective endocarditis.

Syndrome of pericarditis. Pericarditis is an inflammation of the visceral and parietal sheets of the serous membrane of the heart. As a rule, pericarditis is secondary and develops against the background of some other disease: in early childhood, these are respiratory diseases - pneumonia, pleurisy, in children over 5 years old - rheumatism, systemic connective tissue diseases, tuberculosis. Acute pericarditis can occur under the guise of dry (fibrinous) or effusion. With dry pericarditis, there may be a small amount of fluid in the cavity of the heart shirt, which is not determined clinically and does not cause major hemodynamic disorders. The most common and early complaint in incipient pericarditis are stabbing pains in the region of the heart. The pain is aggravated by coughing, movement and occurs due to the friction of the inflamed layers of the pericardium against each other. The intensity of pain varies from mild to severe attacks radiating to the left arm or left shoulder blade. In children under the age of 4–5 years, due to the lack of the ability to localize pain, there are complaints of abdominal pain. In addition to pain in the region of the heart, there may be chills, palpitations, and headache. The general condition worsens, malaise, lethargy appear, the temperature rises. Some children have a dry hacking cough, reminiscent of a cough with pleurisy, a feeling of fear and anxiety appears. These pain sensations are explained by irritation of the intermuscular nerves and the sensitivity of the pericardial sac itself. The main and most reliable sign of dry pericarditis is the pericardial rub. The nature of this sound is compared with the noise of a locomotive, the friction of new skin, the crunch of snow. The pericardial friction noise does not coincide with the heart sounds, has poor conductivity, and increases on inspiration and pressure with a stethoscope. The noise is sometimes so strong that it is determined by palpation. In other cases, it can be gentle, short-lived, auscultated in a limited area. The best place to listen to it is the base of the heart and the area along the left edge of the sternum. Sometimes it can be heard at

the top of the heart. Heart sounds on auscultation are almost unchanged. There may be an increase in heart rate. With effusion pericarditis, as fluid accumulates in the heart shirt, a pattern of cardiac tamponade develops. The pericardial friction noise disappears. The pallor of the skin increases due to anemia (diastole is difficult) and vasospasm. Compression of the superior vena cava leads to the appearance of edema and cyanosis of the face and upper half of the body, to swelling of the cervical veins. In young children, as a result of the compliance of the chest, with the accumulation of a large amount of fluid in the cavity of the heart shirt, a "heart hump" appears. Intercostal spaces are smoothed out. The apex beat becomes weakened, shifts upward into the third or fourth intercostal space and medially from the left border of relative cardiac dullness. During auscultation, heart sounds are weakened, endocardial murmurs weaken until they disappear completely. The heart is forced to contract rapidly to provide blood flow to the periphery, but the left ventricle does not receive enough blood for a normal stroke volume. The nutrition of the heart muscle itself is deteriorating. There is a frequent, small, arrhythmic and paradoxical pulse. There is stagnation of blood in the liver and its increase. Due to obstruction of outflow from the liver, ascites develops. With compression of the inferior vena cava, edema of the lower extremities appears. The subcutaneous venous network develops. Difficulty diastole leads to a drop in blood pressure. With a large effusion, the patient may experience hoarseness or complete aphonia due to compression of the left recurrent nerve.



With the accumulation of fluid in the pericardium, the shape of the cardiac shadow changes. It (the shape) resembles a sphere, a triangle, a trapezoid. ECG with effusion pericarditis is characterized by a decrease in the voltage of the teeth. The S-T interval is displaced above the isoline and arcuately curved, directly passes into a positive T wave.

There may be T-wave inversion in two or all of the standard leads. In children, there is a discordant location of the T wave and the S-T interval in I and III standard leads, as well as in V1, V5 leads.

Syndrome of heart failure. Heart failure syndrome (HF) is a clinical syndrome that can be caused by any disease of the heart that impairs its ability to fill with blood or expel it (the load on the heart exceeds its capacity). Allocate energy-dynamic (according to Hegglin) and hemodynamic, or congestive, heart failure.

Congestive heart failure is divided into acute and chronic. Both acute and chronic heart failure can be left ventricular or right ventricular. More often, insufficiency of both ventricles of the heart develops simultaneously, i.e., total heart failure.

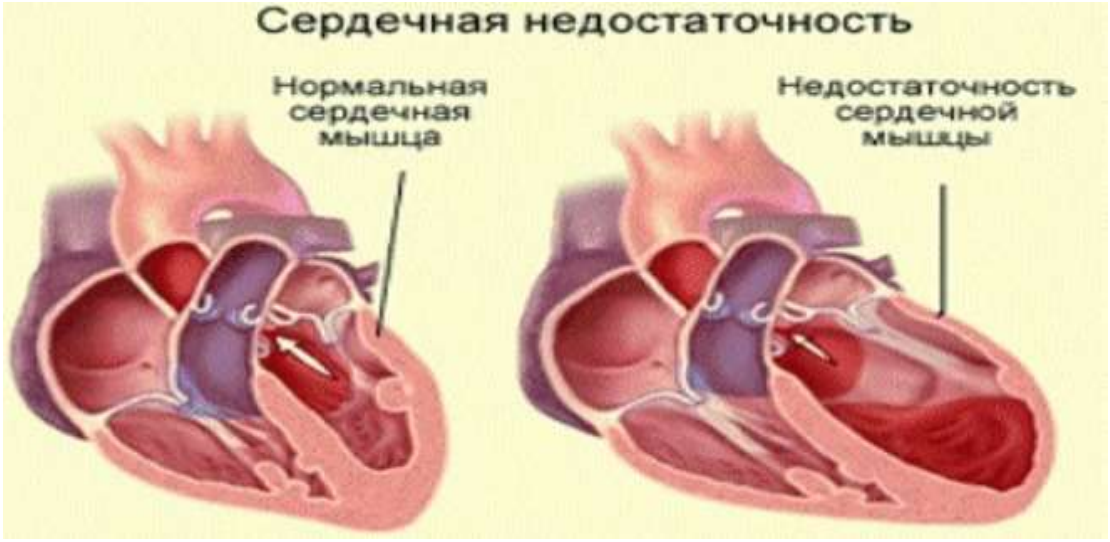


Figure 55. Scheme of heart failure.

Left ventricular heart failure is accompanied by symptoms of congestion in the pulmonary circulation: shortness of breath (tachypnea without increasing the

depth of breathing), cough, sometimes with hemoptysis, cyanosis, pallor of the skin, tachycardia, thready pulse. With percussion, an increase in the size of the heart is determined (mainly to the left), with auscultation - a sharp weakening of tones, a protodiastolic gallop. The clinic of pulmonary edema is characteristic. Hemodynamic changes - an increase in CVP, BCC. On the ECG - levogram, negative peaked T wave in the left chest leads.

Right ventricular heart failure most often develops in nonspecific lung diseases. Accompanied by shortness of breath (increased depth of breathing), rapid progression of cyanosis, swelling of the jugular veins, enlargement of the liver and its thickening, enlargement of the spleen, anorexia, pain in the abdomen and chest, stool disorders, impaired blood supply to the brain (sleep disturbance, headache), development of oligo - and anuria. On percussion, there is an expansion of the size of the heart to the right, on auscultation, a weakening of the heart tones, an accent of the second tone over the pulmonary artery. On the ECG - signs of overload of the right heart. Total heart failure is characterized by a combination of clinical symptoms of right and left ventricular heart failure.

The main symptoms of heart failure by stages are given in Table. 6.

Table 6 Signs and stages of HF in children (according to N. A. Belokon, 1987)

Stage	**Right ventricular**	**Left ventricular**
I	**HF is absent at rest, appears after exercise in the form of shortness of breath and tachycardia**	
IIA	**Heart rate increased by 15-30%. The respiratory rate is increased by 30-50%**	**The liver protrudes 2-3 cm from under the edge of the costal arch**
IIB	**Heart rate increased by 30-50%. RR increased by 50-70%. Possible acrocyanosis, obsessive cough, wet small**	**Liver protrudes 3–5 cm from under the edge of the costal arch, pastosity, swelling of the jugular veins are**

	bubbling rales	**possible**
III	**Heart rate increased by 50-60%. BH is increased by 70–100%. Clinical picture of pulmonary preedema**	**Hepatomegaly, edematous syndrome, hydropericardium, ascites**

Syndrome of arterial hypertension. The syndrome of arterial hypertension (AH) is a symptom complex that reflects a persistent increase in pressure in the arteries. The term essential (primary) hypertension refers to high blood pressure in the absence of an obvious cause of its occurrence. The term secondary hypertension refers to hypertension, which is a syndrome of kidney disease, endocrine pathology, hemodynamic disorders.

Manifestations of the syndrome:

1. Headaches.

2. Signs of "hypertensive encephalopathy" (most pronounced in hypertensive crises): - sharp headaches; - noise in ears; - dizziness (vertigo); - "fog", "shroud", flies before the eyes; - disorientation in space and time; - nausea and vomiting that does not bring relief to the patient; - deterioration of vision; - decrease in memory, attention.

3. Cardialgia or angina pectoris, shortness of breath.

4. Hyperemia or pallor of the skin.

5. Pastosity of the subcutaneous tissue (aldosterone mechanism).

6. Syndrome of hypertrophy and dilatation of the left ventricle.

7. Percussion: expansion of the vascular bundle.

8. Auscultatory: weakening of the 1st tone, emphasis of the 2nd tone on the aorta.

9. High hard hard pulse.

10. Rise in blood pressure. "Inside" this syndrome, it is necessary to be able to tentatively differentiate between essential arterial and symptomatic (eg, renal)

hypertension.

SEMIOTICS OF FUNCTIONAL DISORDERS OF THE GASTROINTESTINAL TRACT IN YOUNG CHILDREN.

The main functions of the gastrointestinal tract are: the secretion of digestive enzymes, digestion of food, the absorption of nutrients, motor activity, the formation of local immunity (the activity of the microflora and the immune system of the gastrointestinal tract). Intestinal dysbacteriosis. The main groups of bacteria in relation to the human body can be considered: pathogenic flora (which always causes disease in an infectious dose); opportunistic flora (E. coli, Klebsiella, Clostridium, Staphylococcus, Candida, Proteus, Enterobacteria); normal microflora (constituting 5–8% of body weight), represented by anaerobes (bifido-, lactobacilli, bacterioids) and aerobes (normal E. coli); transient microflora. The main functions of normal intestinal microflora are: protective, participation in the formation of local immunity and digestion of plant fiber, synthesis of B vitamins, ensuring normal intestinal motility, antiallergic, participation in mineral metabolism and maintaining a normal acid-base composition. Normal indicators of intestinal microflora in children: - normal microflora: anaerobes (bifidobacteria, 109 microbial bodies per 1 g of feces with artificial feeding, 1010 - with breastfeeding), lactobacilli (107 per 1 g of feces and above), bacterioids; aerobes - normal E. coli (107 in 1 g of feces and above); - opportunistic flora: opportunistic E. coli - no more than 10% of the total amount of normal E. coli; Clostridia, Klebsiella, Candida - no higher than 104, Staphylococcus, Proteus - no higher than 103 microbial bodies per 1 g of feces.

Dysbacteriosis is a syndrome that is formed as a result of qualitative and quantitative changes in the normal microbial flora, which entail clinical reactions of the macroorganism and are the result of any pathological processes. They say about intestinal dysbacteriosis if there is a decrease in the level of normal flora and an increase in the level of opportunistic bacteria above acceptable limits, which are observed for a sufficiently long time (more than 3 months) and are accompanied by minimal intestinal dysfunctions (unstable stools - increased frequency, liquefaction

or constipation, flatulence and intestinal colic). It should be remembered that intestinal dysbacteriosis is never accompanied by fever and intoxication, which is how it differs from intestinal infections. When establishing dysbateriosis, diseases with malabsorption syndrome, also occurring with diarrheal syndrome, should be excluded by at least a scatological study.

Physiological (simple) dyspepsia. Physiological, or simple, dyspepsia is characterized by the presence of rapid, liquid, foamy stools mixed with mucus (sometimes green) in infants aged 3-4 days to 2 weeks, less often up to 1 month. May be accompanied by bloating, episodes of anxiety in the child, increased gas discharge. This condition reflects the functional immaturity of the gastrointestinal tract during this period: - the formation of normal intestinal microflora (a temporary predominance of aerobic flora, a relatively large amount of conditionally pathogenic); - transient lactase deficiency; - transient syndrome of impaired digestion of fats; - imperfection of peristalsis at an early age. Over time, as the activity of lactase, proteases and lipases in the intestines of the child increases, as well as after the normalization of the intestinal biocenosis, the phenomena of physiological dyspepsia disappear. Physiological dyspepsia refers to the borderline conditions of the neonatal period.

Infantile intestinal colic. Clinical manifestations of infantile intestinal colic are: 1) attacks of a child's sharp anxiety with a piercing cry, the duration of which can reach several hours; 2) the presence of bloating and some tension in the anterior abdominal wall; 3) reduction or disappearance of symptoms after passing gases or defecation. Infantile colic occurs due to intestinal spasms due to stretching of intestinal loops under the influence of gases, the discharge of which is difficult as a result of impaired intestinal motor function in infants. Motility disorders are due to the physiological immaturity of the CNS. Increased gas production is associated with impaired digestion of lactose and (or) proteins in the intestine due to: - functional immaturity of digestive enzymes (mainly lipases, proteases and lactase); - intolerance to proteins or lactose; - disorders of the intestinal microbiocenosis; - overfeeding of the child, etc. Diagnostic tactics in the presence

of infantile colic should be, first of all, to exclude intestinal intussusception, and then - to establish the cause of colic by questioning the mother in detail, clarifying the allergic history, examining the child and conducting a scatological study.

Functional constipation. The reasons for the high frequency of functional constipation in infants are: artificial feeding, anatomical and physiological characteristics of the intestine, violations of its motor function, in some cases - dysbacteriosis. Diagnostic criteria for functional constipation.

Children and preschoolers for at least 12 weeks have:

1) fragmented, ovine, hard stools in most bowel movements;

2) hard stools two or less times a week;

3) there is no evidence of structural, endocrine or metabolic diseases. Diagnostic criteria for constipation in infants:

1) no stool for 32-48 hours;

2) independent emptying of the intestines one or several times a day, accompanied by severe straining, anxiety, crying; 3) at the same time, the feces are hard, in the form of sheep, in small portions, sometimes with streaks of blood. In children aged 2–4 months, when breastfeeding, in some cases, there is a decrease in stool (from 3–5 times a day to 1 time in 2–3 days), and the stool remains mushy. This is due to the maturation of enzyme systems in the intestines of the child and the maximum digestibility of mother's milk. This condition should not be considered constipation.

Gastroesophageal reflux. Regurgitation (regurgitation) is the reflux of a small amount of gastric contents into the pharynx and oral cavity in combination with the passage of air. Factors predisposing to gastroesophageal reflux (GER) are: a small volume of the stomach and its spherical shape, the esophagus flows into the stomach at a right angle, slowing down its emptying, insufficiency of the lower esophageal sphincter, enzymatic immaturity, immaturity of the neurohumoral regulation of the sphincter apparatus and gastrointestinal motility , inadequate feeding (aerophagia, overfeeding, violation of the regimen, inadequate selection of mixtures. Considering the high risk of gastroesophageal reflux disease (GERD) in

children with regurgitation, it is necessary to be able to distinguish between physiological and pathological GER in infants. Physiological regurgitation (GER) has a rare, not abundant character, occurs no later than one hour after feeding and does not lead to the formation of reflux esophagitis.

Figure 56. Schematic representation of gastroesophageal disease.

Physiological GER with typical manifestations is usually observed in mobile infants under the age of 1 year, and in children there is adequate growth and development, there are no regurgitation during sleep, from the moment the child moves to a predominantly upright position (6–8 months), spontaneous recovery is observed. Pathological GER occurs more than 5 times a day, occurs an hour or later after eating, can occur at night, is profuse and leads to damage to the esophageal mucosa.

Figure 57. Physiological GER

Vomiting is a CNS reflex involving both smooth and skeletal muscle that results in the forceful ejection of stomach contents through the mouth due to coordinated movements of the small intestine, stomach, esophagus, and diaphragm.

SYNDROME OF "UPPER", OR GASTRIC, DYSPEPSIA.

The dyspepsia syndrome is defined as a sensation of pain or discomfort (heaviness, fullness, early satiety and nausea) localized in the epigastric region closer to the midline (Table 7).

Pain syndrome. Features and stages of questioning. Determining the pain syndrome at the time of the diagnostic search in case of suspected gastrointestinal pathology is extremely important. As a result of a detailed questioning of the patient, the doctor is able to determine which part of the gastrointestinal tract is affected, and in accordance with this, competently prescribe a complex of examinations. In table. 8 shows the main characteristics of the pain syndrome in the pathology of the digestive system. dyspeptic syndrome. After clarifying the nature of the patient's pain syndrome, the presence of other complaints of a dyspeptic nature is clarified, which allow obtaining more accurate information about the patient's alleged disease (Table 9).

Table 7

Determination of the main symptoms of gastric dyspepsia

Symptoms	Definition
Pain localized in the epigastric region along the midline	Pain is subjectively perceived as unpleasant sensations, some patients may feel as if "tissue damage". When questioning patients, it is necessary to distinguish between pain and discomfort.
Discomfort	A subjectively unpleasant sensation that is not perceived by the patient as pain, but is described as one of the symptoms listed below.
Early satiety	A feeling of fullness in the stomach that occurs immediately after the start of a meal, regardless of the amount of food taken, as a result of which the patient cannot eat the usual portion of food
Fullness	Unpleasant feeling of constantly filling the stomach with food, which may or may not be associated with eating.
Bloating in the upper abdomen	Feeling of tension (fullness) in the epigastric region, which should be distinguished from visible bloating
Nausea Feeling	"sick" and about to vomit

It should be remembered that, especially at a younger age, GERD may manifest itself with other symptoms or signs that are referred to as the so-called extraesophageal manifestations of the disease (recurrent laryngitis, otitis media, obstructive bronchitis, asthma). Thus, 2 types of GERD are defined: classic, when the symptoms indicate the presence of damage to the esophagus, and latent,

represented by respiratory symptoms without vomiting, especially nocturnal gastroesophageal reflux.

Manifestation of GERD:

1. Classic symptoms: - regurgitation; - nausea and vomiting; - bloating, belching; - heartburn; - epigastric and retrosternal pain; - irritability; – anemia; - hematemesis; - dysphagia, odynophagia; - weight loss, developmental delay.

2. Unusual manifestations: - recurrent aspiration pneumonia; - bronchitis, asthma; - otitis, sinusitis; - laryngitis, laryngomalacia, subglottic stenosis; - stridor; - pharyngitis; - enamel damage, halitosis; - rumination;

3. Functional dyspepsia: - abdominal pain syndrome. Features: pain in the morning, before eating, half an hour after eating, decreasing after eating, aggravated after eating "aggressive" food, aching, seasonality can be observed; - postprandial distress syndrome: nausea, feeling of early satiety, vomiting, belching; - elements of asthenoneurotic syndrome are often observed; - diagnostics - FGDS, ultrasound, examination for Helicobacter pylori and Giardia.

4. Chronic gastritis: - abdominal pain syndrome. Features: pain in the morning, before eating, half an hour after eating, decreasing after eating, aggravated after eating "aggressive" food, aching, seasonality is characteristic; - less often - postprandial distress syndrome: nausea, feeling of early satiety, vomiting, belching; - rarely - asthenoneurotic syndrome; - burdened family history; - diagnostics - FGDS, ultrasound, examination for Helicobacter pylori and Giardia.

5. Peptic ulcer of the stomach and duodenum: - abdominal pain syndrome. Features: pain in the morning, before eating, half an hour after eating, decreasing after eating, aggravated after eating "aggressive" food, aching. The pain is seasonal. hungry pains; Moinigan rhythm; night pains; - less often - postprandial distress syndrome - nausea, feeling of early satiety, vomiting, belching; heartburn; tendency to constipation; - rarely - asthenoneurotic syndrome, rather asthenovegetative; - burdened family history: peptic ulcer of the stomach and duodenum; stomach cancer; - diagnostics - FGDS, ultrasound, examination for Helicobacter pylori and Giardia; - recurrent apnea.

6. Cholelithiasis: - episodes of cramping pain in the abdomen, often in the right hypochondrium, in the presence of gastric dyspepsia; - positive bladder symptoms: Kera, Murphy, Ortner-Grekov, Mussi-Georgievsky; – examination: ultrasound of the abdominal organs (liver, gallbladder, pancreas). Biochemistry: liver tests, amylase, lipase. Coprogram, FGDS; - differentiate with dyskinesia of the gallbladder and spasm of the sphincter of Oddi.

7. Pancreatitis: - abdominal pain syndrome. Specific for pancreatitis is the "girdle" nature of abdominal pain, but this symptom is observed in only 30-40% of cases; the rest of the patients have intense pain in the epigastrium or the left side of the abdomen, appearing after eating or eating fatty foods; - for pancreatitis during the period of exacerbation, repeated vomiting is characteristic, pronounced signs of intoxication syndrome, there may be loose stools; - on palpation of the abdomen, there is pain in the Chauffard zone, De-Jardin and Mayo-Robson points, less often - pain in the Janover zone and the Kutch point; - research - biochemistry, ultrasound, coprogram, computed or magnetic resonance imaging. If necessary, retrograde cholangiopancreatography.

Syndrome of intestinal dyspepsia. The components of the syndrome of intestinal dyspepsia are changes in the frequency and nature of the stool, the presence of pathological impurities in it, bloating, flatulence, difficulty in defecation, pain before, during and after 100 defecation, a feeling of incomplete emptying of the intestine, tenesmus, imperative urges. The most important characteristics for diagnosing both acute (intestinal infections) and chronic (malabsorption syndrome, irritable bowel syndrome, chronic colitis, ulcerative colitis and Crohn's disease) intestinal dyspepsia are such characteristics as changes in the frequency and nature of the stool. stool frequency. In children under the age of one year, the frequency of stool is variable and depends on the nature of feeding. In children older than a year, stools from 2 times a day to 4-5 times a week are considered normal. Options for changes in stool frequency are interpreted as diarrhea (stools from 4–5 times a day or more), frequent stools (stools 3–4 times a day), normal frequency, constipation (stools every other day or less).

The nature of the chair. When determining it, attention is paid to the consistency (the stool can be watery, liquid, frothy, mushy, scanty, formed, compacted, bean-shaped), color (yellow, brown, orange, with an admixture of greenery, like swamp mud, acholic) and the presence of pathological impurities (mucus, pus, blood). The syndrome of malabsorption (impaired absorption) is due to the consequences of limiting the intake of nutrients in the body. It is manifested by diarrheal syndrome, malnutrition, growth retardation, symptoms of polyhypovitaminosis, signs of calcium and iron deficiency. It is observed in cystic fibrosis, lactase deficiency, celiac disease, exudative enteropathy, short bowel syndrome. The syndrome of maldigestion (impaired digestion) develops as a result of violations of the breakdown of nutrients in the gastrointestinal tract and is manifested by intestinal dysfunctions and dysbiosis phenomena. It is observed with violations of the exocrine function of the pancreas and bile secretion, acute intoxication, it can be mild in healthy infants in the first months of life, especially with inadequate artificial feeding. If a patient has the syndrome of intestinal dyspepsia in pediatric practice, differential diagnosis between acute intestinal infections and other chronic diseases that occur with this syndrome, as well as a diagnostic search in the presence of blood in the stool. This is due to the need to quickly prescribe adequate therapy in the first case, and the fact that a number of difficult-to-diagnose and severe diseases occur with symptoms of hemocolitis in the second.

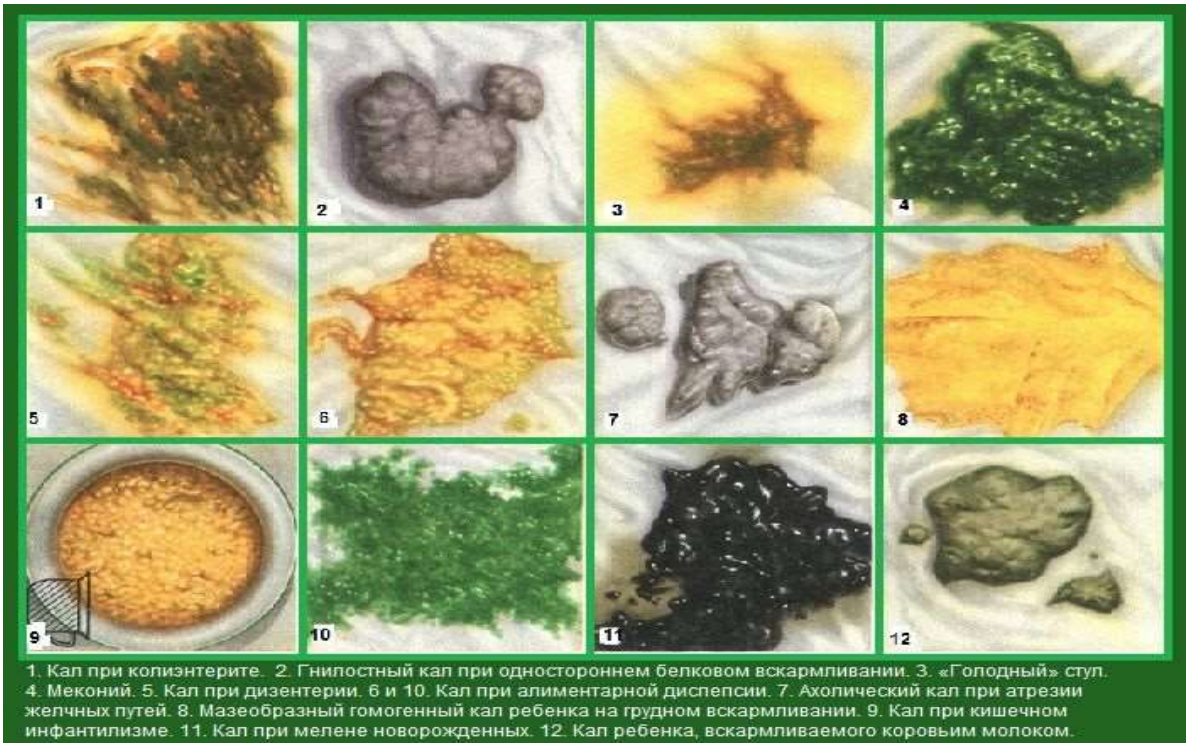


Figure 58. The nature of the chair in various pathological conditions

Acute intestinal infections. In AII, in almost all cases, there is an intoxication syndrome, manifested by fever, vomiting, lack of appetite, pallor, and lethargy of the child. These symptoms are defined as intestinal toxicosis. Anatomical and physiological features of the intestines and kidneys in young children in the presence of repeated vomiting and diarrhea lead to the fact that they easily develop dehydration, accompanied by a decrease in diuresis, increased thirst, lethargy, dry skin and mucous membranes. This life-threatening condition is called exsicosis. With intestinal infections of viral etiology, enteritis syndrome (watery diarrhea) is observed - frequent (from 5-10 times a day) loose or even watery stools with a high risk of developing exsicosis. With intestinal infections of bacterial etiology, colitis syndrome is more often observed - rapid, non-abundant, mushy or loose stools with an admixture of greenery, mucus, and sometimes streaks of blood. In this case, the risk of developing intestinal toxicosis is higher.

The main causes of hemocolitis syndrome:

1. AII: salmonellosis, shigellosis, escherichiosis, campylobacteriosis, amoebiasis, giardiasis, yersiniosis.

2. Hemolytic-uremic syndrome, DIC, hemorrhagic vasculitis.

3. Hemorrhoids.

4. Intestinal polyposis.

5. Tumors of the intestine.

6. Diverticulosis with diverticulitis.

7. Intussusception of the intestine.

8. Chronic inflammatory bowel disease (Crohn's disease and ulcerative colitis).

SYMPTOMS OF LIVER DAMAGE.

Characteristics of the main symptoms in case of liver damage:

1. Pain in the abdomen with hepatic dyspepsia is often aching in nature and is localized in the epigastrium and right hypochondrium, accompanied by a feeling of heaviness in the right hypochondrium.

2. In diseases of the liver, a decrease in appetite is characteristic.

3. In acute hepatitis, there is a change in the color of the skin (jaundice) - from mild icterus of the sclera to severe jaundice up to a change in the color of the palms and feet.

4. In acute hepatitis and obstructive jaundice, discoloration of feces (gray-white color) can be observed.

5. In acute hepatitis, darkening of the urine (beer-colored urine) can also be observed.

6. On palpation of the abdomen, the liver protrudes from under the edge of the costal arch, its size increases according to Kurlov.

7. When performing ultrasound of the liver, diffuse changes in tissues and an increase in their echogenicity are observed.

8. Changes in the biochemical analysis of blood are characterized by an increase in the level of bilirubin (total and bound), AlAT, AsAT, GGTP, thymol test.

9. Positive blood test for markers of hepatitis.

CHARACTERISTICS OF THE BASIC DIAGNOSTIC STUDIES IN THE PATHOLOGY OF THE DIGESTIVE ORGANS.

In the presence of intestinal dyspepsia, it is advisable to conduct a study of feces for dysbacteriosis. The data of a normal stool bateriogram are presented in Table. 10.

Table 10

Normal bacteriogram of feces (study for dysbacteriosis)

Microflora strains	Indicator (number of bacteria in 1 g of faeces)
Bifidobacteria	Not less than 109 –1011
Normal E. coli	Not below 107
Hemolyzing	Not more than 10%
Lactose-negative	No more than 10%
Conditionally pathogenic enterobacteria	No more than 106
Enterococci	No more than 106
Staphylococci	No more than 102 -103
Clostridia	No more than 104
Candida	No more than 104
Klebsiella	No more than 104
Proteus	No more than 104
Lactobacilli	Not below 106
Pathogenic flora	0

MAIN COPROLOGICAL SYNDROMES

Nutrient digestion disorder syndrome is observed in the syndrome of maldigestion, malabsorption, acceleration of peristalsis, disorders of bile secretion and pancreatic secretion and is characterized by the following features: 1.

Steatorrhea (neutral fat + and more, soaps and fatty acids ++ and more). 2. Creatorrhea (muscle fibers ++ and more). 3. Amilorrhoea (starch ++ and more).

Dysbacteriosis syndrome: 1. Digestible vegetable fiber ++ and more. 2. Starch extra- and intracellular + and more. 3. Iodophilic flora+ and more. 4. Candida detection is possible. Indigestible plant fiber can be observed in any amount in healthy patients.

Syndrome of inflammation in the colon: 1. Leukocytes (more than 5 in the field of view). 2. Slime+ and more. 3. Red blood cells. Changes in the pH of feces (5.0 and below) may indicate lactase deficiency, as well as be observed in acute intestinal infections, maldigestion and malabsorption syndrome.

SEMIOTICS OF RESPIRATORY ORGANS DAMAGE.

Respiratory damage is a common pathology in children. When examining a child with a respiratory disease, the following main questions have to be addressed:

1. Is the respiratory lesion the leading one in this patient?

2. Where is the main pathological process localized: in the upper respiratory tract, larynx, bronchi, lungs, pleura, mediastinum?

3. Is the bronchopulmonary process diffuse (common) or local?

4. What type of ventilation impairment prevails - obstructive or restrictive?

5. Is this episode an acute illness or an exacerbation of a recurrent or chronic process?

6. Is the disease associated with an infection, what infection (viral, bacterial, etc.) can be considered the leading one?

7. Does allergy play a role in the development of the disease, what type is it?

8. Is the disease likely to be genetically determined?

9. What is the possible prognosis of the disease for the life of the patient and in relation to the development of persistent changes?

10. What was the effect of treatment in the past? The answer to these questions or part of them can sometimes be obtained already at the first examination of the patient, which makes it possible to study the differential diagnostic circle, thereby determining the necessary additional studies.

The main symptoms that make it possible to suspect a lesion of the respiratory system. When questioning the patient, attention is drawn to the presence of the patient: cough; pain in the chest when breathing; shortness of breath; suffocation; sputum secretion; hemoptysis. During a general examination, a forced position on the side, orthopnea, cyanosis, and the presence of "drum sticks" are noted; when examining the chest - pathological forms of the chest (emphysematous, funnel-shaped, scaphoid, kypholoscoliotic), lagging behind when breathing, quickening or slowing down breathing, pathological breathing rhythm (Cheyne-Stokes, Biot, Kussmaul breathing). During palpation of the chest: changes in voice trembling - weakening or strengthening it. With comparative percussion of the lungs - the presence of a tympanic sound, dull, dull. When conducting topographic percussion - displacement of the lower boundaries of the lungs up, down; limited mobility of the lower edges of the lungs. Auscultation of the lungs reveals changes in vesicular breathing - weakening, strengthening, hard; the appearance of bronchial breathing; the appearance of side respiratory noises: dry, wet rales; crepitus; pleural friction noise.

Pain in the chest. Chest pain in children is much less common than headache or abdominal pain. Pain when breathing in young children is best recognized by external manifestations. Older children report it themselves, albeit to a lesser extent. Sensory innervation of the chest is provided by the intercostal nerves segmentally. Almost half of all sensory fibers approach the diaphragm as part of the phrenic nerve (C3–C5). Pain sensitivity of all organs in the chest cavity is provided only by sympathetic nerves, so pain in the chest wall is perceived as superficial, accurately described, localized and delimited. Visceral pain due to damage to the organs of the chest cavity, on the contrary, often radiates, is dull diffuse and is perceived as coming from the depths. Pain in the chest wall can be grouped according to the following features: - constant pain; - Pain not dependent on breathing - pain that occurs only when breathing; - Constant pain, aggravated by breathing. Pain during movements not related to breathing is caused by damage to the vertebrae, ribs and muscles. If such pain also occurs during breathing, during

coughing, sneezing or laughter, then, in addition to these organs, one should think about damage to the pleura. Children experience their pain sensations incompletely and inaccurately, therefore objective research methods and their consistent implementation are of decisive importance: careful examination, palpation, percussion, auscultation, X-ray examination, complete blood count, tuberculin diagnosis.

Alterations in the upper respiratory tract. Changes in the upper respiratory tract should attract attention in all patients with bronchial pathology. Manifestations of acute respiratory viral infection (ARVI), such as conjunctivitis, rhinitis, pharyngitis, as well as chronic foci of infection (sinusitis, tonsillitis, adenoiditis), may be directly related to deeper processes in the bronchi and lungs. Paleness of the nasal mucosa, hay runny nose (intermittent or persistent allergic rhinitis) are typical for patients with respiratory allergies. Difficulty in nasal breathing is often the result of the growth of adenoid vegetations, less often - the curvature of the nasal septum. The child has a characteristic facial expression, his mouth is ajar, periorbital shadows may be observed, and subsequent deformation of the facial skull is possible. The degree of nasal breathing impairment can be determined by the child's ability to breathe with the mouth closed, and also (in older children) by the speed of the air stream exhaled through one nostril, felt by the researcher's hand. Hoarseness or hoarseness can occur in a variety of conditions. The most pronounced and severe form is aphonia, the main causes of which are: 1. Acute infection of the mucous membrane of the larynx, acute viral laryngitis, "false" croup (viral stenosing laryngotracheitis), diphtheria croup. 2. Chronic infections: chronic sinusitis and bronchitis, tuberculosis of the larynx. Changes in the vocal cords of a different nature are due to: dryness of the respiratory tract with predominant breathing through the mouth; croup syndrome due to severe air pollution, primarily irritating gases; non-inflammatory edema of the respiratory mucosa in diseases accompanied by hypoproteinemia; hypothyroidism, in which hypothyroid swelling of the vocal cords causes a hoarse, low (“grunting”) voice; tumors of the vocal cords, mainly papillomas, nodules,

paralysis of the vocal cords, for example, with damage to the recurrent nerve or functional disorders (phonasthenia). Non-physiological relationships in the formation of sound lead to an overstrain of the vocal cords. The voice, as it were, "intercepts." After a long conversation, hoarseness and fatigue appear.

Psychogenic aphonia. The so-called hysterical aphonia is rarely observed in children and is easily recognized, since the child's voice becomes silent, but clear and distinct sounds are heard when coughing. The causes of psychogenic aphonia must be sought in the life problems that are relevant to the child.

Cough. Cough is a sharp expulsion of air from the lungs and respiratory tract, which before that is delayed by a closed glottis. The volume of the cough depends on the pressure of the exhaled air, and its tone on the properties and characteristics of the walls of the respiratory tract. Cough appears with changes in the pharynx, larynx, trachea, bronchi, and pleura and with irritation of the cough center and external auditory canal. As a reflex act, coughing can be caused by irritation of the endings of the vagus and glossopharyngeal nerves located in the mucous membrane of the respiratory tract: pharynx, larynx, trachea and large bronchi. In the smallest bronchi and alveoli, there are no such endings, so a cough reflex occurs. Coughing is most often and easier caused by irritation of the so-called cough zones: the posterior pharyngeal wall, regio arythaenoidea, glottis and tracheal bifurcation. The pleura is also a significant receptor field. In inflammatory processes of the respiratory tract, the nerve endings are irritated by the swollen mucosa or by the accumulated pathological secretion advanced by the ciliated epithelium to the cough zones. Swelling and accumulation of secretions may be of allergic origin or result from stagnation in circulation. In childhood, foreign bodies in the respiratory tract can often be mechanical irritants, and in other cases, compression of the trachea or vagus nerve by enlarged lymph nodes and tumors in the mediastinum. Rare coughing shocks are physiological, in particular, they can be caused by the accumulation of mucus in the larynx during sleep and the accumulation of mucus from the nasopharynx above the entrance to the larynx. When taking a history of a coughing child, attention should be paid to the

epidemiological environment, since acute febrile illness with cough is characteristic of viral respiratory infections that are epidemiological in nature. It is important to find out whether the disease began with a cough or whether the cough appeared during the development of the disease. It is necessary to determine the characteristics of the cough: dry or wet, paroxysmal, spasmodic, developing during the day or mainly in the evening, when falling asleep, at night, frequent or only coughing, etc. It matters whether the child secretes sputum and whether the cough precedes sputum: sputum without cough is released from the nasopharynx, and sputum from the middle and lower respiratory tract is thrown out with a cough. In most cases, the doctor receives a personal impression of the nature of the cough during the taking of an anamnesis or during the study. If the child does not cough spontaneously, at the end of the study, when examining the oral cavity, it is possible to induce coughing by irritation of the pharynx with a spatula or by light pressure and pressing on the trachea in the area of the jugular fossa. Cough can occur when a foreign body enters. In infants with whooping cough, sneezing can be the equivalent of a coughing fit. Sneezing occurs when the nasal mucosa is irritated. Air flow under increased pressure, as when coughing, exits through the nose. A decrease in the cough reflex can be due to both sensory disturbances and weakness of the muscles that carry out the cough push. In the first case, we are talking about an increase in the threshold of the cough reflex, when a cough is caused only by the accumulation of a significant amount of sputum, which can be judged by a kind of "gurgling" sound during breathing, often heard at a distance. This phenomenon, apparently, lies on the verge of normal, since sputum is evacuated from the upper respiratory tract by more rare cough shocks. A reduced reflex due to a motor disorder is observed in children with a cut in the respiratory muscles and with myopathies. In an acute illness accompanied by a cough, many additional studies are not required, except for the usual detailed blood test, chest X-ray, and paranasal sinuses. However, in diseases with a prolonged cough, it is necessary to conduct a number of studies, such as tuberculin diagnostics, determination of the concentration of electrolytes in sweat, proteinogram,

bronchography, bronchoscopy. Sputum should also be examined (direct microscopy and inoculation on various media). Older children themselves collect sputum in a cup; in small children and infants, sputum for microbiological examination can be taken directly with a swab during discharge from the glottis.

Pharyngeal cough. The accumulation of mucus at the entrance to the larynx or dryness of the membrane of the pharynx causes short, usually repeated cough shocks. They are called coughing, emphasizing the easy character. The cause of coughing can be acute or chronic pharyngitis, a mild form of bronchitis, a fixed habit (like a tic), formed during or after bronchitis and sinusitis.

Simple loose cough. A simple wet cough is a cough of medium volume that occurs as a result of irritation of the bronchial mucosa in bronchitis, sinusitis, bronchiectasis (often there is also a persistent cough resembling whooping cough; in the morning, sputum leaves the mouth full); with congestive bronchitis (HF), smoker's bronchitis; with esophageal-tracheal fistula in newborns. In the presence of such a fistula, the child begins to cough immediately after the first sip of food and coughs at each subsequent feeding. Cyanosis and severe dyspnea may develop. A distinctive feature of a wet cough is its cyclicality, i.e., as if the natural cessation of a coughing fit.

Simple dry cough. A simple dry cough is an uncharacteristic cough of almost constant tone without sputum discharge. Usually such a cough is called irritating, as it is subjectively felt as more intrusive and unpleasant. Dry cough occurs in the initial stage of bronchitis, with laryngitis, laryngotracheitis, spontaneous pneumothorax, aspiration of a foreign body (immediately after aspiration, cyanosis and suffocation develop, subsequently a persistent, sometimes paroxysmal cough resembling whooping cough persists for a long time), when the hilar lymph nodes are affected by tuberculosis or non-Hodgkin's lymphomas, with inflammation of the costal pleura (cough occurs with every deep breath). When moving from cold air to a warm room, coughing can occur in healthy children. The study of the external auditory canal with the help of the auditory funnel is

accompanied by a short cough (physiological reflex of the vagus nerve), a cough occurs with general arousal and the associated increase in breathing.

Interrupted, suppressed (stopped) cough. Sudden cessation and suppression of coughing can occur: with age-related respiratory failure, when, despite severe irritation, the optimal breathing rhythm should remain; with pain sensation of various localization associated with inhalation; with pleuropneumonia, dry pleurisy, fracture of the ribs, fracture in the area of intervertebral joints in the thoracic spine, with inflammatory diseases in the upper abdomen; during severe headache of various origins. Cough with croup. Persistent cough of a peculiar tone and overtone. Laryngeal cough. Differs in a hoarse overtone, it is typical for diseases of the larynx. With diphtheria of the larynx, the cough is almost silent. With viral croup (influenza, parainfluenza, measles, etc.) or other diseases, the cough is hoarse, barking, while the voice is preserved.

Bitonic cough. Deep cough with a double sound: a high whistling tone and a lower wheezing tone during the cough thrust. It is characteristic for the narrowing of the lower respiratory tract with laryngotracheobronchitis, bronchiolitis, limited narrowing of the airways by a foreign body or compression by enlarged paratracheal lymph nodes, goiter and other stenosing processes in the posterior mediastinum.

Paroxysmal cough. It is a sudden series of coughing shocks. Whooping cough is the most prominent example of such a cough. The disease is accompanied by a series of 8-10 short coughing shocks, which are repeated after a deep wheezing breath (reprise) - a sound phenomenon associated with the flow of air through a spasmodic glottis. During an attack, the pressure in the vessels of the head rises, hypoxia develops, the face becomes red or cyanotic, the eyes fill with tears. At the end of the attack, viscous mucus leaves, there may be vomiting. Such attacks are very debilitating for the child. The severity of coughing fits is very individual. The cough is worse and more frequent at night than during the day. There may be a sore on the frenulum of the tongue (cough injury). In the blood - leukocytosis with lymphocytosis. Typical changes on the radiograph are focal-

stretchy shadows in the pericardial region (“basal triangle”). Pertussis-like cough, equally obsessive and acyclic, but not accompanied by reprises, usually indicates the presence of very viscous sputum. The same cough is observed with cystic fibrosis. Its pulmonary manifestations: chronic bronchitis, peribronchitis, sometimes bronchiectasis with sputum, often abundant. In the study of sweat, the content of chlorides is more than 70 mmol / l. In severe purulent bronchitis, sputum is detected, there is no reprise. With bronchiectasis due to chronic bronchitis, chronic pneumonia, with a cystic lung or individual pulmonary cysts (not associated with cystic fibrosis), reprise also does not occur (as opposed to whooping cough). Whooping cough differs from wet cough in the absence of cyclicity.

Psychogenic cough. Cough in a child, especially recurrent, may have a typical genesis. Increased maternal anxiety, concentration and attention to respiratory symptoms may be the cause of the cough reflex in the child. These children have a series of dry, loud coughs in situations where they want to get attention or achieve their goals. At the appointment, they cough before the examination, abruptly ceasing to cough after the anxious expectation of trouble associated with the examination is replaced by calm. A new attack of coughing can be provoked by touching on a topic that is unpleasant for the child (whims, observance of the daily routine) or even simply by starting an abstract conversation, as if not paying attention to the child.

Stridor. Stridor is a breath noise that occurs when air passes through a narrowed airway. Stridor always indicates narrowing of the airways. It can be in the inspiratory phase (inspiratory stridor), the expiratory phase (expiratory stridor), or in both phases of breathing (mixed stridor). In all cases, stridor indicates severe breathing difficulties and is a sign of dyspnea. Cyanosis and impaired respiratory mechanics indicate respiratory failure. Inspiratory stridor indicates a lesion in the region of the glottis or above it, mixed, inspiratory and expiratory, stridor is characteristic of diseases of the vocal apparatus and trachea. It is observed with

damage to the lower respiratory tract. Localization of the narrowing of the airways can be different.

Inspiratory stridor is observed with the following changes in the respiratory tract:

1. Narrowing of the nasal cavity. Noise during stridor resembles sounds during sniffing, occurs with nonspecific rhinitis in infants, syphilitic rhinitis (congenital syphilis) in newborns and children in the first months of life, with blockage of the nasal passages by a foreign body or choanal stenosis.

2. Narrowing of the pharynx. The contraction before inhalation into the larynx causes a peculiar sound similar to snoring. It occurs when the tongue sinks in children in an unconscious state; with a deep location of the tongue due to lower micrognathia, especially with Pierre Robin's syndrome; with abundant accumulation in the pharynx of a secret that impedes the passage of air, which is observed in patients with paralysis of swallowing, retropharyngeal abscess, purulent epiglottitis.

3. Narrowing in the region of the larynx. Characteristic signs - persistent barking cough and hoarseness - occur: - with influenza croup, as well as croup against the background of measles, diphtheria and other diseases; - phlegmonous epiglottitis; - congenital laryngeal and tracheal stridor with softening of the cartilaginous base of the trachea and bronchi and a peculiar stridor sound, reminiscent of chicken bubbling; - rickets (laryngospasm as a manifestation of life-threatening spasmophilia); - as a consequence of traumatic injuries of the larynx (external trauma or intubation, followed by mucosal edema and submucosal hemorrhage).

Mixed (inspiratory and expiratory) stridor may indicate: - tracheobronchitis, including severe laryngotracheitis (viral croup); - diphtheria croup with an abundance of pseudomembranes; - goiter, causing narrowing of the trachea in the form of a saber scabbard; - Volumetric processes in the upper mediastinum, narrowing the trachea; - strictures of the trachea associated with stenosis or atresia of the esophagus, with the consequences of prolonged intubation

(damage to the mucous membrane and cartilage of the trachea); - tracheotomy; - malformations of the aortic arch (doubling of the aortic arch, left-sided origin of the right subclavian artery); - anomalies of the pulmonary trunk (significant expansion); - open ductus arteriosus. Expiratory stridor usually occurs with narrowing of the bronchi, severe spastic bronchitis, bronchial asthma, foreign body aspiration, compression of the bronchi by enlarged lymph nodes, and malignant non-Hodgkin's lymphomas in the area of the roots of the lungs.

Dyspnea (shortness of breath). The concept of dyspnea has many different definitions. In the broadest sense, it means a violation of breathing.

The following forms of dyspnea are distinguished:

1. Inspiratory dyspnea - observed with obstruction of the upper respiratory tract: croup, congenital narrowing of the larynx, foreign body, etc.

2. Expiratory dyspnea - the chest is raised to the top and almost does not participate in the act of breathing. Exhalation is done slowly, sometimes with a whistle. Observed in bronchial asthma.

3. Mixed shortness of breath - expiratory-inspiratory - characteristic of bronchiolitis and pneumonia. This definition includes all types of ventilation abnormalities, all degrees of respiratory failure and other respiratory disorders. Dyspnea can be both a subjective sensation and an objective symptom. In the first case, this is a difficulty in breathing experienced by the patient himself or a feeling of lack of air, in the second - an objective symptomatology for the patient and the doctor. A fundamental factor in the interpretation of each case of dyspnea is the deviation of breathing from the norm. The concept of "dyspnea" does not exclude such a situation when the physiological effect of breathing is achieved and maintained with the help of pathophysiological mechanisms (for example, with the help of tachypnea). The existence of a causal relationship between dyspnea symptoms and certain pathophysiological conditions is still the subject of debate. All currently established relationships between hypoxemia, hypercapnia, ischemia of overactive respiratory muscles and the subjective sensation of increased respiratory work on the one hand, bronchial resistance and the direction of the

respiratory muscles on the other hand, can be used to explain only some, but not all, dyspnea conditions.

Orthopnea - a forced sitting position with an emphasis on the hands (usually behind the body) is taken by patients with a severe attack of bronchial asthma or pulmonary edema to facilitate the work of the respiratory muscles.

Wheezing is a peculiar phenomenon that occurs when exhalation is difficult. The mechanism of its development is associated with the vibration of the lumen of the large bronchi due to an excessive drop in intrabronchial pressure at a high speed of the air stream. Loud wheezing is more often heard in obstructive processes, largely due to bronchospasm, while it is not expressed in bronchiolitis, possibly due to greater narrowing of the small airways. The genesis of moaning breath is completely different. Usually, groaning breathing occurs in children with severe massive pneumonia, it occurs due to difficulty in inhaling as a result of a decrease in lung compliance and pain caused by concomitant pleurisy.

UPPER AIRWAY OBSTRUCTION SYNDROME

Acute upper airway obstruction caused by constriction of the larynx and bronchi is the most common cause of acute DN in children. The following factors predispose to its frequent occurrence: narrow airways, loose fiber of the subglottic space of the larynx, the tendency of children to laryngospasm, relative weakness of the respiratory muscles. In the subglottic space with viral lesions, allergic conditions, trauma, edema quickly occurs and life-threatening stenosis progresses. Against the background of narrow airways in young children, edema of 1 mm leads to a narrowing of the lumen up to 50%. In addition to edema, an important role in the genesis of obstruction belongs to the spastic component and mechanical blockage (foreign body, mucus, fibrin). All three pathological factors are present in upper airway obstruction of any origin.

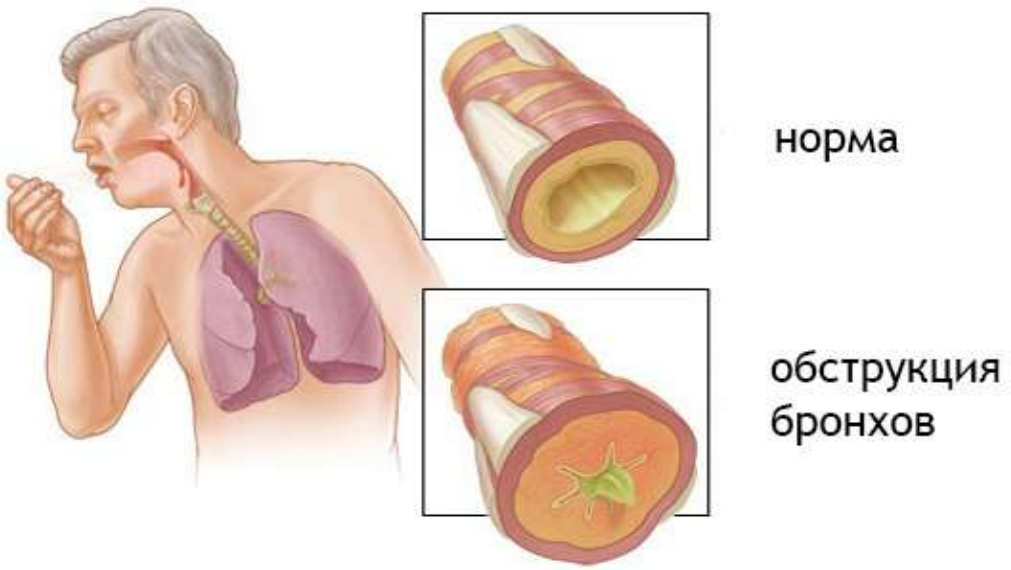


Figure 59. Scheme of bronchial obstruction.

The most common cause of high airway obstruction in children is acute stenosing laryngotracheitis (ASLT), which has a viral (parainfluenza, adenovirus, etc.) or combined viral-bacterial (staphylococcus or E. coli) etiology. Depending on the etiology and the previous disease, one of the forms of OSLT occurs - edematous, infiltrative, fibrino-necrotic (or obstructive).

The edematous form of OSLT usually develops at the onset of ARVI (often parainfluenza), has an infectious-allergic origin and is not accompanied by signs of intoxication. Both a rapid increase in symptoms and a rapid relief of signs of stenosis are characteristic, as well as a good effect when prescribing corticosteroids.

With an infiltrative form, stenosis develops by the 2–3rd day from the onset of ARI, intoxication is moderately expressed. The pathological process is caused by a combination of bacterial and viral infection. Stenosis grows slowly, but progresses to severe degrees, treatment is carried out with antibiotics and inhalations. The obstructive form of OSLT often occurs in the form of laryngotracheobronchitis. The stenosis is caused predominantly by fibrin deposits rather than subglottic narrowing, and the process is a descending bacterial fibrinous inflammation. Treatment is based on the use of antibacterial drugs, sputum thinning therapy, sanitation of the tracheobronchial tree.

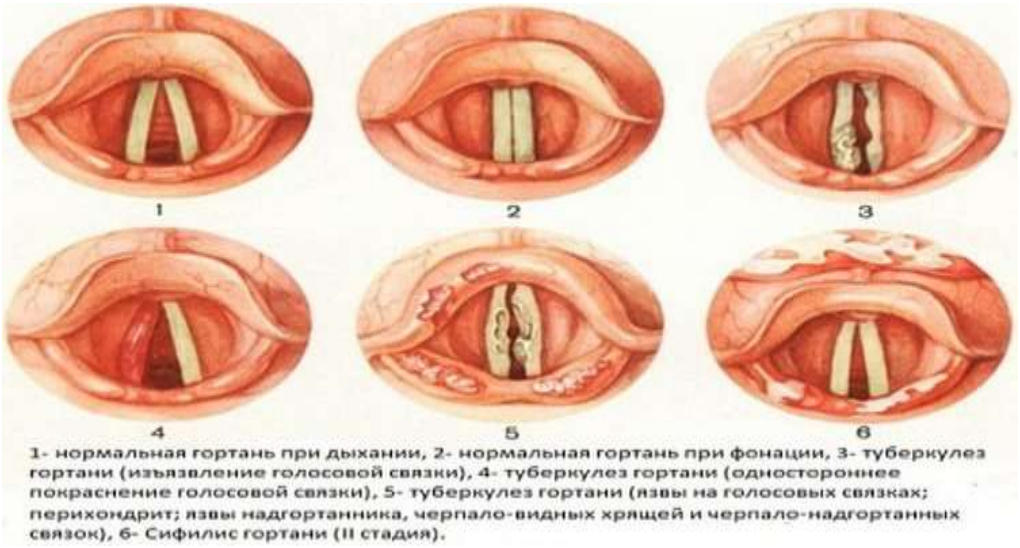


Figure 60. The state of the larynx in various pathologies.

Clinically, 4 degrees of stenosis of the larynx are distinguished:

- I degree (compensated croup) - characterized by hoarseness to aphonia, dry, barking, obsessive, persistent cough. Inspiratory dyspnea, stridor and DN occur in a patient during exercise (screaming, crying, feeding, coughing, etc.). At rest, there is no shortness of breath, tachycardia corresponds to fever;

- II degree (incomplete compensation) - along with hoarseness and a barking cough, stridor breathing is characteristic, audible at a distance, inspiratory dyspnea with the participation of auxiliary muscles in the act of breathing (inflation of the wings of the nose, retraction of the intercostal spaces, soft tissues of the neck), which sharply increases with load. The general state of moderate severity, there is anxiety, severe tachycardia (greater than it should be in terms of fever), increased blood pressure. The gas composition of the blood due to hyperventilation may still be normal (hypocapnia at rest and hypoxemia during exercise). Changes sharply increase during an attack, DN corresponds to the II degree;

- III degree (decompensated croup) - the general condition is severe, anxiety is replaced by periods of adynamia, lethargy, lethargy. Severe DN III degree with perioral and acrocyanosis at rest, periodically turning into generalized cyanosis; both inhalation and exhalation are difficult; in the blood hypoxemia,

hypercapnia, mixed respiratory metabolic acidosis. There are signs of heart failure (cyanosis of the lips, tip of the tongue, nose, fingers, liver enlargement, congestion in the lungs, arrhythmia, decreased blood pressure);

- IV degree (asphyxic) - pale cyanotic skin, periodically increasing cyanosis, arrhythmic, paradoxical breathing, bradycardia, arterial hypotension (i.e., there are signs of DN III and HF II B degree), cardiac arrest and breathing. Only mechanical ventilation in combination with resuscitation measures to maintain cardiac activity can save lives.

Foreign bodies of the larynx and trachea are one of the most common causes of asphyxia and sudden respiratory decompensation. Most often, foreign bodies are aspirated by children aged 1–3 years, boys are twice as likely as girls. The risk of aspiration of small objects, such as seeds, nuts, pebbles, etc., is especially high. When aspiration occurs, food, gastric contents may enter the respiratory tract during vomiting or passive leakage (regurgitation) in children during the first months of life, premature babies, with deep coma. The child can also inhale pieces of solid food and other objects and get a foreign body in the larynx or trachea with lightning-fast asphyxia. In about half of the cases, foreign bodies are localized in the trachea and can move from the subglottic space to the bifurcation of the trachea, causing periodic attacks of suffocation. With the localization of a foreign body in the bronchi, a reflex spasm of the bronchioles can occur, leading to the sudden appearance of signs of bronchial obstruction with a sharp lengthening of the exhalation. A foreign body in the respiratory tract is usually accompanied by the appearance of an acute paroxysmal, often painful cough, suffocation and severe anxiety in a healthy child. Foreign body aspiration is also suggested by the sudden appearance of wheezing or prolonged, unexplained cough, localized atelectasis, or x-ray distention. It should be remembered that the typical complications of foreign body aspiration are: tracheobronchitis, pneumonia, atelectasis and, if they persist for a long time, bronchiectasis. Surrounding adults sometimes note the appearance of sudden shortness of breath or choking, coughing, vomiting, cyanosis, or redness of the face with tears in the eyes,

sometimes with an accuracy of minutes. It is important to find out if small objects, solid foods, etc., were available to the child. True croup in diphtheria is due to the formation of fibrinous films on the vocal cords, manifested by a rough barking cough that loses sonority, complete aphonia and difficulty breathing develop. Often begins with a sore throat with a dense, dirty-gray plaque with an unpleasant odor on the tonsils and a significant swelling of the throat. Characterized by intoxication, enlargement and swelling of the submandibular lymph nodes, swelling of the subcutaneous tissue of the neck. Inspiratory obstruction may be congenital without causing clinical manifestations until the first ARI. Congenital laryngeal stridor is associated with underdevelopment of the tissues of the epiglottis, glottis, larynx, vocal cord paralysis due to birth trauma, and anatomical narrowing of the vocal cords due to stenosis, cyst, or tumor. Croup can also be associated with macroglossia, micrognathia, external compression of tissues in the neck. It is manifested by constant inspiratory sonorous dyspnea, retraction of compliant parts of the chest, which are aggravated by anxiety, screaming. With damage to the vocal cords, hoarseness of the voice is also observed. There is also a group of diseases in which swallowing processes are predominantly disturbed and secondary DN occurs due to damage to the lymphatic apparatus and subcutaneous tissue of the neck. These are pharyngeal and paratonsillar abscesses, infectious mononucleosis, Ludwig's angina. Unilateral swelling of the palatine tonsils, hyperemia, swelling of the palatine arches, along with intoxication, fever, salivation, pain when swallowing, are characteristic of paratonsillar abscess. A retropharyngeal abscess gives similar symptoms, but when examining the pharynx, edema, hyperemia and bulging of the posterior pharyngeal wall are noted. The basis of the treatment of these diseases is antibiotic therapy (usually penicillin), in case of DN - nasopharyngeal airway.

Obstructive difficulty in breathing during sleep. Obstructive difficulty in breathing during sleep is a common complaint of parents. This condition is accompanied by periodic nighttime snoring of the child, breathing through an open mouth, the appearance in a dream of periodic retraction of the intercostal spaces,

supraclavicular fossae, iliac region, enuresis, unusual postures and other sleep disorders. Usually the cause of such disorders are enlarged tonsils of the Pirogov-Waldeyer ring. Funnel-shaped chest, bite defects can also cause obstructive difficulty breathing during sleep. During the day, children may have drowsiness, headaches, absent-mindedness. In their blood, signs of metabolic alkalosis, polycythemia can be detected. The tactics of managing such children is determined in consultation with an ENT doctor.

“Low” airway obstruction. "Low" airway obstruction occurs when the bronchial lumen narrows, creating an obstacle to the passage of the air stream. The syndrome of acute bronchial obstruction (ABO) occurs more easily in young children, in whom the bronchial lumen is significantly narrower than in adults. In the genesis of OBO, edema of the bronchiole wall, obturation of the bronchi with accumulated secretions, mucus, purulent crusts (dyskrinia) and, finally, spasm of the bronchial muscles play a role. The ratio of these components varies depending on the causes of OBO and the age of the child. The most common OBO in children is observed with infectious obstructive bronchitis (bronchiolitis) with ARI, an attack of bronchial asthma and status asthmaticus, congestive left ventricular HF (the equivalent of adult cardiac asthma). In children of the first three years of life, OBO that occurs against the background of acute respiratory viral infections is almost always due to inflammatory edema of the bronchiolar mucosa (bronchiolitis). The primary disease in the first 3–6 months of life is usually associated with rhinosynchytial infection, and at the age of 6 months - 3 years - with parainfluenza. Repeated attacks of OBO in ARVI can be caused by any respiratory virus, since they occur against the background of an already previous bronchial sensitization with the inclusion of reaginic mechanisms. In other words, in these cases, bronchiolitis is combined with bronchospasm. Bronchospasm is always an indispensable component of OBO in children older than 3 years, which indicates the existence of asthma in the patient. The pathological role of dyskrinia (obstruction against the background of accumulation of mucus, desquamated epithelium, fibrin in the bronchi) should be taken into account when OBO develops

by the end of the first week of acute bronchopulmonary disease, especially in frequently ill children with severe concomitant pathology. All children, as a rule, have hypoxemia, which persists for 5 weeks even when the patient's condition improves. In a significant percentage of cases, as a result of increased work of breathing against high airway resistance due to muscle fatigue, the patient develops uncompensated respiratory acidosis with an increase in pCO2 above 65 mm Hg. Art. The terminal stage of any OBO is pulmonary edema due to significant negative intrathoracic pressure and secondary left ventricular heart failure.

Clinical manifestations. The leading symptom of OBO is expiratory, and in children of the first months and years of life, mixed dyspnea. The more severe the degree of obstruction and the more pronounced physical changes in the respiratory system, the more signs of increased work of breathing predominate in the clinical picture. Children of the first years of life, not finding the optimal position for expelling air, worry, rush about, "cannot find a place for themselves." Exhalation is carried out with the participation of auxiliary muscles, and children older than 3 years often take a forced position. Characterized by swelling of the chest, physical signs of increased airiness of the lungs (weakening of breathing and bronchophony, boxed percussion sound). The auscultatory picture differs depending on the predominance of one or another pathophysiological mechanism of obstruction. So, with the predominance of the hypercrine component, mainly coarse, buzzing rales are heard, with the "edematous" variant of OBO with significant extravasation of fluid into the lumen of the bronchi and bronchioles - scattered small bubbling wet rales on both sides. The combination of OBO with primary infectious toxicosis, along with excessive tachycardia (Kishsh toxicosis), widespread finely bubbling wet rales in the lungs, waxy skin, or periorbital edema, makes one suspect bronchial stenosis due to peribronchial edema. It is important to consider that the predominance of dry wheezing during auscultation indicates a narrowing of the bronchial lumen, mainly due to bronchospasm or swelling of the mucous membrane. However, with a pronounced bronchial obstruction, areas of the “silent” lung may appear, over which wheezing is not audible, and breathing is

significantly weakened. In recurrent broncho-obstructive syndrome, a differential diagnosis is made with cystic fibrosis, α1-antitrypsin deficiency, foreign bodies, aspiration pneumonia or bronchitis, and other conditions that cause recurrent broncho-obstructive syndrome. In bronchial asthma in children of the first years of life, bronchospasm is minimally expressed, a violation of bronchial patency occurs as a result of edema of the bronchial mucosa and hypercrinia. An attack is usually preceded by a period of precursors, characterized by a runny nose, cough, lacrimation, and often short-term subfebrile body temperature. During an attack over the lungs, not only dry, but also various wet rales are heard. The older the child, the greater the role of the bronchospastic component in the genesis of the attack. At the same time, the period of precursors is shortened, shortness of breath acquires a distinctly expiratory character, and over the lungs during auscultation, dry wheezing rales prevail over buzzing and moist.

Syndrome of compaction of lung tissue.

Small-focal compaction (lobular, pneumosclerosis). The clinical picture depends on the depth of the seal, their size and number. External examination without visible changes, there may be a lag of the diseased side of the chest in the act of breathing. On palpation of the chest - a slight increase in voice trembling on the affected side. Percussion of the lungs reveals dullness of percussion sound over the affected area. Auscultation of the lungs reveals a weakening of vesicular respiration over the foci of compaction, small bubbling sonorous rales are heard in a limited area.

Large-focal compaction (whole lung, lobe, segment). During the examination, there is a lag of the diseased side of the chest in the act of breathing, increased voice trembling over the affected area during palpation. Percussion of the lungs reveals a dull percussion sound above the compaction zone. During auscultation of the lungs, bronchial breathing is heard above the zone of compaction.

Figure 61. Scheme of macrofocal compaction.

An x-ray examination of the chest with a syndrome of compaction of the lung tissue shows a darkening corresponding to a lobe or segment of the lung, small foci of darkening of various sizes are detected, and an increase in the pulmonary pattern is determined.

Syndrome of the presence of fluid in the pleural cavity. A pleural effusion is an abnormal accumulation of fluid in the pleural cavity. The pleural cavity is the space between the parietal and visceral sheets of the pleura, connecting at the roots of the lungs. The accumulation of fluid in the pleural cavity is called hydrothorax. There may be accumulation of fluid in one or both pleural cavities. Its character can be inflammatory (exudate) and non-inflammatory (transudate). The causes of exudate are inflammation of the pleura (pleurisy) in tuberculosis and pneumonia, pleural carcinomatosis in malignant neoplasms. More often the lesion is unilateral. Causes of hydrothorax, or accumulation of transudate in the pleural cavity, may be congestion in the pulmonary circulation in heart failure or general fluid retention in kidney disease. The process is more often bilateral and is often combined with peripheral edema, ascites, hydropericardium.

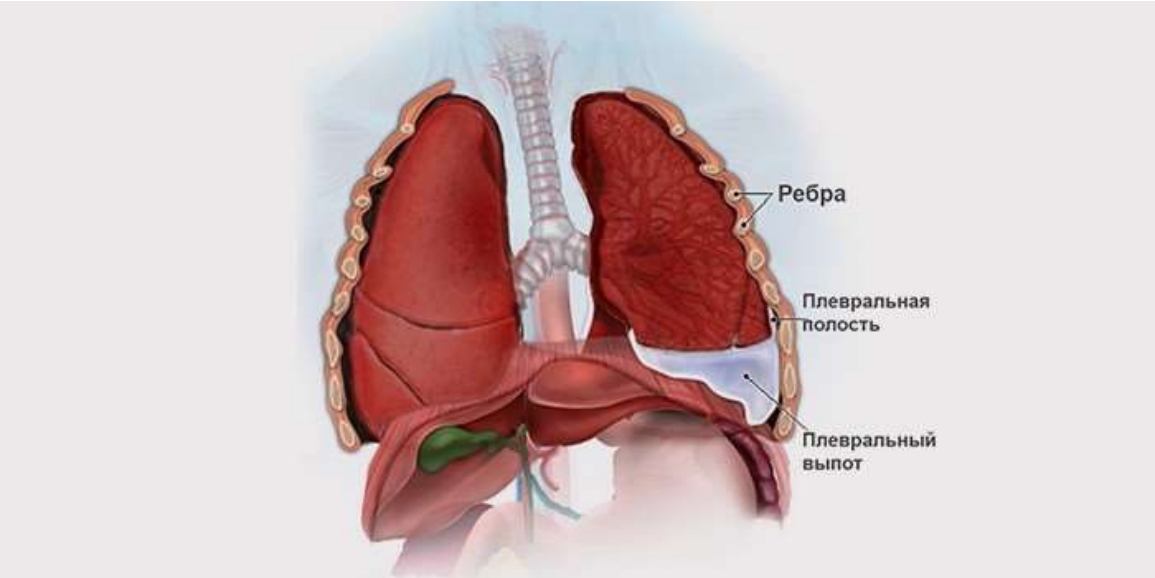


Figure 62. Scheme of the Syndrome of the presence of fluid in the pleural cavity.

Complaints. It is important to assess the general appearance of the patient and his nutrition. The position of the child may indicate irritation of the pleura, since patients with pleural effusion will spare the chest from the affected side. With rapid and significant accumulation of fluid, lung atelectasis and respiratory failure syndrome develop. Patients complain of shortness of breath, aggravated in the position on a healthy side, a feeling of heaviness in the diseased half of the chest.

Inspection. Patients often take a forced position (on the sore side), the affected side may increase somewhat in size, lag behind when breathing, the intercostal spaces are smoothed out, even swell.

Palpation. There is an increased resistance of the intercostal spaces, voice trembling is weakened or absent.

Percussion. With comparative percussion, a dull percussion sound is detected, in the Garland triangle (above - above the compressed exudate of the lung) - bluntly tympanic, in the Rauchfus-Grokko triangle - blunt. With topographic percussion, the upper level of the liquid is in the form of an Ellis-Damoiseau oblique line. Determination of the lower border of the lung and excursion of the lung edge from the affected side becomes impossible.

Auscultation. Breathing over the area of fluid accumulation is weakened or completely absent. In the case of pressing the atelectatic lung to the root directly above the fluid level in a limited space, weakened bronchial breathing can be heard. Bronchophony is negative or weakened, in the zone of bronchial breathing it may increase. Pleural effusion must be differentiated from compaction of the lung tissue, in which dullness of percussion sound and weakening of respiratory sounds will also be noted. The diagnosis can be differentiated by positive bronchophony with a trembling sound (a sign of compaction). Palpation of the trachea and determination of the cardiac impulse may reveal mediastinal displacement in the opposite direction to the lesion.

Syndrome diagnosis. The most important signs of hydrothorax are dull percussion sound over the lower parts of the lungs, lack of breathing and negative bronchophony in the area of dullness. Additional research methods. Radiologically, homogeneous shading of the lung field is determined, mediastinal shift to the healthy side. For diagnostic and therapeutic purposes, a pleural puncture is performed, which makes it possible to determine the nature of the existing fluid.

SYNDROME OF ACCUMULATION OF AIR IN THE PLEURAL CAVITY.

The accumulation of air in the pleural cavity is called pneumothorax. The causes of pneumothorax are varied. So, in the neonatal period, it is more often a consequence of an increase in intrapulmonary pressure, which occurs when coughing, aspiration of mucus from the respiratory tract, as well as during resuscitation with the use of artificial ventilation and forced breathing. Pneumothorax can also occur in the presence of congenital malformations of the lung (cysts, congenital lobar emphysema). In other age groups, pneumothorax may be the result of a breakthrough of air cavities (bulls) and the formation of bronchopleural fistulas. This is most often observed in purulent-destructive processes in the lungs of staphylococcal etiology. Due to infection of the pleura, pneumothorax turns into pyopneumothorax. The occurrence of pneumothorax is

observed with root pneumonia, bronchial asthma, as a result of aspiration of a foreign body, after tracheotomy. In tuberculosis, it occurs as a result of a breakthrough into the pleural cavity of caseous foci or small cavities. By origin, pneumothorax can be spontaneous, traumatic and artificial, produced for therapeutic purposes. There are closed pneumothorax, which does not have communication with the atmosphere, open, freely communicating with it, and valvular, sucking air on inspiration and, as a result, constantly growing.

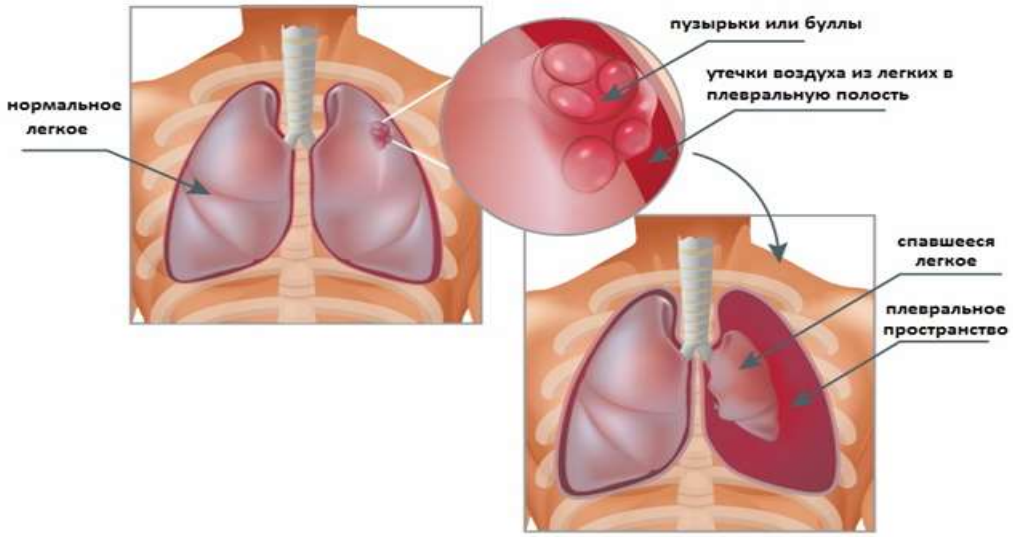


Figure 63. Syndrome of accumulation of air in the pleural cavity

Complaints. At the time of the formation of pneumothorax, the patient experiences a sharp pain in the side, notes a dry cough and severe shortness of breath. With valvular pneumothorax, shortness of breath gradually increases.

Characterized by an acute sudden onset in the midst of full health.

The onset of the disease may be associated with significant physical exertion, vomiting, or as a result of surgical procedures (puncture of veins, arteries). In the anamnesis there are indications of frequent physical overstrain, pulmonary tuberculosis.

Inspection. Possible protrusion of the affected side of the chest, lagging behind when breathing, smoothness of the intercostal spaces.

Palpation. There is no voice trembling on the affected side. At high pressure in the pleural cavity (valvular pneumothorax), the intercostal spaces are resistant.

Percussion. Above the affected half of the chest, a loud tympanic sound is

detected, with valvular pneumothorax - blunt-tympanic. The lower border of the lungs and its mobility are not determined.

Auscultation. Breathing from the affected side is sharply weakened or absent, bronchophony is negative. If the pleural cavity freely communicates with the bronchus, bronchial breathing and positive bronchophony can be heard. There is a shift in the boundaries of the heart and the apex beat to the healthy side. Heart sounds are weakened, tachycardia, pulse of small filling, may be thready.

Diagnosis of pneumothorax. Reliable signs are the lag in breathing of the affected half of the chest, the absence of voice trembling, a loud tympanic sound, and sharply weakened breathing over the affected half of the chest.

Additional research methods. X-ray reveals a light lung field without a lung pattern, closer to the root - a shadow of a compressed lung. The mediastinum in valvular pneumothorax is displaced to the healthy side.

CAVITY SYNDROME IN THE LUNG TISSUE.

The cavity syndrome includes signs, the appearance of which is associated with the presence of a cavity, abscesses, cysts, i.e., formations that have a dense, more or less smooth wall, often surrounded by an infiltrative or fibrous shaft. The identification of this syndrome occurs primarily when using the main methods of examining the patient - examination, palpation, percussion, auscultation. The cavity can be filled with air entirely (an empty cavity) or contain, in addition to air, one or another amount of fluid, remain closed or communicate with the draining bronchus. All this is reflected in the features of the symptomatology, which also depends on the size of the cavity and the depth of its location.

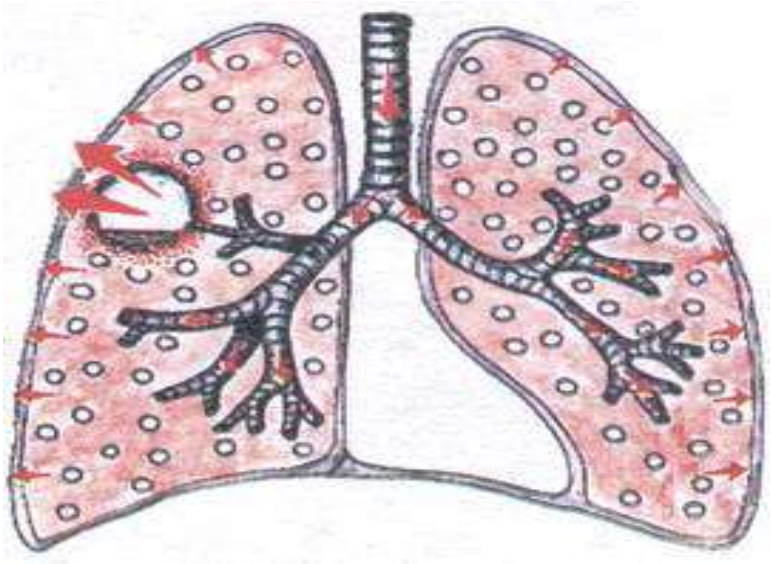


Figure 64. Scheme of cavity syndrome in the lung tissue

Complaints. Worried about cough with purulent sputum in large quantities, often with a full mouth, septic temperature with night pouring sweat (characteristic of lung abscess, bronchiectasis), cough with bloody sputum or streaked with blood (with cavernous tuberculosis, decay of lung tissue). Inspection.

General examination: acrocyanosis, diffuse cyanosis, change in terminal phalanges ("drumsticks", "watch glasses").

Examination of the chest: lagging of the "sick" half of the chest during breathing, tachypnea.

Palpation. Over the projection of the cavity, voice trembling is intense.

Percussion. Above the cavity, a tympanic or dull-tympanic sound is determined. Auscultation. Above the cavity, bronchial breathing or its variety, amphoric breathing, is heard; often medium and large bubbling moist rales.

Additional research methods. Radiographically, against the background of darkening in the lung tissue, a limited enlightenment of a rounded or oval shape is detected, and a characteristic horizontal level of fluid is often determined. Syndrome of increased airiness of the lung tissue (emphysema).

Complaints. Persistent shortness of breath, non-productive cough. Inspection. "Barrel-shaped" chest. Palpation. Voice trembling is weakened on both sides. Percussion. With comparative - tympanic, "box" sound, with topographic -

omission of the lower borders of the lungs, limitation of mobility of the lower edges of the lungs.

Auscultation. Decreased vesicular respiration. Additional research methods. X-ray reveals an increase in the transparency of the lung fields, depletion of the lung pattern. The study of the function of external respiration shows an increase in the residual volume of the lungs, a decrease in VC (restrictive disorders), often obstructive disorders.

Respiratory failure syndrome. Respiratory failure is a state of the body in which the normal gas composition of the blood is not maintained or it is achieved due to more intensive work of the external respiration apparatus and the heart, which leads to a decrease in the functional capabilities of the body.

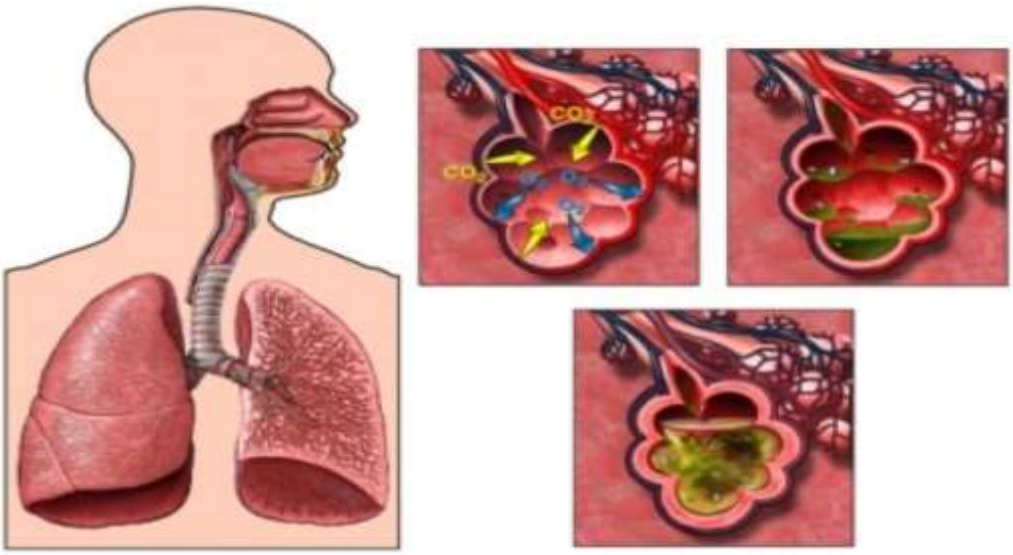


Figure 65. Scheme of respiratory failure syndrome

Normal gas exchange in the lungs is possible with a clear relationship of three components:

1) ventilation;

2) diffusion of gases through the alveolar-capillary membrane;

3) perfusion of capillary blood in the lungs. The causes of DN can be any pathological processes in the body or adverse environmental factors that affect at least one of these components.

There are two groups of DN:

1) with a predominant lesion of the pulmonary mechanisms;

2) with a predominant lesion of extrapulmonary mechanisms.

The first group includes the following pathological conditions:

1. Obstruction of large airways.

2. Obstruction of small airways.

3. Reduction of lung tissue.

4. Violation of the restriction of the alveolar tissue.

5. Violation of pulmonary blood flow.

6. Violation of ventilation-perfusion ratios.

7. Thickening of the alveolar-capillary membrane.

The second group includes the following pathological conditions:

1. Violation of the central regulation of respiration.

2. Damage to the respiratory muscles.

3. Damage to the chest. Depending on the causes and mechanism of DN, there are three types of violations of the ventilation function of the lungs: - obstructive; - restrictive; - mixed.

The obstructive type is characterized by difficulty in the passage of air through the bronchi due to their inflammation, bronchospasm, narrowing or compression of the trachea and large bronchi. Of the phases of respiration, exhalation is predominantly affected. In spirographic study, there is a decrease in maximum ventilation of the lungs and forced expiratory volume in 1 s (functional VC) with a slight decrease in VC.

Restrictive type - ventilation disorders are observed when the ability of the lungs to expand or collapse is limited (hydrothorax, pneumothorax, pneumosclerosis, kyphoscoliosis, massive pleural adhesions, ossification of the costal cartilages, limitation of rib mobility). Of the phases of respiration, inhalation suffers predominantly. In spirographic study, there is a decrease in VC and maximum ventilation of the lungs. The mixed type occurs in long-term pulmonary and cardiac diseases and combines the signs of two types of ventilation disorders (obstructive and restrictive), often with the prevalence of one of them.

There are three degrees of RF:

- I degree - shortness of breath occurs with previously available physical exertion, there is no cyanosis (cyanosis of the nasolabial triangle is possible, tension of the wings of the nose in young children), fatigue, auxiliary respiratory muscles are not involved in breathing, tachycardia, blood pressure is normal, pO2 reduced to 80–65 mm Hg. Art.;

- II degree - shortness of breath occurs with habitual exertion, cyanosis is not pronounced, fatigue is pronounced, with exertion, auxiliary muscles are involved in breathing, blood pressure is increased, euphoria, anxiety are observed, there may be lethargy, adynamia, muscle hypotension. The minute volume of breathing rises to 150-160% of the norm. The respiratory reserve is reduced by 30%, pO2 - up to 64–51 mm Hg. Art., pCO2 is normal or slightly increased (up to 46–50 mm Hg), pH is normal or slightly reduced. When 40% oxygen is inhaled, the condition improves significantly, the partial pressure of blood gases normalizes;

- III degree - shortness of breath at rest, cyanosis, fatigue is pronounced, auxiliary muscles are constantly involved in breathing. Respiratory arrhythmia, tachycardia, the ratio of respiratory rate to pulse rate 1: 2, blood pressure is reduced. Respiratory arrhythmia and apnea lead to a decrease in its frequency. Lethargy, lethargy, adynamia appear; pO2 is reduced to 50 mm Hg. Art., pCO2 rises to 75–100 mm Hg. Art., pH reduced to 7.25–7.20. Inhalation of 40% oxygen does not give a positive effect. Sometimes they distinguish between grade IV DN (hypoxic coma), when the child is unconscious, the skin is earthy, the lips and face are cyanotic, there are cyanotic or cyanotic-purple spots on the limbs and torso. Breathing is convulsive, the mouth is open (the child gasps for air). The respiratory rate decreases and becomes almost normal or even decreases to 8-10 per 1 min due to prolonged apnea. Tachycardia or bradycardia is noted, the pulse is thready, blood pressure is sharply reduced or not detected, pO2 is below 50 mm Hg. Art., pCO2 more than 100 mm Hg. Art.; The pH is reduced to 7.15 or below. The presence of DN and its degree are judged by the severity of such clinical signs as shortness of breath, tachycardia, cyanosis, and by changes in respiratory volumes

and capacities. The data of physical methods of research will depend on the underlying disease with which the development of DN is associated. In the case of hypocapnia (pCO2 below 35 mm Hg) due to hyperventilation, which occurs more often in children than in adults, lethargy, drowsiness, pallor and dry skin, muscle hypotension, tachycardia or bradycardia, alkalosis (pH above 7 ,45), alkaline urine. With an increase in hypocapnia, fainting is possible, hypocalcemia is noted, leading to convulsions.

SEMIOTICS AND MAIN SYNDROMES IN IMPAIRED URINARY SYSTEM ORGANS

Dysuric disorders - violations of the act of urination, which include pain and cramps during urination, frequent or slow urination, nocturnal and daytime incontinence and urinary incontinence. Soreness during urination (algouria) in infants is manifested by anxiety, crying during or immediately after urination.

Pollakiuria - frequent urination in small portions. An imperative urge is the appearance of an irresistible urge to urinate with a sharp shortening of the time interval (up to several seconds) until the obligatory emptying of the bladder. Urinary incontinence is passing urine without the urge to urinate. It can be with diseases of the spinal cord (true) and with malformations of the urinary and reproductive systems (false).

Enuresis is involuntary urination during sleep. With a decrease in urination, the number of urination per day is less than the lower limit of age indicators. Urinary retention (ischuria) is partial and complete (acute and chronic). Partial urinary retention is characterized by incomplete emptying of the bladder, which occurs when there is an obstruction at the level of the bladder neck and urethra that disrupts the passage of urine. In this case, urinary retention occurs, it is carried out in two stages, with an intermittent stream. Acute complete retention may be caused by stone formation, trauma (rupture) of the urethra and other causes, chronic may be the result of trauma, diseases of the spinal cord, etc. Causes of dysuric disorders: congenital and acquired diseases of the spinal cord and brain, peripheral nerves, intramural nerve plexuses ; delay in the development of regulatory systems

of the bladder; microbial-inflammatory processes in the urinary system; malformations of the urinary and reproductive systems; traumatic injuries of the urinary system, ureterocele, epispadias, phimosis, rupture of the bladder, rupture of the urethra, etc. The amount of urine in children depends on age, the nature of food, the volume of fluid taken, physical activity, and ambient temperature. It can be calculated using the formula 1500 ml × (S: 1.73 m 2), where S is the surface of the child's body; for children from 1 to 10 years old according to the formula 600 + {100 × (n - 1)}, where n is the number of years of the child. The ratio of daytime diuresis to nighttime is 3 : 1. The predominance of nighttime diuresis over daytime - nocturia - indicates a violation of kidney function, can be observed when edema converges.

Oliguria - a decrease in the daily amount of urine - is a sign of a violation of either the production or excretion of urine (diuresis less than 0.3 ml / (kg * h). Physiological oliguria is observed in the first 2-3 days after birth due to insufficient lactation. According to the mechanism of occurrence oliguria is prerenal, renal and postrenal.Of the causes of prerenal oliguria, the most common is insufficient perfusion of the kidneys: a decrease in the volume of extracellular fluid as a result of sodium loss (vomiting, diarrhea, increased perspiration at high temperature, shortness of breath, physical exertion, severe sweating), a decrease in BCC (bleeding, hypoalbuminemia, sepsis), decreased cardiac output (myocardial disease, heart defects, pericarditis), kidney vascular damage (renal artery pathology, nephrosclerosis, vasculitis). Renal oliguria is characteristic of kidney diseases: glomerulonephritis, acute interstitial nephritis, acute tubular necrosis, for nephrotoxic substance poisoning, vascular pathology (embolism, infective endocarditis, systemic vasculitis, hemolytic uremic syndrome, etc.). Postrenal oliguria is associated with bilateral obstruction of the urinary tract (ureter obstruction by a stone in urolithiasis, blood clot, tumor, urethral stricture, urethral stenosis, prostate disease, etc.).

Anuria is spoken of when diuresis is less than 0.15 ml / kg • h or its complete absence. Anuria always indicates renal failure. Oliguria and anuria are

differentiated with acute urinary retention, in which there are strong, painful urge to urinate, marked anxiety, and an overflowing bladder is determined by palpation. Acute urinary retention in children is caused by phimosis, balanoposthitis, bladder and urethral stones. The term polyuria is understood as an increase in diuresis by 2 times compared with the norm, or the amount of urine> 1500 ml / m2 / day. A decrease in the water reabsorption coefficient by 1% causes an increase in diuresis by 300–500 ml.

Figure No. 66. Urine color in various kidney diseases

Polyuria is observed under physiological conditions when an excessive amount of fluid is consumed, either out of habit or in connection with mental disorders (diagnosis is helped by a test with a dry diet - the concentration ability of the kidneys during the test is normal). Polyuria is observed in diabetes mellitus, is characteristic of de Toni-Debre-Fanconi syndrome, can be observed in sarcaidosis, multiple myeloma, during the period of convergence of edema in diseases of the heart and kidneys. It can be compensatory in the development of chronic renal failure (polyuric phase) or in recovery from acute renal failure. A change in the transparency and color of urine may be due to excess salt content, cellular elements, mucus and fat (lipuria). If the turbidity disappears when heated, then it is due to an excess of urates. If the turbidity does not disappear when heated, a few drops of acetic acid should be added - the disappearance of the turbidity indicates an excess of phosphates, its hissing indicates the presence of carbonates. When diluted hydrochloric acid is added to the urine, the disappearance of turbidity

indicates the presence of oxalic acid salts. If in this case the urine remains opaque, then we can think that the reason is the presence of cellular elements (detected by microscopic examination of the sediment), uric acid salts, mucus, and fat. Almost colorless light yellow urine is the result of its dilution and is observed with polyuria (diabetes and diabetes insipidus, chronic renal failure, etc.).

Figure No. 67. Urine color in diabetes mellitus, chronic renal failure

Proteinuria is the appearance of protein in the urine, the amount of which exceeds normal values. In the urine of a healthy child, up to 100 mg of protein per day is determined (according to the Lowry method) and up to 30–60 mg / day (with the Geller ring test). The disease is also diagnosed with the help of urinary indicator strips. Proteinuria is represented by various types of plasma proteins (30 types), including albumin, as well as a number of tissue proteins (glycoproteins) secreted by the cells of the tubules and the mucous membrane of the genitourinary organs, including the Tamm-Horsfall uroprotein, the origin of which is associated with cells of the ascending limb of the loop of Henle. Proteinuria occurs alone or in combination with other changes in the urine in the form of erythrocyturia, leukocyturia, cylindruria, bacteriuria, and is also combined with extrarenal symptoms. The protein content in urine depends on: the state of the glomerular filter (endothelium with fenestrae, basement membrane, podocytes), which has a permeability for plasma proteins, determined by the pore size of the basement membrane (2.9 + 1 nm), the molecular weight of the protein (below 65,000–70

000), electric charge and configuration of their molecules; from hemodynamic factors that provide filtration; from the ability of the tubular apparatus to reabsorb proteins from the ultrafiltrate. Normally, almost all of the protein filtered in the Shumlyansky-Bowman capsule - 35-50 g / day - is reabsorbed in the proximal tubules, and only a small part of it is excreted in the urine. In violation of any of these mechanisms, proteinuria of various origins occurs. In the first days of life, the level of protein in the urine exceeds normal values due to increased permeability of the epithelium of the glomeruli and tubules against the background of hemodynamic features (physiological albuminuria). Persistence of elevated levels of protein in the urine after the 1st week of life is considered as a pathological symptom. Depending on the mechanism of occurrence of proteinuria in kidney diseases (renal), it can be glomerular (glomerular), tubular (tubular) and mixed (with a combination of the first two types). Proteinuria is also distinguished prerenal (overflow), postrenal, secretory, pure and functional.

Glomerular proteinuria occurs when the permeability of glomerular capillaries increases due to a violation of their walls, including due to a violation of the electrostatic barrier, an increase in the pore size of the basement membrane. The value of glomerular proteinuria ranges from 0.1 to 20 g/day, represented by albumin, transferrin, β2-microglobulin, γ-globulin. As a result of the loss of plasma proteins in the urine, especially albumin, a decrease in the concentration of protein in the serum, intracapillary oncotic pressure decreases and tissue edema develops. Glomerular proteinuria is typical for primary and secondary glomerulonephritis, including glomerulonephritis with minimal changes in the glomeruli, for amyloidosis of the kidneys, diabetic glomerulosclerosis, thrombosis of the renal veins, etc.

Tubular proteinuria is observed when the functional ability of the tubules (proximal) to reabsorb filtered proteins is damaged and impaired. This type of proteinuria is characterized by a high content in the urine of low molecular weight proteins of the prealbumin fraction, which are easily filtered in the renal glomeruli (β2-microglobulin (molar mass - 11,600), lysozyme (molar mass - 14,000), light

chains of immunoglobulins, ribonuclease, some hormones, etc. .). At the same time, albumin excretion either does not increase at all, or does not increase much. Protein loss is usually small, up to 2 g/day, edema and lipid disorders do not occur, since albumin losses are small. Tubular proteinuria is observed in primary (congenital) and acquired tubulopathies - pyelonephritis, interstitial nephritis, de Toni-Debre-Fanconi syndrome, toxic effects of salts of heavy metals (lead, mercury, cadmium, bismuth) and drugs (salicylates, etc.)

Prerenal proteinuria ("overflow") occurs with increased synthesis and accumulation in the blood plasma of low molecular weight proteins (hemoglobin, myoglobin, immunoglobulin light chains, fibrin degradation products), which pass through an intact glomerular filter in large quantities, and normally functioning tubules cannot ensure their reabsorption. This type of proteinuria is observed in leukemia, malignant lymphomas, multiple myeloma, as well as in massive tissue necrosis (myoglobinuria) and intravascular hemolysis (hemoglobinuria) caused by transfusion of incompatible blood, exposure to hemolytic poisons, medicinal and immunologically mediated effects. Urinalysis also reveals erythrocytes, leukocytes, and sometimes free hemoglobin. Clinically, hemolysis is manifested by the picture of acute renal failure - oligoanuria, arterial hypertension, edema, anemia, bilirubinemia.

Histuria is the appearance of organ-specific tissue proteins in the urine. It is observed in malignant neoplasms of various localization, tissue necrosis.

Secretory proteinuria - increased urinary excretion of Tamm-Horsfall protein secreted by the epithelium of the distal tubules (possibly with acute pyelonephritis, nephrolithiasis) or IgA and IgM secreted through the epithelium of the direct and convoluted distal tubules into the lumen of the latter (possibly with glomerulonephritis).

Functional proteinuria includes orthostatic proteinuria, stress proteinuria, and febrile proteinuria. It is observed in patients with healthy kidneys, low (up to 2 g / day), more often transient, rarely combined with erythrocyturia, cylindruria, leukocyturia.

Orthostatic (or lordotic) proteinuria appears when the child is in an upright position for a long time (when standing, walking) and disappears in a horizontal position. It is seen more often in teenagers. It is believed that orthostatic proteinuria is glomerular, non-selective, its level is usually not more than 1 g / day. To confirm the diagnosis, an orthostatic test is performed. Tension proteinuria occurs with increased physical activity and is explained by the relative ischemia of the proximal tubules during the redistribution of blood flow. The level of proteinuria does not exceed 131 1-2 g / day, is detected in the first portion of urine, disappears with normal exercise.

Feverish proteinuria can develop in children without kidney damage in diseases accompanied by hyperthermia. The appearance of proteinuria is explained by an increase in catabolic processes that occurs at a body temperature of more than 38 ° C, or a transient increase in the permeability of the glomerular filter due to damage by immune complexes. Other changes in the urinary sediment are usually not observed, proteinuria disappears when the temperature returns to normal.

Transient proteinuria is associated with hypothermia, hyperinsolation. Postrenal proteinuria, caused by pathology of the urinary tract and the ingress of inflammatory exudate rich in protein into the urine, is relatively rare in children, it is insignificant in size and is usually accompanied by leukocyturia and bacteriuria. There are also congestive proteinuria that occurs with cardiac decompensation, tumors of the abdominal cavity, etc., usually it is 1-3 g / day, and neurogenic proteinuria - with cerebral injury, meningeal hemorrhage. The last two varieties are combined by the term extrarenal proteinuria. It should be borne in mind that sedimentary protein tests can be false-positive in the presence of certain antibiotics, sulfonamides, and iodine contrast agents in the urine.

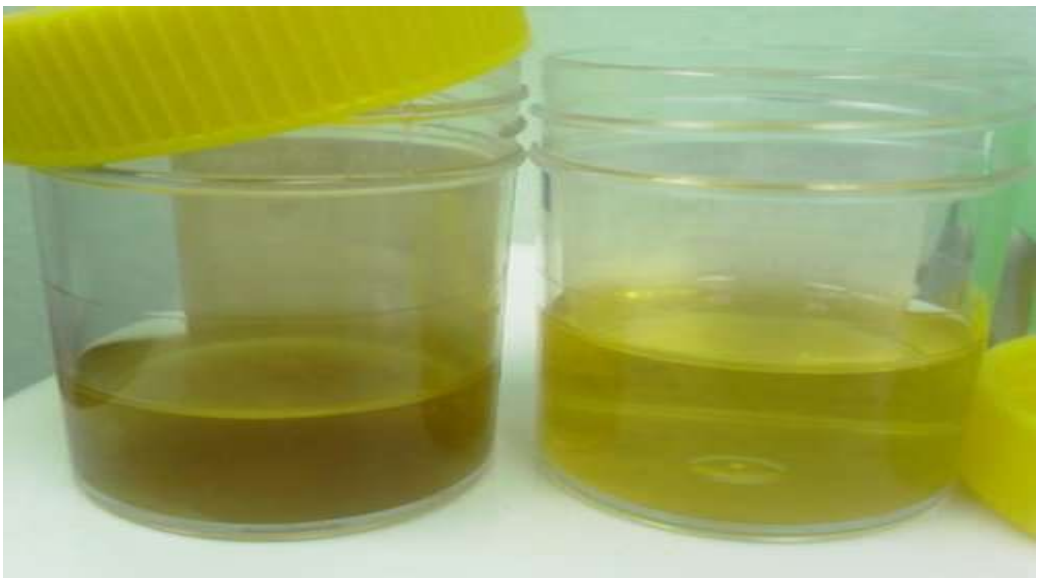


Figure No. 68. The color of urine with proteinuria

Proteinuria can be selective or non-selective.

Selective proteinuria is the penetration into the urine of proteins with only a low molecular weight (<65,000), for example, albumin and fractions close to it (with glomerulonephritis with minimal changes in the glomeruli).

Non-selective proteinuria is characterized by the appearance in the urine along with low molecular weight medium and high molecular weight proteins (α2-macroglobulin, γ-globulin, β-lipoproteins, etc.). The transition of selective proteinuria to non-selective indicates the progression of glomerulopathy, fibroplastic changes in the glomeruli are often determined. According to the severity, moderate proteinuria is distinguished, not exceeding 3 g / day, and high (massive) - more than 3 g / day. The detection of microalbuminuria during repeated urine tests in some cases can be an early sign of damage to the glomerular apparatus in glomerulonephritis, as well as the first sign of kidney transplant rejection. Low proteinuria (300 mg - 3 g / day) is characteristic of tubulopathy, obstructive uropathy, chronic interstitial nephritis, nephrolithiasis, polycystosis, kidney tumors.

Moderate proteinuria is characteristic of acute pyelonephritis, primary and secondary glomerulonephritis, proteinuric stage of amyloidosis, etc. High proteinuria is usually accompanied by the development of nephrotic syndrome (NS), which is characterized not only by proteinuria, but also by hypoproteinemia

(hypoalbuminemia), dysproteinemia, hypercholesterolemia, hyperlipidemia, severe edema. NS is observed in diseases of the kidneys proper (glomerulonephritis, microcystic kidney disease, primary amyloidosis, familial NS), may be incomplete (without edema). NS can also occur in diabetes mellitus, lymphogranulomatosis, secondary amyloidosis, periodic illness, tumors of various localization, and allergic diseases. A special group consists of diseases that occur with hemodynamic disorders (CHD, infective endocarditis, circulatory failure, constrictive pericarditis, renal vascular thrombosis). NS is characteristic of a number of poisonings (with salts of heavy metals, drugs (antiepileptics, bismuth, gold, D-penicillamine, vitamins, etc.), vaccines, sera), and also occurs in infections and parasitic diseases (tuberculosis, chronic active viral hepatitis, syphilis, malaria, etc.). With proteinuria, as a rule, cylindruria is also found. The matrix for the cylinders is the Tamm-Horsfall uroprotein, which coagulates in the lumen of the tubules, and aggregated serum proteins. The components distinguish hyaline, waxy and granular cylinders. The casts are usually of renal origin, rarely from the lower urinary tract. Proteinuria may be isolated or accompanied by changes in urinary sediment. Isolated proteinuria is characteristic of glomerulonephritis with minimal changes in the glomeruli, nephroptosis, de Toni-Debre-Fanconi disease, renal amyloidosis (in the latter case, sometimes in combination with microhematuria). Proteinuria in combination with hematuria occurs in primary and secondary glomerulonephritis, diabetic glomerulosclerosis. Proteinuria, together with neutrophilic leukocyturia, is typical for pyelonephritis, obstructive uropathy. Proteinuria with hematuria and mononuclear leukocyturia is observed in interstitial nephritis, dysmetabolic nephropathies, and kidney tuberculosis.

Hematuria. Hematuria syndrome is diagnosed when there are more than 3 erythrocytes in the field of view in the morning portion of urine, more than 1000 in 1 ml of urine 133 in the analysis of urine according to Nechiporenko (or more than 1,000,000 in daily urine according to Addis-Kakovsky). By intensity, micro- and macrohematuria are distinguished. In the presence of microhematuria, the color of the urine is not changed, erythrocytes are detected only with microscopy of the

urinary sediment (up to 100 per field of view). With gross hematuria, urine has a red or pink color, it can be transparent or cloudy (in the form of meat slops). Renal hematuria is explained by increased permeability of glomerular capillaries, instability of the glomerular membrane, renal intravascular coagulation, and damage to the interstitial tissue. A sign of glomerular erythrocyturia are characteristic changes in the membrane in the form of uneven thickening (up to 80% of erythrocytes). Hematuria can be regarded as physiological when a small number of erythrocytes appear in the urine after exercise (in athletes) or as orthostatic (if the morning portion of urine does not contain erythrocytes). With primary or secondary kidney damage, renal hematuria develops, with diseases of the lower urinary tract - postrenal hematuria.

Figure No. 69. Urine color with hematuria

Hematuria can be isolated or combined with proteinuria, leukocyturia, cylindruria. Bleeding in the area from the renal pelvis to the urethra leads to isolated hematuria without a noticeable increase in the content of protein, cells and casts in the urine. The most common causes of isolated hematuria are stones, trauma, tumors, IgA nephropathy, often tuberculosis, sickle cell anemia, and prostatitis. By nature, hematuria is divided into initial (the appearance of blood in the first portion of urine at the beginning of urination, which indicates damage to the urethra), terminal (hematuria at the end of the act of urination is characteristic of diseases of the bladder) and total (uniform distribution of red blood cells during

the entire act of urination indicates renal origin of hematuria). It should be remembered about the possibility of pseudohematuria, in which the red color of urine may be the result of the use of medications (rifampicin, nitrofurans), eating beets, and the presence of urates in the urine. Red urine may also be associated with hemoglobinuria and myoglobinuria.

Leukocyturia. A sign of leukocyturia is the presence in the analysis of urine more than 6-8 leukocytes in the field of view. As a rule, this is accompanied by an alkaline urine reaction. However, with conventional urine tests, it is not always possible to detect leukocyturia, therefore, in doubtful cases, the study is carried out by special methods, of which the Addis-Kakovsky or Nechiporenko tests are most widely used. For the Addis-Kakovsky test, daily urine is collected and the amount of formed elements in it is determined, taking into account its daily volume. Normally, the number of leukocytes does not exceed 2 million in 1 ml of urine, erythrocytes - 1 million, cylinders - 100 thousand. The method of studying urine according to Nechiporenko is practically more convenient, when an average portion of morning urine is taken for analysis and the number of formed elements is determined in it based on 1 ml; the sample is considered normal if there are up to 2 thousand leukocytes in 1 ml of urine, up to 1 thousand erythrocytes.

Leukocyturia is one of the main signs of urinary infection, including its latent course. However, both general urine tests and Addis-Kakovsky and Nechiporenko tests do not allow us to clarify the source of leukocyturia. Sometimes, to confirm pyelonephritis, urine is examined for "live", active leukocytes (Stenheimer-Malbin cells). Nevertheless, the diagnostic value of Stenheimer-Malbin cells is relative, since sometimes even with active pyelonephritis they are not found, because a certain osmotic pressure and osmotic resistance of leukocytes are important for the detection of these cells. To differentiate purulent inflammation from aseptic inflammation, which is characteristic of diseases such as glomerulonephritis, lupus nephritis, and others, a leukocytogram is determined (the centrifuged urine sediment is applied to a glass slide, stained with hematoxylin-eosin, and the leukocyte formula is calculated in

%). The predominance of neutrophils in the urine sediment is typical for bacterial, purulent inflammation. It should be noted that in girls and women, leukocyturia may not be associated with damage to the organs of the urinary system, this is the so-called false leukocyturia. It is caused by the presence of inflammatory lesions of the genital organs and adjacent areas of the skin.

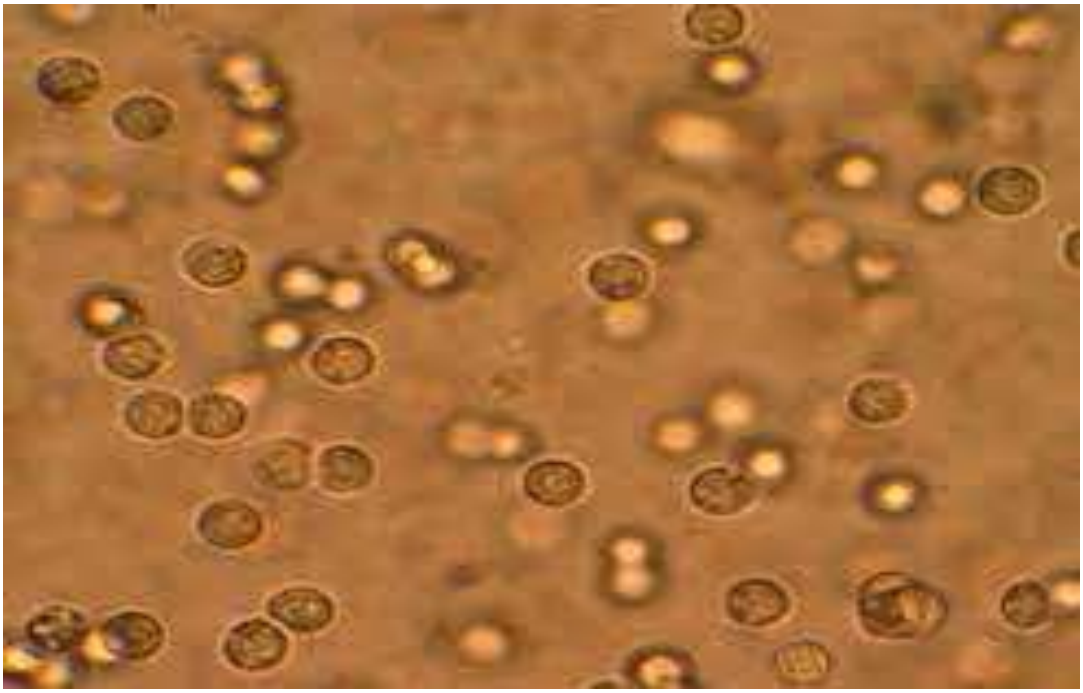


Figure No. 70. Leukocytes in urine under a microscope

That is why urine for analysis should be collected from the middle stream after a thorough toilet, or a two-glass test should be carried out: the predominance of leukocytes in the first portion may indicate false pyuria. Significant leukocyturia (pyuria) is a sign of an inflammatory process in the kidneys or urinary tract (kidney tuberculosis, pyelitis, cystitis, pyelonephritis, etc.). Leukocyturia in the microbial-inflammatory process in the urinary system is usually accompanied by bacteriuria.

Bacteriuria is considered true when at least 100,000 microbial bodies are found in 1 ml of urine during the study of an average portion of freshly passed urine with free urination after a thorough toilet of the external genital organs in a sterile dish, or at least 10,000 in 1 ml of urine obtained by catheterization.

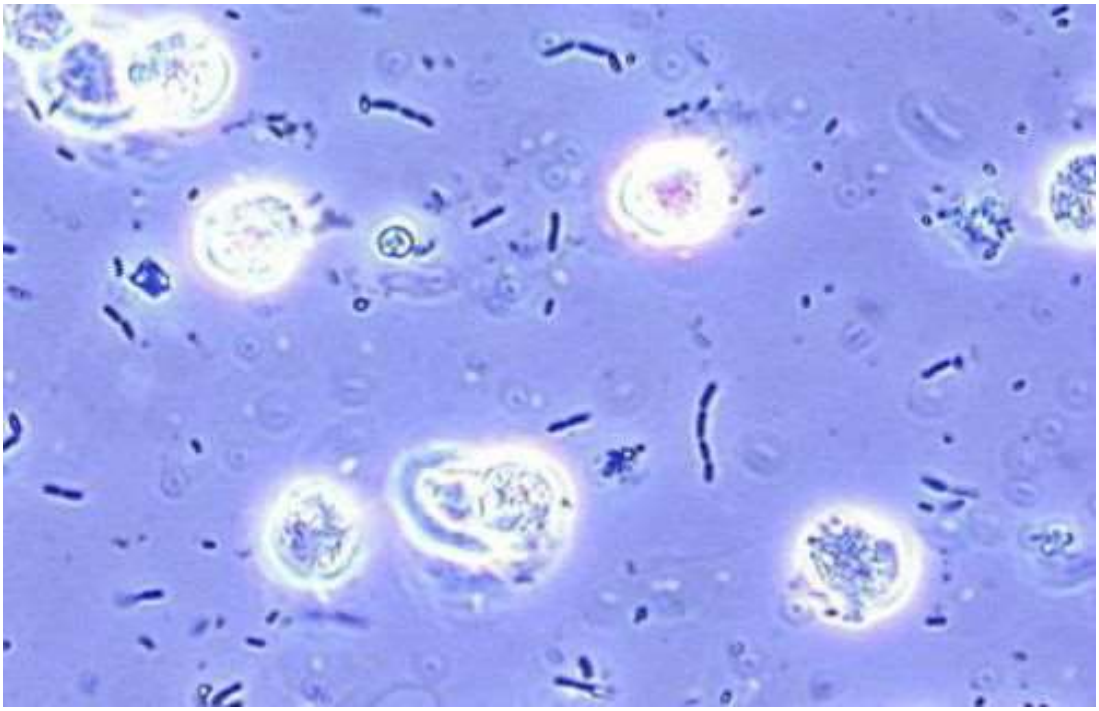


Figure No. 71. Bacteria in the urine.

SYNDROME OF RENAL INSUFFICIENCY.

The syndrome of renal insufficiency includes a set of clinical and laboratory-functional manifestations of a disorder of homeostatic functions characteristic of the kidney. The main manifestations of renal failure are increasing azotemia, dyselectrolytemia, metabolic acidosis, insufficient or, much less often, excessive water excretion. Renal failure can be partial and total. Partial renal failure refers to a persistently pronounced decrease in any kidney function (for example, acidogenesis). With total renal failure, disorders of all kidney functions are observed. It usually develops when only 20% of nephrons retain function. The course of renal failure is divided into acute and chronic. Acute renal failure. OPN is determined by the fact that the existing possibilities of glomerular filtration and tubular functions cannot provide the necessary removal of nitrogenous and other slags, including water. This leads to a profound disruption of water and electrolyte homeostasis. OPN can be observed in glomerulonephritis, hemolytic-uremic syndrome, renal necrosis, in various severe diseases (sepsis and other infections), as well as in the debut of acute glomerulo- and pyelonephritis. AKI occurs when an accidental transfusion of incompatible blood, poisoning with barbiturates, nephrotoxic poisons (compounds of mercury, lead) and antibiotics. Common

causes of AKI are non-renal causes. It is customary to single out this group of causes as "prerenal". They are all similar in one thing - in the occurrence of impaired blood supply to the kidneys, which is possible with any shock, poisoning, blood loss or general illness (HF), leading to a decrease in blood pressure or effective blood flow in the kidneys. From this moment begins already and own kidney damage. The main symptom of acute renal failure is oliguria, turning into anuria, which is accompanied by headache, anorexia, convulsions, thirst, nausea and vomiting. Body weight rapidly increases, peripheral edema appears. There are skin itching, refusal to eat, sleep disorders, stool disorders and abdominal pain are added. Respiration becomes acidotic, blood pressure may temporarily increase, then arterial hypotension and hemodynamic decompensation occur with pulmonary edema or cerebral edema with coma and convulsions. A blood test reveals azotemia, hyperkalemia, and hypocalcemia. With a benign course of acute renal failure, usually after 3-4 days, a polyuric phase occurs, in which a large amount of salts and nitrogenous slags are excreted in the urine. After that, the tubular function is restored to one degree or another.

Chronic renal failure. The distinction between acute and chronic renal failure is based on significant differences in both the clinical picture and the dynamics of impaired renal function. These criteria are: - decrease in endogenous creatinine clearance - 20 (ml / min) / 1.73 m2 or less; - an increase in serum creatinine more than 2 mg%, or 177 μmol / l for 3 months or more. More often, chronic renal failure develops gradually. Initially, its clinical manifestations are not observed, then patients develop moderate thirst and polyuria. Their manifestations can slowly increase, often they are accompanied by pallor associated with anemia and, often, increased blood pressure, nocturia, hypostenuria occur. Subsequently, the density of urine becomes equal to the density of blood plasma, electrolyte disturbances occur (hypokalemia, hyponatremia). Patients are stunted, significantly emaciated, they have general muscle weakness, drowsiness, headache, loss of appetite, dry mouth, weak muscle twitching (hypocalcemia), uremic breath. In the future, a critical picture of uremia sets in with loss of consciousness, significant

disorders in the activity of various body systems (cardiovascular, digestive, etc.) and metabolism. Currently, more and more importance in the development of the clinical picture of true uremia is given not to the level of residual nitrogen (retention of toxins in the body), but to disturbances in electrolyte metabolism and acid-base state.

NEPHROGENIC HYPERTENSION

Nephrogenic arterial hypertension is an increase in blood pressure due to a disease of the kidneys and renal vessels. Nephrogenic hypertension develops as a result of an increase in renin synthesis and a decrease in the activity of angiotensinase, prostaglandin and kallikrein-kinin systems.

Nephrogenic hypertension is divided into three main groups: **1) parenchymal, resulting f**rom unilateral or bilateral lesions of the renal parenchyma of a diffuse nature, observed in glomerulo- and pyelonephritis, kidney tuberculosis, hydronephrosis, polycystic kidney disease, diabetic glomerulosclerosis, nephropathy of pregnant women, systemic connective tissue diseases , amyloidosis;

2) vasorenal, caused by narrowing of the renal vessels due to atherosclerotic stenosis of the renal artery, fibromuscular dysplasia of the renal artery, thrombosis, embolism and aneurysm of the renal artery, anomalies in the development of the renal vessels and aorta;

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2) vasorenal, caused by narrowing of the renal vessels due to atherosclerotic stenosis of the renal artery, fibromuscular dysplasia of the renal artery, thrombosis, embolism and aneurysm of the renal artery, anomalies in the development of the renal vessels and aorta;

3) mixed - the result of damage to the renal parenchyma and changes in the renal vessels with nephroptosis, tumors, kidney cysts, combinations of anomalies of the kidneys and their vessels. The clinical picture in nephrogenic arterial hypertension consists of symptoms characteristic of hypertension and symptoms of kidney damage. Nephrogenic hypertension has a slow (benign) and fast (malignant) form. In benign hypertension, blood pressure is usually stable and does not tend to decrease. Both diastolic and systolic pressure are increased, but more significantly - diastolic. Patients complain of recurrent headaches, dizziness, weakness, fatigue, palpitations, shortness of breath, and discomfort in the region of the heart. The malignant form of hypertension is characterized by an increase in diastolic pressure above 120 mm Hg. Art., sudden and rapidly progressive visual impairment due to the development of retinopathy, patients complain of persistent headaches, often in the back of the head, dizziness, nausea, vomiting. With nephrogenic hypertension, in contrast to hypertension, back pain often occurs - both due to circulatory disorders in the kidney and as a result of the underlying urological disease. It is very important to carefully collect anamnesis, on the basis of which it is possible to suspect the symptomatic nature of hypertension.

For nephrogenic hypertension are characteristic:

- sudden onset; - the appearance of hypertension after acute pain in the lower back, past diseases and injuries of the kidneys, surgical intervention on the kidney;
- the emergence and rapid progression of hypertension in young people;
- malignant course of the disease;
- ineffectiveness of standard antihypertensive therapy;
- lack of hereditary predisposition to hypertension.- the result of damage to

the renal parenchyma and changes in the renal vessels with nephroptosis, tumors, kidney cysts, combinations of anomalies of the kidneys and their vessels. The clinical picture in nephrogenic arterial hypertension consists of symptoms characteristic of hypertension and symptoms of kidney damage. Nephrogenic hypertension has a slow (benign) and fast (malignant) form. In benign hypertension, blood pressure is usually stable and does not tend to decrease. Both diastolic and systolic pressure are increased, but more significantly - diastolic. Patients complain of recurrent headaches, dizziness, weakness, fatigue, palpitations, shortness of breath, and discomfort in the region of the heart. The malignant form of hypertension is characterized by an increase in diastolic pressure above 120 mm Hg. Art., sudden and rapidly progressive visual impairment due to the development of retinopathy, patients complain of persistent headaches, often in the back of the head, dizziness, nausea, vomiting. With nephrogenic hypertension, in contrast to hypertension, back pain often occurs - both due to circulatory disorders in the kidney and as a result of the underlying urological disease. It is very important to carefully collect anamnesis, on the basis of which it is possible to suspect the symptomatic nature of hypertension. For nephrogenic hypertension are characteristic: - sudden onset; - the appearance of hypertension after acute pain in the lower back, past diseases and injuries of the kidneys, surgical intervention on the kidney; - the emergence and rapid progression of hypertension in young people; - malignant course of the disease; - ineffectiveness of standard antihypertensive therapy; - lack of hereditary predisposition to hypertension.

SITUATIONAL TASKS FOR INDEPENDENT TRAINING

Task №1

Patient M., 11 years old, complains of constant dull, aching pain in the right hypochondrium 1-1.5 hours after eating, especially fatty food and after exercise. Sick for the last 1.5-2 months. In addition, he complains of weakness, fatigue, nausea. On examination, the child's condition is satisfactory. The skin is pale, the mucous membranes are pink, the tongue is slightly lined. On palpation of the abdomen, pain in the right hypochondrium is noted. The occurrence and intensification of pain during inspiration during palpation at the point of the gallbladder is noted. Positive symptoms of Ortner, Kera, Murphy are noted. There is no pain on the rest of the abdomen. The chair is issued, the liquid chair is sometimes noted. Your presumptive diagnosis. What research is needed to confirm the diagnosis.

Task №2

An 11-year-old patient was admitted to the gastroenterological department with complaints of loss of appetite, nausea, pain in the epigastric region with irradiation to the right hypochondrium, especially aggravated especially after taking fried, fatty, spicy, salty foods, as well as after nervous tension. And also there is a bitter taste in the mouth, belching. On examination, the child's condition is satisfactory. The skin is pale, dry. The mucous membranes are clean, pale pink in color, the yalyk is slightly lined with white bloom. On palpation of the abdomen, pain is noted, more pronounced in the epigastrium. Symptoms of Murphy, Ortner, Vasilenko are positive. The liver at the edge of the costal arch, painless. Stool without pathological impurities. Urinating freely, painlessly. Urine is light. 1. What is your presumptive diagnosis? 2. Assign a plan of examination and treatment.

Task № 3

A 10-year-old patient was admitted to the gastroenterological department with complaints of loss of appetite, nausea, bitter taste in the mouth, belching, and pain in the right hypochondrium, aggravated after nervous tension, after taking fatty, fried, spicy, salty foods. On examination, the child's condition is satisfactory. Undernourished girl. The skin is pale, dryish. The mucous membranes are clean, pale pink in color, the tongue is lined with a white coating at the root. On palpation of the abdomen, pain is noted, more pronounced in the right hypochondrium. The liver at the edge of the costal arch, painless. The spleen is not enlarged. Symptoms of Mussi, Kera, Ortner, Vasilenko are positive. Stool without pathological impurities, dark brown, dense feces. Urinating freely, painlessly. Urine is light. 1. What is your presumptive diagnosis? 2. Assign a plan of examination and treatment.

Task №4

Patient K., 11 years old. He was admitted to the clinic with complaints of general weakness, fatigue, weight loss. History: underwent surgery for appendicitis at the age of 5. At the age of 4, viral hepatitis B. Did not follow the diet. There were no restrictions on physical activity. Deterioration is not associated with anything.

Objectively: reduced nutrition. FV-15kg. The skin is gray-earthy in color. The sclera are icteric.+ Peripheral nodes are not enlarged. The tongue is covered with a dirty gray coating. The abdomen is soft, palpation moderate pain in the projection of the gallbladder. Liver + 1 cm, the edge is pointed, the surface is smooth. The spleen is not enlarged.

1. What is your presumptive diagnosis?
2. What research needs to be done?
3. Give treatment (with doses).

Task №5

The patient is 12 years old. He was admitted with complaints of weakness, fatigue, loss of appetite, nausea, pain in the right hypochondrium, especially after eating fatty and fried foods. In the anamnesis - a child at the age of 11 suffered from viral hepatitis B, was treated in a hospital. On examination, the child's condition was moderate. Painful facial expression. Tongue coated with white coating at the root. The skin is dry, pale, the sclera is icteric +. The abdomen is painful on palpation, in the epigastrium with irradiation to the right hypochondrium. Liver + 0.5 cm, slightly painful, smooth surface. Chair once a day decorated. In the blood test, ALT - 0.8 mmol / l, AST - 0.5 mmol / l, total bilirubin - 34 μmol / l, direct - 8 μmol / l, indirect - 26 μmol / l.

Your presumptive diagnosis. Plan of examination and treatment.

TESTS

1. Melena happens when:

1) viral hepatitis

2) duodenal ulcer

3) cholecystitis

4) chronic colitis

5) catarrhal esophagitis

6) erosive gastritis

7) diverticulum of the ileum (Meckel's diverticulum)

2. Soreness in the pathology of the pancreas is observed at the point:

1) Mayo-Robson

2) Kacha

3) Kera

4) Desjardins

5) Ortner

3. In the pathology of the gallbladder, positive symptoms are observed:

1) Murphy

2) Kera

3) Ortner

4) Frenicus symptom

5) Trousseau

6) tail

7) Mendel

4. Creatorrhoea occurs with enzymatic deficiency:

1) stomach

2) liver

3) small intestine

4) large intestine

5) pancreas

5. Neutral fat in feces occurs with enzymatic deficiency:

1) stomach

2) liver

3) pancreas

4) small intestine

5) large intestine

6. Fatty acids in feces occur in pathology:

1) stomach

2) liver

3) small intestine

4) large intestine

5) pancreas

7. Creatorrhea is:

1) neutral fat in feces

2) fatty acids in feces

3) muscle fibers in feces

4) grains of starch in feces

5) mucus in feces

8. Signs of chronic constipation are:

1) difficulty defecation

2) lack of satisfaction after defecation

3) stool weight < 35 g per day

4) stool weight < 100 g per day

5) rare bowel movements (after 36 hours)

6) rare bowel movements (after 48 hours or more)

9. The syndrome of "intestinal dyspepsia" manifests itself:

1) diarrhea

2) constipation

3) flatulence

4) rumbling

5) nausea

6) heartburn

7) vomiting

10. Polyfecalia is characteristic for:

1) disaccharidase deficiency

2) celiac disease

3) chronic pancreatitis

4) hepatitis

5) peptic ulcer

11. Pain in the left hypochondrium is observed when:

1) damage to the stomach

2) diseases of the pancreas

3) lesions of the left (splenic) curvature of the colon

4) diseases of the diaphragm

5) liver diseases

6) lesions of the mesenteric (mesenteric) lymph nodes

12. Pain in the umbilical region is characteristic of diseases:

1) 12 duodenal ulcer

2) small intestine

3) mesenteric lymph nodes

4) oil seal

5) liver

6) esophagus

7) pancreas

13. Pain in the right iliac region is characteristic of all diseases, except:

1) appendicitis

2) esophagitis

3) pancreatitis

4) tiflita

5) Crohn's disease

6) sigmoiditis

14. Pathological increase in appetite can be observed in the following diseases, except:

1) thyrotoxicosis

2) diabetes

3) duodenal ulcer

4) chronic atrophic gastritis

5) chronic colitis

6) erosive esophagitis

SYMPTOMS AND SYNDROMES OF THE CARDIOVASCULAR SYSTEM

1. What are the characteristics of Fallot's tetrad?

1) lag in physical development

2) cyanosis

3) right ventricular hypertrophy

4) dextroposition of the aorta

5) left ventricular hypertrophy

2. What signs are typical for mitral stenosis?

1) left atrial enlargement

2) an increase in the amplitude of the R wave in lead V5-V6

3) diastolic murmur at the apex

4) "clapping" I tone at the top of the heart

3. What clinical signs are typical for children with aortic coarctation?

1) differences in the physical development of the upper and lower half of the body

2) the presence of pulsation of blood vessels in the lower extremities

3) increased blood pressure in the upper limbs

4) listening to systolic murmur in the interscapular region

5) lowering blood pressure in the legs

4. Specify the complaints typical for an older child with aortic coarctation:

1) headache

2) dizziness

3) weakness in the legs

4) tendency to frequent respiratory diseases

5) nosebleeds

5. List the signs characteristic of mitral valve insufficiency:

1) enhanced apex beat

2) the border of relative cardiac dullness is extended to the left

3) the noise increases in the position of the child on the left side

4) weakened II tone on the pulmonary artery

5) increased amplitude of the R wave in leads V5-V6

6. What congenital heart defects in children are accompanied by blood shunt from left to right?

1) ventricular septal defect

2) coarctation of the aorta

3) atrial septal defect

4) open ductus arteriosus

5) all of the listed vices

7. List the complaints characteristic of Fallot's tetrad:

1) shortness of breath

2) papular rash on the skin

3) cyanosis

4) lag in physical development

5) cyanotic-dyspnea attacks

8. The best place to listen to systolic murmur with a high ventricular septal defect is:

1) apex of the heart

2) second intercostal space on the right side of the sternum

3) second intercostal space to the left of the sternum

4) IV intercostal space at the left edge of the sternum

5) base of the xiphoid process

9. What kind of heart murmur is typical for patent ductus arteriosus?

1) prolonged systolic murmur at the apex of the heart

2) diastolic murmur at the apex of the heart

3) systolic murmur in the II intercostal space to the right of the sternum

4) systolic-diastolic murmur in the II intercostal space at the left edge of the sternum

5) diastolic murmur in the II intercostal space to the right of the sternum

10. Select the signs characteristic of vagotonia:

1) lowering blood pressure

2) bradycardia

3) increase in blood pressure

4) sweating

5) fainting

11. Plain radiographs e chest left contour of the heart is formed:

1) aortic arch

2) pulmonary artery

3) left atrium

4) left ventricle

5) right ventricle

12. Which of the following is typical for acute right ventricular failure?

1) liver enlargement

2) general edema

3) pulmonary edema

4) increase in blood pressure

5) oliguria

13. The ductus botulinum is obliterated at ... a month of extrauterine life:

1) first

2) second

3) fourth

4) fifth

5) sixth

14. What signs are typical for mitral stenosis?

1) cyanotic flush of cheeks

2) an increase in the amplitude of the R wave in lead V5-V6

3) mitral click

4) on palpation, presystolic trembling ("cat's purr")

5) clapping I tone

15. What signs are characteristic of functional noise:

1) do not extend beyond the heart

2) short in duration

3) soft in timbre

4) extend beyond the region of the heart

5) decrease under load

16. Organic noise differs from functional noise in the following features:

1) rough, "blowing" in timbre

2) occupy most of the systole in duration

3) spread well throughout the region of the heart and beyond

4) decrease when vertical

5) increase with physical activity

17. Specify the best place to listen to systolic murmur in atrial septal defect:

1) apex of the heart

2) II intercostal space to the right of the sternum

3) xiphoid process

4) II intercostal space to the left of the sternum

5) interscapular region

18. List the defects in which there is hypertrophy of the left ventricle:

1) mitral valve insufficiency

2) stenosis of the aortic mouth

3) aortic valve insufficiency

4) pulmonary artery stenosis

5) Fallot's tetrad

SKIN AND SUBSKIN SYMPTOMS

2. Vasculitis-purple type of rash is typical for:

1) hemophilia

2) hemorrhagic vasculitis

3) thrombocytopathy

4) thrombocytopenia

5) Rendu-Osler disease

3. Dysuric syndrome includes:

1) frequent urination

2) slow urination

3) pain in the lumbar region

4) pain when urinating

5) burning sensation when urinating

2. Pyuria syndrome is:

1) leukocytes in the urine

2) red blood cells in the urine

3) white blood cells and bacteria in the urine

4) red blood cells and bacteria in the urine

5) protein in the urine

3. Oliguria is:

1) an increase in the daily amount of urine

2) decrease in the daily amount of urine

3) no urine output

4) daytime diuresis is higher than nighttime

5) nocturnal diuresis is higher than daytime

4. Acute renal failure is characterized by:

1) azotemia

2) hyperkalemia

3) oliguria

4) metabolic acidosis

5) vomiting

5. When palpation of the abdomen in children, you can feel the kidneys:

1) more often right

2) in young children

3) in children with reduced nutrition

4) in overnourished children

5) in normosthenics

6. Pollakiuria is:

1) infrequent urination

2) frequent urination

3) painful urination

4) urinary incontinence

5) difficulty urinating

7. The transparency of urine is disturbed by excessive content in the urine:

1) salts

2) cellular elements

3) slime

4) fat

5) bilirubin

8. Urine of the color of meat slops in children is observed with:

1) gross hematuria

2) pyuria

3) the presence of salts

4) microhematuria

5) glucosuria

9. Red urine in children is observed in the following pathological conditions:

1) nephrolithiasis

2) pyelonephritis

3) kidney injury

4) kidney infarction

5) glomerulonephritis

10. Brown urine is observed when:

1) bilirubinuria

2) methemoglobinuria

3) porphyrinuria

4) proteinuria

5) in healthy children under 3 years of age

11. Urine becomes alkaline when:

1) her prolonged standing

2) inflammatory processes of the urinary tract caused by gram-positive flora

3) the predominance of plant foods in the diet

4) the predominance of meat food in the diet

5) hypokalemia

12. Physiological hypo- and isosthenuria is observed in:

1) newborns and children of the first year of life

2) children from 1 to 3 years old

3) children from 3 to 7 years old

4) children from 7 to 10 years old

5) children after 10 years

13. Daily excretion of protein in the urine is equal to:

1) 30-60 mg / day.

2) 100-150 mg / day.

3) 310-30 mg / day.

4) 100-200 mg / day.

5) 200-300 mg / day.

14. Oliguria is observed with:

1) insufficient fluid intake

2) feverish conditions

3) vomiting, diarrhea

4) cardiovascular and acute renal failure during the period of edema formation

5) impaired iron absorption

15. Facies nephritica is:

1) dark circles under the eyes

2) "purple glasses" around the eyes

3) erythema on the face in the form of a "butterfly"

4) pallor of the face

5) puffiness of the face

17. **The main symptoms of acute renal failure are:**

1) oliguria, turning into anuria

2) headache

3) anorexia

4) convulsions

5) vomiting

18. Chronic renal failure manifests itself:

6) anemia

7) increased blood pressure

8) nocturia

9) hypostenuria

10) none of the above

19. Nephritic variant of edematous syndrome includes:

1. weight gain

2. swelling (pastosity) under the eyes

3. moderate swelling of the legs

4. anasarca

5. hydrothorax

20. Nephrotic variant of edematous syndrome includes:

1. swelling (pastosity) under the eyes

2. ascites

3. hydrothorax

4. severe swelling of the face, limbs

5. swelling of the anterior abdominal wall

21. Oliguria is the amount of urine in a child:

1. under the age of 1 year less than 1 ml / kg / h

2. under the age of 1 year less than 0.5 ml/kg/h

3. older than 1 year less than 0.5ml/kg/h

4. older than 1 year less than 0.3ml/kg/h

5. under the age of 1 year less than 4 ml / kg / h

22. The criteria for respiratory failure are as follows:

1) cyanosis

2) shortness of breath

3) violation of the rhythm of breathing

4) tachycardia

5) cough

23. Surfactant is produced by:

1) mast cells

2) lymphocytes

3) goblet cells

4) macrophages

5) type II alveolocytes

24. Bronchial breathing is heard when:

1) segmental and lobar pneumonia

2) atelectasis

3) significant bronchospasm

4) bronchitis

5) massive tuberculous infiltration

6) foreign body of the bronchus

25. Crepitus is formed in:

1) larynx

2) trachea

3) bronchi

4) bronchioles

5) pleural cavity

6) alveoli

26. Types of shortness of breath in children include:

1) inspiratory

2) expiratory

3) mixed

4) superficial

5) Shika's shortness of breath

27. Crepitus is heard in the following diseases:

1) lobar pneumonia

2) exudative pleurisy

3) pulmonary edema

4) focal pneumonia

5) the initial stage of tuberculous infiltration of the lungs

6) congestion in the lungs

28. Displacement of the mediastinum in the direction of the lesion can be observed with:

1) hemothorax

2) pneumothorax

3) hydropneumothorax

4) lobar emphysema

5) lung atelectasis

29. True croup is observed in:

1) diphtheria of the larynx

2) acute respiratory viral diseases

3) bronchitis

4) pneumonia

5) pleurisy

30. False croup most often occurs when:

1) diphtheria of the larynx

2) acute respiratory viral diseases

3) bronchitis

4) pneumonia

5) pleurisy

31. The maxillary sinuses in children are well developed:

1) at birth

2) by 1 year

3) by 7 years

4) by 2 years

5) by 6 months

32. Harsh breathing is heard when:

1) atelectasis

2) significant bronchospasm

3) exudative pleurisy

4) bronchitis

5) bronchopneumonia

6) pneumothorax

33. Weakened breathing is heard when:

1) bronchitis

2) laryngitis

3) pharyngitis

4) exudative pleurisy

5) rhinitis

6) significant bronchospasm

7) pneumothorax

34. Wet rales are formed in:

1) larynx

2) trachea

3) bronchi

4) alveoli

5) pleural cavity

35. Complete cessation of mobility of the lower edge of the lungs occurs when:

1) emphysema

2) flatulence

3) chronic pneumonia

4) exudative pleurisy

5) liver enlargement

36. Tympanic shade of percussion sound is noted in the following cases:

1) emphysema

2) bronchial asthma

3) obstructive bronchitis

4) focal pneumonia

5) pneumothorax

37. The nature of shortness of breath in pneumonia:

1) inspiratory

2) expiratory

3) mixed

4) Shika's shortness of breath

38. Shortening of percussion sound is observed in the following diseases:

1) pneumonia

2) atelectasis

3) pleurisy

4) bronchiolitis

5) primary tuberculosis complex

39. Weakened breathing is observed in the following conditions:

1) paresis of the respiratory muscles

2) foreign body of the bronchus

3) significant bronchospasm

4) exudative pleurisy

5) emphysema

6) lobar pneumonia

7) obesity

40. A hoarse cough is observed in the following diseases:

1) laryngitis

2) diphtheria of the larynx

3) lobar pneumonia

4) swelling of the larynx

5) laryngospasm

6) papilloma of the larynx

41. Spasmodic cough is observed in the following diseases:

1) cardiomegaly

2) whooping cough

3) foreign body

4) obstructive bronchitis

5) cystic fibrosis

42. Bitonal cough is observed in the following diseases:

1) tuberculous bronchoadenitis

2) laryngitis

3) mediastinal lymphosarcoma

4) mediastinal pleurisy

5) cardiomegaly

43. Voice trembling is weakened in the following diseases:

1) exudative pleurisy

2) hydrothorax

3) hemothorax

4) severe pneumosclerosis

5) pneumothorax

6) massive lung atelectasis

44. Rigidity of the chest increases with the following diseases:

1) ossification of the cartilage of the ribs

2) extensive pneumosclerosis

3) emphysema

4) exudative pleurisy

5) bronchitis

45. Lung volumes are measured using:

1) spirometer

2) spirograph

3) pneumotachometer

4) peak flowmeter

5) oximeter

46. Obstructive type of ventilation disorders is observed in the following diseases:

1) foreign body of the bronchus

2) bronchiolitis

3) obstructive bronchitis

4) exudative pleurisy

5) bronchial asthma

6) stenosing laryngitis

47. During an attack of bronchial asthma percussion sound over the lungs:

1) clear, pulmonary

2) dumb

3) tympanic

4) boxed

5) metal

48. Restriction of lung mobility is typical for the following diseases:

1) emphysema

2) pneumosclerosis

3) exudative pleurisy

4) lobar pneumonia

5) focal pneumonia

6) hydrothorax

7) adhesive pleurisy

49. Strengthening of bronchophony is noted in the following diseases:

1) polysegmental pneumonia

2) compression atelectasis

3) large bronchiectasis

4) large lung abscess

5) lobar pneumonia

50. On the basis of what test can the most correct diagnosis of respiratory failure be made?

1) history

2) physical examination of the patient

3) X-ray examination of the chest

4) spirographic study

5) studies of arterial blood gases (ABG)

51. Which of the following clinical signs are typical for exudative pleurisy?

1) pain when bending over to the healthy side

2) displacement of the mediastinal organs in a healthy direction

3) box sound on percussion

4) vesicular breathing on auscultation

5) wet cough

6) dullness of percussion sound

7) weakening of breathing

52. A child has shortness of breath, dry wheezing. What is the localization of the lesion in the respiratory tract in this situation?

1) large bronchi

2) middle bronchi

3) small bronchi

4) pleura

5) alveoli

6) larynx

53. Paradoxical breathing is most often observed in:

1) pneumothorax

2) emphysema

3) laryngospasm

4) pneumonia

5) atelectasis

53. Paradoxical breathing is most often observed in:

1) pneumothorax

2) emphysema

3) laryngospasm

4) pneumonia

5) atelectasis

54. Mediastinal shift to the healthy side can be observed in:

1) hemothorax

2) pneumothorax

3) lung atelectasis

4) hydropneumothorax

5) lobar emphysema

55. Increased voice trembling is noted with:

1) exudative pleurisy

2) severe pneumofibrosis

3) large cavity

4) hydrothorax

5) lung abscess

56. Expiratory dyspnea is observed in:

pneumonia

bronchial asthma

pleurisy

bronchiolitis

obstructive bronchitis

laryngotracheitis

57. Inspiratory dyspnea is observed when:

1) true groats

2) bronchitis

3) retropharyngeal abscess

4) laryngitis with stenosis of the larynx

5) a foreign body in the larynx or trachea

6) pneumonia

58. The state of orthopnea is noted in the following respiratory diseases:

1) pleurisy

2) an attack of bronchial asthma

3) pneumonia

4) Hamman-Rich syndrome

5) laryngotracheitis

6) bronchiolitis

59. Reducing the volume of one half of the chest can be observed with:

1) pneumonia

2) extensive pneumosclerosis

3) complete closure of the pleural fissure

4) lung atelectasis

5) exudative pleurisy

6) obstructive bronchitis

60. Rubbing noise of the pleura:

1) heard on exhalation

2) heard on inspiration

3) heard on inhalation and exhalation

4) disappears after coughing

5) does not disappear after coughing

6) increases with pressure with a stethoscope

7) weakens when pressed with a stethoscope

61. Wet rales:

1) heard on inspiration

2) heard on exhalation

3) heard on inhalation and exhalation

4) do not change localization after coughing

5) after coughing, change localization

6) after coughing can intensify

7) may disappear after coughing

62. The displacement of the upper border of the lungs upward and the increase in the Krenig fields is noted with:

1) pneumonia

2) bronchitis

3) emphysema

4) pneumosclerosis

5) wrinkling of the tops of the lungs

6) an attack of bronchial asthma

63. Low standing of the diaphragm is noted when:

1) emphysema

2) focal pneumonia

3) tension pneumothorax

4) atelectasis of the lungs

5) massive pleural effusion

64. High standing of the diaphragm is noted when:

1) emphysema

2) paresis of the diaphragm

3) atelectasis of the lungs

4) pleural effusion

5) hypoplasia of the lung lobes

65. Shortening of percussion sound and bronchial breathing is observed in:

1) focal pneumonia

2) segmental and polysegmental pneumonia

3) atelectasis

4) in areas of the lung above the pleural effusion

5) bronchial asthma

66. Shortening of percussion sound and weakening or disappearance of respiratory sounds over any part of the lung occurs when:

1) atelectasis

2) bronchial asthma

3) obstructive bronchitis

4) exudative pleurisy

5) hydrothorax

67. The reasons for the development of Kussmaul breathing can be:

1) end-stage heart failure

2) diabetic coma

3) end-stage renal failure

4) severe acetonemic vomiting

5) brain damage of a different nature

68. The reasons for the development of Cheyne-Stokes respiration can be:

1) brain tumor

2) end-stage heart failure

3) diabetic coma

4) end-stage renal failure

5) severe acidosis

6) meningitis or encephalitis

SYMPTOMS AND SYNDROMES OF THE DAMAGE OF THE musculoskeletal system

2. With rickets, the following changes in the bones of the skull are observed:

1) osteomalacia

2) craniotabes

3) pronounced frontal tubercles

4) pronounced parietal tubercles

5) Olympic forehead

3. Decreased muscle tone is manifested in:

1) belly enlargement

2) pronounced lordosis of the lumbar spine

3) pterygoid scapulae

4) violation of posture

5) athetotic position of the hands

4. Signs of hypertonicity of the leg flexors in children during the first months of life are:

1) some limitation of movements in the joints

2) hip abduction less than 75 degrees to each side

3) extension of the leg at the knee joint with the thigh bent at a right angle less than 150 degrees

4) hip abduction 75 degrees to each side

5) all of the above

5. Hypokinesia is a factor:

1) reducing the level of children's health

2) contributing to the development of obesity

3) leading to weight loss

4) contributing to the development of autonomic dysfunction

5) improving the health of children

6. A feature of the children's skeleton is:

1) relatively large thickness of the periosteum

2) large volumes of intraosseous spaces

3) high functional activity of the periosteum

4) intensive blood supply

5) small volumes of intraosseous spaces

7. At what age does growth lag become most noticeable in case of pituitary dwarfism?

1) during the neonatal period

2) in 2-3 years

3) at 4-5 years old

4) at 6-8 years old

5) in puberty

8. Congenital primary hypothyroidism is characterized by:

1) low birth weight

2) prolonged jaundice

3) low timbre of voice

4) increase in the level of TSH in the blood

5) persistent constipation

9. With thyrotoxicosis in a child, you can find:

1) irritability

2) constipation

3) weight loss

4) tachycardia

5) tremor

10. Parathormone is an antagonist of what hormone?

1) TSH

2) thyrocalcitonin

3) thyroxine

4) cortisol

5) everything is wrong

11. Almost all effects of somatotropic hormone (GH) on tissues are mediated by:

1) thyrocalcitonin

2) insulin-like growth factors and their transport proteins

3) parathyroid hormone

4) adrenaline

5) receptors

12. Biological effects of thyrotropic hormone:

1) stimulates the growth of the thyroid gland

2) does not affect the growth of the thyroid gland

3) stimulates the secretory function of the thyroid gland

4) stimulates the accumulation of iodine by the thyroid gland

5) stimulates the secretory function of the parathyroid gland

13. Diseases caused by excessive secretion of pituitary hormones include:

1) Itsenko-Cushing's disease

2) true (central) precocious puberty

3) gigantism

4) nanism

5) hypopituitarism

14. Diseases caused by insufficient secretion of pituitary hormones include:

1) Itsenko-Cushing's disease

2) central hypothyroidism

3) gigantism

4) nanism

5) hypopituitarism

15. Waterhouse-Frideriksen syndrome is caused by:

1) toxic damage to the adrenal medulla

2) an infectious process in the adrenal glands

3) hemorrhage in the adrenal glands

4) the presence of petrificates in the adrenal glands

5) all of the above

16. Chronic adrenal insufficiency is characterized by:

1) weight loss

2) hypokalemia

3) muscle weakness

4) arterial hypotension

5) hyperpigmentation

17. Diabetes is characterized by:

1) hyperglycemia

2) glycosuria

3) hypoglycemia

4) polyuria

5) polydipsia

18. With premature sexual development, puberty occurs at the age of:

1) in girls and boys under 11 years old

2) in girls under 10 years old, in boys up to 11 years old

3) in girls up to 9 years old, in boys up to 10 years old

4) in girls and boys up to 8 years

5) in girls up to 8 years old, in boys up to 9 years old

19. Which of the following is a physiological feature of a newborn:

1) low digestive capacity of phagocytic cells

2) low activity of the complement system

3) low ability to produce interferon

4) low level of immunoglobulin synthesis

5) high level of immunoglobulin synthesis

GLOSSARY

An abscess is a delimited purulent-inflammatory process of fatty tissue.

Agglutinins, hemagglutinins are protein antibodies that promote the connection (gluing) of corpuscular particles in the body to each other. They are present in the blood plasma and determine the belonging of the blood to a particular group.

Agenesis (underdevelopment) of the corpus callosum is a malformation of the brain, characterized by the complete or partial absence of the corpus callosum, which connects the 4 lobes of the brain. The reasons can be different - genetics, ecology. Agenesis of the corpus callosum can lead to serious problems in the development of the child, however, in some cases, with a mild degree of agenesis, the child may develop normally. Present in 0.05-0.7% of the population.

Agranulocytosis (aleukia, neutrophilia, granulophthisis) is a sharp decrease in the number of leukocytes, leading to increased susceptibility to bacterial and fungal infections.

Platelet aggregation - connection of platelets with collagen (connective tissue protein). The process is extremely important for blood clotting, wound healing.

Adenoiditis is an inflammation of the adenoids, a glandular tissue located at the back of the throat, above the soft palate. In this case, there is an increase in the volume of the tonsil-shaped glands of the pharynx.

Adenoids - growths of the nasopharyngeal tonsil, located in the upper part of the pharynx. When inflammation causes numerous complications in children: otitis media, laryngitis, sinusitis, pharyngitis, rhinitis, bronchitis. In this case, the child often has difficulty in nasal breathing, he breathes through his mouth. There is a long, untreated runny nose.

Adenoma is a benign epithelial tumor, the cells of which form glandular-like structures.

Adenopathy is a disease of the lymph nodes of inflammatory, infectious or tumor origin.

Adipocide is a cell whose main role is to store fat in the body. Adipocytes form adipose tissue.

Accommodation of the eye is an adaptation of vision, a change in the curvature of the lens of the eye under the action of the ciliary muscle, which makes it possible to obtain a clear image on the retina.

Acromegaly is a disease characterized by morphological changes, namely: hypertrophy of the arms, legs and head, associated with disorders of the heart.

Actinomycosis is a chronic specific inflammatory disease with lesions of various tissue structures and organs, characterized by an undulating long course.

Alkaptonuria - insufficiency of homogentisic acid oxidase; tissue pigmentation and arthrosis are characteristic.

An allergen is any substance that can cause an allergy in a person predisposed to it.

Allergy is a state of the human body, manifested in the painful intolerance of any substances, the appearance of an allergic reaction. The physiological

mechanism of an allergic reaction is associated with the formation of specific antibodies, which leads to a decrease or increase in the sensitivity of the body. Allergy is often manifested by significant irritation of the mucous membranes, skin rashes, general poor health and malaise, etc.

An allergological test is a test for the detection (determination) of a substance that causes a painful condition in a person (a painful reaction of the body).

Alopecia, baldness - a strong loss of hair in whole strands.

Albuminuria is the appearance of albumin protein in the urine of a pregnant woman. The indicator should be monitored monthly and can serve as a sign of a pregnancy complication: preeclampsia, urinary infection, etc.

Pulmonary alveoli - cavities located at the end of the bronchioles (branching of small bronchi).

Alveoli are cavities located at the ends of the bronchioles.

Aldolase is an alphafetoprotein, a protein present in fetal tissues. In adults, it is present in the blood in very small (trace) amounts. It can begin to be produced in the liver and in the blood during inflammation of the liver (hepatitis), some types of cancer and at the beginning of myocardial infarction.

Alzheimer's disease is a chronic neurological disease. It is characterized by irreversible intellectual impairment. Leads to dementia.

Amenorrhea is the absence of menstruation for a long time. It can be caused by anatomical, biochemical, genetic, physiological or mental disorders in the body.

Lactational amenorrhea is the physiological cessation of menstruation during breastfeeding.

Amyloidosis (amyloid degeneration) is a pathology of unclear etiology (dysproteinosis), characterized by extracellular accumulation of amyloid in tissues and organs; leads to sclerosis, atrophy, loss of functions.

An amino acid is an organic acid that makes up proteins. In total, there are 20 amino acids in the body that form protein structures.

Amniocentesis is a puncture of the fetal bladder, in which a small portion of amniotic fluid is taken. Allows you to determine the malformations of the fetus.

Analgesia - provoking the loss of pain sensation with the help of painkillers.

The anus is the opening that ends the rectum (the final, lower lobe of the large intestine). The anus is equipped with sphincters that allow it to in the normal state to be closed and to keep the feces inside the intestines. Opens during bowel movements.

Anatomy is a science that studies the structure and spatial interaction of various organs and tissues that make up the body of a living being.

Angina is an infection of the tonsils and throat of viral or bacterial origin (differentiated only by the results of bacteriological culture). There are Vincent's angina (ulcerated, ulcerated angina) and angina, in which the tonsils are covered with a whitish coating. Some bacterial sore throats caused by group A streptococci, if not completely cured, can lead to complications in the form of acute articular or cardiac rheumatism. Recurrent tonsillitis suggests the presence of a persistent focus of infection in the tonsils.

Angiogenesis is the phenomenon in which new blood vessels form from existing vessels.

Angioma is a vascular tumor, a malformation of the arteries and veins. It can be flat (without reliefs) or bumpy (embossed). Angiomas disappear with pressure, then re-stain instantly. Most of the time they don't pose any danger. Angiomas appear in 10% of young children and disappear, as a rule, by the age of 10. Papillary angiomas can be removed with a laser, which is safe and does not require the use of anesthesia.

Angiomatosis (hemangiomatosis) is the general name for diseases characterized by excessive proliferation of blood vessels.

Diabetic angiopathy is a complex of vascular diseases associated with diabetes mellitus.

Andropause is a natural contraction of a person's sexual functions after a certain age.

Aneurysm - a local expansion of the lumen of a blood vessel or heart, or a tumor containing blood, directly associated with the lumen of an artery.

Anemia is a decrease in the amount of hemoglobin in the blood. The main cause of anemia in pregnant women is a lack of iron in the body. With anemia, red blood cells are affected, the main component of which is hemoglobin, a protein that contains iron and ensures the delivery of oxygen to the lungs to body tissues. Anemia can be caused by genetic factors, malnutrition, or blood loss.

Anesthesia - general or local loss of sensation during the introduction of anesthetics. General anesthesia (general anesthesia) is accompanied by loss of consciousness.

Anxiolytic - a drug used to treat anxiety in its various manifestations, one of the types of tranquilizers.

Anorexia is a deviation in eating behavior, the main symptom of which is the almost complete lack of appetite. An anorexic person drastically reduces his diet, which leads to numerous health problems. In children, anorexia often occurs in adolescence and is due to psychological problems.

Antibacterial - a substance that can fight bacteria.

Antibiotics are a class of medicines that have an antibacterial effect. They help the immune system fight bacteria in the body. Should not be used without a doctor's prescription.

Antidepressants are medicines designed to combat depression. Currently used to correct mood, allowing you to reduce mental stress.

Antioxidants are substances of natural or artificial origin that block free radicals in living organisms. It is believed that this prevents the aging process of cells. Present in foods - vitamins C, E, carotenoids (pigments containing carotene).

Antiseptics are a class of medicines designed to fight bacteria on the surface of the body. Used, for example, to clean small wounds.

Antibodies are immunoglobulins that result from the body's reaction to certain particles and alien microorganisms. Antibodies play an important role in immunity.

Antiphospholipid syndrome - a syndrome that includes arterial and venous thrombosis with immune thrombocytopenia; detect autoantibodies to cellular phospholipids (for example, cardiolipin).

Apathy - a deep state of physical or mental fatigue, characterized by complete indifference to any desires or experiences.

Apnea is a temporary stoppage of breathing. Often occurs in premature infants with an immature respiratory system.

Apoplexy is a stroke.

Apparatus - a set of organs that perform the same physiological function.

Appendicitis is an inflammation of the appendix, a small process located at the end of the colon. Removal of the inflamed appendix is the most common abdominal surgery. Appendicitis is extremely rare in children under 3 years of age and in pregnant women.

Arachnodactyly (spider fingers) - a narrow long palm with long fingers.

Cardiac arrhythmia - a violation of the heart rhythm, changing its frequency, regularity and intensity of the pulse.

An artery is a vessel that carries blood from the heart to the tissues.

Arthritis is any inflammatory disease, acute or chronic, that affects the joints.

Arthrography - x-ray of the joints.

Arthrosis is a mechanical disease of the joints.

Archaic (primary) reflexes - reflexes characteristic of newborns under the age of 3 months. Their presence is one of the signs of good neurological and muscular development. There are several varieties of such reflexes: sucking reflex, Moro reflex (protection reflex), search reflex, automatic stepping, swallowing reflex, crawling reflex, grasping reflex.

Asthenic syndrome (asthenia) is a condition that has the following symptoms: increased fatigue, frequent mood swings, irritable weakness, hyperesthesia, tearfulness, autonomic disorders, sleep disorders.

Asthenia - impotence, a state of general weakness, characterized by a decrease in the functional capabilities of the body, not associated with fatigue from physical exertion and does not disappear during rest.

Asthenovegetative syndrome (vegetoasthenic syndrome) is a syndrome that includes asthenia with vegetative disorders, predominantly of a vagotonic nature.

Asthma is a disease of the lungs and bronchi. Characterized by difficulty breathing, changes in respiratory rhythm, wheezing when breathing, sometimes chronic cough and complications from the heart, the most dangerous of which is tachycardia. Asthma can be chronic, allergic, or stress-related. Asthma treatment varies depending on the extent of the disease and its nature.

Neonatal asphyxia - respiratory failure in newborns.

Ascites - dropsy of the abdominal cavity, the accumulation of fluid in the abdominal cavity.

Atheroma - a cyst of the sebaceous gland, resulting from the difficulty of removing the contents through the duct of the gland.

Atherosclerosis - sclerosis of the arteries, hardening of the walls of the arteries.

Atresia is a developmental anomaly: the complete absence of the lumen of a hollow organ.

Atresia of the biliary tract, biliary atresia in children is the almost complete absence of outflow of bile from all hepatic bile ducts. An extremely dangerous liver disease in young children. Occurs at birth and is manifested, among other things, by prolonged cholestatic jaundice in newborns.

Atrophy - a decrease in the weight and size of any organ, tissue or limb due to insufficient nutrition of cells or immobilization (immobility).

Autism is a psychotic condition that includes various developmental disorders, the general result of which is the inability to communicate with other

people and social adaptation, withdrawal into oneself. Childhood autism can suddenly appear before the age of 2-3 years. The autistic child loses control with reality, shows disinterest in relation to the environment. Childhood autism is a serious deviation.

Autoimmune diseases are diseases characterized by the production of antibodies that react with body tissues.

Aphasia is a partial or complete loss of the ability to speak and understand someone else's speech.

Aphthae, oral ulcers - superficial lesions of the oral mucosa.

Achondroplasia, chondrodystrophy is a congenital disease characterized by the cessation of growth of the bones of the limbs as a result of the lack of production of growth hormone.

LIST OF USED LITERATURE

1. Baranov, A. A. Pediatric rheumatology: a guide for doctors / A. A. Baranov, L. K. Bazhenova. Moscow: Medicine, 2002. 335 p.

2. Belyaeva, L. M. Diseases of the joints in children and adolescents / L. M. Belyaeva. Minsk: BelMAPO, 2006. 60 p.

3. Gintera, E. K., Puzyreva V. P. Brief edition of the national guide "Hereditary diseases": Publisher: GEOTAR-Media, 2017.- 464 p.

4. Geppe, N. A. Childhood diseases: textbook / Geppe N. A. - Moscow: GEOTAR - Media, 2018. - 760 p.

5. Dedov, I. I. Diabetes mellitus in children and adolescents: a guide / Dedov I. I., Kuraeva T. L., Peterkova V. A. - 2nd ed. , revised and additional - Moscow: GEOTAR - Media, 2013.

6. Mazurin, A. V. Propaedeutics of childhood diseases / A. V. Mazurin, I. M. Vorontsov. St. Petersburg: Folio, 2000. 538 p.

7. Melnikova, I. Yu. Children's gastroenterology / ed. Melnikova I. Yu. - Moscow: GEOTAR-Media, 2019. - 480 p.

8. Kalmykova, A. S. Propaedeutics of childhood diseases: textbook / ed. Kalmykova A. S. - Moscow: GEOTAR-Media, 2018. - 768 p.

9. Kalmykova, A. S. Polyclinic and emergency pediatrics: - Moscow: GEOTAR-Media, 2020. - 968 p.

10. Kildiyarova, R. R. Laboratory and functional studies in pediatric practice / Kildiyarova R. R. - Moscow: GEOTAR-Media, 2017. - 176 p.

11. Samsygina G.A. Cough in children. - Moscow: GEOTAR-Media, 2021. - 376 p.

12. Shabalov N. P. Childhood diseases. Volume 1.2. 8th edition. - Moscow: GEOTAR-Media, 2018. - 976 p.

13. Bazhenov N.P. "Neonatology". Moscow, 2004 yil. (Electronic version.)

14. Krasnov A. F. “Nursing”. Textbook for students of faculties of higher nursing education of medical universities. Moscow, 2000, volume 1-2.

15. Karimjonov I.A. “Neonatologyda Hamshiralik Ishi”. Tashkent, 2009 yil. (electronic version)

16. 5. Tulchinskaya V. D., Sokolova N. G., Nikovtsova N. M. “Nursing in Pediatrics”. Rostov-on-Don, 2001.

17. Shabalov N. P. “Children's diseases”. Textbook, Moscow, 2000.