**Topic:** Anatomical and physiological characteristics of the respiratory, cardiovascular, digestive and urinary system in children.

**Content topics:**

**Anatomical and physiological features of respiratory organs in children**

The airways are divided into three sections: the upper (nose, throat), medium (larynx, trachea, bronchi), bottom (bronchioles, alveoli). By the time the baby is born their morphological structure is not perfect, what connected and functional features of the respiratory system. Formation respiratory ends to an average of 7 years of age, and later only increase their size. All children airways are much smaller and narrower clearance than adults. The mucosa is more subtle, delicate, easily damaged. The glands are underdeveloped, IgA and surfactant products is negligible. Submucosal layer friable, contains a small amount of elastic and connective elements, many vascularized. Cartilaginous skeleton airway soft and pliable. This reduces the barrier function of the mucosa,

The nose and nasopharyngeal space in young children are small. The nasal passages are narrow, thick shell (lower developing to 4 years of age), so even a slight redness and swelling of the mucosa cause obstruction of the nasal passages, causing shortness of breath, sucking bother. With paranasal the time of birth only haymorovi formed (developed by 7 years of age). Etmojidalna, sfenoidalna and two front sinuses complete their development before the age of 12, 15 and 20 years respectively.

Nososlizna short duct located close to the corner of the eye, its valves are underdeveloped, so infection can easily penetrate the nose in the conjunctival sac.

Throat relatively wide and low. Yeystahiyev (auditory) tube connecting the nasopharynx and tympanic cavity, short, wide, straight and arranged horizontally, which facilitates the penetration of infection of the nose to the middle ear. In the throat situated lymphoid ring Pirogov-Valdeyera, which includes 6 tonsils, palate 2, 2 Trumpet 1 1 nasopharyngeal and tongue. An examination of the oropharynx uses the term "mouth". Ziv - this anatomical formation, surrounded by the bottom of the tongue, the sides - the tonsils and brackets at the top - the soft palate and tongue, rear - the rear wall of the oropharynx, front - Oral.

Epiglottis neonatal relatively short and wide, can cause functional narrowing the entrance to the larynx and the emergence stridoroznym breathing.

The larynx in children is higher and longer than in adults, has liykopodibnu shape with sharp narrowing in the area pidzv'yazkovoho space (newborn to 4 mm), which is gradually increasing (at the age of 14 to 1 cm). Glottis is narrow, it is easy to tired muscles. Voice communications thick, short, mucous membrane is very delicate, loose, vascularized considerably rich in lymphoid tissue, which easily leads to swelling of submucosal membrane during infection and respiratory syndrome occurrence rump.

The trachea is relatively greater length and width liykopodibnoyi form contains 15-20 cartilaginous rings very mobile. The walls of the trachea, soft, easily spadayutsya. The mucosa soft, dry, well vascularized.

By the time the baby is born bronchial tree is formed. Dimensions bronchial rapidly increasing in the 1st year of life and during adolescence. they also form a cartilaginous rings, which are in early childhood have locking plates are connected by
fibrous membrane. Cartilage bronchi very flexible, soft, easy shifting. Bronchi in children is relatively wide, the right main bronchus is almost a direct continuation of the trachea because it is often detected foreign objects. For the smallest bronchi characterized by sheer narrowness, which explains the occurrence of obstructive syndrome is in young children. Mucous major bronchi is covered with ciliated ciliated epithelium, which acts as a clearing bronchi (mucociliary clearance). Incomplete myelination vagus nerve hypoplasia and respiratory M' muscles contribute to the lack of cough reflex in young children or very weak cough jerks. Accumulated in small bronchi mucus clogs them and easily leads to atelectasis and infection of the lung tissue.

The lungs in children as in adults, with segmental structure. Segments are separated by a thin connective membranes. The basic structural unit of the lung - acinus, but the terminal it bronchioles end no tassel alveoli, both adults and bag (saccus), the "lace" edges which gradually forming new alveoli guestrooms in infants 3 times less than in adults. With age increases the diameter of each alveoli. Simultaneously increases vital capacity of the lungs. Interstitial lung tissue loose, rich in blood vessels, tissue contains little connective tissue and elastic fibers. In this regard, the lung tissue in children during the first years of life more full of blood, less airway. Underdevelopment flexible framework leads to emphysema and atelectasis. The tendency to atelectasis also occurs due to deficiency of surfactant - a film that regulates alveolar surface tension and stabilize the amount of terminal respiratory spaces, ie the alveoli. Synthesized surfactant alveolocyte type II and appears in fetal weight at least 500-1000 g The lower the gestational age of the child, the greater the deficiency of surfactant. It surfactant deficiency underlies the lack of smoothing lungs in premature infants and the occurrence of respiratory distress syndrome.

The main functional respiratory physiological features such children. Breathing frequent in children (compensating small volume of breathing) and superficial. The frequency is greater the younger the child (physiologic shortness of breath). Newborn breathes 40-50 times for 1 min, a child aged 1 year - 35-30 times for 1 min, 3 years - 30-26 times for 1 minute, 7 - 20-25 times for 1 min, 12 - 18-20 times for 1 min, adults - 12-14 times for 1 min. Accelerate or slow breathing ascertain if breathing frequency deviation from the average by 30-40% or more. Newborn respiratory TIMING MISTAKES short stops (apnea). Mostly diaphragmatic breathing, with 1-2 years of age he mixed with 7-8 years - girls - chest, boys - typhoid. Respiratory lung capacity, the smaller the youngest child. Minute object ' Capacity breathing also increases with age. However, this figure relative to weight gain in infants 2-3 times higher than in adults. Vital lung capacity in children is much lower than in adults. Gas exchange in children more intense due to the rich vascularity pulmonary circulation high speed, high diffusion capacity.

**APF cardiovascular system**

The cardiovascular system delivers oxygen and nutrients to all organs and tissues and removing carbon dioxide and other metabolic products and thus participate in maintaining the constancy of the internal environment. The cardiovascular system in children of all ages has a number of differences that affect its function. This determines the need to study the anatomical and physio-logical features of this system in children medical students.
Anatomical and physiological features of the cardiovascular system in children of all ages

- Anatomical Heart: baby lies more cranial due to the high standing of the diaphragm.
- Considerably larger volume of the heart relative to the chest volume.
- Spherical shape of the heart.
- Right and left ventricles approximately the same volume and wall thickness at the time of birth.
- After the birth of the rapidly growing left heart due to increased vascular resistance and blood pressure.
- The growth rate of major vessels is less than the growth rate of the heart.
- Newborn ratio of the diameter of the pulmonary artery and aorta: Another (aorta - 16 mm, pulmonary artery - 21 mm); aged 10-12 years is the same diameter, and the aorta in adults is always larger than the pulmonary artery (aorta - 80 mm, pulmonary artery - 74 mm).
- Newborn Blood vessels have thin walls, they are not sufficiently developed muscle and elastic fibers.
- Newborn clearance corresponding arteries and veins of the same.
- The capillaries in children are well developed, wide and relatively short.
- Pulse: children of all ages more often than adults, due to enhanced metabolism and the late development of vagal innervation of the heart.
- Blood pressure in children less than adults. For approximate calculation of blood pressure (mm Hg. Art.) in children older than 1 year can use the formula:

  \[
  \text{systolic blood pressure} = 90 + 2n, \\
  \text{diastolic blood pressure} = 60 + 2n, \\
  \text{where} \ n - \text{age children in years.}
  \]

Anatomical and physiological characteristics of the digestive system in children

In the embryonic period is the main power histiotrofne embryo (secret lining of the uterus, bladder material yolk).

On the 2-3 th month of fetal development begins hemotrofne power by transplacental transport of nutrients. From 16-20 weeks begin to operate their digestive organs that are beginning amniotrofnoho supply.

Depending on the formation of certain enzyme systems supply the fruit starts to get enterally protein, glucose, water, minerals and others. Rate differentiation and maturation of the digestive system is growing rapidly, but the time of birth are stored relative immaturity of the system. Laktotrofne power is the most important stage adaptation of the newborn child; it can solve the contradiction between the needs of a large and rapidly growing body of low functional unit of the distance digestion.

All parts of the digestive system adapted to infants breastfeeding mother's milk. Mouth child in the 1st year of life is relatively small, relatively large tongue, palate flattened. Well defined body fat cheeks, valykopodibni thickening of the gums, cross-folds in the mucous membrane of the lips, which are also important for the act of sucking. Oral mucosa dry, rich in blood vessels, very sensitive. The secretion of saliva provide submandibular, sublingual, parotid gland and numerous small. In the first 3 months of life saliva secretion is negligible, but under its influence in the
mouth already begins the digestion of carbohydrates and casein coagulation of milk. The esophagus is liykopodibnu shape, its length is equal to half the length of the body of the newborn (10 cm); adolescents up to 25 cm. The stomach of a newborn baby round his capacity 30-35 ml, aged 7-11 years, stomach similar in shape to the stomach of an adult, its capacity increases to 1020 ml. Motor function of the stomach is made up of peristaltic movements and periodic disconnections and zmykan home. The acidity and enzymatic activity of gastric glands low, but 1/3 fat (lipase emulsified milk) is hydrolyzed in the stomach under the action of gastric lipase. In the stomach, partially hydrolyzed protein is mainly due to proteolytic enzymes such as chymosin (renin labferment, rennet) hastryksyn; absorbed small amount of salt, water and glucose. Histological differentiation of gastric lasts until the end of the 2nd year of life. Motor function of the stomach is made up of peristaltic movements and periodic disconnections and zmykan home. The acidity and enzymatic activity of gastric glands low, but 1/3 fat (lipase emulsified milk) is hydrolyzed in the stomach under the action of gastric lipase. In the stomach, partially hydrolyzed protein is mainly due to proteolytic enzymes such as chymosin (renin labferment, rennet) hastryksyn; absorbed small amount of salt, water and glucose. Histological differentiation of gastric lasts until the end of the 2nd year of life.

The pancreas is a major digestive gland and its secretion increases rapidly especially after the introduction of feeding (feeding) and reaches the level of an adult at the age of 5 years. The main pancreatic juice enzymes, trypsin, chymotrypsin, diastase, amylase, lipase, phospholipase, inkretornyy insulin.

Liver newborn has a relatively large size of 4-4.4% of body weight, well vascularized, has not developed enough connective tissue and poorly differentiated segments functionally immature. Well manifest function and not hlikohenutvorennya - detoxification. The liver is involved in the processes of digestion, blood circulation and metabolism. Bile in the first months of life formed in small quantities, contains little bile acids (which sometimes leads to steatorrhea in infants), a lot of water, mucin, pigments; in infants as much urea. It also contains more tauroholevoyi acid than hlikoholevoyi that enhances its antibacterial properties, stimulates the secretion of the pancreas, increases peristalsis of the colon.

Guts baby infants relatively longer than adults, they are 6 times greater than the length of her body. The mucous membrane of the intestines tender, rich villi, blood vessels, cellular elements. Lymph nodules are well developed. The cecum and appendix moving, descending colon longer than upward. The rectum is relatively long, is poorly fixed and submucosal mucous membranes. Guts child perform
digestive, motor and suction function. Intestinal juice less active compared with the juice of an adult, he has slabkokyslu or neutral reaction, then - alkaline. Contains enterokinase enzyme, alkaline phosphatase, amylase, lactase, Maltase, invertase, then - lipase. Hydrolysis products formed as a result cavity (of the distance) and diaphragm (the wall) etching, absorbed all the small intestine, Unlike adults. Of great importance in childhood is intracellular digestion with a slight shift in milk lactoglobulin unchanged in the blood. In the colon, water is absorbed, formed stool, mucus secretion occurs. The feature of the intestines in children is relatively weak but long mesentery, which creates favorable conditions for the development of intussusception.

**Anatomical and physiological features of urinary system in children**

The relatively large size of the kidneys and the shorter length of the lumbar spine causing low topographical placement of the kidneys in children during the first years of life. The upper pole renal located at XI-XII thoracic vertebra and the lower - level lumbar IV, ie below the iliac crest. In infants kidneys are more mobile, due to poor development navkolonyrkovoyi fat. In the first years of life have kidney lobular structure. Bowls relatively wider kidneys, ureters away from them at right angles. More tortuous ureter slightly hypotonic and have a relatively large diameter. The bladder in infants is above symphysis, he later lowered into the pelvis. The urethra in women of all ages is shorter and wider than boys.

These morphological features of the urinary system in children are prerequisites for the possible development of microbial inflammatory diseases of the urinary system, and determine the interpretation of a number of instrumental methods of research and diagnostic studies.

The secretion of urine release it in alantoyisnu and amniotic fluid occurs in the antenatal period. At this stage urine hypotonic relative to plasma, low in uric acid, urea, chloride. After birth the kidney is the main organ that provides vital consistency internal environment. In infants concentration decreased renal function. The low density of urine associated with a small diameter glomeruli, reduced production of antidiuretic hormone, underdevelopment osmorehulyatoriv, functional deficiency epithelium distal tubules and others.

Total diuresis in infants 2-8 times higher thanin older children. He is 80-90 ml per 1 kg of body weight in the first months of life and about 50 ml per 1 kg of body weight in children 8-10 years of age. Given these characteristics, newborns and young children is recommended to administer about 200 ml of liquid per 1 kg of body weight. However, despite the increased diuresis, the child can not quickly compensate for excessive input fluid can cause anxiety, vomiting, diarrhea, polyuria, convulsions. In addition, high tubular reabsorption (99,4-100%) leads to a low rate of excretion of chlorides, which leads to the deposition of sodium chloride in the tissues, reducing filtration decrease in urine output. Therefore, the introduction of excess sodium chloride may be accompanied by significant violations diuresis even anuria, edema, so-called salt fever.

Imperfect mechanisms of reabsorption of water and sodium in the distal tubules enhanced functional immaturity processes secretion of hydrogen ions and ammonia in this section tubular apparatus, so that there can be conditions for the development of severe metabolic acidosis. During the final maturation and differentiation of
morphological structures of the kidney (aged 5-7 years) urine in their functional performance close to that of an adult.

**Topic:** Vitamins and their importance for development. Semiotics hypovitaminosis and hypervitaminosis children.

**Content topics:**

Clinic and diagnosis of hypovitaminosis. Participation vitamins in many metabolic processes leads to the development of systemic deficiency disorders in various organs and tissues. In this clinical signs of deficiency of certain vitamins are numerous, varied and often nonspecific. Common symptoms typical of deficiency of different vitamins are presented in Table 1

<table>
<thead>
<tr>
<th>The manifestations of vitamin A deficiency</th>
<th>vitamins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pale skin</td>
<td>C, A, PP, folic acid, B12, biotin</td>
</tr>
<tr>
<td>Dry skin</td>
<td>C, A, B6, biotin</td>
</tr>
<tr>
<td>Seborrhea</td>
<td>A, B2, B6, PP</td>
</tr>
<tr>
<td>Acne, boils</td>
<td>A, B6, PP</td>
</tr>
<tr>
<td>Dry hair loss</td>
<td>A, B6, biotin</td>
</tr>
<tr>
<td>Nausea</td>
<td>V1.V6</td>
</tr>
<tr>
<td>State of the gastrointestinal tract: dyspepsia, diarrhea, motility</td>
<td>A, PP, folic acid, B12</td>
</tr>
<tr>
<td>conjunctivitis</td>
<td>A, B2, B6</td>
</tr>
<tr>
<td>Susceptibility to infections</td>
<td>C, A</td>
</tr>
<tr>
<td>Fatigue, weakness</td>
<td>C, A, E, B1, B2, B12</td>
</tr>
<tr>
<td>Irritability</td>
<td>C, B1, B6, B12, PP, biotin</td>
</tr>
<tr>
<td>Insomnia</td>
<td>B6, PP</td>
</tr>
<tr>
<td>Violation of Twilight</td>
<td>A B2</td>
</tr>
<tr>
<td>Stomatitis</td>
<td>B2, B6</td>
</tr>
<tr>
<td>Anemia</td>
<td>B6, B12, folic acid</td>
</tr>
<tr>
<td>Tendency to hemorrhage</td>
<td>C, E, K</td>
</tr>
</tbody>
</table>

**Table 1**

**Risk for the occurrence vitaminodefisytynih states children are:**
- young children and teenagers during the most intensive growth;
- children involved in sports (with high physical activity);
- sick children (acute infectious disease of viral or bacterial origin, pathology of the cardiovascular system, gastrointestinal tract, etc.);
- sick children who have been taking some drugs;
- children from families with low socio-economic level.
Detection of vitamin A deficiency in children based on clinical and laboratory diagnosis. Clinical features Consequences deficient states on vitamins and options for laboratory diagnosis of vitamin presented in Table. 2

**Table 2**

**Clinical features Consequences deficient states on vitamins and options for laboratory diagnosis of vitamin in the body**

<table>
<thead>
<tr>
<th>Name Vitamin</th>
<th>Clinical Features Consequences Deficient States</th>
<th>Laboratory Diagnosis of Deficiency States</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vitamin C (ascorbic acid)</td>
<td>• Violation of the synthesis of connective tissue and blood vessel fragility leads to abnormal bleeding, frequent bruising, bleeding gums and inflammation, stiffness and joint pain (due to bleeding in joints) • Extraneous keratin in hair follicles makes the skin rough Slow healing Weakness, rapid fatigue Anemia The psychological / neurological symptoms, including irritability, depression, hypersensitivity and individual changes • Reduced immunity and increased risk of infections</td>
<td>Vitamin C in plasma or less than 0.006 g / L-hour urine - less than 0.005 g. The content of ascorbic acid in leukocytes or less than 114nm / 10ya (Vyhhu coat), in mononuclear leukocytes or less than 25 mg / 108. Resistance by capillary A.I.Nesterovym - more than 15 petechiae.</td>
</tr>
<tr>
<td>Vitamin B1 (thiamine)</td>
<td>Violation of perception and reflexes (polyneuritis, paresthesia) Uneven gait, imbalance (ataxia) Mental retardation, problems with learning and memory, headaches, insomnia. Individual changes (depression, irritability, emotional lability)</td>
<td>Vitamin B1 plasma - less than 14.8 mmol / L-hour urine - less than 10 mg. The content of pyruvic acid in plasma - more than 0.144 mg / l hour urine of more than 30 micrograms. Reduced activity of erythrocyte transketolase (ETKA) and their stimulation tiaminpirofosfatazoyu</td>
</tr>
<tr>
<td>Muscle weakness</td>
<td>Cardiomyopathy, tachycardia, dyspnea</td>
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<td></td>
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<tr>
<td>(especially in the calf muscles)</td>
<td>Anemia</td>
<td></td>
</tr>
<tr>
<td>Violations of energy production, increased fatigue</td>
<td>Violation of fusion proteins (collagen)</td>
<td></td>
</tr>
<tr>
<td>Slow healing of wounds</td>
<td>Low resistance to infection</td>
<td></td>
</tr>
<tr>
<td>• Loss of appetite, constipation</td>
<td>(TPF) - ETKA level of less than 5 units. / Nmol hemoglobin and more than 16% increase after adding TPF</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Vitamin B2 (riboflavin)</th>
<th>Vitamin B2 hour urine - less than thirty micrograms or less than 125 mg 1 gcreatinine. The content of riboflavin levels less than 200 nmol / L, erythrocyte - less than 15 mg / dL.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Zaid, cracks and crusts in the corners of the mouth (angular stomatitis), dryness, redness of the lips (heyloz). Smooth bright red (fuksynovyy) tongue</td>
<td>Determination of glutathione reductase (an enzyme that depends on riboflavin) in red blood cells after stimulation and flavin adenine dinucleotide - if the ratio before and after activation exceeds 1.2</td>
</tr>
<tr>
<td>• Vascularization (redness) of the cornea, burning eyes, excessive tearing, sensitivity to light, conjunctivitis, blepharitis.</td>
<td></td>
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<tr>
<td>• Anemia with decreased production of red blood cells</td>
<td></td>
</tr>
<tr>
<td>• Irritability, drowsiness</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>PFA vitamin (niacin or nicotinic acid)</th>
<th>The content of methyl nicotine amide, hour urine - less than 4 mg in hour - less than 0.3 mg. Content-pyridine nucleotides (NAD and NADP) is less than 0.3 mg in 1 ml.</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Red, cracked, scaly sealed shkira.sho in areas sensitive to sun exposure, such as elbows, knees, back of the neck and arms, shoulders (pigment dermatitis)</td>
<td>Niacin levels - less than 30 mmol / l</td>
</tr>
<tr>
<td>• Inflamed, painful, swollen cracked tongue, burning sensation in the mouth, salivation.</td>
<td></td>
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<tr>
<td>• Reduced secretion of the digestive tract, loss of appetite, bloating,</td>
<td></td>
</tr>
<tr>
<td><strong>Vitamin B12 (tsianokobalam-in)</strong></td>
<td><strong>Vitamin B12 plasma levels below 10 pmol / L. Bold methylmalonic acid excretion - more than 5 mg / ml creatinine. Defining index hipersehmentatsiyi neutrophil nuclei - the ratio of neutrophils with more than 5 sehmantamy the number of neutrophils with less than four segments: the level of 30% indicates a deficit.</strong></td>
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</tr>
</tbody>
</table>
| vomiting, diarrhea without mucus and blood.  
  • Fear, anxiety, fatigue, irritability, headaches, insomnia, emotional lability, lethargy |                                                                                                 |
| **Vitamin BC (folic acid)**       | The content of folic acid in blood plasma - less than 4.8 nmol / L, erythrocyte orless 312 nmol / l. Defining index hipersehmentatsiyi neutrophil nuclei-neutrophil ratio of more than 5 segments in the number of neutrophils with less than four segments: the level of 30% indicates a deficit. |
| • Deterioration of cell replication leads to atrophy and inflammation of the mucous membranes of the mouth and digestive tract, reducing the absorption of nutrients, anorexia and weight loss  
  • megaloblastic anemia, accompanied by weakness, shortness of breath, decreased concentration  
  • neuropathy, paresthesia  
  • Reduced production of platelets may increase the risk of abnormal bleeding |                                                                                                 |
| • atrophy of the epithelium of the gastrointestinal tract, reduced absorption of nutrients, diarrhea, anorexia, weight loss, glossyt, stomatitis, gingivitis, ulcerative gastritis, enteritis  
  • macrocytic megaloblastic anemia, fatigue, weakness, shortness of breath  
  • Reduced production of platelets increases the risk of bleeding  
  • Violation of the formation of white blood cells weakens the immune response to infection  
  • Elevated levels of homocysteine in the blood increases the risk of |                                                                                                 |
| Vitamin B6 (pyridoxine) | • dermatitis (red, covered with scales, fat, itching skin, especially around the nose, mouth, ears, genital area)
• Cracks in the corners of the mouth and lips (heyloz)
• Anemia mikrotsytarna
• Reducing the function of white blood cells, reducing the production of antibodies
• Atypical electroencephalogram, seizures, convulsions, paresis, peripheral neuritis
• Depression, irritability, anxiety, headache, insomnia
• May increase cholesterol and low-density cholesterol, reduced high-density cholesterol
• may increase the risk of calcium kidney stones |
| --- | --- |
| Vitamin H (biotin) | • Anorexia and vomiting
• myalgia, skin hypersensitivity
• Luschastyy dermatitis with gray skin tones
• Hair loss and baldness
• The weakening of immunity
• Changes in mental status, depression, fatigue, exhaustion, anxiety. The lag in development (in children with hereditary disorders of biotin metabolism) |
| Ksanturenuriya more 0.005 gday Level 4 pirydoksynovyi acid-hour urine less 0.0005h. Green urine with addition of 10% ferric chloride.
When Wb-dependent convulsions withdrawal EEG after intramuscular administration of 100 mg of pyridoxine.
Plazmapirydoksal-5-phosphate orless 30 nmol / L. Vitamin B6 plasma - less than 40 nmol / L.
Index alanintransaminazy erythrocytes - the ratio of more than 1.25. |
| Biotin serum - less 1.02 nmol / L.
Biotin in urine - less than 31 nmol / L. |
| Vitamin B5 (pantothenic acid) | • Vomiting and stomach pain  
• Fatigue, headaches, insomnia, irritability  
• Numbness and a feeling of "heartburn" at the bottom of the shin and feet  
• arthralgia, myalgia, muscle cramps  
• Anemia  
• dull hair, baldness  
• Reduced immunity  

The content of pantothenic acid in the blood less than thirty micrograms / ml. Withdrawal of pantothenic acid in the urine of more than 1 mg / day. |
|---|---|
| Vitamin A (retinol) | Dryness, redness, itching (xerosis) of the conjunctiva, cornea  
• Failure of adaptation in low light (night blindness / day-blindness)  
• Dry, rough, itchy skin rash often with pustular  
• Dry and brittle hair and nails  
• Loss of taste and appetite, anosmia  
• Tendency to stone formation, interstitial renal lesions, hyperoxaluria  
• Hipoatsydnyy gastritis, diarrhea syndrome, delayed weight gain and psychomotor development  
• Slow growth defect formation epiphysis bones and tooth enamel  
• Increased susceptibility to infection  

Vitamin A in serum - less than 0.7 mmol / l. Carotene content in blood serum - less than 1.0 mmol / l. Dark adaptation in rapeseed below normal. |
|---|---|
| Vitamin D (holekaltsy-Férolles) | • Slow growth and development (children begin to crawl and walk later)  
• Irritability  
• Rickets  
• Slow change of teeth and enamel formation  

The level of 25 (OH) - holekaltsyferolu plasma norm 25-310 nmol / l. The level of 1,25 (OH) 2 cholecalciferol plasma norm 48-100 pmol / L |
| Vitamin E (tocopherol) | • Violation of the integrity of erythrocyte membranes leading to hemolysis and anemia  
• degeneration of nerve cells  
• muscle weakness  
• myocardial dystrophy | The level of vitamin E in blood plasma - less than 0.5 mg/L higher percentage hemolysis in 0.12% solution of hydrogen peroxide.  
Level isomers plasma tocopherols - alpha-tocopherol levels less than 10 mmol/l |
| Vitamin K | Bleeding for vascular-platelet type  
• Deterioration of bone mineralization | The level of vitamin C in plasma or less 0.4 nmol/l.  
The increase in prothrombin time, low vitamin- K-dependent blood factor, and C and B-protein osteocalcin, the appearance of a blood vitamin inactive precursors - K-dependent factors (RIUKA) |

*Micro and macro are entirely exogenous origin* And their only food source is, because the body is not able to synthesize. One of the features of the child's body - high speed metabolism, and therefore the need for macro and micronutrients in children can not be satisfied only with food. The main micro and macro that affect the functioning of the immune system include iron, zinc, magnesium, manganese, copper, molybdenum, vanadium, nickel, boron, fluorine, cobalt.

*zinc deficiency* leads to disruption of both nonspecific and specific defense mechanisms microorganism. Zinc plays an important role in maintaining the balance between cellular and humoral immunity. Zinc deficiency leads to inhibition of Th1-response of the immune system by reducing the production of interferon-γ, TNF-α, interleukin (IL) 2 while maintaining pressure synthesis of IL-4, IL-6 and IL-10 production by mononuclear cells. Filling zinc deficiency contributes to the restoration of the immune system, reduce respiratory and enteric infections in children.
iron deficiency can lead to anemia, lowering resistance to infectious agents, suppression of cognitive function. Iron deficiency in the body leads to cellular immunity expressed abuse due to the fact that iron ions play a leading role in the mechanisms that regulate the functional activity of T-lymphocytes.

Magnesium is an essential component of enzyme systems involved in protein, carbohydrate and fat metabolism. Participation magnesium is necessary for the proper functioning of the immune system.

Copper actively involved in the functioning of the central nervous and immune systems. The presence of copper deficiency with infectious and inflammatory diseases accompanied by reduced production of IL-2 T cells and Th1-response activity.

Boron is not only a necessary component of the calcium-phosphorus metabolism, but also an important factor in the functioning of the immune system.

deficiency of selenium can lead to activation avirulentnyh Coxsackie virus strains, infection which leads to the development of myocarditis.

Manganese is an activator of many enzymes involved in the biosynthesis of proteins, DNA, RNA and carbohydrate metabolism.

The use of fluorochemical drug plays a crucial role in the prevention of dental caries in children due to the fact that fluorine stabilizes the calcium in the bones. Fluoride, cobalt, molybdenum, vanadium, nickel mechanisms involved in immune defense.

With a lack of cobalt, which is part of the vitamin B12 molecule, there is weakness and decreased appetite.

Molybdenum deficiency leads to intolerance of certain amino acids, the emergence of irritability, tachycardia, "night blindness" and serious neurological disorders.

Vanadium helps reduce cholesterol levels in serum. With sufficient nickel providing the body with glucose and cholesterol in serum is maintained within the age norm.

Thus, vitamins, micro and trace elements play a significant role in the life of a child. The need for vitamins and trace elements depends on the age, the characteristics of the biological state of metabolism and the particular period of life, sex, physical activity, presence of chronic diseases and other factors. The most at risk of deficiency states children during critical periods of growth and development, weakened by malnutrition and recurrent or chronic illness.

Prevention and correction of hypovitaminosis:
1. The full and varied diet is the basis for the prevention of vitamin deficiencies in children of all ages.
2. treatment of various systemic diseases, especially the treatment of digestive tract pathology. Particular attention intestinal dysbiosis correction.
3. Prevention vitaminodefitsitnyh conditions recommended products preventive purposes (vitamins and vitamins and minerals, dietary supplements). Typically prophylactic administration of at least 3-month course of vitamins and mineral complexes year.
4. For the prevention and treatment of conditions vitaminodefitsitnyh recommend vitamin preparations - mono or complex vitamin and mineral preparations.
**Topic:** Hereditary and congenital diseases of bronchopulmonary system in children.

Content topics:

**Bronchiectasis** - acquired disease with chronic suppurative local process (purulent endobronchitis) in bezpovorotno modified (extended, deformed) and usually functionally disabled bronchi, manifested mainly in the lower parts of the lungs.

**Etiology.** Polyetiological disease. The following main factors leading to the formation of bronchiectasis:

1) and postnatal congenital malformations of the lungs;
2) non-specific inflammatory recurrent respiratory disease;
3) children's infectious diseases (measles, whooping cough);
4) foreign bodies tracheobronchial tree;
5) tuberculosis;
6) some hereditary diseases and systemic lesions (cystic fibrosis syndrome Kartahenera, alpha, hammahlobulinemiya);
7) bacterial destruction of lungs.

**Pathogenesis.** The major importance in the pathogenesis of bronchiectasis is the combination and interaction of two factors: inflammation and violation of the drainage function of bronchi. This is confirmed by morphological picture remote parts of the lung with bronchiectasis. It is characterized by growing degenerative and atrophic changes of all structural elements lungs. The result of these processes - easing bronchial tone, decrease in contractile ability of the walls and peristalsis. The constant increase in internal pressure during bronchial cough helps further expand the bronchi, joining a secondary infection entails peribronhialnoy sclerosis accumulation of purulent sputum.

Important role in causing atelectasis bronchiectasis play various origins (born, aspiration, pneumonic, with foreign bodies, tumors of the bronchus, cystic fibrosis, bronchial compression of the lymph nodes).

VA Klymanskyy (1975) attaches great importance to the formation of bronchiectasis blood circulation in the pulmonary system. Anhipulmonohrafiya allowed to study the pathogenesis component. Narrowing of the pulmonary arteries and their branches leads to chronic inflammation in the lungs. Do not rule out the relationship nasopharynx diseases (rynynosusopatiy) with bronchiectasis. Concomitant nasal and lung inflammation occurs jet swollen lymph nodes submandibular area, neck, mediastinum, and bronchopulmonary paratahealnyh. Enlarged lymph nodes are not only a source of infection, but also a factor that gives blood flow in the lungs causes the development of chronic inflammation.

Thus, the pathogenesis of bronchiectasis - a whole series of pathological processes that interact and influence each other.

**Classification.** There are primary bronhoektaziyi as an independent nosological form (bronchiectasis) and secondary displays bronhoektaziyi as a complication of other diseases (tuberculosis, abscess, staphylococcal lung destruction and so on.). Used in this country in the literature, the terms "bronhoektaziyi" "bronchiectasis", "bronchiectasis" should be considered synonymous.
The literature provides many different classifications of chronic inflammation in the lungs. The most modern is the following classification of bronchiectasis:

2. The shape, cylindrical, saccular, kistovydni.
3. Distribution: unilateral, bilateral (extensive, neobshyrni) - iz indicating segments.
5. During the course, with exacerbations (often rare).

Clinic, diagnostics. Since bronchiectasis - a chronic disease, the clinical picture depends not only on the extensiveness of lesion severity and prevalence of bronchitis in unaffected areas of the lungs, but also the availability of exacerbation or remission. Over the past 15-20 years running rare form, accompanied by cough and plenty of fetid purulent sputum more common small form with moderate symptoms. This is a bona fide conservative treatment of pneumonia, timely readjustment of foci of infection in the nasopharynx, the general clinical examination of children, prevention activities undertaken in children's institutions.

Bronchiectasis - a disease of childhood. Most often it occurs in children up to 2-5 years. Congenital bronchiectasis occur at the 1st year of life. In most cases parents are associated with the onset moved to the first months of life with pneumonia, followed by frequent SARS, bronchitis, measles, whooping cough. Children often catch cold with fever, exacerbation occur regularly in spring and autumn. These children all the time, even in remission, coughing, especially in the morning vidkashlyuyuchy purulent sputum. Overall condition may be little affected, but in advanced process sooner the symptoms of intoxication. Children suffering from bronchiectasis, quickly tired, lagged behind in the development of peers. Hemoptysis them is rare, it is more typical for bronchiectasis motivated by foreign bodies. With limited bronhoektaziyah dyspnea at rest usually do not bother with bilateral lesions of severe shortness of breath during exercise and small even at rest. Some children complain of headaches, bone. This usually occurs in children with adverse pathological processes in the nasopharynx. In periods of exacerbation increases the amount of sputum, cough constant throughout the day, increasing shortness of breath and starts, the body temperature rises to subfebrile figures. During the examination of the child pay attention to lag in its development, pale skin. But in recent years, children can meet outside if at all healthy. Changes fingers as drumsticks and watch glass found in patients with congenital bronchiectasis and in cases that went too far. with bilateral lesions of severe shortness of breath during exercise and small even at rest. Some children complain of headaches, bone. This usually occurs in children with adverse pathological processes in the nasopharynx. In periods of exacerbation increases the amount of sputum, cough constant throughout the day, increasing shortness of breath and starts, the body temperature rises to subfebrile figures. During the examination of the child pay attention to lag in its development, pale skin. But in recent years, children can meet outside if at all healthy. Changes fingers as drumsticks and watch glass found in patients with congenital bronchiectasis and in cases that went too far.
One of the important diagnostic features of chronic lung disease is a deformity of the chest. More common asymmetrical distortion, retraction of the chest on the background of the lesion, its lag in breathing, some omission blades, ribs convergence, narrowing of the intercostal spaces, scoliosis. Deformation of the chest most pronounced in atelektatychnych bronchiectasis. When percussion is as the most significant changes is determined at atelektatychnych bronchiectasis - reveal shortening percussion sound. In emphysematous bronchiectasis percussion sound box with color. If you can not find atelectasis shortening of percussion tones in cases where compensatory increased "emphysematous" areas of the lungs cover it. With extensive atelectasis percussion show bias toward boundaries of the heart of the process. Auscultation often more permanent changes. A characteristic is the presence of wet variegated krepliyuchy and wheezing after expectoration of sputum them becomes smaller. In young children with complete bronchial obstruction with thick sputum can not always hear the wheezing. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrycasty wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. sputum expectoration after they become
smaller. In young children with complete bronchial obstruction with thick sputum can not always hear the wheezing. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrychastyh wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. Sputum expectoration after they become smaller. In young children with complete bronchial obstruction with thick sputum can not always hear the wheezing. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrychastyh wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrychastyh wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrychastyh wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrychastyh wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. It is necessary to try to cause such child cough pressing a spatula on the root of the tongue or a light pressing on the trachea. It is characteristic that more wheezing tapped morning. In acute auscultative changes are characterized by an increasing number of small- and wet serednopuhrychastyh wheezing can hear the dry whistling wheezing. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi. Breathing over the area lesions usually weakened. Listening to the constant rattle, their localization in stable remission may indicate bronhoektaziyi.

On the basis of the relevant medical history, examination, physical information can assume that the child bronchiectasis. Clarify diagnosis is possible only with the full examination of the child.

When viewing radiographs important to assess the dynamics of the process, objective opinion can make only with images taken in remission. You can find direct and adverse radiological manifestations. For bronchiectasis is characterized by atelectasis, ring, cellular shades. In cylindrical bronchiectasis without atelectasis can detect small lung pattern deformation, tyazhystist roots of the lungs. Side feature may be the second circuit in the heart atelektatychnyh bronchiectasis lower lobe of the left lung. Suspicion of the process medium particles or reed segments shown on lateral radiographs.

Remember that even in the absence of marked changes in children with typical radiograph history and clinical signs set forth above, as shown bronholohichne examination. It must begin with bronchoscopy. Review of the tracheobronchialichne tree to exclude or detect the presence of defects and abnormalities of the trachea and
bronchial foreign body or its consequences, determine the status of the bronchial lumen (subject to compression from the outside), the state of the mucosa, the presence, nature of mucus, its predominant localization. Bronchoscopy allows for cytological and bacteriological examination of sputum to determine the nature and prevalence endobronchitis. Children are more common catarrhal, purulent, fibrinous-ulcerous bronchitis, the foreign bodies - and hemorrhagic granulation. The prevalence may be local (within the share segment).

The main method to establish the diagnosis of bronchiectasis is Bronchography. Bronchography - contrast examination of the bronchial tree. Most children Bronchography performed under general anesthesia with the use of muscle relaxants. Only in some older children can not do it under local anesthesia. Currently used water-soluble contrast (propilyodol). To obtain high bronhohramy, good filling segmental bronchi before bronchography necessary to reorganize the hard tracheobronchial tree, children with a lot of phlegm its repeated several times with the introduction of proteolytic enzymes. In children with bilateral bronhoektaziyamy prior bronchography appropriate to reorganize the bronchi via percutaneous mikrotraheostomiyi (introduction of thin polyethylene catheter during bronchoscopy controlled by eye puncture the trachea or bronchus partial). Daily administration of an antibiotic solution to the enzyme stimulates coughing and sputum bronhohramy us to obtain better quality. It is obligatory to perform bidirectional bronchography. Alternately contrast fill each lung perform bronhohramu each lung in two projections. Fill lung contrast possible through the bronchoscope or endotracheal tube, controlling by means of EOC. After aspiration of contrast filling the second lung. Daily administration of an antibiotic solution to the enzyme stimulates coughing and sputum bronhohramy us to obtain better quality. It is obligatory to perform bidirectional bronchography. Alternately contrast fill each lung perform bronhohramu each lung in two projections. Fill lung contrast possible through the bronchoscope or endotracheal tube, controlling by means of EOC. After aspiration of contrast filling the second lung. Daily administration of an antibiotic solution to the enzyme stimulates coughing and sputum bronhohramy us to obtain better quality. It is obligatory to perform bidirectional bronchography. Alternately contrast fill each lung perform bronhohramu each lung in two projections. Fill lung contrast possible through the bronchoscope or endotracheal tube, controlling by means of EOC. After aspiration of contrast filling the second lung.

Bronhohram assessment to determine the location of bronchiectasis, their prevalence, form. Acquired bronchiectasis can be cylindrical, baggy, mixed, often combined with deforming bronchitis.

An important feature of the cylindrical bronchiectasis is that the periphery is not narrowed bronchi (as normal), and expand and do not change their diameter, terminal bronchi departments not kontrastuyutsya. In baggy bronchiectasis terminal units and extend "cut off."

Most localized bronchiectasis in the lower lobe and left lung reed segments, the lower and middle lobes of the right lung. Localization of bronchiectasis in the upper lobes often occurs in the presence of congenital pathology or tuberculosis. For deforming bronchitis, bronchial characteristic change direction, their tortuosity, convergence and inequality circuits without significantly expanding the bronchial lumen.
The changes identified during anhipulmonohrafiyi children with bronhoektaziyamy depend on the depth and prevalence of pathological process in the bronchi and lung parenchyma. The slightest change in deforming bronchitis note: some narrowing of the arteries subsegmental and their deformation. The biggest changes are in the baggy and kistovydnyeh bronhoektaziyah. In these cases, a sharp narrowing of the arteries and partial amputation of the distal segmental shafts. Bold contrast capillary-venous or mesh defined by "silent zone". Particularly important angiographic study if extensive bronchial lesions (more than 9 segments). The complete absence of blood flow in the affected areas of the lungs indicates the failure of functionally and the need to remove these plots are only pockets of intoxication.

In assessing the pulmonary circulation attaches great importance radionuclide scanning. In bronhoektaziy in the area of the lesion seen on skanohrami reduce accumulation radiopreparatu or complete lack compared to healthy areas.

Functional study of cardiac and pulmonary circulation show that children change degree determined by the amount and duration of disease lesions.

ECG in patients with damage not exceeding one particle, shows no disorders bioelectrical activity of the heart muscle. If the lesion has spread to a number of shares of one or both lungs, there is a high occurrence of sharp teeth P vnutrishnoperedserdnoyi abuse and intraventricular conduction, myocardial metabolic changes.

Rheographic assessment of pulmonary hemodynamics in children with chronic bronchitis and deforming bronhoektaziyeyu one particle shows no significant circulatory disorders regardless of age and disease duration. For bilateral bronchiectasis process in the lungs along with a decrease in the intensity of pulmonary circulation disorders observed pronounced tone of precapillary vessels.

**Treatment** bronchiectasis surgery. The very presence of the disease is an indication for surgical treatment. But it is not limited to the operation. Conservative treatment is used as a preoperative preparation and postoperative period. Then-rehabilitation patients spa treatment.

Indications for surgery, the optimum time to carry it out is determined in each case individually. Child's age is not an obstacle for the operation. Detection of a child, even one year of life, atelektatychnyh bronchiectasis involving clinical pulmonary suppuration, is an indication for surgery.

Relative contraindications occur in children with cylindrical bronhoektaziyamy individual segments - with no signs of chronic suppurative inflammation.

Absolutely contraindicated deal with bronchiectasis in children with diffuse Bilateral bronhoektaziyamy, with lesions over 12-13 segments, and in severe cardiopulmonary disease, amyloidosis.

A comprehensive preoperative preparation with bronchiectasis aimed primarily at the maximum rehabilitation tracheobronchial tree and all related inflammatory foci (nasal cavity, paranasal sinuses). For sanitation trachea and bronchi using bronchoscopy, which is repeated 2-3 times. During bronchoscopy aspirating purulent sputum, washed tracheobronchial tree isotonic sodium chloride with mucolytics and enzymes administered antibiotic sensitivity based flora. If no effect after 2-3 sanatsiy in children with bilateral bronchiectasis extensive localization before surgery performed percutaneous catheterization of the trachea (mikrotraheostomiyu). Having a catheter in the trachea to stimulate coughing by periodically introducing several
milliliters of isotonic sodium chloride 4-6 times or more per day) 2-3 times a day administered antibiotics, mucolytic drugs. The duration of this catheter - 14-15 days. Assign inhalation, exercise therapy, postural drainage, broncho and mucolytic drugs, vitamins, desensitizing agents. When local forms when bronchitis is localized within the same lobe, spending long readjustment is needed. Limited 1-2 bronchoscopy, a complex exercise, inhalation, postural drainage.

The amount of resection is determined according bronchography and refine during surgery. Performed resection of the lobe, several segments of two light particles (at right location), very rarely have to remove the entire lung-pulmonoktomiya. At bilateral bronchiectasis localization operation is performed in two stages with an interval of 6-12 months.

In addition to replenishing the treatment of postoperative blood loss, correction of electrolyte, acid-base status important is the maximum smoothing left parts of the lungs. To do this, during the operation in the left pleural cavity drainage, with which you can maintain negative pressure. For this purpose, by active aspiration (10-15 cm of water. C.) Aspirating the contents of the pleural cavity for 2-3 days. After complete drainage smoothing lung removed. It is also important to stimulate a child's cough, sputum from the oropharynx suck, and, if necessary, and the trachea and bronchi, thus providing airway. These measures prevent the occurrence of postoperative complications such as atelectasis. It is important that these tasks are well-anesthesia. Apply topical administration 0,25-0,

Used as antibiotic therapy, antihistamines, vitamins, cardiac glycosides, physiotherapy, breathing exercises, massage.

Further, patients needing rehabilitation under the supervision of a pediatrician, pulmonologist. At 6 months after surgery they need sanatorium treatment, clinical supervision lung and thoracic surgeon.

Primary Prevention of bronchiectasis (bronchiectasis) is preventing the proper and timely treatment of acute infectious respiratory processes in children. Prevent exacerbation help seasonal influenza vaccination. To prevent the formation and progression of bronchial obstruction syndrome matters eliminate pathogenic risk factors obstructive pulmonary pathology (tobacco smoke, aggressive factors of production), and guidance that defines prognosis of the disease

**Williams Syndrome Campbell**

The frequency of the syndrome, according to our data, up 2.5% of children hospitalized pulmonological hospital with chronic lung disease. The cause of the disease - underdevelopment bronchial cartilage rings of 3-8 orders of magnitude. Distal zone on lesions of cartilage appears again, in addition, cartilage is stored in the corners of the bifurcation of the bronchi. These changes lead to a sharp increase in respiratory bronchi mobility, complicating their treatment because of the inefficiency of coughing that leads to the formation of chronic inflammation.

In most cases the defect is common bilateral in nature, but may be limited. It is more common in boys. The first signs of the disease in almost all patients are in the first three years of life as pneumonia or bronchitis, which flows continued with obstructive syndrome, so that 15% of patients wrongly diagnosed asthma. Further there is a constant wet cough with purulent sputum, chest deformity, transforming terminal phalanges. Delayed physical development is found in half of patients.
Auscultation listened scattered wet, preferably serednomihurtsevi wheezing in both lungs and mainly dry whistling wheezing exhale, lengthening the exhalation. In remission decreases the number of wheezing and obstructive syndrome often disappears.

Radiologically visible coarse gain and deformation of lung pattern, often ring-shaped or oval sealed with clarifying the walls (bronchiectasis). Pnevmosklerotychni localized changes in both upper and lower lobes of the lungs.

Bronchography detects local expansion, mainly subsegmental or segmental bronchus, bronchial contrast over the distal extension there often. When using a contrast agent that lingers on the walls of the bronchi may exercise their descending on inspiration and expansion exhale.

In acute bronchoscopy are sharp bronchial mucous congestion and a large number of viscous purulent secretions occlusive segmental bronchi and subsegmental. A common characteristic of suppurative endobronchitis for patients with bilateral lesions of the lungs. On examination of the bronchi half of the patients seen various anomalies of the structure of the bronchial tree, such as tracheomalacia (10%), expansion gaps large bronchi (16%), increased respiratory mobility segmental bronchus (25%), abnormal branching bronchial tree (10%), less - narrowing of the large bronchus. Under the influence of treatment, most patients eventually endobronchitis marked decrease in activity and, often, strengthen cartilage skeleton bronchi. Tsytohramy bronchopulmonary washings exhibit a pronounced neutrophilia (80%) and reducing the number of alveolar macrophages.

For this kind of abuse defects characteristic of obstructive ventilation type - a symptom air traps caused by bronchial expiratory kolabuvannyam in the samples with forced breathing. The collapse of the bronchial lumen during inspiration leads to a brief cessation of airflow that spirohrami displayed as plot curves plateau at FEV and IAL (Fig. 1). These changes cause hypoxemia in 20% of hypercapnia PaCO2 above 40 mmHg. c. 40% of patients. Often spirographic appears the phenomenon of "wavy exhale."

With bronchial secretions often planted Haemophilus influenzae (42%), Streptococcus pneumoniae (26%) and Staphylococcus aureus (26%).

The feature of the immune status of children is higher than in children with chronic pneumonia severity hiperimunohlobulinemiyi G, as well as higher levels of secretory immunoglobulin A. Differential diagnostic wide range Syndrome - is cystic fibrosis, primary immunodeficiencies, asthma, and other common malformations of bronchus, chronic aspiration syndrome.

Forecast serious, due (half of patients) with progression and the development of pulmonary hypertension and the formation of pulmonary heart.

Treatment only conservative, its main objectives - fighting purulent endobronchitom, prevention of pulmonary heart.

lung cysts

There acquired and congenital lung cysts that are air or fluid-filled cavity. Acquired cysts formed in destructive pneumonia. Congenital (true) cyst arising from the breach of the bronchi (bronchial cyst) or dysplasia of the pulmonary parenchyma. Localization bronchogenic cysts depends on the time of the violation. With the
formation changes early эмбрионаеза cysts are located within the mediastinum (in the trachea, esophagus or main bronchi). In more recent violations cysts are внуритшнлехенево. Bronchial (bronchogenic) cyst containing elements in their bronchial wall structures; epithelium lining the cyst, capable of producing a liquid. Cysts are lined with pulmonary alveolar epithelium origin and located in the peripheral regions of the lungs. Alternatively cystic degeneration described "cellular lung" of many small cavities. In some cases, the cyst lumen communicates with the bronchi.

Congenital lung cysts often combined with other malformations of the lungs, they can be single and multiple (polycystic), asymptomatic and detected during random X-ray. Children, especially young children, the disease often occurs as recurrent pneumonia in the same area of the lung.

Physical changes in uncomplicated cysts are absent, the pneumonic exacerbations usually are marked shortening of percussion sound, relaxed breathing and the relative small amount of wet wheezing. In remission satisfactory condition of patients, symptoms of intoxication and decline in physical development there. Congenital cysts often localized in the upper lobe of the left lung, at least in other shares. In some children bronchoscopy detected эндобронхит. In some cases, abnormal distribution of the bronchi are confirming the genesis of congenital cyst.

In addition to acute, protracted and chronic inflammatory changes, lung cysts can become strained, sometimes coming into the pleural cavity to form the air - or pneumoempyema have severe weather, especially in children during the first months of life. The hard cyst usually occurs on the background of pneumonia or SARS. Leading role in its pathogenesis plays formation valve mechanism resulting эндобронхита causing bronchial stenosis. This problem is especially dangerous in young children because of the development of severe respiratory failure, which is a threat to life.

X-rays air cysts appear as clearly delineated single or multiple pulmonary unchanged at clarifying the field or on the background of enhanced and / or deformed lung picture. When stratification cavities each other affected areas of the lung is сотовидный. In pneumonic exacerbations of radiographically detected pattern inflammatory infiltration of lung tissue within the segment or part of the extension root of the lung due to enlarged lymph nodes. Reverse slow growth and often, especially when multiple cysts remain stable changes radiographically as крупноволносцевых shadows of different shapes. Swollen lymph nodes in conserved. In bronchography lung cysts контрастуются relatively rare. According to our observations,

Suspected pulmonary cyst (cyst) occurs when a recurrence of pneumonia in the same regions of the lungs in the absence of reasons such as immune deficiency, cystic fibrosis, aspiration of food. However, air cyst found in the X-ray is not always possible, more reliable CT.

Differential diagnosis requires exclusion of cavitation, the bronchogenic cysts - cancer.

Treatment of inflammatory exacerbations same as pneumonia. At relapse or development of valve mechanism illustrated surgery.

Kartahenera syndrome - a syndrome of ciliary dyskinesia
Wada development triad of symptoms: the opposite of positions, bronchiectasis and chronic sinusitis. At the core lesion of the respiratory tract is a hereditary defect ciliary epithelium - no pens in dineinovyh eyelashes ciliary epithelium. Because they contain ATP, providing the movement of cilia, eyelashes still in these patients (ciliary dyskinesia syndrome or estate cilia). The result is stagnation of secretions in the airways, infection and formation of chronic inflammation. Dysfunction ciliary epithelium combined estate of sperm in men and dysfunction of the epithelium of the fallopian tubes in women. Kartahenera syndrome is a special case of ciliary dyskinesia syndrome, which some patients are not accompanied by a reversed internal organs.

Clinical manifestations usually appear early in life. After repeated respiratory diseases, bronchitis and pneumonia symptoms are chronic bronchopulmonary process. Typical restless also be subject to treatment difficult nasal lesions (recurrent sinusitis, rhinitis, adenoiditis). In some patients formed deformity of the chest and change the terminal phalanges. The main type of pulmonary fibrosis changes are limited to the deformation of the bronchi, often bilateral. A typical spread purulent endobronhit having intrusive flow. Patients with the syndrome described Kartahenera other defects (heart, kidney, polydactyly) and hypofunction of endocrine glands.

Ciliary dyskinesia in the lack of feedback that the positions of the detected repeated as bronchitis and pneumonia, development of chronic bronchitis, but many patients with lung pathology rough does not develop, which is clearly associated with a lower degree of dysfunction of cilia.

The diagnosis of the presence of the reverse arrangement is not difficult, in the absence of it can be confirmed by electron microscopy mucosa biopsies or bronchial congestion and mobility study cilia in phase-contrast microscope. As screening can be used saharynovyy test. It is time evaluating moving grains of saccharin placed on the lining of the nose to the nasopharynx (the examinee marks the appearance of sweet taste); normally this period is less than 30 minutes, the ciliary dyskinesia - much longer.

Treatment involves the repeated postural drainage of lifelong patient and energetic antibacterial treatment of pulmonary exacerbations and sinusytu. Indications for surgical treatment are extremely limited due to the prevalence of underlying defect and the consequent possibility of progression after resection of the lungs; remove experiences are the most affected areas of bronchiectasis light.

Symptom McLeod: characterized by a constant cough with sputum, bronchial episodes, one-sided process, often in the left lung, spasmolytic refractory to therapy, X-ray - unilateral pulmonary dystrophy (decrease with an increase of light airiness) biasing the shadow of the mediastinum.

Primary pulmonary hypertension. According to WHO recommendations of this disease are conditions in which increased pressure in the pulmonary artery and isolated myocardial hypertrophy of the right ventricle is not associated with other congenital disorders of the heart and lungs. In literature, the name of the disease Aerza. The cause is unknown and assume the role of inflammation and thrombosis of small branches of the pulmonary artery, pulmonary fence preservation of fetal hemodynamics of disease progression from the first day lives. Ting dominant inheritance, hereditary defect of fibrinolysis. For clinics are common dyspnea,
cyanosis, pain in the heart, chest, dizziness, coughing up blood, increased 2 tone of the pulmonary artery. Differentiated from congenital heart disease, kollagenozah, chronic bronchopulmonary diseases. Treatment - beta-blockers, aminophylline, corticosteroids.

Symptom Hammena-Rich similar to chronic pneumonia following features: continuous dry cough, intermittent fever, shortness of breath. Differences: 1. Diffuse alveolar interstitial fibrosis and vnutrishnoalveolyarnyy, especially around the vessels, bronchial and pleural changes. 2. X-ray - fibrosis, emphysema different size and intensity focal shadows, mostly in the lower and middle sections of the lungs. 3. Persistence of radiological changes. 4. Lack of effect of antibiotics. 5. Resistance to therapy. 6. The box percussion sound, dry or wet sounding variegated wheezing.

MUCOVISCIDOSIS (Kistofibroz pancreas) - Universal ekzokrynopatiya genetically determined - the most common inherited disorder of childhood. Clinical manifestations of the disease associated with dysfunction of the exocrine system, which determines changes in the organic and inorganic components secrets. Secret exocrine glands increased viscosity, making it difficult to evacuate it leads to blockage of ductless followed by secondary changes, most pronounced in bronchopulmonary and gastrointestinal tract. Often in the clinical impression of the respiratory tract in cystic fibrosis dominant manifestation of chronic bronchitis, but sometimes watching acute onset of pneumonia development that becomes recurrent prolonged duration. Sometimes recurrent pneumonia accompanied by abscess formation. Bronchopulmonary changes tend to progress, develop pneumofibrosis diffuse, emphysema, and bronchiecstasy common bronhiole-, cysts, atelectasis, restricted areas pneumosclerosis. Progressive diffuse chronic bronchopulmonary process of increasing obstruction leading to the formation of pulmonary hypertension and pulmonary heart. To severe complications include pneumothorax, pneumoempyema, hemoptysis and pulmonary bleeding. Frequent sinusitis. Fermentative pancreatic insufficiency, malabsorption syndrome, retarded physical development, changes in the liver, diabetes, eye impression - other manifestations of the disease. Diagnosis is based on a combination of clinical manifestations of the disease (intestinal and respiratory syndrome) and increased 2-5 times the level of electrolytes note. The concentration of chlorides note equal to or above 60 mM / l (navazhtsi weight at least 100 ml) during pilokarpinovoho test indicates cystic fibrosis. Sample Shvahmana (renthenoplivchastyy test) - determination of trypsin in fecal masses in a dilution of only 1 to 5, at a rate of 1:100. When ultrasound of the pancreas can sometimes find it zoom seal. Modern methods of screening: VM = sensitive test at a concentration of albumin in meconium than 20 mg / g dry matter test - immunodiffusion radial method for the quantitative determination of albumin in meconium. ICT test - determining the level of trypsin in dry blood spots by radionuclide assessment trypsynonodibnoyi immunoreactivity. at a rate of 1: 100. When ultrasound of the pancreas can sometimes find it zoom seal. Modern methods of screening: VM = sensitive test at a concentration of albumin in meconium than 20 mg / g dry matter test - immunodiffusion radial method for the quantitative determination of albumin in meconium. ICT test - determining the level of trypsin in dry blood spots by radionuclide assessment trypsynonodibnoyi immunoreactivity. at a rate of 1: 100. When ultrasound of the pancreas can sometimes find it zoom seal. Modern methods of screening: VM = sensitive test at a concentration of albumin in
meconium than 20 mg / g dry matter test - immunodiffusion radial method for the quantitative determination of albumin in meconium. ICT test - determining the level of trypsin in dry blood spots by radionuclide assessment trypsyanonodibnoy immunoreactivity.

Before the appointment of medical therapy ascertain possible allergic reactions to medications relatives of the child, child's susceptibility to allergic reactions.

It is important to promptly appoint adequate antibiotic therapy based etiological factor and the nature of the pathological process and general condition of the patient. The effect of antibiotic therapy depends on correctly matched the dose and route of administration of antibiotics during treatment. In most cases, a mild pneumonia treated with antibiotics for 7-10 days. After reaching effect (temperature drop, stop progression of clinical data and auscultation) continue antibiotic treatment for another 2-3 days. In general, the duration of antimicrobial treatment is determined by the patient. The criterion for discontinuation of antibiotics is the total elimination of clinical and diagnostic signs of pneumonia.

In severe pneumonia therapy should begin with parenteral antibiotics, but should firmly know that when clinical improvement of the patient should switch to shorter periods possible at the oral route taking the same drug (sequential therapy).

The main groups of antibiotics that can be used (in ranzhyrovanni order):
- semisynthetic penicillins,
- semisynthetic penicillins with clavulanic acid,
- cephalosporins,
- macrolides,
- aminoglycosides II-III generation (use gentamicin undesirable due to the lack of sensitivity to the antibiotic pneumococcus)
- derivatives of metronidazole (metronidazole, Metrogyl etc.)
- in some cases severe course of life-threatening - fluoroquinolones (appointed by children of 12 years).

To start empirical treatment of medium and medium-severe pneumonia in children can recommend the following scheme:
- Beta-lactam antibiotics (penicillins, penicillins, by clavulanic acid, cephalosporins or carbopinemy) plus macrolides.

Antibiotic therapy should be administered at the earliest possible timing, which will reduce mortality. By using a combination of antibiotics should be considered synergy and antagonism of their action.

Full effect of antibiotic - lowering body temperature to below 38° C for 24- 48 hours. in uncomplicated and 2-4doby - with complicated pneumonia improving the general condition of the patient, recovery of appetite, dyspnea reduction, normalization of laboratory parameters of blood. In this situation does not change antibiotic and parenteral drug substitute for oral administration. If no pharmacy enteral forms antybiotykiv- full course of antibiotic therapy is brought parenterally. Partial effect - saving fever longer than recommended improving the clinical and radiological absence of negative dynamics; change the antibiotic is required.

Lack of effect - preservation of fever in deterioration of growth of pathological changes in the lungs during physical examination and radiography in the lungs (the appearance of new foci of infiltration of the merger, the occurrence of pleural-
pulmonary complications). You need to replace the antibiotic. It is desirable to make based on the results of bacteriological examination of sputum..

The indication for use of other antibiotics have no clinical effect of the drug of first choice for 48-72 hours at 36-48 hours uncomplicated and complicated with pneumonia and the development of adverse drug reactions. Benchmarks for withdrawal of antibiotics, in addition to positive clinical dynamics, there is a tendency to normalization of X-ray pictures of blood parameters.

Prevention of complications of antibiotic therapy necessarily involves the appointment of vitamins, prolonged use of broad-spectrum antibiotics - the appointment of probiotics (multiprobiotics).

Expectorants on the mechanism of action: mukotsiliarnoho shown to improve clearance of tracheobronchial tree and antitussive agents - prolonged dry unproductive cough.

I. Drugs that stimulate coughing (sekretomotorni, rehidranty):
   A) reflex action: plant (Althea roots, grass and ran Ledum, dyv'yasyla rhizome and roots, leaves mother and stepmother, many others), semisynthetic and synthetic products and medicines based on medicinal plants;
   B) drugs resorptive action (potassium iodide, sodium bicarbonate, essential oils, etc.);

II. Preparations thinning bronchial secretions (bronhosekretolytichni, mucolytic):
   A) proteolytic enzymes - trypsin, chymotrypsin;
   B) Synthetic mucolytics - ambroxol hydrochloride, acetylcysteine, carbocisteine.

III. Antitussive drugs - pakseladyn, glauvent, tusyn, kodterpyn.

Remember that antihistamines are prescribed in some cases with severe eksudatovym component because they have a "drying" effect on the bronchial mucosa, increase non-productive cough, dangerous in case of an already viscous nature of the secret.

Antipyretics prescribed:
- Children under the age of 3 months at body temperature above 38oS - if febrile seizures in history,
- patients with temperature above 39-39,5oS,
- the deterioration of the child, the appearance of shaking, pale leather covers and other manifestations of toxicity - "pale version rush" along with antispasmodics peryferichnoyi action.

As antipyretics in children paracetamol and ibuprofen use, TSOH1,2 group. Appointment of aspirin is undesirable due to its hepatotoxic effects.

Physiotherapy treatment. INacute period physiotherapy treatment begins and using electric field UHF. Assign 5 - 7 sessions. Longer courses are not recommended because it can promote pnevmosklerotychnyh changes in lung tissue. UHF not appoint destructive pneumonia. Successfully used microwave therapy - effects of microwave (MW). Radiating power - 10 - 15 W, duration of procedure - 5 - 7 minutes. Sessions - 10 - 12. Unlike UHF microwave electric field does not act on the entire body and locally on the inflammatory site. Young children used inductothermy (with the use of low-power UHF). Used Power Output - 30 - 40 W, duration of session - from 5 to 10 minutes. (Depending on age). Course10-12 treatment sessions.
At the end of the UHF and microwave spend 10-15 amplipulsoforezu. Most amplipulsoforez use of nicotinic acid, calcium chloride or sulfate, copper, magnesium and eufillina. In preference expressed in lung fibrosis shows the use inductothermy, diathermy, amplipulsoforezu 3% solution of potassium iodide lidasa. Children with rickets during recovery prescribe a course of general ultraviolet irradiation (starting at 1/4 biodoses with an increase by the end of the course to 2 - 3 Biodoza). When purulent lesions formed in the lungs, spendtreatment UHF amplipulsoforez of platifillin and staphylococcal antyfahinom or proteolytic enzymes.

Therapeutic begins immediately after normalization of temperature or reduce it to subfebrilnoyi, exit child the state of toxicity and includes the appointment of special breathing exercises and chest massage (vibro massage, cupping massage).

It is expedient to heat wet inhalation and inhalation of mucolytics, herb, saline or inhalation of essential oils.

Restorative therapy. Aimed at improving defenses in a period of improvement and recovery, reduces the possibility of complications of pneumonia. With this in mind each child (especially young children) after acute period should be designed course adaptogens in combination with a multivitamin.

When antibiotics (especially broad spectrum antibiotics) is mandatory appointment of vitamins C, A, E, B orally. After finishing a course of antibiotics prescribed probiotics in case of disorders of the gastrointestinal tract and the presence of dysbiosis.

Immunotherapy, cardiovascular drugs, hepatoprotectors, infusion therapy, diuretics appointed by clinical indications.

**Topic:** Functional and organic diseases of the hepatobiliary system in children.

**Content topics:**
- Functional disorders of the biliary tract (FRBT) - functional motility disorders of the gallbladder (ZHМ) and (or) the tone of the sphincter apparatus due to inconsistent, untimely, insufficient or excessive contraction of the gallbladder and (or) sphincter apparatus. Polyetiological disease is genetically determined.

**Classification of functional disorders of the gallbladder and sphincter of Oddi** (Rome III consensus)

E. Functional disorders of the gallbladder and sphincter of Oddi

E1. Functional disorders of gallbladder

E2. Functional biliary sphincter of Oddi disorder

E3. Functional pancreatic disorder of the sphincter of Oddi

1. **Localization:**
   - gallbladder dysfunction.
   - sphincter of Oddi dysfunction.

2. **Etiology:**
   - A. Primary.
   - B. Secondary.

3. **Functional status:**
   - A. Hypofunction hyperfunction or gallbladder.
   - B. spasm or lack of sphincter of Oddi.
Clinical forms FRBT:
- hyperactivity, hypertension
- hyperkinetic-hypotonic
- hypotonic-hypokinetiс
- hypokinetiс-hypertonic

(MB variants combined with normal sphincter kinetics or gallbladder)

**Primary research:**

*Laboratory:* coprogram; biochemical serum, cholesterol, alkaline phosphatase, HHTP, bilirubin total and fractions, AST, ALT, amilyzy and lipase. Microscopic and biochemical bile (if biliary sludge).

*Instrumentation:* dynamic ultrasound cholecystography - ehosonohrafichne study to determine the functional status of the gallbladder and sphincter of Oddi (such as functional disorders of the biliary tract.)

According to the testimony: FYEHDS, w / cholecystography, fractional duodenal intubation, bacteriological, biochemical and microscopic examination of bile.

**The symptoms, syndromes, physical status.**

*Anamnesis* - disease duration of more than 3 - months, hereditary predisposition, anomalies of the gallbladder and bile ducts.

Complaints on hyperfunctionPain in the right upper quadrant, navel attacks, in 20-30 minutes after eating, physical or emotional stress, loss of appetite, fatigue, emotional lability.

Complaints on hypothyroidism: Dull, aching pain in the right upper quadrant after eating 60 - 90 min., Exercise; nausea, bitter taste in the mouth, fatigue, emotional lability, dizziness and others.

*Physical status* Pain on palpation in the right upper quadrant, navel, possible positive bladder symptoms with hypokinetic type - increase in liver size (soft, movable, painless, declining rapidly after application holekinetiki), perhaps - distal hyperhidrosis, pathological dermographism, susceptibility to hypertension, functional systolic murmur.

**Clinical syndromes** : Dyspeptic, pain, asthenic-vegetative, cholestatic.

**Diagnosis:** FRBT diagnosis (dyskinesia or dysfunction of the biliary tract) is determined by a set of complaints, anamnesis clinical, laboratory and instrumental methods.

**Laboratory studies:**

1. Coprogram - raising neutral fat, a significant increase in the number of fatty acids, intracellular starch, cellulose (lack zhovchoviddilennya).
2. Biochemical serum - raising cholesterol, alkaline phosphatase, HHTP, total bilirubin through direct fraction (subject to change with hypothyroidism gallbladder dysfunction SFO). Indicators of amylase / lipase within normal limits.

**Instrumental research:**

1. ultrasound.

To assess motor function of the gallbladder and sphincter of biliary tract using dynamic ultrasound cholecystography using holekinetychnoho breakfast (egg yolks, sorbitol solution, a solution Hofitol). Measuring the volume of the gallbladder (ZHM) is performed on an empty stomach and after stimulation at 5, 15, 30, 40, 60 minutes. With D & C cholecystography registered phase gallbladder. The first phase lasts for
4-6 min., Due to the state of the SFO, the extension of this phase indicates spasm SFO. The second phase lasts about 15 min., Caused as SFO, Extent ZHM reduced by 29-31%. The third phase lasts 30 min., Caused by the sphincter as Lyutkensa, Extent ZHM zmenshuyetssya further 30-35%. The fourth phase - ZHM further reduction of 33% - 65% compared to the Initial Extent. In hyperkinetic type of dysfunction ZHM gallbladder volume reduced more than 65% for 60-90 minutes. after taking holekinetiki; at hypokinetic type - less than 33%.

2. duodenal intubation, followed by biochemical studies (concentration of total bile acids, cholesterol, phospholipids, bilirubin determination index litohennosti) and bile microscopy (detection of cholesterol crystals, calcium bilirubinatu) to assess litohennosti bile.

3. holetsystopankreatohrafiya Endoscopic retrograde (ERCP) - extension of the common bile duct of 10 mm and a delay in the overall contrast bile duct (ZZHP) over 45 minutes. suggests toning SFO. A dysfunctional disorders to clarify the SFO and diagnostics in mechanical deterred ZZHP.

4. FEHDS - evaluation of the mucosa of the esophagus, stomach, and 12 duodenal ulcer; duodenal papilla.

**Diagnostic criteria for functional disorders of the gallbladder:**

Biliary pain combined with normal levels of liver enzymes, conjugated bilirubin, amylase / lipase (if possible hypokinetic type of dysfunction Moderate biochemical cholestasis syndrome - increased serum cholesterol, alkaline phosphatase, HHTP, total bilirubin through direct fraction); gallbladder dysfunction on the results of ultrasound.

**Diagnostic criteria for functional biliary disorders SFO:**

Biliary pain combined with normal levels of amylase / lipase; possible increase in transaminases, alkaline phosphatase, direct bilirubin fraction, time attacks associated with pain; ZZHP expansion after the simulated test fatty food - a sign of dysfunction SFO (hypertonicity).

**Diagnostic criteria for functional pancreatic disorder:**

Epigastric pain combined clinical signs of pancreatic cancer; possible biochemical signs of cholestasis syndrome manifestations.

**Characteristics of treatment.**

1. Dietary treatment:
2. Drug therapy.

A. hyperactivity (hypertonic) type:
- sedatives
- holespazmolicychni drugs
- choleretic
- physiotherapy:
- balneotherapy
- phytotherapy (zhovchohonni collection, mainly choleretic action).

B. hypokinetic (hypotension) type:
- Tonic preparations
- choleretic
- holekinetiki
- prokinetic:
• rinse 2 times a week number 5 - 7 in the form of hypotonic mineral water (average mineralization) 100 - 200 ml per admission, 25% solution of magnesium sulfate to 20 - 50 ml, 10-20% solution of sorbitol or xylitol 50-100 ml.
• physiotherapy, electrophoresis of magnesium sulfate in the right hypochondrium, sinusoidal modulated currents with a solution of mud, electrical gallbladder;
• balneotherapy: mineral water of average mineralization and the average gas saturation (Luzhanska, Morshyn, Glade Kvasova etc.) 3-5 ml per 1 kg of the reception, 3 times a day for 1 month ;
• phytotherapy (zhovchohonni meeting with choleretic, holekinetychnoyu action).

**Length of hospital treatment:** 2 weeks (possible treatment the conditions of a day hospital or outpatient).

**The goal of treatment:**
1) Kupiyuvaty symptoms of functional disorders of the biliary tract.
2) Normalize kinetics tonic function of the biliary tract.

**Requirements for treatment outcomes:** Absence of clinical signs and ehohrafichyh signs FRBT.

**Medical observation.** 3 years beyond the admissible period. With clinical supervision can be removed in the absence of lesions after laboratory and instrumental examination.

Overview pediatric gastroenterologists 2 times a year, pediatrician - 2 times a year; otolyarynholoh, dentist - if necessary.

**Chronic cholecystitis. Code C 81.1**
- Code C 81.1 - chronic cholecystitis
- Code C 83.0 - cholangitis
- Code C 83.0 - chronic holetsystoholanhyt (a form of inflammatory disease biliary tract localization, which occurs mainly in children)

**Definition:**
Chronic cholecystitis - gall bladder disease, which is based inflammatory changes in the gallbladder wall of various etiologies.
Chronic cholangitis - a chronic relapsing inflammatory disease of the bile ducts.
Chronic holetsystoholanhyt - Polyetiological chronic inflammatory disease of the gallbladder and bile ducts, combined with functional disorders of the gallbladder and bile ducts, changes in physical and chemical properties and biochemical structure of bile.

**Classification** chronic cholecystitis (with Nohaller AM)
1. According severity:A) mild b) moderate in) heavy form.
2. For stage diseaseA) exacerbation, b) incomplete clinical remission, c) remission (stable, unstable).
3. In the presence of complicationsA) not complicated, b) difficult.
4. The nature of the courseA) recurrent b) monotonous, c) which alternates.
Primary study.

Laboratory: Clinical blood test to determine the level of serum cholesterol, alkaline phosphatase, bilirubin (total and fractions), AST, ALT, HHTP, amylase / lipase "C" -reaktyvnho protein (CRP).

Instrumental: Ultrasound gallbladder and biliary tract, liver and pancreas; duodenal intubation, followed by microscopic and biochemical studies of bile. FEHDS, ERCP (when indicated).

The symptoms, syndromes, physical status.

Complaints: pain in the right upper quadrant, sometimes in the epigastrium, umbilicus (usually after physical or emotional stress, errors in diet, intercurrent diseases), possible irradiation in the right shoulder, right shoulder; nausea, vomiting bile, bitterness in the mouth, belching, violations stool (tendency to weaken or constipation), chronic nonspecific signs of intoxication.

Physical status: coated tongue, palpation pain in the right upper quadrant, epigastrum, umbilicus, positive bladder symptoms, increased liver size (no more than 3 cm from the edge of the rib, liver firm, not reduced after taking holekinetiki) moderate chronic nonspecific symptoms of intoxication.

Clinical syndromes: Dyspeptic, pain, asthenic-vegetative, cholestatic, chronic nonspecific toxicity.

Diagnosis.

Laboratory research:

1. Clinical analysis of blood - neutrophilic leukocytosis may be insignificant, the tendency to accelerate ESR (an exacerbation);
2. Biochemical serum - Moderate cholestasis syndrome (high cholesterol, HHTP, alkaline phosphatase, a tendency to increase total bilirubin through direct fraction), the top level of performance standards AST, ALT (acute stage, the stage of incomplete clinical remission), positive "C" - reactive protein (exacerbation); amylase / lipase serum within normal limits.
3. Microscopic examination of bile - indicators of inflammation (increased content of epithelial cells, leukocytes in the portions B and C, crystals components of bile -nepryami signs of exacerbation or incomplete clinical remission); detection of vegetative forms of protozoa.
4. Biochemical bile - increasing the concentration of free bile acids, cholesterol, bilirubin, bile lipids portions B and C in inflammation.
5. Bacteriological study of bile - the identification of pathogens and determine its sensitivity to antibiotics (prolonged course with frequent exacerbations).
6. Ultrasound - compaction and thickening of the walls of the gallbladder (2 mm), "sludge" in the gall bladder, the presence perefokalnoho inflammation of the liver parenchyma.

Characteristics of therapeutic measures:

In acute:

1. Bed rest for a period of exacerbation (3-5 days), then -schadnyy.
2. Diet within the table number 5 on Pevzner.
3. Antispasmodic therapy (see. Section FRBT)
4. Antibiotic therapy in acute (when expressed intoxication and inflammatory responses in peripheral blood):
• drugs present in the bile in high concentrations: penicillin. Antibiotics are appointed by the age standard doses.
• drugs that accumulate in the bile in sufficient concentrations to effect treatment, furazolidone (age dose).

The course of antibiotic therapy 7 - 10 days.

Given the bacteriological examination of bile antibacterial and antifungal agents appointed on the basis of sensitivity.

5. Symptomatic therapy - depends on the type of functional disorders of the biliary tract that accompany chronic holetsystoholanhyt; with antibiotic therapy can be performed simultaneously with the appointment of sedative and antispasmodic drugs (if necessary); enzymes; antacids, choleretic and holekinetiki appointed after completion of antimicrobial therapy.

6. Physiotherapy - depending on the type of functional disorders of the biliary tract outside the period of exacerbation; (Section FRBT).

7. Balneotherapy - for the same reasons.

Incomplete clinical remission: appointed by type of treatment of functional disorders of the biliary tract.

Length of hospital treatment: 3 weeks may permanently hold the initial rate, and then - in a day hospital or outpatient.

Clinical supervision.
Clinical supervision - 3 years beyond the period of exacerbation. With clinical supervision can be removed in the absence of lesions after complete laboratory - instrumental examination.

Antirecurrent treatment 2 times a year. The amount of treatment depends on the period of the disease and the condition of the child (diet, nutrition, herbal medicine, balneotherapy, physiotherapy, exercise therapy).

Spa treatment is recommended after 6 months after the exacerbation. Recommended balneohryazovi resorts (Truskavets, Transcarpathian group of resorts, etc.).


Content topics:
Irritable bowel syndrome (IBS)
Code for ICD-10: K58.
Irritable bowel syndrome (SEC) - functional intestinal disorder manifested abdominal pain and / or defecation disorders and / or flatulence.

Roman clinical criteria considered three options:
• prevalence of constipation;
• with predominant diarrhea;
• with pain and abdominal discomfort.

Chronic and / or recurrent functional disorders of the distal intestine of at least 12 weeks during the last 12 months, which is manifested by pain and / or discomfort in the abdomen, going after defecation, and are accompanied by changes in the
frequency and stool consistency, combined for 25% of the time the disease at least two of the symptoms of bowel dysfunction:
- Change stool frequency,
- konsystentsiyi feces,
- aktu defecation (mandatory actions, tenesmus, the need for additional efforts during defecation, feeling of incomplete bowel movements after it)
- vydilennyam mucus in the feces,
- meteoryzmom.

**Etiology and pathogenesis.** One of the main factors involved in the pathogenesis of IBS are disorders of the nervous and humoral regulation of motor function of the intestine.

**Leading clinical symptoms and syndromes in SEC:**

**Pain:**
- pain localized around the umbilical area, the right and left iliac areas
- increases after eating, with anxiety, emotional arousal during menstruation
- relieved by defecation
- there is no night

**Dyspeptic syndrome:**
- fragile stool
- excrement mixed with mucus
- often flatulence

**Hastrokolitychny syndrome:**
- compelling urge to defece immediately after eating

**Asthenic-neurotic syndrome:**
- characterized by headache (similar to migraine)
- feeling "coil" swallowing
- inability to sleep on the left side
- sleep disturbances
- state of fatigue
- mood lability
- general domestic concerns
- breach of condition not related to the quality of food
- urination disorders

**Laboratory and instrumental diagnostics.**
- General and biochemical analysis of blood - no changes coprogram - kashytsepodibnyy or diarrhea to the first portion of thick, mucus +, ++, ++++, blood - no,
- Sigmoidoscopy (colonoscopy), pain during insufflation air mucosa of normal appearance, often flushed with reinforced vascular figure, the imposition of mucus, high rigid folds with increased physiological sphincter tone.
- Iryhohrafiya: uneven filling colon, spastic phenomena in nyshodyaschomu departments and sigmoid colon area segmentation, deep, often haustration fast release of contrast and appearance of symptoms "cord."
- Additional research method - manometry: at bolonnomu roztyazhenni rectal pressure change rates.

**Differential diagnosis:** Constipation and encopresis organic origin, chronic colitis, allergic, medykoamentozni.

**Irritable bowel syndrome (IBS)**- is a diagnosis of exclusion, which requires a lot of tests and differential diagnosis of various diseases. Crucial in the examination of patients with colonoscopy and data proctosigmoidoscope

Particularly alarming are the symptoms of anxiety, which exclude the diagnosis of IBS:
- fever
- admixture of blood in the stool
- unwarranted weight loss
- gastrointestinal disorders, interrupting sleep
- changes in blood count (anemia, leukocytosis, neutrophilia, accelerated ESR)

**Basic principles of treatment of IBS in children:**

- kupiyuvaty pain and dyspeptic syndromes
- to eliminate violations of the motor-evacuation function of the intestine,
- correction of psycho-vegetative disorders.

**Correction food:** Children should limit intake of fatty and gas-forming foods, sometimes - excess fat, with constipation - on the contrary, increase the amount of dietary fibers (cereals, corn, beets, carrots, pumpkin, fruit, dried fruit, prunes). When pain syndrome and flatulence food should act only boiled or stewed in the future, a combination of raw and cooked vegetables and fruits chosen individually. When diarrhea is excluded milk, raw vegetables and fruits, in severe diarrhea food fray. From food exclude products individually poorly tolerated children.

**Pharmacotherapy with IBS in children:**

**In IBS with constipation mostly:**

- medicines that increase the amount of feces and facilitate defecation (lactulose preparations of psyllium)
- prokinetic
- enzymatic preparations containing bile and hemicellulose
- pre- and probiotics
- therapeutic agents (antidepressants) after consultation psychiatrist

**IBS with diarrhea usually:**

- tools with binders, and Obducing sorbentni properties, and when they are ineffective - drugs that reduce intestinal motility and tone
- pre- and probiotics
- enzymatic means
- therapeutic agents (antidepressants), after consulting a psychiatrist.

**IBS usually with abdominal pain and flatulence:**

- selective antispasmodics
- medicines that reduce flatulence
- pre- and probiotics
- psychotropic drugs (antidepressants) after consultation psychiatrist
**Functional constipation** - bowel dysfunction, which manifests itself in increasing intervals between acts of defecation (compared with individual physiological "norm") and its insufficient emptying.

The main causes of constipation is a disorder of motor activity of intestinal muscles, weakening the urge to defecate, or change the structure of the intestine closest to him of which interfere with the normal progress of the intestinal contents if any inconsistency between the volumes of the colon and intestinal contents.

*Risk factors for constipation in infants are:*
- genetic predisposition;
- poor nutrition nursing mothers;
- perinatal hypoxic-ischemic central nervous system (CNS ISU);
- muscular hypotonia (rickets, iron deficiency, hypothyroidism);
- intolerance to cow's milk protein;
- insufficient drinking regime;
- early (up to 3 months. life) switch to bottle-feeding;
- the use of infant formula high in iron;
- frequent changes mixtures for feeding.

The diagnosis of functional constipation criteria set under the Rome Consensus III (2006):

*Diagnosis establish the presence of children under 4 years of age for 1 month. at least 2 of the following signs:*
- two or less bowel movement per week
- at least 1 episode of fecal incontinence per week after mastering hygiene practices
- availability episodes delay defecation
- the presence of painful bowel movement or solid stool
- the presence of large quantities of faeces in the rectum
- the formation of "fecal stones" that can impede defecation

*The presence of the above signs accompanied:*
- irritability
- decrease in appetite
- feeling of early satiety

*These symptoms disappear immediately after defecation*

Experts who worked on the creation of the Rome criteria III (2006) separated functional disorders as a separate group of childhood diseases with two subgroups:
- disease of infants and young children;
- diseases of children and adolescents.

In the 1st subgroup isolate functional constipation, and 2nd - irritable bowel syndrome.

*Diagnostics* based on the need to review the anus, digital research anus rectoscopy, ultrasound colon, bowel radiography, scatological and bacteriological examination of feces.

*Differential diagnosis* performed with nonspecific bowel disease, irritable bowel syndrome, with abnormalities of the gut.

*Treatment* aimed at eliminating pain, correction of motor disorders and recovery reflex to defecate. To do this:
Correction food. In the nutrition of children living on breastfeeding, the importance of proper nutrition is the mother hold early introduction of fruit juices. When you start eating solid food should be given plenty of fluids and fiber. Children who are bottle-fed, appoint a mixture containing dietary fiber, gum, lactulose, probiotics, Nutrilon comfort Nutrilon 1 and 2-Semper Bifidus, Detolakt-bifidus.

For older children with hipomotorny dyskinesia intestine diet should include fruits and vegetables, berries, mostly cheese, prunes or apricots, apples, oil crops (olive, corn) on an empty stomach or stirring into yogurt (at night), buckwheat, oats, barley porridge honey (2-3 times a day). To get morning toilet must fulfill the morning "ritual": fasting drink room temperature water and vegetable oil. Showing refreshing mineral water with middle and high salinity.

Diet for hyper dyskinesia of the colon includes boiled vegetables, pay attention vegetable fats. Show the warm mineral waters with low salinity level. Now, recognizing the role of diet with constipation, proved that inadequate intake of dietary fiber (cellulose, lignin, gums) increases the risk of constipation. Therapeutic effect of constipation give wheat bran, seaweed, flax seed, and drugs from the group of dietary fiber: laminaryd, mukofalk.

Drug therapy
An important trend in treating children with Federal Law is to reduce gas formation and elimination of intestinal colic. For this purpose, simethicone preparations, means which enable mechanically evacuate feces (vapor tube, enema, glycerin candles, massage anus). However, these methods should not be used daily. It means "first aid".

Children with CNS ISU should be performed treatment with pediatric neurologists using vascular, nootropic drugs, vitamins of group B.

In spastic constipation in infants use within 3-5 days antispasmodics, contact heat, warm baths, candles with extract of belladonna, papaverine, novocaine.

Due to the fact that functional motility disorders of the colon caused by secondary and changes of nerve and / or humoral regulation, the appointment of treatment should always take into account the state of the autonomic nervous system and features a psycho-emotional sphere of the patient. Usually used means to stimulate gut motility, including prokinetic use drugs lactulose.

Non-medicament methods Influence: herbal medicine, physiotherapy, reflexology, massage and physiotherapy.

Ulcerative colitis
Code according to ICD-10: K51.

Ulcerative colitis (UC)- a chronic inflammatory disease of unexplained origin, which is clinically characterized by recurrent course, with periods of bloody diarrhea and diffuse-pathological inflammation in the colon. Inflammation is the prevalence of proximal rectum and rectal limited tovstokieshechnoyu and mucous.

The clinical picture. The disease develops gradually. The primary feature of most patients is the appearance of bright red blood drawn or mushy stools, some patients - gastrointestinal dysfunction for months prior to the release of blood.

Leading clinical symptoms and syndromes in NEC:

Pain:
- pain is cramping in nature
- pain occurs during defecation or before eating, often has no connection with food
- localized in the abdomen, left hipohastralniy area or around the navel
- pain accompanied by tenesmus

**Dyspeptic syndrome:**
- bloody diarrhea (frequency of which depends on the severity of the disease)
- flatulence
- nausea
- loss of appetite

**Intoxication syndrome:**
- presence of fever
- weakness, fatigue

Sometimes, along with a typical beginning of ulcerative colitis in children noted unusual expression, masking important disease. These symptoms include joint injuries, rarely - rash anulyarnoyi as nodular or erythema.

There are rapid, acute, chronic and recurrent course torpid form disease.

**Fulminant form** lasts several days and often fatal complications through early development.

Acute, chronic, recurrent and torpid form differentiated by the severity of symptoms. They may be accompanied by various local and systemic complications that are life threatening (perforation, pyosepticemia, anemia, dystrophy).

Severity distinguish mild, moderate and severe form.

In mild general condition of patients is not disturbed appetite remains, no shortage of weight gain, ESR within normal limits or slightly elevated. The level of total protein is not reduced, a small note hypergammaglobulinemia. Stool infrequent - 3-5 times a day, mushy with little admixture of mucus and blood. When proctosigmoidoscope show moderate activity process with distal lesions.

Children with pomirnotyazhkovu form of the disease noted violation of general condition, emptying 5-8 times a day, liquid, mixed with blood, mucus, pus. Laboratory method can determine the increase in ESR tends to shift leykohramy left anemia.

In severe stage of the disease patients general condition greatly disturbed, weakness, dizziness, loss of appetite, pain syndrome. Stool more than 10 times a day, smelly, liquid, mixed with blood, mucus, pus, mostly at night. Body weight is reduced by 20-25%. In the blood, noted a significant increase in erythrocyte sedimentation rate, leukocytosis with stab shift, toxic granularity of neutrophils, severe anemia, hypoproteinemia, dysproteinemia. The data show total colonoscopy bowel syndrome with maximum activity.

**Complication.** The most dangerous complication is perforation of the bowel. Local complications: massive intestinal bleeding, acute toxic dilatation of the colon, cracks and abscesses in anorectal area. With long-term course of the process (over 10 years) with total defeat there is a risk of malignancy, to detect any required annual endoscopic examination with biopsy.

**Diagnostic algorithm:**
Complete blood count (ESR determination, the number of red blood cells and hemoglobin, white blood cells, platelets) - often in severe
Total protein and protein fractions - often in severe
Blood Sugar -dvichi
Liver and kidney tests - often in severe
C-reactive protein (quantitative determination) - often in severe
Blood group and Rh factor disposable
Coprogram - twice
Repeated sowings fecal pathogens and worm eggs (to exclude infectious or parasitic nature)
Urinalysis - once
Endoscopic examination of the morphological study of biopsy specimens - "gold standard" - made in all cases to verify the diagnosis.
Morphological examination of biopsy samples, mostly reveals inflammatory infiltration of the mucosa, sometimes - submucosa, mucosal edema and hemorrhage in its ranks, krypt- abscesses, superficial ulcers, loss of cell bokalopodibnyh
- ECG once
- X-rays of the chest - once
- ultrasound of the abdomen - once

Treatment. Treat patients with ulcerative colitis in the hospital only. Treatment should be comprehensive, aimed at compensation of disturbed metabolic processes, eliminating the complications and improve the regenerative processes in the wall of the colon. We recommend bed rest. Assign diet number 4, dairy-free.

Drug therapy: Azo compounds sulfonamides purpose of salicylic acid reduced doses. These drugs include salazopirydazyn, salazodymetoksyn, sulfosalazyn, salazopiryn. Begin with a dose of 48 g per day. The course of treatment lasts 4-6 months.

According to the testimony appoint anhioprotektory, hormones and anabolic steroids.
Upon detection of metabolic and electrolyte imbalance conduct infusion therapy, including iron supplements to combat anemia, sometimes - red cell mass.
Local therapy is the use of microclysters salazopirydazynom, hydrocortisone, sea buckthorn oil, vinilinom, irradiation intestinal mucosa heliyneonovym laser.
Surgery indicated in severe, life-threatening complications and the ineffectiveness of complex conservative treatment.
Forecast depending on the severity of the disease, the nature of complications and efficacy of adjuvant therapy.

Crohn's disease
Code according to according to ICD-10, K-50.
Crohn's disease (HC) - a chronic relapsing disease of the digestive tract. Characterized by granulomatous inflammatory and ulcerative lesions of various parts of the entire digestive tract.
The etiology and pathogenesis is not completely clarified. Assume the role of viruses, bacteria and other psevdotuberkuloznyh.
Inflammatory infiltration initially localized in the submucosal layer and gradually spreads to all layers of the intestine. Typical oseredkovist lesions with fuzzy borders between healthy and affected areas.

Among the cellular elements in the infiltration area is dominated by lymphocytes and, to a lesser extent, neutrophils and plasma cells.

*The clinical picture of* ahvoryuvannya *beg* in gradually or sharply. In the initial period is characterized by pain. Its intensity varies - from mild to attacks. The pain associated with the consumption of food or bowel movements, diarrhea is rare, emptying mushy, with large admixture of mucus and pus.

Palpable show soldered conglomerates of several intestinal loops. In blood observed shift to the left, anemia, hypoproteinemia, electrolyte disturbances.

*Diagnosis.* Diagnosis is determined on the basis of history, clinics, results of laboratory and instrumental studies. During kolonofibroskopiyi mucosa looks stohanoyi blankets with a matte surface, vascular pattern is not defined in the future there aftopodibni elements with some superficial ulcerations and fibrin coating. In phase cracking determine single or multiple deep longitudinal ulcer defects mucosa looks like "paving".

*Differential diagnosis.* Differentiate from ulcerative colitis.

*Treatment.* Diet № 4. Assign salazopirydazyn (sulfosalazyn, salazopryn), corticosteroids, metronidazole, immunosuppressive, bacterial agents, symptomatic agents.

Surgical treatment is indicated in the perforation, profuse bleeding, intestinal obstruction.

**malabsorption syndrome.**

*Malabsorption syndrome (CMA)*- a set of clinical symptoms caused by disorders cavity, the wall, membrane transport and digestion in the small intestine, leading to changes exchangesubstances. In children, the most common forms of SMA are disaharidaznaya failure (including lactose intolerance) and celiac disease (gluten intolerance).

Malabsorption syndrome can be congenital and acquired.

The term "malabsorption syndrome" currently has more than 70 diseases and syndromes, which creates significant classification difficulties.

Acquired malabsorption syndrome observed in children with enteritis Viplaix disease, intestinal limfanhiektaziyeyu, short bowel syndrome, malignant tumors of the small intestine, chronic pancreatitis, cirrhosis of the liver. In 3% of patients with acquired malabsorption syndrome manifested allergy to cow's milk protein.

*Primary malabsorption* genetic changes resulting from the structure of small intestinal mucosa and genetically caused fermentopathy: deficiency in the mucous membrane of specific enzymes. This damages the absorption of sugars and amino acids such as tryptophan, protein crops (wheat, barley, rye, oats) - Gluten determined nespriymannya disaccharides.

*Secondary malabsorption* occurs on a background of chronic enteritis, Crohn's disease, Viplaix, exudative enteropathy, diverticulitis, if resection and tumors of the small intestine, pancreas disease and hepatobiliary system, amyloidosis, scleroderma, agammaglobulinemia, abetalipoproteinemiyi, lymphoma, heart failure, disorders of mesenteric circulation, tyrotoksykozu, hypopituitarism, poisoning, blood loss, vitamin deficiency, radiation injuries.
**exudative enteropathy** - a pathological condition characterized by loss of plasma proteins through the gastrointestinal tract and is accompanied by symptoms of impaired intestinal absorption.

There are primary and secondary forms of exudative enteropathy. Primary - abnormalities of the lymphatic system, and intestinal limfanhiektaziya chylangioma. Secondary causes of exudative enteropathy - celiac disease, gastroenteritis, intolerance to cow's milk protein, Viplaix disease, lymphoma, tuberculosis mesadenitis more. In these diseases tends to be "Escherichia limfanhiektaziya" which appears morphologically expansion of lymphatic vessels, interstitial edema. As a result, developing lymphatic stagnation with increasing hydrostatic pressure and protein intake in the lumen of the intestine with subsequent loss of stool and development of severe hypoproteinemia.

The disease often develops sharply after a year of life, the child is shown a lag in physical development, puffy syndrome, diarrhea, steatorrhea, severe hypoproteinemia. Hypoproteinemia developing very quickly because albumin, which synthesizes liver without replenishing lost from the bloodstream. Lack of protein in serum, it is the head of a factor of edema syndrome and nephrotic a pseudo-character. The degree of its severity varies - from small displays to anasarca. Increased plasma protein loss through the gastrointestinal tract and leads to a state hipohammahlobulinemiya a sharp decline in all classes of immunoglobulins, and therefore the children there is a tendency to chronic course of infection. Exudative enteropathy can run acutely or chronically transient.

**Main symptoms and syndromes in exudative enteropathy:**

**Syndrome trophic disorders:**
- drunk, height, proportions, trophic skin changes, gradual disappearance of subcutaneous fat layer, lower turgor, muscle mass.

**Syndrome of functional changes in the central nervous system:**
- changes in emotional tone, neuro reflex irritability, muscle hypo- and dystonia, hyporeflexia, lagging rates of psychomotor development, disturbed sleep, thermoregulation.

**Syndrome lowered food tolerance:**
- loss of appetite anorexia, weight curve flattening, dyspeptic disorders (regurgitation, vomiting, unstable emptying), reduced enzyme secretion and digestive tract function.

**Syndrome reduce reactivity immunobiologichnoyi:**
- frequent infectious and inflammatory diseases, erased or atypical course, toxic and septic conditions, secondary immunodeficiencies, violation of hematopoiesis.

**Dysaharydazna failure** and malabsorption of sugars - symptoms of gastrointestinal disorders due process violation disaccharide hydrolysis and transport in the intestinal mucosa.

**Laktazna failure**
Code according to according to ICD-10: E73


*Laktazna failure in children* - inbD-borne deficit halaktozidhidrolazy responsible for the metabolism of lactose-free food that spadkuyetsya for utosomno recessive pattern.

**Classification:**
- Primary (type Durand, type Holzel, primary lactose intolerance late-onset);
- Secondary (unfolds against the backdrop of chronic diseases, especially those that lead to atrophic processes mucosa of the small intestine);
- Constitutional (associated with the natural decline of enzyme activity after the introduction of baby foods diet).

**The main clinical symptoms and syndromes:**

**Pain:**
- abdominal pain, aggravated after taking milk

**Dyspeptic syndrome:**
- loss of appetite
- regurgitation, vomiting
- flatulence
- diarrhea with frequent watery feces foam

**Functional changes in the central nervous system:**
- changes in emotional tone
- lagging behind the pace of psychomotor development
- sleep disturbances

**Laboratory and instrumental diagnostics.**
Clinical analysis of blood - can thrombocytosis.
Clinical analysis of urine - proteinuria possible, leukocyturia, cylindruria.
Biochemical studies of blood, may increase cholesterol, ALT, AST.
Biochemical urine laktozuriya, hiperaminoatsyduriya.
Glycemic curve flattening under load lactose.
Coprogram: fecal acidic reaction (pH less than 5.5).
X-ray examination of the abdomen, excess liquid and gas in the lumen of the small intestine, it dyskinetychni disorders zmazanist relief mucosa.

Histochemical study of biopsies of the small intestine: a sharp reduction of galactose bD hydrolase.

Provocative test: the deterioration of the child (the appearance of diarrhea) after administration of lactose in the background to improve lactose diet.

**Characteristics of therapeutic measures.**

**Diet:** Complete exclusion from the diet lactose. Symptomatic therapy: adsorbents, enzyme preparations, vitamin and others.

Length of hospital treatment. Individually, to eliminate diarrhea.
Requirements for the results of treatment: removal of the clinical manifestations of the disease.

**Celiac disease**

Code according to according to ICD-10: K90.0
According to current data, one of the most common options malabsorption syndrome is celiac disease - a chronic immunodependent, genetically determined disease that is characterized by stable (life) nesprymannyam gluten (prolamin and glutenin) and structurally similar proteins of cereals to the development of non-specific lesion of the small intestine, chronic inflammation, atrophy mucous membrane of the small intestine and consequent malabsorption syndrome and the full restoration of the mucosa after excluding food gliadin wheat, oats and analohich them he fractions (rye - sekalin, barley - hordeyin).

The main clinical symptoms and syndromes in celiac disease:

**Lead is a gastrointestinal syndrome:**
- loss of appetite;
- nausea and vomiting;
- resistant to therapy diarrhea (watery bowel movements, light brown, gray or fat and foam with a distinctive unpleasant smell);
- possible constipation;
- polyexcrements and steatorrhea;
- hepatomegaly;
- increase in stomach

**Syndrome of functional changes in the central nervous system:**
- changes in emotional tone
- lagging behind the pace of psychomotor development
- sleep disturbances

**Syndrome trophic disorders:**
- weight loss or lack of increase
- degeneration, thinning of the subcutaneous fat basis
- a sharp decline in tissue turgor
- hipoproteininemichni swelling
- dry skin
- degenerative changes and brittle bones
- hair loss
- follicular hyperkeratosis
- breach of the evening
- cheilitis, hlossyt
- paresthesia loss of sensitivity;

**Rahitopodibnyy syndrome:**
- osteopenia, osteoporosis, pathological fractures
- decreased muscle tone
- bone pain, convulsions
- defects in tooth enamel, large cavities

**Hemorrhagic syndrome:**
- increased bleeding, epistaxis
- ferro- anemia, f oliyevo- and B12 deficiency

**Laboratory and instrumental diagnostics:**
complete blood count of reticulocytes;
serum iron, ferritin, blood cholesterol, total protein and protein fractions, serum immunoglobulins;
urinalysis;
coprogram, bacteriological examination of feces;
Ultrasound of the liver, biliary tract, pancreas;
esophagastroduodenoscopy with biopsy of the mucous membrane of the distal duodenum or empty intestines and histological examination of biopsy.

The diagnosis of celiac disease can be confirmed by serological methods, defining antigliadin antibodies (AGA IgG and AGA IgA) or antyendomizialni antibodies (EMA IgA).

Mainly in the treatment of celiac disease is strict adherence to a gluten-free diet.

Cystic fibrosis
Code according to according to ICD-10: E84

Cystic fibrosis - multi-tag hereditary disease that develops against the backdrop of products ekzokrynymy vital organs glands secretion viscosity increased with the development of secondary changes mainly in the respiratory and digestive system.

Clinical and physical status. Flutuence, vomiting mixed with bile mekonialny ileus, prolonged cholestatic jaundice in the newborn period, violations stool, slow weight gain (usually at birth), loss of appetite, loss of rectal "salty taste" by kissing, dehydration and "heat stroke" in hot weather, abdominal pain, intestinal obstruction, liver cirrhosis with portal hypertension syndrome manifestations of diabetes, chronic sinusyty, retarded sexual development, male sterility, diseases of the respiratory system (be repeated onhity and pneumonia, cough with thick purulent sputum, shortness of breath, respiratory failure).

Leading symptoms and syndromes:
Gastrointestinal syndrome:
- rich fat smelly ointment chair
- vomiting, flatulence
- ileus may develop at an early age, rectal prolapse
- hepatomegaly
- increase in stomach

Broncho-pulmonary syndrome:
- compulsive cough bronhoreya, shortness of breath
- Often the disease broncho-pulmonary system
- progression of chronic bronchopulmonary process with diffuse pneumofibrosis, bronchiectasis, cysts, atelectasis, limited areas pneumosclerosis
- chest deformity

Syndrome trophic disorders:
- weight loss or lack of increase, dystrophy
- thinning of the subcutaneous fat basis, a sharp decline tissue turgor
- thin limb deformation of the terminal phalanges as "drumsticks"
- degenerative changes and brittle bones, hair loss

Laboratory and instrumental diagnostics:
complete blood count, hemoglobin reduction;
coprogram: steatoreja 1st degree (high content of neutral fat), X-ray film positive test;
sudoriferous test: the high content of Na and Cl in sweat (more than 70 mg / dL);
Biochemical analysis of feces, a sharp increase in the total fatty acids trylehtritserydov, a sharp decline trypsin;
duodenal contents drastic reduction of pancreatic enzymes (trypsin, lipase, amylase)
  Ultrasonography of the abdomen - increase the size, compression, fibrosis of the pancreas; seals, liver fibrosis, manifestations of cholestasis, cirrhosis;
  reduction of fecal elastase-1;
  RÖ-obstezhenny GIT: change the size, shape and position of the duodenum, small intestine dyskinesia, rough terrain mucous, a large amount of mucus in the intestine, ileostaz;RÖ-hrafiya chest, bronchiectasis, fibrosis, areas of emphysema, atelectasis, bronchitis phenomenon or pneumonic lesions, perhaps - destructive changes;
  histological study of biopsies of the small intestine, bokalopodibnyh increase in the number of cells in the mucous membrane of the small intestine; histological examination of liver biopsy, focal or diffuse protein and fatty degeneration of hepatocytes, cholestasis phenomenon, fibrous transformation, biliary cirrhosis;
  DNA testing of the identification of two mutations of cystic fibrosis transmembrane regulator protein.

Treatment.
Correction food: the daily calorie content by 20 - 40% higher than normal due to the protein, limiting fat, extra salt introduction. Pancreatic enzymes (pancreatic enzymes preferably in the form of gastroresistant granules and microspheres) - the dose is determined individually, depending on the severity of malabsorption syndrome, but no more than 10 thousand. - 15 thousand. Units. lipase in1 kg weight per day. Prevention of liver cirrhosis, choleric and hepatoprotectors (ursodeoxycholic acid (ursofalk suspension, silymarin and others). Saving lung function - in marked changes in the respiratory system - lung transplant, Organocomplexes "lung-heart-liver".

Also abdominal pain may accompany with AGR:
✓ intestinal abnormalities;
✓ food allergies and intolerance to cow's milk protein;
✓ ulcerative colitis;
✓ Crohn's disease;
✓ Whipple's disease.

Diarrhea, combined with repeated vomiting can occur Whenthis diseases:
✓ Food allergies and intolerance to cow's milk protein;
✓ Zollinger
✓ Ellison (hastrynomy);
✓ intestinal abnormalities;
✓ abetalipoproteidemiya;
✓ deficit transkobalamin II.

Diarrhea combined with skin manifestations:
✓ Food allergies and intolerance to cow's milk protein;
at enteropatychnomu akrodermatyti;

**Differential diagnosis of diseases with malabsorption syndrome in children**

<table>
<thead>
<tr>
<th>The main precipitating factors of the disease</th>
<th>Dysaharydazna failure</th>
<th>Cystic fibrosis</th>
<th>Celiac disease</th>
<th>IBS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastrointestinal symptoms</td>
<td>The apparent deterioration after eating foods containing sugar, including milk</td>
<td>Often manifests in infancy in connection with the transition to mixed feeding baby</td>
<td>Communication with the introduction of a hliadinovmisnyh food products (semolina, herculean, oatmeal, etc.), perhaps with a latent period of 1 - 2 months</td>
<td>Deterioration chair against a background of a stressful situation</td>
</tr>
</tbody>
</table>

- **Dyspeptic syndrome**
  - At the heart failure is disaharidnqh secretory diarrhea - sparse foam with sour smell feces
  - Stool often in large volume, mushy, zlovonni, steatorrhea
  - There is osmotic diarrhea associated with a decrease in overall suction surface of the mucosa of the small intestine - expressed polyexcrements often gray, greasy stools
  - Alternating constipation and mushy stool or chair epizodykashkopolobnoho

- **Pain**
  - Abdominal pain that appears or is enhanced in patients receiving carbohydrates, including dairy products
  - Abdominal pain of varying intensity and localization
  - Abdominal pain of varying intensity and localization
  - Abdominal pain appears or increases against the backdrop of stress; relieved by defecation; Character not at night

The main purpose of treatment of diseases of the bowel, accompanied by malabsorption syndrome is to create conditions for better digestion and absorption of food. This correction is paramount child nutrition.

When fermenting dyspepsia limit the intake of carbohydrate (sugar, honey, jam) and products that are easily fermented (high in starch crops). When putrefactive dyspepsia limit the use of protein foods - meat, poultry.
Bread recommended only white in the form of dried or crackers. Useful jelly, omelets, steamed dish of minced meat and fish. During exacerbation should be limited to vegetable and fruit products, eliminate those that increase flatulence (peas, beans, cabbage). With the subsiding exacerbation expanding range of products. The diet should contain adequate amount of protein consumed as foods with low-fat meats, fish, cheese, eggs. Shown digestible protein soybeans. Animal fats should be used is limited. The diet must contain foods rich in calcium (cheese, cheese) and potassium (raisins, bananas, apricots). Dairy products are appointed for portability. It is best to include in the diet of dairy products (yogurt, sour milk, yogurt, bifidoprodukty).

When installing any enzyme deficiency exclude a product for which insufficient enzyme digestion. Thus, the shortage of lactase exclude milk. If gluten intolerance excluding cereals, but you can eat rice, corn starch, potato. Accurately determine which enzyme is not enough, you can at a special examination prescribed by pediatric gastroenterologists.

In allergic enteritis must complete exclusion plausible Product-allergen, such as strawberries, eggs. Difficult to choose a diet with a sensitivity to food additives (colorants, emulsifiers, preservatives). In such cases, it is recommended to use fresh ingredients in kind and the exclusion of so-called "obligate" allergens - chocolate, citrus, seafood.

Prevention of chronic diseases of the intestine is respect for the culture and food, timely treatment of acute intestinal infections and food poisoning. With early diagnosis and correct treatment prescribed in children does not develop malabsorption syndrome.

**Topic: Cholelithiasis in children.**

Content topics:

Gallstone disease (GSD) - a disease hepato-biliary system caused by the violation of cholesterol metabolism and (or) bilirubin, which is characterized by the formation of concretions in the gall bladder and (or) in the bile ducts.

**II. Diagnostic criteria:**

Medical history, clinical and paraclinical examination.

1. Asymptomatic form - accidental finding. Concretions located in the "silent" zone - the bottom of the gallbladder.

2. Biliary colic (connected with promotion of stone or spasm obturation biliary tract):
   - Leading symptom - pain -
   - in the area piloroduodenalnoy
   - in the right upper quadrant
   - acute or obtuse
   - irradiation in the right shoulder, lower back
Dyspeptic signs -
- decreased appetite
- nausea
- vomiting (possible bile)
- unstable stool, constipation often
- Asthenovegetative syndrome -
  - mood lability
- headache
- trouble sleeping
- mramornist skin
- Red dermographism
- Objective data:
  - possible ikterychnist sclera, skin
  - moderate jaundice, itchy skin without
  - emptying neokrasheni
- On palpation -
  - Protective tension of the front wall bryushnoyi
  - pain in the right upper quadrant
- Presence of symptoms: Ortner-Grekov - pain when tapped on the edge of the right edge; Zakharyin-Ged - Zone hyperesthesia skin in the right upper quadrant; Kera - palpable tenderness in the area of the gall bladder, especially when insufficiently; Murphy - severe tenderness in the gallbladder deeply insufficiently
- Complications:
  - calcification concretions
  - cholecystitis - fever, right upper quadrant pain, vomiting, not bringing relief
  - reactive hepatitis - increase in liver size, increased performance cytolysis, hamahlobulinemiya
  - stenosis of the common bile duct - severe jaundice of the skin, vomiting, severe pain

2. Laboratory tests:
- blood test - leukocytosis, elevated ESR
- Analysis of urine - increased content urobiline
- coprogram - without change
- total protein - without change
- total bilirubin with factions - moderate hyperbilirubinemia
- ALT, AST - a possible slight increase
- cholesterol and alkaline phosphatase - increased
- lipid metabolism (triglycerides, phospholipids, fatty acids neeteryfitsyrovani) increased.
- Instrumental methods:
  - Ultrasound - the main method of detection of concretions in the gall bladder and ducts;
  - X-ray examinations (oral intravenous cholecystography) - renthenkontrasnyh presence of concretions in the gallbladder. Contraindications contrast radiographic
studies of the biliary system: jaundice, severe allergic reactions (idiosyncrasy to iodine, renal failure);
- CT of the abdomen (more accurately detects the density of concretions);
- ERCP - suspected bile duct stones.

III. Basic principles of treatment (C):
The task of therapy zhovchnokamyanyoi disease:
- preventing the migration of stones and connected with these complications;
- reducing litohennosti bile;
- the elimination of metabolic disorders.

Treatment of latent or oligosymptomatic zhovchnokamyanyoi forms of the disease:
- Diet - table number 5 (by Pevsner) with the exception of cholesterol rich foods;
- Henoterapiya:
  a) drugs ursodeoxycholic acid (Ursofalk, Ursohol, Ursosan) at 10 mg / kg body weight per day [daily dose is divided into 2 doses (in the evening should be given daily doses 2/3)] for 6 - 24 months);
  c) preparations henodezoksyyholevoiy acid (Henofalk) at a rate of 15 mg / kg body weight per day (children over 12 years) for 3-24 months. The drug is taken once in the evening;
  d) hepatoprotectors (Silibor, Lioliv, Hofitol, Darsil) with reactive hepatitis after treatment of biliary colic.

Contraindications to conservative treatment:
§ acute inflammatory diseases of the gallbladder and bile ducts;
§ stones the size of more than 2.0 cm and their renthenopozytvnist;
§ nonfunctioning gallbladder;
§ ulcer;
§ chronic pancreatitis;
§ concomitant diabetes.

Treatment of biliary colic:
- antispasmodic - peripheral anticholinergics M:
  0.1% solution of atropine sulfate 0.2% solution platifillin hydrotartrate. For enhancement of M-holynolitykiv additional drugs used myotropic - 2% solution of papaverine hydrochloride 4% solution Nospanum;
- narcotic analgesics (50% solution analhinu 5% solution of tramadol) with a strong and constant pain;
- antibiotics (ampicillin, oxacillin, dondomitsyn, erythromycin, etc.) In the gallbladder inflammation, fever and laboratory signs of inflammation (leukocytosis in violation to the left).

Surgery (Laparoscopic cholecystectomy) only for special indications - not functioning gallbladder, common bile duct calculus, gangrene of the gallbladder.

Clinical supervision gastroenterologist 4 times a year. After the elimination of concretions 2 times a year.

Content topics:

Acute pancreatitis

In childhood acute pancreatitis is polyetiological disease. The main reasons for its development in children:

✓ trauma;
✓ infections (viruses, mumps, rubella, Coxsackie B, measles, infectious mononucleosis, herpes, cytomegalovirus, influenza, bacterial infection, mycoplasma, pseudotuberculosis, dysentery);
✓ worms;
✓ drugs (azathioprine, furosemide, sulfonamides, tetracycline, estrogen, 6-mercaptopurine, sulfasalazine, corticosteroids, metronidazole, nitrofurans, calcium, indomethacin, rifampicin, isoniazid, salicylates, cyclosporine) inflammatory and obstructive processes biliary tract (malformations of the common bile duct and major duodenal papilla);
✓ systemic connective tissue disease (systemic lupus erythematosus, nodular peripheral arteritis;
✓ metabolic diseases (diabetes, congenital hypertriglyceridemia);
✓ juvenile tropical pancreatic syndrome;
✓ allergy

Classification of acute pancreatitis

1. Interstytialnyy (swelling and hemorrhagic)
2. Necrotizing (pancreatic necrosis)
2.1. Complications of early pancreatic necrosis, shock, liver failure, disseminated intravascular c m, bleeding, diabetes.
2.2. Complications of pancreatic later: psevdokysty pancreatic abscesses, cellulitis, fistulas, peritonitis.

Clinic

Acute pancreatitis in children is often the type of interstitial flow. The painful abdominal syndrome is a major clinical manifestation of pancreatitis. The pain is intense, paroxysmal, shingles is localized in the middle of the epigastric region and left of the midline. Twinge triggered by errors in diet or arises after suffering bacterial and viral infections. The duration of pain lasting 1-24 hours. Pain decreases in supine position on the left side or stomach.

Dyspeptic syndrome is characterized by constant nausea, vomiting, not bringing relief. In exocrine pancreatic insufficiency maldyhestiyi develop signs: heavy, frothy, semi-liquid feces and flatulence. Syndrome of intoxication: skin pallor, tachycardia, headache, irritability, fever with high febrile digits.

During the examination of the child's condition significantly impaired by intoxication, pain syndrome, raising the temperature to febrile digits. The skin is pale, grayish-cyanotic, peryorbitalnyy cyanosis, disturbance of microcirculation in a petechial rash. On palpation of the abdomen, epigastric muscle tension (symptom Kert), pain in the left costal-vertebral angle (symptom Mayo - Robson), pain in the right costal-vertebral angle (symptom Desjardins) tensions left oblique abdominal
muses as painful strand, increasing pain at the point of fixing swinging. In many patients the pain Chauffard triangle (Fig. 15.3). For the differential diagnosis with the disease stomach and intestine performed by palpation of the pancreas grotto (three positions).

**Diagnostics**

1. **Laboratory methods exocrine pancreatic function:**
   - Clinical analysis of blood: leukocytosis, shift to the left, lymphocytosis, accelerated ESR;
   - determining the content of pancreatic enzymes in the blood (amylase, lipase, trypsin) and urine (amylase, lipase). In acute pancreatitis, amylase in blood and urine may be increased by 5-10 times;
   - determining the level of trypsin in blood serum. Increased levels of trypsin in the blood indicates an acute disease;
   - determining elastase 1 in feces. Elastase-1 - a proteolytic enzyme of the pancreas, which does not change its structure as far as the passage through the gastrointestinal tract. These circumstances suggest that the concentration of elastase-1 in fecal masses reflects the degree of exocrine pancreatic insufficiency. Normally the activity of elastase-1 in children aged 1 month is more than 200 mg / g of feces. Index of less than 100 mg / g of feces indicates a severe degree of pancreatic insufficiency;
   - lipidohrama. This method allows to determine the overall total amount of fat in the feces considering exogenous fat. Normally the amount of fat excreted in feces which shall not exceed 10% of the fat that comes from food. The method can be used to clarify the nature of steatorrhea, evaluating the effectiveness fermentoterapiya;
   - scatological study. Makroskopiya - evaluation of the appearance of stool: polyexcrements, gray excrement, zlovonnnyy smell. Microscopic examination are the following symptoms:
     - Steatoreja - the presence of fecal masses neutral fat (steatorrhea and degree); fatty acid soaps (steatorrhea II level); neutral fat, fatty acids, soaps (steatorrhea III level);
     - Creatorrhea - the presence of fecal masses large number of muscle fibers (++ or +++)
     - Amylorrhea - the presence of fecal masses large numbers of starch grains.

2. **Laboratory studies of endocrine pancreatic function:**
   - determination of fasting blood sugar;
   - glucose

3. **Instrumental methods:**
   - ultrasound, sensitivity is 70%. With it showed an increase in size gland parenchyma heterogeneity of structure, lower echogenicity of the parenchyma due to swelling and inflammation, enlargement pancreatic duct due to hypertension;
   - the gold standard in the diagnosis of pancreatic diseases is endoscopic retrograde holetsystoholanhiopankreatohrafiya. This method can detect pancreatic duct stenosis, identify structural changes Strait kaltsynaty and protein plugs;
   - imaging (computed, magnetic resonance) can detect change the size and structure of the pancreas, the presence of cysts, foci of necrosis.

**Differential diagnosis of acute pancreatitis is often carried out with:**
- acute diseases of the abdominal cavity (acute cholecystitis, pancreatitis, appendicitis, perforative stomach ulcer, intestinal obstruction, etc.).
- Diseases of the heart and blood vessels (myocardial infarction, thrombosis, mesenteric vessels).
- diseases of the chest (pneumonia, pleurisy) (Table. 1).

**Table №1**
Differential diagnosis of acute pancreatitis

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Acute pancreatitis</th>
<th>Acute cholecystitis</th>
<th>Perforative ulcer</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beginning</td>
<td>Acute</td>
<td>Gradual</td>
<td>Acute</td>
</tr>
<tr>
<td>The nature of pain</td>
<td>Dumb, intense</td>
<td>cramping</td>
<td>Acute</td>
</tr>
<tr>
<td>Localization of pain</td>
<td>Above the navel, in the left upper quadrant</td>
<td>In the right upper quadrant</td>
<td>Epigastric in the right abdomen</td>
</tr>
<tr>
<td>The irradiation of pain</td>
<td>In the lumbar region</td>
<td>On his right shoulder, shoulder blade</td>
<td>In the middle of the back</td>
</tr>
<tr>
<td>Anamnesis</td>
<td>Errors in diet</td>
<td>Errors in diet</td>
<td>Ulcerative history</td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>Vomiting without relief</td>
<td>Nausea</td>
<td>Nausea, vomiting fold</td>
</tr>
<tr>
<td>Palpation</td>
<td>Symptom Kert, sore point de Jardin</td>
<td>Tension in the right pidreber &quot;her, point Kera</td>
<td>Stress fracture around the abdomen</td>
</tr>
<tr>
<td>Percussion, auscultation</td>
<td>Flatulence, absence of intestinal noises</td>
<td>intestinal atony</td>
<td>Symptom Jobert &quot;deathly silence&quot;</td>
</tr>
<tr>
<td>Positive symptoms</td>
<td>Mayo-Robson, Resurrection</td>
<td>Murphy Myusi, George</td>
<td>SHCHetkina-Blumberg</td>
</tr>
<tr>
<td>Blood test</td>
<td>Polycythemia, leukocytosis, hyperglycemia, increased amylase, hypocalcemia, hypercoagulable</td>
<td>Leukocytosis, bilirubinemia</td>
<td>Leukocytosis</td>
</tr>
<tr>
<td>urine</td>
<td>Diastazuriya, hyaline cylinders</td>
<td>The positive reaction to the bile pigments</td>
<td>hyaline cylinders</td>
</tr>
</tbody>
</table>

Differential diagnosis of acute pancreatitis

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Acute appendicitis</th>
<th>Intestinal obstruction</th>
<th>mesenteric infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beginning</td>
<td>Gradual</td>
<td>Gradual</td>
<td>Acute</td>
</tr>
<tr>
<td>-----------</td>
<td>---------</td>
<td>---------</td>
<td>-------</td>
</tr>
<tr>
<td>The nature of pain</td>
<td>Permanent, increasing gradually</td>
<td>Colicky, colicky</td>
<td>Standing, cramping, increase the attack</td>
</tr>
<tr>
<td>Localization of pain</td>
<td>Right iliac area</td>
<td>In the lower abdomen</td>
<td>Diffuse in the lower abdomen</td>
</tr>
<tr>
<td>Anamnesis</td>
<td>Epigastric pain of transition in the right iliac area</td>
<td>Eating foods with lots of fat, heavy meals</td>
<td>Diseases of the heart, blood vessels</td>
</tr>
<tr>
<td>Nausea, vomiting</td>
<td>Perhaps the single vomiting</td>
<td>Nausea, vomiting, often with unpleasant odor</td>
<td>Nausea</td>
</tr>
<tr>
<td>Palpation</td>
<td>Defense in the right lower abdomen</td>
<td>Defense limited</td>
<td>Defense limited</td>
</tr>
<tr>
<td>Percussion, auscultation</td>
<td>-</td>
<td>Metal-tympanic sound</td>
<td>Metal-tympanic sound of blunting zone</td>
</tr>
<tr>
<td>Positive symptoms</td>
<td>Symptom SHCHetkina-Blumberg</td>
<td>Symptoms Val, Kivulya, Sklyarov</td>
<td>Symptom Mondor</td>
</tr>
<tr>
<td>Radiography</td>
<td>-</td>
<td>Bowls Kloybera</td>
<td>-</td>
</tr>
<tr>
<td>Blood test urine</td>
<td>Leukocytosis</td>
<td>Leukocytosis</td>
<td>Leukocytosis</td>
</tr>
</tbody>
</table>

*Food poisoning* in addition to the relevant history, characterized by less intense abdominal pain, compared with pancreatitis, the pain has herpes nature accompanied by enhanced intestinal motility and diarrhea. Diastase urine increased. No specific symptoms of pancreatitis.

*Nyzhnochastikova pneumonia.* The disease begins with acute fever, chills, pain in the left side of the chest. Pain increases when coughing, which is dry at first, then after 2-3 days, there is phlegm. The patient is spared the affected side. On examination - redness of face, shortness of breath. Chest behind in breathing. Auscultation - weakened breathing hard. In the blood - hiperlykotsytoz. Urine does not indicate increasing diastase. There are no symptoms of pancreatitis phase flow. X-ray can detect fire eclipse.

Myocardial infarction is localized pain in the epigastric, often has herpes character, he characterized collapse, shock, intestinal paresis, shortness of breath, cold sweat. But in myocardial infarction show a sharp general weakness early in the disease, there is no increase of diastase and urine amylase levels. Negative symptoms Mayo-Robson, the Resurrection, Kert, Mondor, Gray-Turner, Halstead, coulaines. ECG data - splitting the T wave, lower voltage always speak in favor of myocardial infarction.
Treatment

A particularly important role in all phases of patient treatment acute pancreatitis plays diet. In the first 3 days dosed shown hunger and drink, providing pancreas physiological rest. Standing nasogastric tube suction of gastric contents. While improving the state diet very gradually expand, first introduced pureed oatmeal in water, then milk, slimy soup, tea, biscuits, scrambled eggs, then cheese, milk pudding, jelly, milk, white bread. As the severity of pain recommend a diet - table number 5 on Pevzner with reduced fat to 55-60 g per day of carbohydrates to 250-300 g per day and increase protein to 80th120 ga day through dairy products, eggs, low-fat varieties of meat and fish. Eliminates sokohinni and Bile products: raw fruits and vegetables, juices, vegetable oils, meat broth, spicy dishes, mushrooms, coffee, chocolate. The following foods that contain natural inhibitors of enzymes - egg whites, oatmeal, soybeans, potatoes. The diet should be strictly observed for 6 months.

1) Symptomatic treatment of pain:
   - Pain Therapy begins with the appointment of narcotic analgesics (paracetamol, metamizole sodium) in the absence of contraindications - NSAIDs (ibuprofen);
   - if necessary (severe, persistent pain) - narcotic analgesics short courses (tramadol);
   - antispasmodics (mebeverin, drotaverine, papaverine).

2) The sphincter of Oddi dysfunction correcting tone - Assign selective spazmolityniv (mebeverin).

3) If symptoms of exocrine insufficiency software functions are assigned polenzyme drugs (pancreatin) at 25,000 - 40,000 U lipase to the main meal and 10,000 - 20,000 U lipase on minority meal. Algorithm purpose replacement enzyme therapy is given in Annex 2.

4) To reduce external secretion software (creating "functional rest" software) appointed:
   - proton pump inhibitors in the standard dose 2 times a day;
   - octreotide (in complicated CPs).

5) To correct vitamin deficiency appointed vitamins (mono and combined): menadione / fitomenadion, retinol, ergocalciferol, tocopherol, multivitamin complexes that contain these vitamins.

6) the treatment of exacerbations and complications of CP in a hospital designated infusion therapy substitutes and perfusion solutions for the purpose of detoxification:
   - electrolytes in combination with other drugs: Sodium lactate sorbitol + + + Sodium chloride, calcium chloride, potassium chloride + + Magnesium chloride / v - 400 ml per day;
   - blood protein fractions: albumin solution humans 10% solution - 100 ml / in a day;
   - carbohydrates: glucose 5 - 10% solution - 500 ml / in a day.

7) If necessary, reduce the risk or treat bacterial complications XII appointed antibacterial drugs used to treat infections of the gastrointestinal tract, taking into account the type of possible pathogen that infects Software: carbapenems, fluoroquinolones, cephalosporins III - IV generations nitroimidazole derivatives.
8) In secondary pancreatitis that developed due to other digestive diseases, treatment of the underlying disease is appointed by the current medical and technical documents.

**Chronic pancreatitis**

*Chronic pancreatitis* Usually the result of acute pancreatitis, so most of the etiological causes of the last full can be attributed to the causes of chronic pancreatitis. In children, chronic pancreatitis develops against the backdrop of digestive diseases, especially bile ducts and duodenum.

Exacerbation of chronic pancreatitis develops gradually and is characterized by latent course in which there are symptoms of exocrine insufficiency, bloating, polyexcrements, emptying fat, shiny, with scatological study steatorrhea exhibit varying degrees. Against the backdrop of increasing the number of enzymes in the blood, there is pain that is caused vnutrishprokopokovoyu hypertension perypankreatychnym inflammation. Clinical manifestations exacerbation of chronic pancreatitis - namely pain, dyspeptic symptoms, symptoms of chronic toxicity The external review - identical clinical acute pancreatitis. On palpation the abdomen observed diffuse pain, most pronounced in the under-umbilical area and the projection area of the head and body of the pancreas. Positive symptoms: Mayo - Robson, Kacha, Desjardins,

Recurrent option chronic pancreatitis characterized by alternating periods of exacerbation and remission.

**Diagnostics**

1. **Laboratory methods exocrine pancreatic function:**
   - determining the content of pancreatic enzymes in the blood (amylase, lipase, trypsin) and urine (Amylase, lipase). Normal activity of enzymes in the blood and urine do not exclude zahostrennyahronichnoho pancreatitis. In this case provokatsiynitesty diagnostic use. Tests conducted with the use of drugs that stimulate cancer diyalnistpidshlunkovoyi, secretin, pankreozyminu, Proserinum, glucose.

   *Prozerynovovyy test* - definition content in urine amylase activity after stimulation of the pancreas Proserinum.

   *The test of glucose* (Double load of glucose) is registered amylase levels when administered glucose stimulates the synthesis of pancreatic juice.

   *Renthenoplivkovyy test* - approximate method for determination of trypsin activity in feces.

   *Yodlipolovyy test*. The principle of the method is that adopted yodlipol inside, consisting of fatty substances (lipolu) and iodine under the action of pancreatic lipase converted into free iodine released in the urine;
   - determining the level of trypsin in blood serum. Lowering blood levels of trypsin suggests chronic disease;
   - determining elastase 1 in feces. Evaluation of fecal elastase-1 helps only in defining the severe lack of exocrine function that does not correlate with the severity of morphological changes in chronic pancreatitis;
   - lipidohrama. This method allows to determine the overall total amount of fat in the feces considering exogenous fat. Normally the amount of fat excreted in feces...
which shall not exceed 10% of the fat that comes from food. The method can be used to clarify the nature of steatorrhea, evaluating the effectiveness fermentoterapiya;

- scatological study. Makroskopiya - evaluation of the appearance of feces, in lifekaliya, gray excrement, zlovonnyy smell. Microscopic examination are the following symptoms:
  
  Steatoreja - the presence of fecal masses neutral fat (steatorrhea and degree); fatty acid soaps (steatorrhea II level); neutral fat, fatty acids, soaps (steatorrhea III);

  Creatorrhea - the presence of fecal masses large number of muscle fibers (++ or +++)

  Amylorrhea - the presence of fecal masses large numbers of starch grains.

- "Gold standard" assessment of the pancreas is the secretin-pankreozymovyy test (SPT). This is determined by the level of bicarbonate and enzymes after intravenous administration of secretin and pankreozyminu (direct stimulation of the pancreas). In addition, for the assessment of pancreatic cancer, especially for the diagnosis of a serious condition (cystic fibrosis), the pankreolaurylovyy test (PLT) - indirect stimulation of the pancreas.

2. Laboratory studies of the endocrine function of the pancreas:

- determination of fasting blood sugar;
- glucose

3. Instrumental Methods:

- ultrasound. Chronic pancreatitis exhibit heterogeneity roses division echo signals in the tissue of the pancreas, pancreatic duct expansion, increase in size, increased echogenicity of the parenchyma, inequality external contours, the presence of calcifications or cysts;

- imaging (computed, magnetic resonance) can detect change the size and structure of the pancreas, the presence of cysts, foci of necrosis, endoscopic retrograde pankreatoholanhiohrafiya can detect pancreatic duct stenosis, identify structural changes Strait kaltsynaty and protein plug. Differential diagnosis spend with chronic digestive diseases: chronic gastroduodenitis, chronic cholecystitis, bowel disease.

Treatment of exacerbation of chronic pancreatitis conducting a similar treatment of acute pancreatitis:

- elimination of pain a functional peace pancreas reduce its secretory activity;
- correction of exocrine insufficiency.

Clinical supervision. In remission after discharge from the hospital under the supervision of the child is transferred to a gastroenterologist. Particular attention is paid to dietary regime - a diet number 5 within 6-12 months after the exacerbation. In the first year after exacerbation child examined each month. Once in 3 months necessary to conduct blood chemistry and determination of amylase in blood and urine. Clinical examination is compulsory component preventive treatment, which is carried out 2 times a year, within 4-6 weeks and is substitution fermentoterapiya (controlled coprogram), physiotherapy (paraffin, ozocerite baths, inductothermy) herbal medicine. The successful result in the prevention of relapse of chronic pancreatitis giving spa treatment (Truskavets, Morshyn, Mirgorod)

Pancreatic cysts
Pancreatic cysts - fluid-filled cavity lesions, breaking pancreatic epithelium lined inside (true) or no epithelial lining (false). Acute pancreatitis uskladnyuetsya cyst in 3.5-11% of patients. Much more often (25% of cases) cyst formation after injury appears pancreas. Congenital and parasitic cysts account for about 3% of all cysts.

**Clinical symptoms.**

Clinical signs of pancreatic cysts significant inherent polymorphism. It is caused by etiological factors, mechanisms kistoutvorennya differences, the localization of cysts in the abdomen.

With small size cysts clinical signs. Symptoms cyst caused mostly basic disease against which it originated, the presence of the cyst and its complications. Signs of recurrent chronic pancreatitis dominate the clinical picture of cysts formed due to inflammation. Persistent symptoms are typical of pancreatitis pain localized in the epigastric and left subcostal areas radiating to the lumbar area and left shoulder, often zoster character. The pain is constant, dull sometimes arching. This is due to the increase in size of the cyst and compression bodies that surround it. Strong, growing pain occurs when the cyst complications, suppuration, hemorrhage in its cavity, cysts in the next breakout body or in the free abdominal cavity.

Constant and characteristic feature is the presence of tumor cyst formation that is palpable in the abdomen. Dimensions cyst in a short time can then be increased then decreased. With the rapid and significant increase in the size of the cyst revealed signs of acute pancreatitis. Reducing cysts caused by partial zporozhnennyam it through the ductal system cancer; while improving the condition of patients.

Manifestations endocrine gland failure are nausea, vomiting, regurgitation, defecation disorders. Typical signs are unstable character of stool (constipation and diarrhea), the presence in the feces undigested food.

Patients concerned about the general weakness, fast fatigue, weight loss, lack of appetite. As the cysts increase in size there are signs of compression of the neighboring organs and anatomical lesions, intestinal obstruction, jaundice, segmental portal hypertension.

**Laboratory and instrumental methods of diagnosis:**

1. Complete blood count, urine, feces, coprogram;
2. Biochemical analysis of blood (sugar, bilirubin, total protein, residual nitrogen, urea, creatinine, alkaline phosphatase, electrolytes);
3. glycemic, hlyukozurychnyy profile;
4. Ultrasonography of the abdomen
5. X-ray of the chest;
6. CT scan of the abdomen;
7. EFHDS;
8. ERCP;
9. Duodenohrafiya on a background of artificial hypotension;
10. Laparoscopic ultrasound pankreatohrafiya;

**Differential diagnosis.**

When pancreatic cysts should be a differential diagnosis of:
1. Tumors of the stomach.
2. tumors (cysts) of the liver.
3. gallbladder hydrops.
4. pancreatic cancer.
5. splenomegaly.
7. renal tumors.
8. aneurysm abdominal aorta.

*Tumors of the stomach.* Any damage to the stomach, usually palpable tumor antrum and lesser curvature. This is a manifestation of cancerous diseases, anemia, cachexia, effects of stenosis, dysphagia. When X-ray showing signs of straight walls, including changes in the mucous relief. These feature different from "filling defects" caused by compression of the stomach wall pancreatic cyst.

*Liver tumors.* Often in the left lobe. Assist to conduct a differential diagnosis, the results of ultrasound, isotope scan of the liver and selective angiography.

*Dropsy gallbladder.* The final diagnostic methods should be considered as ultrasound and X-ray study of the biliary tract.

*Cancer of the pancreas.* Sometimes reaches a considerable size at which they can be palpated. They usually accompanied by severe pain, common symptoms of cancer disease. When cancerous lesions of the pancreatic head syndrome occurs early in obstructive jaundice. The final diagnosis is verified, computed tomography, ultrasound and relaxation duodenohrificiya.

*Splenomegaly.* Difficulties arise when intimate joined capsule cysts of the spleen. In such cases, even when laparoscopy is not always possible to clearly verify the concerned authorities in the wide infiltrative-adhesive conglomerate. The most reliable method of diagnosis is selective angiography, ultrasound and computed tomography.

*Cancer of the colon.* Large tumors located in the colon area splenic angle, accompanied by severe violation of permeability of the colon. The final diagnosis is made using irrigoskopiiya and ultrasound.

*Tumors of the kidneys.* (Hipernefroma, hydronephrosis). In order to verify the diagnosis of urological necessary to conduct complex studies (excretory urography, retrograde pyelography, renoskanuvannya, US).

*Aneurysm of the abdominal aorta.* It differs from the presence of cysts side ripple palpation. Sometimes aneurysm listen on typical noise. The final studies for final diagnosis should be considered aortography, ultrasound, computed tomography.

**Topic:** Intestinal toxicity syndrome in children with eksykozom.

Content topics:

*Intestinal toxemia* - a severe form of acute indigestion, accompanied by intoxication, dehydration, violation hemodyna-Economy, the central nervous system, kidneys and liver. Intestinal toxemia and eksykoz (dehydration) observed mostly in acute intestinal infections in young children, but the risk of development is also in infants with malnutrition, overheating, SARS when there is vomiting and watery bowel movements; is associated with high permeability intestine for toxic substances and products infringed splitting nutrients tendency of the body to generalized reactions intoxication, stress and imperfect regulation of water and electrolyte metabolism.
In normal excretion of water and electrolytes from the digestive tract is small, so that their stool stands no more than 5% of the daily volume of faeces. When digestive disorders these losses greatly increased. Renal function is broken quickly and does not provide enough water and salt reabsorption in the tubules (even in normal circumstances they lack sensitivity to aldosterone), resulting in kidney child "bad save water." The smaller the child, the greater her overall water content as a percentage of body weight (newborn - 80% child 5 years of age - 62%), including extracellular fluid (newborn - 40- 50%, 5-year-old child age - 22%). Extracellular fluid less tightly fixed that promotes intensive metabolism in healthy children.

The main clinical signs eksykozu and the need for fluid to combat dehydration

<table>
<thead>
<tr>
<th>The degree and type eksykozu</th>
<th>Clinical signs</th>
<th>The amount of liquid, in milliliters per 1 kg body weight per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>And the degree of (water deficit) - an acute shortage of liquid (5% of body weight)</td>
<td>Moderate dry skin and mucous membranes of moderate retraction Fontanelle, thirst, anxiety. The level of sodium in blood serum - 134,5 ± 3,6 mg / dl (normal - 140 mg / dL). The level of potassium - 4.0 mmol / L ± 0,2 mmol / L (normal - 5.0 mmol / l)</td>
<td>130-170</td>
</tr>
<tr>
<td>AP degree (isotonic) - acute fluid deficit (5-10% of body weight)</td>
<td>Acute onset, severe dry skin and mucous membranes without retraction Fontanelle, oliguria, fever, thirst, anxiety child tachycardia. Level sodium serum raised to 151-155 mg / dL. Potassium level decreased to 3,3-3,5 mmol / l</td>
<td>175</td>
</tr>
<tr>
<td>NO degree (soledefitsytnyy, hypotonic) - an acute shortage liquid (10% of body and more)</td>
<td>The main clinic - lack of fluid in the bloodstream (hypovolemia) due to loss of electrolytes during vomiting, diarrhea. Start gradually, mucous membrane is not very dry, skin cold, with marble pattern, wet. Sclerema, Fontanelle retraction body temperature is normal or low-grade,</td>
<td>220</td>
</tr>
</tbody>
</table>
lethargy, refusal to drink, paresis intestines, bloating, oliguria. Low level of sodium to 119 mmol / L potassium - to 3.1 mmol / L. Hematocrit - 0,39-0,55 (normal - 0,27-0,43). Anhidremichnyy can develop hypovolemic shock syndrome trombohemorahichnyy

Basic principles of oral rehydration
When the disease mild and moderate rehydration therapy conducted by oral administration of glucose-saline solutions, "Hlyukosolanu" "Tsytrahlyukosolanu" "rehydron" "Ora-fly". In the absence of these drugs can be used sugar-salt solution (1 teaspoon salt, 8 teaspoons of sugar, 5 cups of drinking water, add 1 teaspoon of baking soda).

Primary rehydration (within 4-6 hours) directed at correcting fluid and electrolyte deficits that occurred before treatment; Fractional held portions, often - 2-4 teaspoons of solution every 10-20 minutes. Subsequent rehydration aimed at maintaining and restoring the loss of water and salt ongoing maintenance of current needs in a liquid suspension to diarrheal syndrome and restoring fluid and electrolyte balance. Every 4-6 hours the child is introduced as a solution, as it has lost during this time, given the amount of food that a child receives during food restriction. The inability to accurately count after each bowel movement introduced the approximate amount of liquid: children under 2 years - 50-100 ml after 2 years - 100-200 ml of water and salt solution. If desired, a child can be digested drink water, tea, broth rodzynkovym, karotynovoyu mixture broth blueberries and others. In conducting oral rehydration to children the first 3 months of life, with malnutrition, drinking is always injected with saline (in the ratio 1: 2) for the prevention of hypernatremia.

The scheme of remedial measures:
- cleansing enema (digested water, pale pink solution of potassium permanganate, 1% solution of soda);
- depending on the degree eksykozu start putting salt solutions inside (I-II degree - rehydron, oralit etc.) and intravenous (III degree). Introduction isotonic sodium chloride and 5% glucose solution in the ratio 1: 3 (children under 1 year) or 1: 2 (children after a year). Dehydration should begin therapy with the introduction of 3% sodium bicarbonate at a dose of 5 ml per 1 kg of body weight;
- In the absence of vomiting - the introduction Enterosorbents (smectite, enterosgel et al.);
- antibiotic therapy for persistent intoxication syndrome (ampicillin, gentamicin, ceftriaxone, etc.);


Content topics:
Acute renal failure (ARF) - a syndrome characterized by the sudden and rapid decline in glomerular filtration and is clinically decrease in urine output, violation azotovydilnoyi function, fluid and electrolyte and other exchange lesions of virtually all organs and systems.

**Etiology.**

<table>
<thead>
<tr>
<th>prerenal</th>
<th>renal</th>
<th>Postrenalni</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypovolemia</td>
<td>Glomerulonephritis</td>
<td>obstructive uropathy</td>
</tr>
<tr>
<td>(Blood loss, burns, loss of fluids through the gastrointestinal tract, kidney and adrenal gland with loss of salt hepatorenalnyy syndrome);</td>
<td>(pilislyastrepto-coccal, lupus, idiopathic), anaphylactoid purpura, localized intra-vascular coagulation, thrombosis, renal vein necrosis of the medulla, the effect of salts of heavy metals, pharmaceuticals, chemicals, hemoglobin, myoglobin, ischemia, tumors, kystoz, uric acid nephropathy, hereditary nephritis.</td>
<td>Pelvis-ureter segment</td>
</tr>
<tr>
<td>Hypotension</td>
<td></td>
<td>ureterocele, tumors, bladder-ureter reflux, stones, blood clots.</td>
</tr>
<tr>
<td>(Septicemia, DIC, hypothermia, heart failure, hemorrhage);</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypoxia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Pneumonia, impaired respiratory syndrome).</td>
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<td></td>
</tr>
</tbody>
</table>

**Clinic and over.**

*And the stage. Elementary*

Duration - 6-24 hours. The clinical picture is dominated by symptoms of the underlying disease and decrease in urine output.

II stage. Olihoanurichna

Duration - from a few days to 3 weeks. The clinical picture - the defeat of almost all organs and systems: oligoanuria, azotemia, encephalopathy, electrolyte and acid-base disturbances, changes in the cardiovascular system (expanding the boundaries of the heart, tachycardia, arrhythmia, pericarditis), violation of the respiratory system (dyspnea, interstitial pulmonary edema), changes in the gastrointestinal tract (dry mouth, nausea, vomiting, diarrhea, abdominal pain, intestinal paresis), skin manifestations (dryness, ikterychnist, hemorrhagic petechiae, ecchymosis), changes in the urinary system (swelling, back pain ).

The third stage. Recovery diuresis

Duration - 5-15 days. It is noted a gradual increase in urine output or a sudden increase in the urine.

The fourth stage. Recovery

Duration - 6-24 months. Slow recovery of partial kidney function, normalization of water-electrolyte and acid-base balance. The disappearance of the symptoms of organs and systems.

Diagnosis.

*Blood:* Hb and morphology of erythrocytes, leukocytes and their formula, platelets, K +, Na +, bicarbonate, protein, albumin, creatinine, blood gases, blood cultures for sterility.

*Urine:* an.sechi general, urine microflora, osmolality, Na +, urea, creatinine.

*Functional methods:* X-ray of the chest, abdomen X-ray inspection, X-ray bone, knee, ultrasound of the kidneys and bladder.
Differential diagnosis. It is necessary to conduct differential diagnosis between functional and organic renal failure. The most common cause of functional renal failure is acute geodynamics disorder caused by hypovolemia, decreased blood pressure, circulatory centralization compensatory followed by a decrease in renal blood flow. Increased aldosterone excretion in functional LV increases the reabsorption of sodium, which excludes damage to the tubules, whereas the organic ARF these conflicting data - transport function tubules significantly affected. In some cases, the functional tests:

- of vasodilator drugs (aminophylline) - after the introduction of reduced urine output, which excludes organic ARF;
- appointment of provocative and alkaline water load - w / 2% of body weight (0.85% district sodium chloride, 10% of the district of glucose with insulin); if under the influence of the child intended infusion hourly urine output reaches the age norm, and the relative density decreases to 1010-1015, it indicates the functional ARF;
- appointment of high doses saluretics. Lack diuretic response characteristic organic ARF.

ARF should be differentiated from acute decompensation of chronic renal failure, which is latent, acute glomerulonephritis and shvydkoprohresuyuchym, recurrent chronic pyelonephritis.

Resuscitation measures:

<table>
<thead>
<tr>
<th>State</th>
<th>Correction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Circulatory</td>
<td>- plasma or saline solution - 20 ml / kg intravenously.</td>
</tr>
<tr>
<td>collapse</td>
<td>- Dopamine - 1.10 mg / kg / min., Intravenous calcium gluconate (10%) 0.5 ml / kg, intravenously, Calcium-rezonium - 1 g / kg / day, per os or per rectum, insulin - 0 1 U / kg and glucose - 0.3 g / kg, intravenously.</td>
</tr>
<tr>
<td>Hyperkalemia</td>
<td>- Calcium gluconate (10%) - 0.5 ml / kg intravenously.</td>
</tr>
<tr>
<td>Seizures</td>
<td>- Magnesium sulfate (50%) - 0.1 ml / kg intramuscularly.</td>
</tr>
<tr>
<td>Hypocalcemia</td>
<td>- Diazepam - 0.25 mg / kg intravenously.</td>
</tr>
<tr>
<td>Hipomahnezemiy a</td>
<td>- Calcium gluconate (10%) - 0.5 ml / kg intravenously.</td>
</tr>
<tr>
<td>Acidosis</td>
<td>- Sodium bicarbonate - 2 mg / kg intravenously.</td>
</tr>
<tr>
<td>Hypertension</td>
<td>- Hidralazin - 0.2 mg / kg intravenously.</td>
</tr>
<tr>
<td>Edema</td>
<td>- Furosemide - 1-5 mg / kg intravenously.</td>
</tr>
<tr>
<td>hyperphosphatemia</td>
<td>- Alyudroks - 1 ml / kg / day, per os.</td>
</tr>
<tr>
<td>Sepsis</td>
<td>- Antibiotics intravenously.</td>
</tr>
</tbody>
</table>

Indications for hemodialysis:

- Fluid overload with pulmonary edema and daily gain in body weight of more than 5.7%;
- Uremic intoxication (urea serum - more than 24 mg / dL, creatinine - 500 mmol / l. The daily increase in serum urea at 21,4-28,5 mmol / (L x day) and creatinine 180-440 mmol / (l x d);
- Nekorehuyema hyperkalemia - more than 7.5 mmol / L;
- Acidosis that is difficult to correct;
Absence of positive dynamics on the background of conservative treatment (anuria over 2 days);
The increase in neurological symptoms and convulsions.

**Forecast.** Mortality - 20-40%. Lower mortality extracorporeal detoxification methods.

**Clinical. Rehabilitation.** The duration is 3 years. Review child after 1 month. after discharge from hospital, after - after 3 months., further - every 6 months. controlled Al. blood biochemical tests, performance tests and Reberga Zimnitskiy. Optional radiographic studies carried out but not earlier than in 1-2 years after discharge.

**Chronic renal failure** (CRF) - non-specific syndrome that develops as a result of hereditary, congenital and acquired diseases of the kidneys, resulting in progressive loss of nephrons and the stroma of the steady decline in the ability to perform kidney homeostatic functions.

**Etiopathogenesis.** Drawbacks number and structure of renal cystic dysplasia, malformations pyelocaliceal complex and ureter, renal vein thrombosis, hemolytic uremic syndrome, primary glomerulonephritis and secondary (against the background of diffuse connective tissue diseases).
The development of renal failure due to the decrease in mass due to functioning nephrons glomerular sclerosis, tubular atrophy and interstitial sclerosis.

**Classification.**
I. tubular kidney failure (normal creatinine in the blood).
II. Total kidney failure.
And II - the concentration of creatinine 170-440 mmol / l;
II b - 441- creatinine concentration of 880 mmol / l;
II in - the concentration of creatinine than 880 mmol / l.

And the stage. Compensated. Volume renal function 80-50% of normal.
II stage. Subcompensated. Volume renal function 50-30% of normal.
The third stage. Decompensated. Volume renal function less than 30% of normal.
The fourth stage. Terminal. Residual renal function is less than 5%.

**Clinic.**

<table>
<thead>
<tr>
<th>Syndromes</th>
<th>Clinical manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Violation of growth and development</td>
<td>Hipostatura. The absence or underdevelopment of secondary sexual characteristics. Decreasing supply and growth.</td>
</tr>
<tr>
<td>Azotemia (uremia)</td>
<td>Asthenia, anorexia, neurological disorders, Gastroenterocolitis, pericarditis.</td>
</tr>
<tr>
<td>Anemia</td>
<td>Lethargy, pale skin, degenerative changes in the organs.</td>
</tr>
<tr>
<td>Fluid and electrolyte imbalance</td>
<td>Clinical syndromes, depending on the preferences hiperkaliemiyi, hipokaltsiemiyi, hiponatriemiyi, edematous syndrome.</td>
</tr>
<tr>
<td>Diagnosis and treatment of early stages of CRF is aimed at:</td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>- identify risk groups and ongoing clinical supervision for children with chronic kidney disease (CKD);</td>
<td></td>
</tr>
<tr>
<td>- Assign specific therapy aimed at correcting the underlying cause that led to the development of chronic kidney disease;</td>
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</tr>
<tr>
<td>- timely surgical treatment of patients with congenital abnormalities of the urinary tract (in most cases prevents the development of renal failure);</td>
<td></td>
</tr>
<tr>
<td>- detection and treatment of opportunistic diseases;</td>
<td></td>
</tr>
<tr>
<td>- delay the loss of kidney function - renoprotektsiya;</td>
<td></td>
</tr>
<tr>
<td>- prevention and treatment of CKD complications or manifestations (such as hypertension, proteinuria, anemia, acidosis, delayed physical development, skeletal deformation rahytopodibni);</td>
<td></td>
</tr>
<tr>
<td>- beginning preparations for the treatment of terminal renal failure (creatinine more than 0.35 mmol / l).</td>
<td></td>
</tr>
</tbody>
</table>

The plan of remedial measures

1. Correction / liquidation proteinuria, ACE inhibitors, Sartany, moksonydin, felodipine, diltyazem, lerkandypin; glycosaminoglycans; amino acids).
2. The correction reduced the relative density of urine (Fanconi syndrome): ACE inhibitors; Sartany; moksonydin, felodipine, diltiazem, lercanidipine, glycosaminoglycans.
3. Correction hypertension to normal age parameters: ACE inhibitors; Sartany; moksonydin, felodipine, diltiazem, lercanidipine.
4. Correction of anemia to normal age parameters (over 120 g / l), iron, folic acid, erythropoietin.
5. Correction to the growth of normal age-related values (growth hormone).
6. Correction of the end products of protein metabolism to normal (sorbents, ketosteryl + nyzkobilkova diet, the latter only at stage 4 CRF).
7. Correction of calcium-phosphorus metabolism and hyperparathyroidism (calcium + vitamin active metabolite D).

**Indications kidney transplantation:**
- the age of 3 years with an increase in neurological symptoms, delayed growth, progressive osteodystrophy;
- age 3 to 7 years old - growth delay, uremia that symptomatic, significant osteodystrophy with symptoms of uremia;
- age 7 to 15 years - Clinic uremia, creatinine clearance less than 5 ml / (min. · 1,73 m 2), progressive osteodystrophy, psychosocial problems.

**Topic: Tubulopatiyi children. Interstitial nephritis.**

Content topics:

- **Tubulopatiyi** - a large group of illnesses which are based on the infringement kanaltsiyevoho transport of organic substances and electrolytes.

**Etiology.**
- hereditary disorders tubular transport systems;
- acquired metabolic disease due to the disorder of metabolism out of the nephron;
- changes in the structure tsytomembran dysplasia;
- inflammatory kidney disease.

**Classification.**

<table>
<thead>
<tr>
<th>Localization of lesions</th>
<th>Tubulopatiyi primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>- proximal tubules</td>
<td>The disease where Toni-Debre-Fanconi, hlyukozaminovyy diabetes, glycosuria, phosphate diabetes iminoatsyduriya (tsystinuriya, imunohlitserynemiya disease Hartnapa, hlytserynuriya, renal tubular acidosis type</td>
<td>Tsystinoz syndrome Lowe, tyrozynemiya, galactosemia, glycogen storage disease, poisoning, heavy metal salts, Lysol, tetracycline, Wilson disease, primary hiperparatireoidizm, hypophosphatasia, celiac disease, primary hyperoxaluria,</td>
</tr>
</tbody>
</table>
- The total damage to the tubular device
- Renal diabetes insipidus, renal tubular acidosis, and type pseudohypoaldosteronism.
- Chronic renal failure, Fanconi nephronoptyz
- Diabetes, kantynuriya.

**Phosphate-diabetes** - linked dominant X-linked or autosomal dominant disorder with profound disorders of phosphorus-calcium metabolism; tubulopatiya with advantage in clinical symptoms skeletal abnormalities.

**Эtiolohiya pathogenesis.** Believed that the phosphate-enzymatic processes affected by diabetes foreground vitamin D to the active substance or hormone receptor reduced sensitivity to intestinal epithelium of these metabolites. Reabsorption of calcium in the intestines is reduced.

**Diagnostic criteria:**
2. Delay height, curvature of the legs, skeletal deformity;
3. Hypophosphatemia, hipokaltseemiya, increased alkaline phosphatase levels;
4. Giperfossaturiya, aminoacidurija;
5. Increased parathyroid function.

**Differential diagnosis:** vitamin D-resistant rickets (beginning in 1.5-3 months, the first clinical manifestation in the form of changes in the central nervous system - irritability, restlessness, muscle hypotonia, kraniotabes, frontal, parietal hillocks, rachitic "chotky" and "bangles" deformation of the feet, systemic osteoporosis); hypophosphatasia, celiac disease.

**Therapeutic tactics:**
1. Diet: increasing foods rich in calcium.
2. General activities (gymnastics, massage, walking).
3. Vitamin therapy D2. The starting dose of 10.000-25.000 IU / day. Duration 4-6 weeks. Once - in addition designate another 10.000-15.000 IU / day to increased levels of phosphate in the blood.
4. The mixture Albright - orally.

**Renal tubular acidosis** - tubulopatiya margin of clinical abnormalities of the skeleton.

<table>
<thead>
<tr>
<th>Age</th>
<th>Type and</th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 months. - 2 years</td>
<td>Delayed growth, changes in rahitopodibni bones crisis dehydration and polyuria, nephrocalcinosis, tuhovuhist, heterochromia iridum iris, interstitial nephritis, pyelonephritis.</td>
<td>Delayed growth, rahitopodibni changes in the bones, vomiting, fever, polyuria, polydipsia, nephrocalcinosis.</td>
</tr>
</tbody>
</table>

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**Renal tubular acidosis**

<table>
<thead>
<tr>
<th></th>
<th>Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>3 - 18 months.</td>
</tr>
<tr>
<td>Clinic</td>
<td>Delayed growth, changes in rhahitopodibni changes in the bones, vomiting, fever, polyuria, polydipsia, nephrocalcinosis.</td>
</tr>
<tr>
<td>Paraclinica</td>
<td>Hypokalaemia, metabolic acidosis, hiperkaltsyuriya, hiperkaliiyuriya, alkaline urine pH.</td>
</tr>
</tbody>
</table>
Osteoporosis, valgus deformity of the bones. Nephrolithiasis.

| Differential diagnosis | Likavannya amphotericin B, nephrocalcinosis due to: hiperkaltsyemiyi, hiperparatyreoidizmi, hypervitaminosis D; obstructive uropathy; Ehlers Danlos Syndrome--; to lulolom intoxication. | Tsystinoz, galactosemia, poisoning, heavy metal salts, hiperparatyreoidizm vitamin D-deficient rickets, a disease Lee tyrozynoz, Wilson's disease, hiperimunohlobulinemiya. |

**Therapeutic tactics:**
2. General activities (gymnastics, massage, walking).
3. Correction of acidosis, sodium bicarbonate, and type - 1-3 mEq / kg / day, type II - 5-15 mEq / kg / day in 3-4 hours.
4. Correction of hypokalaemia: 7.5% solution of potassium chloride at a dose of 2 mg / kg / day.
5. Correction hipokaltsyemiyi 10% solution of calcium gluconate intravenously 10 ml.
6. Vitamin therapy D2 - at a dose of 50,000 IU / day.

**The disease where Toni-Debre-Fanconi** - tubulopatiya margin of clinical symptoms skeletal abnormalities.

**Diagnostic criteria:**
2. The delay physical and mental development rahytopodibni changes in skeletal bone pain;
3. Muscle and hypotension;
4. Thirst, vomiting, constipation, polyuria, dehydration symptoms;
5. Hypokalaemia, metabolic acidosis;
6. Phosphaturia, aminoaciduria, glucosuria, proteinuria, hypo, izostenuriya.

**Pattern inspection:**
- General clinical blood;
- levels of total and free calcium, phosphorus, alkaline phosphatase, pH, serum bicarbonate; 1.25 (OH) D, 25 (OH) D serum;
- Serum PTH levels;
- level tubular reabsorption of phosphate (KRB), glomerular filtration rate (GFR) (defining correlation KRF / GFR);
- X-ray bones;
- densitometry bones;
- Ultrasound of the kidneys;
- CT and MRI;
- consulting podiatrist, dentist, neurologist.

**Differential diagnosis:** rickets, phosphate diabetes, renal tubular acidosis, osteopathy with chronic renal failure, glycogen storage disease, tsystinoz.

**Therapeutic tactics:**
1. Diet: potato-cabbage diet, alkaline drinking.
2. General activities (gymnastics, massage, walking).
3. Vitamin therapy D2. The starting dose of 10,000-25,000 IU / day. Duration 4-6 weeks. Once - in addition designate another 10,000-15,000 IU / day to increased levels of phosphate in the blood.
   Mahurlit 0.5-2 g - 3 times a day, or blemaren - to prevent nephrolithiasis

**Topic: Acute heart and vascular insufficiency in children.**

Content topics:

Heart failure (HF) is called heart failure to provide blood flow that meets the metabolic needs of the body.

This - multifactorial disease in which a primary dysfunction of the heart leads to a number of hemodynamic, neural and hormonal adaptive responses aimed at maintaining circulation in accordance with the requirement of working classification of acute heart failure determining clinical options such as left ventricular, right ventricular, and arrhythmogenic total. There is also a classification under which isolated systolic, diastolic heart failure and combined. Systolic heart failure resulting from myocardial damage or cardiac overload. Overloading can be caused by pressure (eg aortic stenosis) or be voluminous (eg, ventricular septal defect). Diastolic heart failure form may be due to a violation of relaxation processes myocardial diastole phase (eg, obstructive cardiomyopathy), decreasing about

The main causes of acute left ventricular heart failure is a disease of the myocardium (acute carditis, cardiomyopathy, etc.). Reducing the contractile function of left ventricular myocardium, which emerged sharply, leading to reduction in stroke volume and cardiac blood. The heart is notable to pump the necessary amount of blood to the periphery. Aggravated course of acute left ventricular failure and the fact that unstruck the right ventricle pumps blood to the left, which can translate venous return in the adequate cardiac output. In the cavity of the left ventricle (LV) diastolic retained residual blood volume, and by increasing the amount of the increases in it and diastolic pressure. Rising pressure in the left ventricle leads to increase it in the left atrium and the development of stagnation in the venous vasculature of the pulmonary circulation (passive, venous, retrograde hypertension in the pulmonary circulation). In the crowded blood of the pulmonary veins and capillaries sharply increased hydrostatic pressure. Pathophysiological prerequisite of acute left ventricular heart failure is extremely unfavorable conditions of flow in the coronary vessels of the left ventricle, which is only a phase of diastole and is intermittent in nature, as opposed to blood flow in the coronary vessels of the right ventricle. As a result, any decrease in cardiac output leads to a clear reduction in coronary blood flow and further reduce contractile ability miokarda.

Rozvynutyy syndrome "small release" of the left ventricle leads to reduced systemic blood flow and related circulatory hypoxia, which acts as a trigger activation sympathoadrenal systems, which is a protective and adaptive reaction to a stressful situation. Activation of this system leads the release of catecholamines, and as a result there is a generalized vasoconstriction, increased contractility of the myocardium, tachycardia develops.
All of this maintains blood flow at a certain level. This protective mechanism has little spare capacity and quickly exhausts them. Hypoxia - trigger not only sympathoadrenal system, it activates the release of biologically active substances (histamine, serotonin, kinins, prostaglandins), under which there is a spasm of vessels of the pulmonary circulation, which further increases the hydrostatic pressure in them increases the permeability of capillaries. Increased hydrostatic pressure in the vessels of the pulmonary circulation promotes cardiac asthma and pulmonary edema. Equally important protective compensatory mechanism in acute and subacute heart failure is to enhance myocardial contraction according to the Frank-Starling law, the essence of which is that the power of heart rate depends on the initial length of myofibrils, muscle contraction strength is determined by the degree of stretching directly before the reduction. This mechanism is very important in increasing overload (increased venous return to the heart). Klinichno is a large pale skin (vasoconstriction of peripheral vessels) and tachycardia. Pulse thready, weak content or even poorly defined. Dyspnea provided left ventricular heart failure is characterized by accelerated breathing without noticeable changes in its depth and rhythm. Shortness of cardiac origin, usually inspiratory,

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By percussion showed an increase in heart size at the expense of the left ventricle, which is confirmed by echocardiographic and radiologic studies. Auscultation of the heart weakened sharply, and over the top and the tone can not listen at all. Its disappearance is the result of the weakening of muscle tone and valve components, due to the weakening of the contractile ability of the myocardium, a significant increase in left ventricular cavity and stretching the fibrous ring of the left atrioventricular opening. The appearance of systolic murmur from the epicenter of the apex of the heart associated with the development of relative failure of the left atrioventricular valve. There is emphasis II tone above the base of the heart. Typically, listen protodiastolichnyy (ventricular) gallop, caused by the emergence of third ton in early diastole. Pathological gallop developed at a time when the blood increased rapidly enters the left ventricle contractility of which significantly znyzhena. Dlya acute left ventricular heart failure is characterized by increasing bcc, due to the following factors: 1) the movement of fluid from the interstitial space (reduction of hydrostatic pressure); 2) the release of additional blood volume of the depot; 3) increasing the reabsorption of sodium and water in the proximal renal tubules. Increasing BCC to a certain extent be regarded as a protective compensatory
mechanism occurring in response to insufficient release of blood from the left ventricle. However, the more the BCC, the greater the burden falls on the affected serse.Na ECG voltage can be lowered, but in most cases it high. Registered livohrama, often negative T wave in the left chest, and in the second standard and avL leads. Moreover, the gradual disappearance of the negative T wave indicates a favorable course of myocarditis and effectiveness likuvannya.Po the progression of heart failure, pressure increases not only in the pulmonary veins and capillaries, but in arterioles. Increased hydrostatic pressure and capillary permeability causes extravasation liquid part of blood in the interstitial space (interstitial edema) develops clinical cardiac asthma. This occurs when the hydrostatic pressure in the pulmonary capillaries increases to 28-30 mm Hg. c., that he urivnyuyetsya oncotic pressure of the blood. In the prodrome cardiac asthma patients complain of general weakness, headache, feeling of tightness in the chest, increase shortness of breath, dry cough. This wheezing in the lungs does not listen. Clinic cardiac asthma is characterized by attacks of breathlessness inspiratory arising on a background of heart inspiratory dyspnea. The cause of asthma is inspiratory interstitial nabryak.Pid an attack of asthma patient becomes restless, covered with cold sweat, suffering facial expression. The attack often starts with a cough, shortness of breath and then increases that goes into breathlessness. Increased blood pressure, there is increasing dyspnea and tachycardia, increased respiratory work and supporting muscles. Prysmokuvalna action forced inhalation is increased blood supply vessels of the pulmonary circulation. Thus, increasing the load on the heart and reduces its effectiveness. Growing central cyanosis. Pulse weak filling, can be determined alternating pulse, blood pressure decreased more without changing although there may be a tendency to increase. Increasing hypoxia and acidosis, in turn, further aggravating the heart and reduce the effectiveness of drug terapiyi.Prystup cardiac asthma can last from several minutes to several hours. It usually begins at night, due to several reasons: 1) reducing the sensitivity of the central and autonomic nervous system during sleep, it impairs gas exchange in the lungs, there is a spasm of bronchioles and blood extravasation of fluid into the interstitial space first, then the alveoli; 2) increased vagal tone at night; 3) increase gipervolemii horizontal body, accompanied by an increase in BCC and impaired blood flow to the left shlunochka.Podalshe progression of heart failure increases the permeability of capillaries, not only, but also the alveolar-capillary membrane. Against the background of reduced oncotic blood pressure occurs proprivannya of its liquid, proteins and even formennyh elements in the alveoli. If proprivannya blood proteins formed foamy liquid, which is made up of bubbles. It covers the entire surface of the alveoli. These processes help to reduce vital capacity, disruption of the alveolar-capillary gas exchange, change in the activity and development of surfactant atelectasis. Described pathophysiological mechanisms leading to the development of acute respiratory failure. It joins the already-formed heart failure. The patient developed pulmonary edema leheniv.Nabryak - the most severe manifestation of left ventricular heart failure. The condition of patients deteriorates, breathlessness increases, there is a choking breath (due to proprivannya liquid part of the blood into the alveoli). For children there is a feeling of fear, they are covered with cold sweat. At this stage, attached to hypoxia circulatory hypoxia caused by deterioration of gas exchange in the lungs, that is hypoxic hypoxia. The sharp deterioration in gas exchange involves a reduction in the
formation of oxyhemoglobin and increasing the number of reduced hemoglobin in the blood, leading to an increase in central cyanosis. The long wet cough with sputum (fluid from the alveoli). Above mixed wet listen lungs wheezing. Foaming in the alveoli dramatically impairs reproduction and activity of surfactant. This stage of acute heart failure requires complex resuscitation to improve myocardial contraction, increased cardiac output, elimination or reduction of hypoxia, improved coronary krovoplynu. Dlya differentiating cardiogenic pulmonary edema and nekardiohennoho, about the question of volume and rate of infusion therapy nowadays there are recommendations for measuring wedge pressure, often in practice adults. This corresponds diastolic pressure in the left atrium, and in clinical practice allows, while stroke volume measurement, to assess left ventricular overload. However, it is a very difficult diagnostic procedure that requires catheterization pulmonary arteriyi. Pry emergency care in children with acute left ventricular heart failure should provide enhanced position in bed, sometimes using venous harnesses on the lower limbs. Are effective defoamers with oxygen (30% ethanol solution through a mask or antyfomsylantu 10% solution), the testimony - mechanical ventilation, intravenous furosemide, 2-5 mg / kg infusion 2 Eufillina 4% solution of 1 ml / year of life (no more than 5 ml), steroids (prednisone 5-7 mg / kg) promedol. Pry hypokinetic type of circulatory disorders is important intravenous sympathomimetic amines (dopamine, dobutamine) or cardiac glycosides (digoxin). In hyperkinetic type of circulatory disorders ganglioplegic (Pentamino, benzoheksoniy or arfonad) used very carefully, slowly drip carefully monitored blood pressure in the ICU. Perhaps the very careful use of peripheral vasodilators (nitroglycerin sublingual or intravenously) in the intensive care unit. Mandatory correction of acid-base balance. Traditionally used kardiometabolity ( "polarizing" mixture Panangin, fosfaden kokarboksilaza, etc.).

<table>
<thead>
<tr>
<th>Вік</th>
<th>Тотальна доза наслічення діоксину</th>
<th>Добова підтримуюча доза діоксину, яка призначається у 2 прийоми на добу</th>
</tr>
</thead>
<tbody>
<tr>
<td>Недоношені новонароджені</td>
<td>20-30 мкг/кг</td>
<td>15-25 мкг/кг</td>
</tr>
<tr>
<td>доношені новонароджені</td>
<td>25-40 мкг/кг</td>
<td>20-30 мкг/кг</td>
</tr>
<tr>
<td>11 мс.-2 роки</td>
<td>30-60 мкг/кг</td>
<td>30-50 мкг/кг</td>
</tr>
<tr>
<td>2 роки-дорослі</td>
<td>30-40 мкг/кг</td>
<td>25-35 мкг/кг</td>
</tr>
<tr>
<td>максимально</td>
<td>0,75-1,5 мг</td>
<td>0,5-1,0 мг</td>
</tr>
</tbody>
</table>

Acute right ventricular heart failure occurs in pathological conditions accompanied by sudden restriction of blood flow in the pulmonary circulation. Such pathological conditions observed in a fit of severe asthma, lung atelectasis, hydrothorax, trachea and bronchi obstruction foreign body, pulmonary embolism, respiratory distress syndrome in infants. The development of acute right ventricular heart failure characteristic heart defects with reduced pulmonary blood flow. It may occur with rapid blood transfusion citrate when both were administered calcium and novocaine in case of rapid intravenous hypertonic solutions, as well as radio-opaque substances that cause vascular spasm of the pulmonary circulation and increase their opir. Hostra right ventricular heart failure develops suddenly. A patient with ' is shortness of breath, changing the frequency, rate and depth of breathing, subjectively perceived as
shortness of breath and difficulty breathing. The child complains of chest compression sensation, pain in the heart, severe weakness. Rapidly growing central cyanosis. The patient is covered with cold sweat. CVP increased significantly, there are signs of stagnation in the large circulation, swollen neck veins, quickly increased liver that becomes painful by stretching hlisonovyi capsule. Pulse weak filling, developed tachycardia. Cardiac weakened boundaries relative cardiac dullness shifted to the right, there is an emphasis II tone of the pulmonary artery. Fast development of stagnation in a large circle leads to functional failure of the kidneys. Biochemical studies confirmed serum renal failure and liver. Using X-ray studies indicate an increase in right ventricular and pulmonary artery expansion cone. Marked ECG characteristics of acute overload right sections sertsysa.Prohnoz acute right ventricular heart failure is extremely serious. A possible consequence is the sudden death because the patient needs emergency treatment dopomohy.U it is the main treatment of the underlying disease, which complicated the development of acute right ventricular heart failure: myotropic antispasmodics with congenital heart defects with reduced pulmonary circulation; while pulmonary embolism - heparin, fibrinolytic agents, embolectomy; In severe fit of asthma - glucocorticoids, bronhospazmolityky on the testimony - the removal of foreign body airway others. Cardiac glycosides may increase the clinical manifestations of right ventricular heart failure worsen prognosis, so their use should be very oberezhnym.Zdiysnyuyetsya oxygen therapy, if indicated - mechanical ventilation, intravenous loop diuretics (furosemide), carried out a correction of acid-base and electrolyte and water balance. Perhaps the very careful use of peripheral vasodilators (nitroglycerin or sodium nitroprusside intravenously) in the department reanimatsiyi.Takym way, based pharmacotherapy of acute heart failure in children are beta-agonists (dopamine, dobutamine), cardiac glycosides (digoxin), diuretics (furosemide) [2 -5, 9].Isnuyut recommendations for intravenous amrinone - phosphodiesterase inhibitor that increases contractility of the heart ' Yazoo and causes vasodilation. In its hemodynamic effect it is like dobutamine. Cardiac glycosides enhance myocardial contractility without any noticeable increase in oxygen consumption (due to slowing heart rate), stimulates anaerobic metabolism, increase respiration rate of the tissue, normalize the formation of ATP, improve the use of energy-rich phosphates. Therapeutic doses of cardiac glycosides cause a number of specific effects of the cardiovascular system, positive inotropic, negative chronotropic, positive or negative dromotropic batmotropnu action. At present, among the preferred cardiac glycosides digoxin. Table 1 shows the dosage of digoxin in children of different viku.Sered diuretics are the most effective use of furosemide. The drug is administered intravenously initial dose of 1 mg / kg, the maximum single dose - 2 mg / kg. The maximum dose - 6 mg / kg.
Great prevalence of resuscitation and intensive therapy at present become kardioinotropni sympathomimetic agents, the use of which is carried out in order to maintain perfusion and oxygenation of vital organs and tissues of the child's body, increased cardiac output and optimize its rozpodilu. Dlya proper use of modern sympathomimetic remember main effects of stimulation of adrenergic receptors, beta-1, increased heart rate and myocardial contractility; Beta 2: bronchodilation and vasodilation; Alpha: vasoconstriction; dopamine: renal vasodilation and mesenteric arteries, increasing diurezu. Rozpovsyudzheny scheme is the use of dopamine (dopamine) via infusion pump in pediatric intensive. Preparation has the following effects: low doses (0.5-5 mg / kg / min.) Cause dopaminergic effects, increase urine output; medium dose (5-15 mg / kg / min.) cause beta-1 adrenergic effect, have positive inotropic and chronotropic action; high doses (> 15 mg / kg / min.) with alpha-adrenergic effect and causing systemic vasoconstriction. Effects of dopamine starts quickly, within 2-4 min. After the introduction, and ends in 5-10 minutes. after vvedennya. Adrenalinu (epinephrine) effects inherent to stimulate beta 1, beta 2 and alpha receptors. It is used in children with heart failure after failure of other drugs, stopping cardiac activity. However, be aware of the side effects of this drug - tachycardia and increased afterload. Dosage - intravenously 0.1-1 mcg / kg / min., At resuscitation bolus 100 mg / kg (1 ml / kg district 1: 10,000). medium dose (5-15 mg / kg / min.) cause beta-1 adrenergic effect, have positive inotropic and chronotropic action; high doses (> 15 mg / kg / min.) with alpha-adrenergic effect and causing systemic vasoconstriction. Effects of dopamine starts quickly, within 2-4 min. After the introduction, and ends in 5-10 minutes. after vvedennya. Adrenalinu (epinephrine) effects inherent to stimulate beta 1, beta 2 and alpha receptors. It is used in children with heart failure after failure of other drugs, stopping cardiac activity. However, be aware of the side effects of this drug - tachycardia and increased afterload. Dosage - intravenously 0.1-1 mcg / kg / min., At resuscitation bolus 100 mg / kg (1 ml / kg district 1: 10,000). medium dose (5-15 mg / kg / min.) cause beta-1 adrenergic effect, have positive inotropic and chronotropic action; high doses (> 15 mg / kg / min.) with alpha-adrenergic effect and causing systemic vasoconstriction. Effects of dopamine starts quickly, within 2-4 min. After the introduction, and ends in 5-10 minutes. after vvedennya. Adrenalinu (epinephrine) effects inherent to stimulate beta 1, beta 2 and alpha receptors. It is used in children with heart failure after failure of other drugs, stopping cardiac activity. However, be aware of the side effects of this drug - tachycardia and increased afterload. Dosage - intravenously 0.1-1 mcg / kg / min., At resuscitation bolus 100 mg / kg (1 ml / kg district 1: 10,000). medium dose (5-15 mg / kg / min.) cause beta-1 adrenergic effect, have positive inotropic and chronotropic action; high doses (> 15 mg / kg / min.) with alpha-adrenergic effect and causing systemic vasoconstriction. Effects of dopamine starts quickly, within 2-4 min. After the introduction, and ends in 5-10 minutes. after vvedennya. Adrenalinu (epinephrine) effects inherent to stimulate beta 1, beta 2 and alpha receptors. It is used in children with heart failure after failure of other drugs, stopping cardiac activity. However, be aware of the side effects of this drug - tachycardia and increased afterload. Dosage - intravenously 0.1-1 mcg / kg / min., At resuscitation bolus 100 mg / kg (1 ml / kg district 1: 10,000). high doses (> 15 mg / kg / min.) with alpha-adrenergic effect and causing systemic vasoconstriction. Effects of dopamine starts quickly, within 2-4 min. After the introduction, and ends in 5-10 minutes. after vvedennya. Adrenalinu (epinephrine) effects inherent to stimulate beta 1, beta 2 and alpha receptors. It is used in children with heart failure after failure of other drugs, stopping cardiac activity. However, be aware of the side effects of this drug - tachycardia and increased afterload. Dosage - intravenously 0.1-1 mcg / kg / min., At resuscitation bolus 100 mg / kg (1 ml / kg district 1: 10,000). have positive inotropic and chronotropic action; high doses (> 15 mg / kg / min.) with alpha-adrenergic effect and causing systemic vasoconstriction. Effects of dopamine starts quickly, within 2-4 min. After the introduction, and ends in 5-10 minutes. after vvedennya.
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Porivnyalna characteristic action of beta-adrenergic dose dopamine and dobutamine presented in Table 2. Very important information is that dobutamine improves not only as dopamine, on the contrary, lowers blood pressure in the pulmonary capillaries, which is extremely important in terms of Supervision of children with bronchopulmonary disorders, impaired heart and great vessels, transient neonatal pulmonary hypertension. The drug can be used in combination with sympathomimetic dopaminom. Vnutrishnovenq infusion should be carefully monitored, monitoring, heart rate, ECG, blood pressure, urine output, peripheral blood, pH, pO2, SatO2. Dobutamine is not used in patients with hypertrophic obstructive cardiomyopathy, significant aortic stenosis, severe pericarditis, atrial peredserd.Ratsionalnyu is the following method of calculation when administered dobutamine (using infusion-perfusion pump type "Liniomat") [10] and. To calculate the amount of the drug dobutamine (mg) that should be added to the base solution for infusion (saline), using the formula: 6 x desired dose in mg / kg / min. / Desired infusion rate in ml / hour x body weight in kg = mg dobutaminu. II. To calculate the amount of drug (in ml) that should be added to 100 ml of saline formula is used: mg dobutamine that calculated above / drug concentration in mg / ml = ml dobutaminu. Pry sympathomimetic application should remember these rekomendatsiyi- warning: 1) to ensure adequate fluid balance, 2) to correct acidosis,
3) use only the large central veins or 4) follow the extravasation 5) closely monitor the speed and duration of the infusion.

**Topic: Juvenile rheumatoid arthritis and reactive arthritis in children.**

Content topics:

**Juvenile rheumatoid arthritis (JRA)** - a chronic inflammatory autoimmune disease of the connective tissue, mainly affecting the joints on the type of erosive-destructive progressive polyarthritis and belongs to the multifactorial polygenic inherited diseases, of which there are infectious, hereditary and external factors, including immunogenetic.

In Ukraine, the CIS remains in force, the term "juvenile rheumatoid arthritis", which is classified according to ICD 10 to XIII class of diseases of the musculoskeletal system and connective tissue Subdivision general poliartropatiyi (MO8). Active is the classification Dolgopolov A. et al (1980), according to which, taking into account clinical and anatomical characteristics were identified:

- suhlobova form of JRA (with no eye disease or eye damage) zvariantom course: arthritis, olioartryt, monoaarthryt;
- suhlobovo-visceral form of the current options, restricted vistseryty syndrome Still, alerhoseptychnyy syndrome;
- YURA combined with rheumatism, systemic connective tissue diseases (SZST).

Depending on the presence or absence of rheumatoid factor selected RF-positive and RF-negative JRA; the course of the disease - a rapidly progressive, slowly progressive, with no significant progression.

The current classification proposed by the American Rheumatology Association (ARA), depending on the characteristics debut JRA identifies three forms (variants) of the disease:

- System option.
- Articular option (seronegative and seropositive)> 4 joints.
- Olihoartykulyarnyy option:
  a) occurs in girls at an early age with the presence of ANF (antinuclear factor), absence of rheumatoid factor (RF), HLA B27 high risk of eye damage;
  b) more often in boys begins in middle and older age, mainly affecting the lower limbs, lack of, the ANF (-).
  c) in children of all ages with the absence of RF, ANA, HLA-B27.

It should be noted that the European School of Rheumatology uses the term "juvenile chronic arthritis" (Juha), which combines:

- polyarticular arthritis, seropositive option (actually JRA);
- pautsiartykulyarnyy arthritis (olioartryt);
- systemic arthritis;
- juvenile ankylosing spondylitis;
- psoriatic arthritis;
- arthritis associated with inflammatory diseases of the intestines (inflammatory bowel disease - IBD).

This classification adheres to Moscow, Kharkov school pediatricians.
So (Juha) - heterogeneous group of childhood diseases with different etiopathogenesis and immune-genetic origin, different nosological identity and different prognosis. These diseases integrate tendency to chronic, often progressive course that has a significant impact on the quality of life of the child and the high probability of early disability. Juvenile rheumatoid arthritis (JRA) is a share in the structure of Juha and the structure of rheumatic diseases is 10%.

In 1994, under the auspices of WHO and international antirheumatic league (International League Against Rheumatism - ILAR) by key (R. Petty, TR Southwood, J. Baum, D. Glass et al.) Proposed new classification criteria for chronic inflammatory diseases of the joints children adopted at a meeting of the Standing Committee of pediatric Rheumatology in 1997 in the city. Durban (South America).

It was decided to abandon the terms of JRA, Juha and call all chronic inflammatory joint disease in children juvenile idiopathic arthritis (JIA).

JIA can be defined as arthritis of unknown causes with the beginning of 16 years of age, disease duration ≥ 6 weeks, to the exclusion of other diseases. The term JIA is essentially equivalent to the definition Juha. However, the term JIA are increasingly part of the foreign medical practice, it is only valid for international congresses in special medical literature. Nosological out the following forms:

1. System arthritis.
2. polyarthritis, of (+) - seropositive.
3. polyarthritis, of (-) - seronegative.
4. Olihoartryty.
5. Ekstenzuyuchi (progressive) olihoartryty (with the involvement of one to four joints in the first 6 months of disease).
6. Arthritis with enteritis.
7. psoriatic arthritis.

However, the new classification of JIA is not perfect, as not taking into account the type of debut disease. The authors illogical term "juvenile rheumatoid arthritis". The classification is not final and requires further improvement.

Clinic. Clinical manifestations of JRA is quite varied and depend on the age, sex, provoking factors, the characteristics of the further development of the pathological process. According Fujikava S. (1997), A. Lukyanov (2002), almost 20% of cases of children with juvenile rheumatoid arthritis, the onset of the disease diagnose joint - visceral form, 30% - poliartrytychnyy option and joint forms observed in 50% of rheumatoid arthritis.

In most cases, the clinical picture is determined joint damage that have synovialnyu shell. Disease onset MB slow, hardly noticeable, with the appearance of minor pain and swelling in a joint, usually the knee and ankle. After 1-2 weeks or 1-2 months. pathological process develops in a different symmetrical joint. In addition to pain and limitation of movement in the joint children observe general indisposition, weakness, decreased body weight appears subfebrilitet, ESR increases to 20-25 mm / h. Later in the process involves other joints. Defeat 2-4 joints - olihoartryt (pautsiartryt). Engaging in the process of joint ≥ 5 shows the development of arthritis. This subacute disease occurs in children after five years, mostly schoolchildren. In children who are sick at an early age acute onset of fever, arthralgia marked with lesions ≥ 4 joints, including small joints of the hands and feet. Against fever appear polymorphic rash, swollen lymph nodes, liver and spleen. Children take a forced...
situation, become inactive, appear painful contractures. Quite often in girls early years JRA occurs in the form monoartrytu (mostly knee) and accompanied by eye disease - uveitis.

So, arthralgia, swelling, morning stiffness and joint dysfunction - the main complaints of patients. First pain occur with movement, further spontaneous (at rest). Swelling of joints symmetric determine its synovial hypertrophy and accumulation of fluid, providing soft tissue of joints tistuvatu consistency, increased local temperature, pain. This is not typical erythema. Morning stiffness is a diagnostic value, if it takes an hour. Arthralgia most intense in the second half of the night, in the morning, at night and in the evening is simultaneously MB muscle cramps, paresthesias. Inflammatory changes in joints prior myalgia, neuralgia, moderate arthralgia, bursitis, tenosynovitis. Symptoms initial period not always pathognomonic.

Joint damage in the early stages of JRA MB unstable and undergo their own without specific treatment. But after a short time arthralgia are constant and intense multiple developing joint damage. Appear local signs of inflammation, stiffness, dysfunction.

Acute onset JRA is not always accompanied by a clear joint damage. The leading symptom MB intermittent fever with a steady rise in the morning, stable polymorphic rash and arthralgia in the large joints - knee, ankle-step, hip.

Articular syndrome manifested all the signs of local inflammation, pain or slight pain, swelling, change in shape joint, local temperature increase. However olhioartykulyarnomu variant of the disease is not characteristic marked hyperemia skin joints. Children often affects the knee, ankle, foot, radio-carpal, elbow joints, proximal interphalangeal joints 2, 3, 4 fingers slightly less - metacarpal phalangeal, and joints of the feet, hipbone, the joints of the cervical spine, temporal - nyzhnoschelepni. Children often (80-90%) affected knee joints. Clinical manifestations honitu largely depend on the severity of the process and the age of patients. Serous membranes (including synovial joints) in young vikubahato vascularized. Because inflammatory reaction in them accompanied by considerable exudation. At the same time, the acute onset of the disease, regardless of age can also be accompanied by a pronounced exudative component, particularly the knee. An indication of the presence of fluid in the knee joint is a vote elbow and severe swelling in the area of the upper pole joint and liquid filling external and internal twisting bags. Exudation in the cavity of the joint causes a sharp pain and limitation of movement in the joint. This defines the first painful contracture, which prolonged the time fixed contracture of muscle. and severe swelling in the area of the upper pole joint and liquid filling external and internal twisting bags. Exudation in the cavity of the joint causes a sharp pain and limitation of movement in the joint. This defines the first painful contracture, which prolonged the time fixed contracture of muscle. and severe swelling in the area of the upper pole joint and liquid filling external and internal twisting bags. Exudation in the cavity of the joint causes a sharp pain and limitation of movement in the joint. This defines the first painful contracture, which prolonged the time fixed contracture of muscle.

In subacute onset of the disease exudative changes less pronounced: a small swelling on the upper side of the upper pole and twisting. Movement moderate joint pain, limited. Changing pace, there is morning stiffness. The last symptom often
occurs in older children in the morning of 10-15 minutes. to 1-1.5 hours. With long-term course of developing joint deformity, muscle atrophy hips and legs, damaged tendons and ligaments. All this leads to distortion subluxation of the tibia - valgus deformity that changes the course of the child.

Ankle joint and foot joints affects children quite often, especially in early childhood. Swelling is observed in the area of foreign and domestic lodyzhok and in front of the medial joint department (perhaps caused by inflammation of the tendon). In young children (especially girls) are often affected paw and paw-phalangeal joints. This foot has a cushion shape, disturbed gait. This may wonder and proximal interphalangeal joints, often II, III, IV, stop. Fingers swollen, red, painful movements in them. In children less than adults, there are gross subluxation ("fabulyarna" deviation). However mono- oligoartrity ankle joints in young age girls are often combined with rheumatoid uveitis, which for a short time leads to a sharp decrease in visual acuity.

Radio-carpal joint and joints of hands. The defeat of the joints in children ranks third frequency in children with JRA. Still syndrome are included in the process at an early stage in conjunction with other peripheral joints. Initially, the outer surface of the joint appear limited "tistovatist" swelling caused tendosynoviyitamy muscles flexors and extensors. The joint fusiform dials, movements, it is limited and painful. Brush gradually deviates outward, forming ulnar deviation. The latter is not always matched with similar changes in the metacarpophalangeal joints, although the latter is also affected. Often the process involved metacarpal phalangeal joints, II-IV fingers, swelling on the outer and inner surfaces. On the back surface of the congestion occurs, followed by pigmentation. Most affected and proximal interphalangeal joints of the fingers. Rapidly developing muscle atrophy brush. In teenage girls formed persistent strain - sustained flexion of the proximal and distal interphalangeal joint extension. This is one cause of disability children.

Cervical spine. Engaging in the process of the joints of the cervical spine - one of the earliest manifestations of JRA. Inflammation develops between the vertebral joints having synovial lining. Children have complaints of pain and limitation of movement in the cervical spine, head takes a forced position - tilted forward or sideways. Defeat neck joint in JRA is an unfavorable prognostic criteria, as there is a possibility of further development of generalized arthritis or one of the systemic forms of the disease. Early involvement in the process of the joints of the neck with long-term disease leads to restriction of movement when turning heads is only possible with the trunk. This shift characteristics vertebrae as paresthesias of the face and extremities, nystagmus, dysarthria observed.

Elbow joint is involved with poliartritychnomu variant of the disease. In the absence of external changes observed limit flexion and extension. Flexion contracture at the elbow joint and simultaneous changes hands and wrists sharply limit the ability to self and the child becomes disabled. In the presence of exudative component increases elbow becomes painful. Bursitis have sustained recurring nature and are torpid in treatment. Prolonged disease (> 10 years) developing ankylosis of the elbow with complete loss of function.

Hip joint in children with JRA affects more frequently than in adults. This to some extent explains the tendency of children to exudation. This pain of varying intensity in the groin (sometimes in the buttocks), lameness and complete loss of
opportunities to walk. Defeat hip evidence of Fanconi syndrome -articular or
generalized form of the disease. Visl era syndrome, Fanconi often a aseptic necrosis
of the femoral head, due to the frequent development of vasculitis in this form of the
disease. With the development of aseptic necrosis of the femoral head in joint pain
intensity increases, limited movement, marked temperature reaction. Perhaps the
complete destruction of the head and closing the joint space with the formation of
ankylosis.

Oral-temporal joint. This localization is quite typical for JRA, especially the
syndrome and Still onset of the disease at an early age. Children Complaints of pain
when chewing, sometimes radiating to the ear, limited mouth opening. It slows
the growth of the lower jaw, resulting in developing common form of deformation of
facial proglathism.

The shoulder joint. Involved in the process is not often, mostly girls with a
generalized form of the disease. while there is swelling in the anterior-lateral area of
the shoulder caused by bursitis. Movement joint pain and limited. The presence of
fluid in the joint can be detected by palpation.

December-clavicular joint is affected rarely. Swelling and soreness little effect on
joint function.

Articular syndrome in JRA has some features depending on the clinical form of
motion, age, sex of the child. Articular form of the disease with subacute beginning
accompanied by the development of arthritis, mainly affecting the knee and ankle
joints. Later lesions often joins the wrist and elbow joints. The process progresses
moderately dominate productive change. Radiologically determined mostly II-III.
(For Shteynbrokkerom). In acute disease earlier this variant more involved in the
process of radio-carpal, metacarpal phalangeal-interphalangeal joints and hands, and
Atlanto-oktsyptalnnyy and maxillo-temporal joints. Thus for 2-3 years in 30% of
patients progressing destructive changes in the joints with the development of
erosion. When systemic forms of articular syndrome also has its own peculiarities.
Alerhoseptychnyy option usually begins with a large persistent arthralgia (knee,
hipbone) and medium (ankle-foot, radio-carpal, elbow) joints with no visible changes
in them. The duration of arthralgia are different - from several weeks to several
months. Eventually joined exudative and productive changes in the joints with the
rapid development uzur and erosions, sometimes aseptic necrosis of the femoral head.
Most viewed articular syndrome syndrome Still. It is very fast-paced generalized
articular syndrome with lesions of hand joints, feet, cervical spine, jaw-temporal and
large joints. The initial exudative phase fairly quickly (a few months) changing
productive processes, erosion and destruction of cartilage, leading to early ankylosis,
often in the elbow and radio-carpal joints

The boys less pronounced exudative component dominated productive-degenerative changes in the joints of the lower extremities (hip, knee, ankle stepped
joints feet) of in serum rarely defined.

Girls in the early stages of the disease prevails exudation in the joints of the upper
extremities - radio-carpal, elbow, small joints of the hand, Russia is rarely positive
(20%). In mono olioahrtryti (10%) girls discovered early age eye disease (uveitis),
combined with the detection of serum antinuclear factor and antibodies to DNA.

The latter determines the loss of mobility of the joints. Polyarthritis the first days
of the disease is one of the major symptoms of the syndrome Still. Already 2-3, the
Disease may develop destructive changes of cartilage, bone erosion, in some cases - ankylosis of small joints of the wrist bones is determined by X-ray.

- Fever, hectic, sometimes up to 39-40 °C, lasts 3-4 weeks. With the progression of joint syndrome reduces fever, although some can be observed fever, often with skin rash.

- The rash often dribnotochkovi (Scarlatiniform), less korepodibni (macular-papular) are unstable, disappearing along with the normalization of body temperature.

- Fast-paced cachexia.

- Generalized lymphadenopathy, particularly increased lymph nodes and inguinal pidkryltservi.

- Pericarditis usually occurs with small serous effusion in the pericardial cavity and in most patients found only in the instrumental study.

- In this form of the disease often develop secondary amyloidosis (20%), mainly affecting the kidneys. Thus patients within 2-3 years can be stored selective proteinuria, and 4-5, it becomes permanent.

- The condition of the syndrome Still progressing rapidly.

- High activity of the laboratory criteria: erythrocyte sedimentation rate 60 mm / hr., Raising all classes of Ig (especially IgG), an increase of SRP. Most tendency to neutropenia than leukocytosis, anemia progresses rapidly hipsohromna.

**Features paraclinical investigations.**

Laboratory findings with JRA often reflect the presence and severity of inflammation and thus is a nonspecific inflammatory process and are therefore not specific. The exception is rheumatoid factor (RF). Children discover Russia not more than 15-20%. Cases and twice more in the synovial fluid than in serum.

**Total blood.**

One of the most important indicators are increased ESR and largely depends on the form of the disease. Since the mono- and olihoartri ESR does not exceed 30-35 mm / hr., With arthritis and systemic form of JRA - increased to 50-60 mm / h. The number of white blood cells also depend on the form of the disease. In olihoartri not exceed the number of leukocytes 8-10h10^9 / l. Larger changes in leucogram observed in systemic forms of the disease. Still syndrome is characterized by rapid progression of anemia with a slight leukocytosis and leukopenia and with increasing ESR to 40-50 mm / h. For alerhosepychnoho syndrome is typically marked leukocytosis to 20-30h10^9 / l neutrocytosis to 70-80% and stab shift (10-12%). ESR can reach 50-60 mm / hr., The number of platelets - 400-500 thousand. It is important to note, between the size and activity ESR process is not always a direct correlation. In some patients on background therapy following positive clinical dynamics and the ESR value remains high, indicating incomplete remission.

In the active phase of JRA patients increased levels of serum Ig all fractions (A, M, G), most increases the level of IgG. Increased IgG observed in the later stages of the disease and is an indirect criterion of inflammation and erosions. However, these figures reflect the nature of the pathological process in general.

**C-reactive protein (CRP SRP)** - one of the non-specific indicators of activity of rheumatoid process and fluctuations reflect its degree. Increased CRP occurs in 60-70% of cases among patients. CRP is produced in hepatocytes under the influence of IL-6, whose synthesis is induced by IL-1 and FNO-α. This origin has amiloyid- ny protein.
Rheumatoid factor (Russia) - an antibody produced by a certain population of plasma cells of the synovial membrane. In response to the synthesis of other populations of IgG, changing under the influence of an unknown antigenic stimulus. RF is an antibody to the Fc-fragment of IgG. Rose Vaalera reaction is positive with titer 1:32, latex test - at tytril 40. Russia is a highly sensitive and specific test disease, but serum is determined 15-20% in synovial fluid in 30-35% of cases. In all cases, the RF belong to the class IgM (IgM -RF).

Antinuclear factor (ANF) - antibody solid nucleus of cells, determining its use as a screening test for the diagnosis of SLE. Patients with JRA find it relatively rarely, and it indicates the severity of the process. However, there is a group of patients with frequent detection of ANF - girls up to 4 years with mono- or oligoarthritis and accompanying uveitis. Detection they ANF indicates high risk eye damage.

Radiologically changes in JRA as with RA, assessed by O. Steinbrocker (1949) and identifies four stages of the disease:

And art. - osteoporosis, mainly epiphyseal;
Second century. - osteoporosis and primary destruction of cartilage, joint space narrowing;
III. - severe cartilage and bone destruction, bone erosion;
Fourth century. - Symptoms III. and ankylosis.

It should be noted that in one patient different joints have different radiographic changes. However, in assessing the gravity take note of any changes in the maximum joint.

Activity process within the set three levels: low (I), moderate (II), high (III). The dynamics of the main syndromes and process activity during the first three years evaluating the criteria for fast, slow and little progressive course.

The degree of activity of JRA:

And art. (Minimum activity). Minor joint pain, morning stiffness in the morning to 60 min., Minor exudative changes in the joints, increased local temperature or normal. ESR increased to 20 mm / hr., L-norm α2-globulin content increased to 12%, SRR + HRT moderately elevated.

Second century. (Average activity). Pain in the joints as during movement and at rest, stiffness held noon, marked limitation of movement pain in the joints, moderate stable exudative symptoms. Moderate hyperthermia skin over the joints. Vague symptoms of internal organs ESR: 25-40 mm / hr., 10-15 h109 L / L level of α2-globulin increased to 15%, SRR ++, elevated levels of sialic acid, fibrinogen.

III. (High activity). Severe pain alone expressed exudative reactions in joints (redness, swelling, hyperthermia), stiffness throughout the day, marked limitation of movement. Signs vistserytiv (pleurisy, pericarditis, nephritis, etc.). Febrile body temperature. ESR ≥ 40 mm / hr., L> 15-20 h109 / L, α2-globulin ≥15%, SRR +++ sharply elevated levels of fibrinogen, sialic acids.

In practice for the diagnosis of JRA used as diagnostic criteria for rheumatic American Association (APA):

- onset before 11 years of age;
- the defeat of two or more joints, characterized by swelling or effusion are at least two signs: limiting function, tenderness, local temperature rise;
- the duration of the joint changes ≥ 6 weeks;
- the exclusion of all other rheumatic diseases.

Based on criteria developed by ARA classification JRA.

**Clinical onset JRA.**

**Polyartritychnyy option** in the first 6 months. develops in 30-40% of cases. System lesions are not present. This is typical:
- acute or subacute debut;
- the disease is more common in girls than in boys;
- affects 5 or more joints, large joints;
- are involved in the process of the joints of the cervical spine, lumbar-sacral, temporal-mandibular joint;
- rheumatoid rash usually absent, but MB erythema of palms and soles;
- subcutaneous nodules are sometimes found in RF-positive patients;
- eye disease is rare;
- arthritis can be a chronic relapsing and easier - intermittent (prolonged remissions);
- in seropositive patients are at high risk of erosive arthritis;
- in seronegative patients the disease is less manifest and often turns into adult form of RA.
- RF-find factor in 10-20% of cases, ANF - at 20-40%.

Olihoartrity occurs in half of patients and characterized by lesions 4 joints in the first 6 months. disease.

**Options:**
1. Olihoartrity and anterior uveitis.
   - occurs predominantly in girls begins at an early age;
   - asymmetry of joint damage;
   - mild course lesions of the knee, etc. may be affected. joints;
   - rarely is affected spine;
   - systemic lesions are absent or expressed only slightly;
   - uveitis occurs in 20-30% of cases, can lead to blindness;
   - antinuclear antibodies found in the blood in 60% of cases and is a risk factor for uveitis common antigens HLA B27 (-) and are HLA DR5, DRW8;
   - seronegative option;
   - favorable course and only 1/3 of patients, there is a transformation of arthritis.

2. Olihoartrity spine and joints of the lower extremities.
   - occurs mainly in boys during adolescence;
   - frequent lesion of the sacroiliac joints, asymmetric lesions of the hip and knee joints;
   - acute anterior uveitis, flows easily, there is no risk of blindness;
   - seronegative (RF-) nDNK not find antibodies in 50% of cases -HLA B12 +;
   - further transformation in ankylosing spondiloartrity disease Reiter.

**Still syndromenow quite rare (12-16%) with JRA, mostly in preschool children. It is characterized by:**
- often sharp manifest than subacute beginning;
- in early disease affects both large and small joints, including small joints of hands and feet;
- typical for this variant of the disease is cervical spine lesions and temporal-mandibular joints. Swollen joints, sharp pain spontaneous, quickly develop painful contractures;
- quickly appear and systemic complaints pozasuhlobovoi destruction;
- hectic fever;
- maculopapular rash;
- generalized lymphadenopathy;
- hepatosplenomegaly;
- polyserositis;
- carditis, pankardyt, aortyt (10%);
- in some cases, arthritis and lower spondiloartryt develop a few weeks or months of fever and extraarticular manifestations;
- seronegative, in most cases, seropositive;
- find HLA B8, DR4, DW7.

**syndrome Felti** combines syndromokompleks Jura - splenomegaly - neutropenia. Developed with significant disease duration. At the same time notes bw loss, generalized lymphadenopathy, skin pigmentation and ulceration of the lower leg.

Radiological findings reveal erosion of the articular surfaces. In 90% of cases are RF, antinuclear antibodies. Leukopenia may progress to complete agranulocytosis. Along be thrombocytopenia, hemolytic anemia.

**Alerhoseptychnvy syndrome (syndrome of Fanconi-Visslera)** known since the 60th century. a systemic form of JRA, which is typical for:
- acute manifest beginning;
- constant high fever. The latter appears in the morning, starting from 5-6 hours. after a severe chill. Duration of fever 3-4 hrs., Following hectic further lowering the temperature of pouring sweat. Getting fever often coincides with the appearance of skin rash;
- polymorphic allergic rashes are torpid course and can be stored for months. Localized on the trunk extensor surfaces of the extremities, polymorphic, groups on the sides of the chest, thighs. The typical rash is linear (lines 1-2 cm)
- lymphadenopathy;
- articular syndrome develops later (after a few weeks or months, even years 2-3).
- A peculiar localization is articular lesions, knee, ankle, foot, often hipbone, very rarely - joints of hands and feet. Pretty soon they develop destructive changes - easing cartilage, bone erosion. This process is progressing rapidly, causing considerable destructive processes.
- Severe neutrophilic leukocytosis with a shift to the left.
- Cirrhosis and elevated ESR.

**Diagnosis. Criteria for early diagnosis of JRA.**

**Clinical signs:**
- Arthritis $\geq$ 3 months.;
- arthritis of other joints that occurs after 3 months. after the first defeat;
- symmetrical lesions of small joints;
- effusion in the joint cavity;  
- joint contracture;  
- effusion in the joint cavity;  
- tendosynovit, bursitis;  
- regional muscular atrophy;  
- morning stiffness;  
- rheumatic eye damage;  
- rheumatoid nodules;  
- lesions of the cervical spine.

**Radiographic signs:**
- osteoporosis, bone structure drbnokistozna restructuring epiphysis;  
- joint space narrowing, erosion of the articular surfaces ankylosis;  
- Bone growth disorders;  
- lesions of the cervical spine.

**Laboratory criteria JRA.**

**Hemography:**
- leukocytes within the normal range, rarely moderate leukocytosis; develops with prolonged duration of radiation;  
- eosinophilia (at vistserytah, vasculitis);  
- thrombocytosis, thrombocytopenia rare;  
- anemia complicated origins, reticulocytes within normal values, MB hemolysis;  
- elevated ESR.

**HRT:** Increased content always SRR, sialic acid seromucoid, fibrinogen.

**Proteyinohrama:** Elevated levels of α2 and γ-globulin fractions.
- **Russia:** Ig M, rarely Ig A, Ig E, Ig G, Ig D.  
  - functional activity of mononuclear phagocytes, increased synthesis of IL-1β, FNO-α.  
  - Increased concentration of prostaglandin E2 in serum.

**Immunogram:** T lowered maintenance, lack of suppressor function (SD8 +) dominated helper (CD4 +). Increased concentration kriohlobuliniv (often mixed character, especially when vistserytah), elevated levels of Ig A, Ig G, Ig M, CIC.  
- Positive results synovial biopsy.

**The criteria for clinical remission.**
- morning stiffness duration 30 min .;  
- general condition is satisfactory;  
- no pain in the joints with active and passive movements;  
- no synovitis;  
- no inflammatory changes in periarticular tissues;  
- ESR ≤ 10 mm / h.

**Differential diagnosis** JRA conducted with rheumatism, juvenile ankylosing spondiloartrytom (YUAS), SLE, SSC, YUDM, arthritis non-rheumatic nature - septic and infectious (tuberculosis, brucellosis), disorders of the musculoskeletal system noninflammatory genesis (arthropathy in genetic and metabolic diseases, dysplasia, osteochondropathy) , lesions of the joints in hematologic diseases.  

In rheumatoid arthritisthere is a direct relationship with streptococcal infection, most of the articular syndrome combined with carditis. Joint damage is symmetric,
the volatile nature monoartryt rarely seen without deformation characteristic of rheumatic joints, muscle atrophy, morning stiffness of joints and degenerative changes. The cell composition of blood, inflammation biochemical tests are not informative for the differential diagnosis of these two diseases.

YUAS - diagnostic criteria (for Hermish-Partenkirchen).
1. Major (great) criteria:
   - oligoartryt asymmetric (<5 joints) mainly the lower extremities in the first three months;
   - enthesisopathy and other tendon connection for symptoms;
   - Pain in the lumbar spine or Sacre ilealniy region;
   - acute iridocyclitis;
   - non-destructive nature of joint damage (except tarzytu and koksytu);
   - relatively benign course with a tendency to develop long (years) remissions.

2. Additional (small) criteria:
   - polyarthritis (> 5 joints) at the onset of the disease;
   - male;
   - onset in children after six years;
   - HLA B27-positivity;
   - familial aggregation in HLA B27-associated diseases.

To Yusa flow is characterized by the following:
   - development of peripheral arthritis in most children, a few years prior shock spine, eyes;
   - oligoartryt arthritis or limited;
   - asymmetry of joint damage;
   - primary lesion joints of the lower extremities;
   - combined with enthesis and other symptoms of tendon-ligament;
   - non-destructive nature of joint damage (except tarzytu and koksytu);
   - relatively benign course with a tendency to develop long (years) remissions.

Peredplyusy joint damage occurs to the formation of ankylosing tarzytu - typical clinical symptom YUAS. This kind of joint damage and tendon-ligaments of the foot and clinically severe deformation of the tarsus significant changes in the skin over it, combined with a marked ahilobursytom, pid'p'yatkovym bursitis, tenosynoviyitamy areas of foreign and domestic lodyzhok. Violated pohodka to complete loss of the bearing capacity of the limb. Tarzyt radiologically evident osteopenia, bone erosion of the articular surfaces of the tarsus sometimes in combination with bone growths and periosteal layers, and with prolonged duration - the development of joint ankylosis tarsus.

Characteristic lesions are YUAS finger joints and the development of erosions in the distal stop, usually at the site of attachment of the joint capsule (this option entezopatiy).

Prior to the enthesisitis refer Dactylitis, expression of which is "sosyskopodibna" finger deformity due to concurrent inflammatory lesions joint and tendon-ligament apparatus that is characteristic of arthritis. However, it may be observed and in YUAS. Sacroiliitis develops, usually in adolescence. In adolescent girls may develop Yusa-like syndrome, arthralgia benign, intermittent episodes of synovitis), peripheral
articular syndrome, multiple localization enthesisopathy no signs of exudation, pain in
the back. This is the result of neuro-humoral disorders and is completely reversible.

Uveitis develops acutely, with bright clinical symptoms (pain, redness eye, photophobia), occurs predominantly in older children, mostly boys.

During prolonged and sustained fever should be excluded septic, infectious
process, cancer. At that crucial blood cultures, serological studies, research
myelogram, ultrasound of internal organs.

Acute local symptoms limited number of joints, especially in combination with
signs of periarticular inflammation, osteomyelitis requiring exclusion.

In recent years, increasingly find original lesion hands in a significant
deforation of small joints, especially 2-4 proximal interphalangeal joints without
local and humoral manifestations of inflammation and bone changes. This is a
manifestation of impaired collagen metabolism and is genetically determined.
Deformation of the small joints of hands MB consequence of increasing epiphysis
and is a feature of the Constitution (epiphysial dysplasia).

Arthralgia MB hypermobility syndrome on the background of the joints, flat feet,
constitutional dysplastic changes the axis of the limbs (varus, valgus deviation), with
osteocondropathy that can occur with benign secondary synovitis.

Select the lower extremities arthritis on a background of Crohn's disease, non-
specific ulcerative colitis, Volyn NGO.

In the last decade increasing relevance of jet (acute and chronic) arthritis (CEA)
associated with various infections. The structure of CEA rheumatic diseases in
children under 14 years are ~ 50% in adolescents - 37%. Previously prevailed among
the causes intestinal infection CEA, the end of 80 years, the role of acute and chronic
chlamydial infection (XI) caused by Cl. trachomatis, and in recent years dominated
Cl. pneumoniae. This is to some extent due to the epidemic of chlamydia Chlamydia
is sviti.Spryatlyvymy to all people. Previous infection does not provide immunity
continue living. Chlamydia peculiar tropism for epithelial conjunctiva of the eyes,
lungs and urinary system. They are characteristic of intracellular parasitism.
Diagnosis XI always indicates infection. Increasing incidence of chronic persistent
chlamydial inektsiyi.

With purposeful examination of children with disorders of the joints (JRA, Yusa,
Juha) XI detected in 80% of cases, most often in children with JRA (Cl. Rneumoniae
- 93%). These children history retrospectively diagnosed XI starting from peri,
intrapartum period, later half of them observed recurrent respiratory infections,
microbial and inflammatory diseases of the kidneys, urinary tract infection (30%),
recurrent conjunctivitis (30%).

Results of clinical Jesse articular syndrome associated with XI, in all forms of
lymphoma have common properties:
- asymmetric exudative arthritis, mainly affecting the joints of the feet;
- continuously relapsing course, significant joint effusion in the cavity;
- no morning stiffness, pain;
- Minimum breach affected joints;
- not progressive radiographic changes;
- torpidist to antibiotic therapy in most patients.

Joining XI in patients with JRA changed the nature of joint syndrome relapses
and recurrences of arthritis actually occurring type olihoartrtyu, exacerbation occur
regardless of baseline therapy 1-1.5 months. Changes are characterized by a pronounced exudative component, mainly in large (knee) joints, no morning stiffness, pain and functional impairment.

Reiter's syndrome is characterized by classic tetrad of symptoms that combines arthritis, conjunctivitis, urethritis, Keratoderma. Development Reiter syndrome accompanied by fever, intoxication, increased acute inflammatory tests, ESR, CRP, of γ-globulin, sialic acids complement others.

Children develop Reiter's syndrome has certain characteristics:
- varies in time all the appearance of symptoms;
- stertist extraarticular clinical manifestations;
- equivalent urethritis in boys is balanitis infected synechia, phimosis;
- girls - vulvovaginitis, cystitis, leukocyturia, microhematuria;
- catarrhal conjunctivitis is often quickly treated, prone to relapse rarely find uveitis with flabby course.

Chronic CEA in the XI characterized by lack of acute inflammatory changes penchant for symmetrical joint syndrome rarely have relapses entire triad of symptoms. This set of clinical and radiological data does not allow vyryfikuva diagnosis of JRA. It should be noted that some children, particularly boys HLA B27-positive, further developing Yusa.

Given XI role in the development and maintenance of inflammatory changes in children with arthritis, including JRA, all children must examine the presence XI. Examination and treatment of family members are also subject to a sick child.

**Treatment.**

The main goal of treatment of JRA - a suppression of inflammatory and imunosupresornoyi pathogenesis of the disease, reduction and elimination of systemic manifestations, thereby preventing cartilage destruction, disability patients iatrogenic complications, quality of life of patients.

Treatment should ensure maximum recovery of joint function, reduce inflammatory synovitis, slowing the progression of destructive changes in the joints.

In the treatment of JRA monotherapy is not possible because of unknown etiology and pathogenesis is a complex disease. Thus, treatment of JRA is complex and combines various groups of drugs.

In the treatment of juvenile rheumatoid arthritis in children using nonsteroidal anti-inflammatory drugs (NSAIDs) with relatively rapid effect and basic therapy for a long time. A special place in the treatment of JRA take CC are used now mostly in systemic forms of the disease.

NSAIDs combine a large group of drugs. Traditional nonsteroidal drugs - salicylates, phenylbutazone, ibuprofen, naproxen, ketoprofen, diclofenac sodium, indomethacin, piroxicam - is a non-selective against COX-1 and COX-2 and determine the risk of adverse reactions. Recent expressed as follows: erosive and ulcerative processes of the stomach and intestines, gastrointestinal bleeding, a tendency to water retention, sodium tissues, increased creatinine in serum, increased blood pressure, the development of interstitial nephritis and renal capillary necrosis. It can also develop toxic hepatitis, "aspirin" asthma. Moreover, among NZPZ ibuprofen is less toxic, highly toxic diclofenac sodium - indomethacin, meklofenamat, ketoprofen.
Currently, we favor selective COX-2 inhibitors: meloksikamu (mova-forest) and nimesulide. They are characterized by high anti-inflammatory activity and minimal side effects. In recent years, a new generation of drugs using COX-2 inhibitors - tselekobsib and rofenobsib - they are more pronounced inherent selectivity to COX-2 and minimal complications from the gastrointestinal tract.

In the treatment of NSAIDs should consider the following:
- half of children with juvenile rheumatoid arthritis, a positive clinical effect occurs within two weeks of starting treatment, so the time to assess the effect of their use does not exceed 30 days;
- NZPZ combine irrational, since it increases the risk of gastrointestinal complications, renal disorders of hemostasis;
- Absolute contraindications for the appointment of NSAIDs is erosive and ulcerative processes of the digestive tract, severe heart, lung, renal failure, elevated serum creatinine;
- the use of highly selective inhibitors of COX-2 is associated with a high risk of thrombosis, in particular myocardial infarction.

NSAIDs with JRA are symptomatic and do not prevent disease progression. Reducing the intensity of autoimmune reactions (the basis of JRA) is achieved using basic therapy (DMARD - disease-modifying anti-rheumatic drugs).

To the basic therapy drugs include:
- quinolone derivative, Immunosuppressants (methotrexate, cyclosporin B, azathioprine) sulfosalazin. In addition, the group of drugs refer leflyunomid basic therapy, etanercept, infiksamit, minocycline, imunoabsorbsiyo staphylococcal protein A (as recommended by the APA, 2002 "Guidelines for the treatment of RA").

Currently, no single provision of basic starting drug therapy. In mild forms of juvenile rheumatoid arthritis (articular form in the early phase of disease with minimum and average activity) often prescribe aminohinolony (delagil, plakvenil) having a soft imunosupresornu action, binding of DNA and RNA, disrupting their metabolism. Therapeutic effect develops slowly (1-1,5 months. From start reaching a peak after ≥ 6 mo.).

Delagil assign 4 mg / kg bw once a day (at night), but not ≥ 250 mg after meals. Plakvenil prescribe once 8 mg / kg bw not ≥ 400 mg. In patients with arthritis and systemic forms of the disease with these drugs do not give the desired effect. Side effects in the treatment of quinoline drugs:
- retinopathy with loss of vision;
- leucopoenia, thrombocytopenia;
- photosensitivity, dermatitis, pigmentation disorders of the skin, hair, iris, alopecia;
- reduced secretion of gastric juice;
- myopathy;
- diarrhea;
- myasthenic syndrome.

When severe, progressive generalized versions steroyidozalezhnynh course JRA is an immnosuppressant drug of choice. The nature of highlight antimetabolites (methotrexate, azathioprine), which block the synthesis of nucleic acids and alkylating agents (chlorbutin, tsyklofosfatyd) that can denature nucleoproteins. Drug of choice in this group is methotrexate - folate antimetabolites which reduces the rate
of growth of actively proliferating tissues, decreases the synthesis of antibodies, Russia, normalizes the synthesis of inflammatory and immunoregulatory cytokines in slow destruction of bone and development of osteoporosis. These properties provide methotrexate high efficiency and good tolerability. Methotrexate in JRA prescribe once a week from 2.5 to 7.5 - 10 mg / m2 and Adolescents maximum dose of 15 mg / m2. The therapeutic effect of taking immunosuppressants achieved in 60-70% of patients resistant to other forms of treatment. However, quite often (40-50%) of developing side effects.

The advantage of methotrexate is as follows:
- The possibility of long-term use in standard doses with no adverse effects;
- efficiency in oligo-, polyarticular JRA;
- suspension of cartilage and bone destruction, often only in reducing clinical disease activity and laboratory parameters;
- well protysupresyvnymy combination with other drugs.

Disadvantages methotrexate therapy:
- no effect on the activity of T-lymphocytes (does not block their clonal expansion);
- imunosupresornyy insufficient effect at low doses;
- increased risk of side effects at doses ≥ 12 mg / m2 per week;
- insufficient effect of severe systemic JRA;
- not eliminate koksyt;
- not stimulate the repair of cartilage and bone in aseptic necrosis of femoral head;
- Tolerance may develop after a positive dynamics.

Complications methotrexate therapy, toxic hepatitis, amyloidosis progressing the development of cancer, sepsis, infertility.

Indications of methotrexate in the treatment of JRA:
- Still syndrome moderate;
- polyarticular of (-) and Russia (+) JRA;
- pausiatrykulyarnyy option 1 type.

This methotrexate dose is 10-12 mg / m2 per week.

cyclosporin_A(Sandyman, Neoral) TsyA sufficiently effective drug therapy and the base is selective in Immunosuppressants beloved dose. It blocks proliferation of T cells through inhibition of the synthesis of IL-2, blocking antigen presentation by macrophages T-lymphocytes, inhibits the synthesis of pro-inflammatory cytokines (IL-6, FNO-α, FNO-β, IL-8), antibody B-lymphocytes). By blocking the synthesis of proinflammatory cytokines, reduces the resorptive activity of calcitriol, PTH and prostaglandin E2. It has a direct effect on chondrocytes, osteoblasts, blocking the synthesis of them stimulants resorption (FNO-α, prostaglandin E2). As a result of these processes TsyA prevents the development of cartilage and bone destruction, stimulates regeneration and growth processes. TsyA dose - 2,5-4 mg / kg DD in two stages (under the control of renal function, including serum creatinine).

Benefits TsyA:
- no effect on macrophage function, infectious complications;
- tsytopenichnych no reactions;
- suspend destruction of cartilage and bone;
- Stimulation repair femoral head in aseptic necrosis;
- effectiveness in severe systemic JRA;
- the possibility of long-term treatment with no side effects;
- the possibility of treatment without NSAIDs;
- abolition of the Civil steroyidozalezhny patients.

Disadvantages TsyA:
- less ability to inhibit the function of macrophages than T lymphocytes;
- lack of effectiveness in JRA from Russia (+);
- Activity insufficient effect in most patients with the syndrome and Still alerhoseptychnym JRA.

Indications for use TsyA:
- Still syndrome;
- alerhoseptychnyy syndrome;
- koksyty, the threat of aseptic necrosis of femur head;
- aseptic necrosis of the femoral head bone.

Currently, developed a program of monotherapy and combined therapy using drugs base:
- rising - "step up" from a gradual increase in the dose and number of drugs;
- downward - "step down".

According to ARA is the most optimal combination of TsyA plakvenilom MT or less effective - the MT with leflyunomidom, plakvenilom.

Indications of Civil systemic effects in the treatment of JRA:
- highly active form of systemic manifestations and multiple lesions of joints;
- specific eye disease;
- inefficiency NSAIDs and basic drugs in severe autoimmune shifts, acute immune crises.

Using GC based on the pronounced anti-inflammatory and immunosuppressive effects, the main mechanisms which are:
- stabilization of lysosomal membranes and cell organelles, prevent the allocation of cytotoxic enzymes;
- decrease vascular permeability, improve microcirculation, inhibition receipt of leukocytes and mast cells in the area of inflammation;
- inhibition of activation of enzymes "arachidonic cascade": phospholipase Aα, COX-2, lipooksyhenazy;
- inhibition of lipid peroxidation;
- inhibition of metalloproteinases genes excretion;
- inhibition of synthesis and the final effect of humoral mediators of inflammation;
- stimulation apoptosis of T and B lymphocytes.

So, all GK affect the pathogenesis of the disease and are versatile, fast strong anti-inflammatory effect. However GC therapy leads to the development hormonorezystentnosti, hormonozalezhnosti, severe, often irreversible side effects.

For the treatment of JRA use mainly two drugs - prednisone and methylprednisolone (metipred, solyumedrol). Dose choose individually at a rate of 0.5-1.5 mg / kg m.t.d.d. The full dose 1-1.5 months taken to stabilize the pathological process, and continue to slowly reduce the maintenance dose of 1/4, 1/8 tab. once in 4-5 days) 5 mg DD At present the benefits of minimal doses of GC - ≤0,3 mg / kg
To prevent withdrawal of the Civil introduce intra - in the most painful joints no more than 2-3 times in one joint. Using methyl prednisolone and diprosan.

Severe JRA is an indication for individual treatment. In severe disease:
- onset before the age of five;
- Systemic JRA debut variants;
- debut on the type of seropositive RA;
- oligoartrykulyarnoho debut on the type of option type 2;
- rapid formation of symmetric generalized syndrome;
- continuously relapsing course;
- the growth of functional failure in the first 6-12 months. from the onset.

This intensification of treatment includes:
- plasmapheresis (4-10 sessions);
- methylprednisolone pulse therapy 5-15 mg / kg bw

Schemes pulse therapy:
- 1 gram of the drug per day;
- 3 grams within three days;
- 1 gram once a month for 6 months;
- 1 or 3 grams per 1 gram of methylprednisolone and cyclophosphamide;
- pulse therapy + = synchronous plasmapheresis therapy;

- / v input immunoglobulin (VVIH) 0.5-1.0 g / kg bw three times a day in combination with a broad spectrum antibiotykmky re-monthly or at intervals of 3-6 months. This makes it possible to reduce the dose GC. Properties VVIH: neutralization of virus and inactivation of complement, phagocytosis stimulation, neutralizing autoantibodies and autoantigens, control inflammation by binding to basophils and mast cells, stimulation of proliferation, differentiation uu T-suppressors.

Indications VVIH:
- the drug of choice in combination with methylprednisolone pulse therapy, intraarticular injection GC and broad-spectrum antibiotics in treating alerhosepsystu;
- "Therapeutic cities" of the appointment of immunosuppressive drugs to the appearance of their therapeutic effect in systemic JRA;
- use at the onset of severe systemic manifestations of JRA.

Treatment - Immunosuppressants, imunosupresorna combination therapy.

In recent years, there are reports of new treatments JRA:
- antyfaktor tumor necrosis (anti FNO-α): remikeyd, etanercept;
- dissolved recombinants receptor FNO-α, linked protein Fc fragment IgG;
- in the introduction of anti-CD4 monoclonal antibody in combination with methotrexate;
- Chicken collagen type II orally;
- metalloproteinase inhibitors (selective drugs with chondroprotective action);
- terozynkinazy cytoplasmic inhibitors (decrease osteoclast activation, synoviotsytiv);
- modulators of the immune response: antibodies to surface molecules T cells, cytokines, etc.;
- genetic engineering.

Over the past 10 years successfully used systemic enzyme (vobenzim, flohenzym) based on their anti-inflammatory, antykoahulyatsiynoyi action, the ability to normalize metabolic disorders.

The Institute PAG used to treat JRA omega-3 polyunsaturated fatty acids (n "eikonal equation" domestic "Tecom" French "Maher"). Anti-inflammatory effect caused by the decrease produktsiyiIL-1, IL-2, FNO peripheral blood mononuclear stabilizing effect on endogenous proteins, inhibition of proliferation of T-lymphocytes tsytiv.

Local treatment:
- application of the 50% DMSO district combined with aminophylline, nicotinic acid, heparin;
- NSAID application in the form of ointments, gels, creams.

Physiotherapy: UFO, hydrocortisone phonophoresis, electromagnetic fields and ultra-zverhvy sokyh frequency variable magnetic field of high frequency, paraffin, ozokeritotherapy.

**Topic:** Violations heart rhythm and conduction in children.

Content topics:

**The main causes of cardiac arrhythmias:**
1) cardiovascular system - myocarditis, heart disease, cardiomyopathy, pericarditis, cardiac tumors, myocardial degeneration, injury of the heart;
2) Psychogenic arrhythmia, diseases of the nervous system, pathology of the spine.
3) Vistserokardialni effects - a disease of the gastrointestinal tract, respiratory system.
4) Diffuse connective tissue disease.
5) Intoxication (exogenous and endogenous origin);
6) Violation of fluid and electrolyte balance;
7) Genetically determined syndrome (WPW-syndrome, sick sinus syndrome, prolonged QT et al.).

**Mechanisms of arrhythmias.**
There are several theories:
- theory of re-entry;
- Theory currents injury;
- theory phase of increased excitability;
- paraarytmiy theory;
- Theory "Moon";
- Theory lability assimilation rate;

**Classification of arrhythmias and conduction electrocardiogram (Kushakov NS, NB Zhuravlev, a modification Murashko VV, 2000).**

I. Violation formation rate
A. Violation of automaticity of sinus node (nomotopni arrhythmias).
1. Sinus tachycardia.
2. Sinus bradycardia.
3. Sinus arrhythmia.
4. Sick sinus syndrome.

B. ectopic (heterotopic) rhythms due to the predominance of ectopic automatism centers.
   1. Slow (replacing) complexes and rhythms:
      and) atrial;
      b) AV-out connection;
      in) ventricle.
2. The migration of supraventricular pacemaker
3. Accelerated ectopic beats (tachycardia neparoksyzmalni):
      and) atrial;
      b) AV-out connection;
      in) ventricle.

B. Ectopic (heterotopic) rhythms, preferably not involve a violation of automatism.
1. Extrasystole (atrial, with AV connections, ventricular).
2. Paroxysmal tachycardia (atrial, with AV connections, ventricular).
3. Atrial flutter.
4. Flashing (atrial) fibrillation.
5. Blink and flutter (fibrillation) ventricles.

II. Violation conductivity.
1. Sinoatrial (SA) block.
2. Vnutrishnoperedserdna blockade.
3. Atrioventricular block (I, II, III degree).
4. Intraventricular blockade (blockade branches bundle branch block):
   and) one branch (monofastsykulyarni);
   b) two branches (bifastsykulyarni);
   c) three (tryfastsykulyarni).
5. Ventricular asystole.
6. Pre-excitation syndrome:
   and) syndrome WPW;
   b) syndrome reduced PQ (R) = syndrome CLC (LGL).

III. Combined arrhythmias.
1. Parasistoliya.
2. Эктопичні rhythms blockade exit.
3. AV dissociation.

Diagnostics
   The main method of diagnosis of arrhythmias and conduction remains electrocardiogram. Another diagnostic method is a method of assessment Holter monitoring daily variability of heart rate.
   The algorithm of differential diagnosis of tachycardia
1. Detection of tachycardia (heart rate is determined).
2. Definition of complaints and anamnesis, allowing to confirm or exclude symptomatic tachycardia.
3. Assessment of the central nervous system, respiratory and circulatory system.
4. Detecting signs SMSV and CHF.
SMSV due to inefficiency diastole, reduced stroke volume and cardiac circulation. Clinically characterized by arterial hypotension and signs of circulatory centralization (symptom of "white" spots, oliguria, decompensated lactic acidosis).

CHF is characterized by small overload and / or systemic circulation. Congestion in the pulmonary circulation determined by the appearance of shortness of breath, wheezing humid in the lower regions of the lungs and in the future - the development of pulmonary edema. Stagnation in the large circulation characterized by the appearance of peripheral edema, increased liver size, development of ascites and hydrothorax.

5. ECG examination, if possible - ECG monitoring.

6. In patients with clinical signs of heart failure or SMSV, regardless of its cause tachycardia and required urgent hospitalization of the child in the ICU.

**Paroxysmal tachycardia.**

**Supraventrylyarna**

Characterized by increased heart rate to 160-200 beats per minute, sudden onset and sudden termination of the attack. Maybe atrial and atrioventricular, it is difficult to differentiate in the stratification of P wave to the other elements of the ECG.

Diagnosis. During the attack excitation pulses can come from a variety of ectopic cells, sinus-atrial zone atrium (atrial form PT), atrioventricular node (AV-form PT). Duration of attack from several seconds to several hours or even days. With an increase in heart rate of 220-250 per minute more developed SMSV.

NB! If the duration of the attack more than a day developing CHF!

In the form of atrial PT ECG recorded a number of consecutive atrial beats (at least 4-6 with a frequency of more than 160 per minute). prong P various forms of (+, -) or not detected. QRS complex is not changed. Can stratify incomplete transient AV-block I-II levels.

In atrioventricular form PT -QRS complex is not changed. P negative prong recorded by the complex QRS. Maybe AV dissociation due to retrograde atrial block.

**Algorithm for urgent action**

**Step A**

1. Lay the child in a horizontal position and provide fresh air.
2. Reflex techniques increase vagal tone performed in children over 3-4 years:
3. Sedatives, Corvalol, valokordin, valerian.
4. Asparcam (Panangin).

If no effect - hospitalization in children kardiorevmatolahichne compartment depth examination and selection of therapy. Step B

The control heart rate, blood pressure, ECG monitoring.

In the absence of effect of reflex techniques and sedatives used antiarrhythmic drugs in that order.

1. Izoptyn (verapamil) 0.25% solution in / in slowly (no dissolution) in age doses: In the absence of the effect of the introduction izoptynu can re-enter after 15-20min. **Izoptyn form suitable for supraventricular PA aberrant ventricular complexes.**
2. Panangin (introduced after izoptynu).
3. ATP
4. Aymalin (hilurytmal)
5. Digoxin
Shown with a decrease in blood pressure and pumping function of the myocardium. Digoxin is contraindicated in the form of supraventricular PA aberrant ventricular complexes.

6. When clinical signs of heart failure - diuretics: furosemide (Lasix), veroshpiron in age doses.

In the absence of effect of the antiarrhythmic therapy - transfer the patient to the ICU or kardiorevmatolohichnyy center stage in which exercise (minutes).

**Ventricular paroxysmal tachycardia**

Clinically characterized by increased heart rate to 140-250 beats per minute, sudden onset and sudden end attack. With an increase in heart rate over 120-140 beats per minute SMSV develops. The ECG is recorded several successive ventricular beats (more than 5). Wide QRS complex (more than 0.1 seconds), twisted, T waves discordant main prongs complex QRS. R prong rarely detected through layers of other elements of the ECG. For the duration of ventricular PT can be "volley" or continuous-recurrent, mono- or politopnoyu. Politopna or chaotic ventricular PT is dangerous for the development of ventricular fibrillation. Urgent measures Phase A and B

Due to the rapid development of life-threatening conditions (ventricular fibrillation, heart failure) stages of emergency aid on time should be as short and sick child should be hospitalized in the ICU urgent. Prior to hospitalization of the patient is necessary to purchase urgent measures to attack. 1. Lidocaine. If no effect can be repeated administration of lidocaine after 5-10 minutes in the half dose.

2. novokainamid. At the same time to prevent hypotension in / introduced mezatona solution.

Step B is carried out in the ICU.

**Urgent measures the syndrome of Morgagni-Adams-Stokes**

Step A

In the prehospital ensure free access of air to the respiratory tract baby, if possible, carry oxygen therapy.

2. Atropinu sulfate

In the absence of the effect of atropine can be used eufillin

3. Provide urgent hospitalization of the child in the ICU.

4. Perform ECG differential diagnosis form for bradycardia.

5. With the development of asystole - of cardiopulmonary resuscitation. Step B is carried out kardioreanimatsiynomu department or missing. In the absence of the effect of those measures and maintaining SMSV, signs of heart failure, recurrent attacks of Morgagni-Adams-Stokes - advice surgeons to address the urgent surgical implantation of an artificial pacemaker (pacemaker) in terms of cardiac center.

While improving the state of the sick child and stabilize the heart rate within the age norm shows a cardiologist consultation and transfer of specialized kardiorevmatolohichne department to clarify the clinical diagnosis and subsequent forms bradyarrhythmias etiopathogenetical treatment.
Topic: Hypertension.

Content topics:
Hypertension occurs in 1-3% of children and adolescents. Blood pressure in children gradually increases with age. At birth, the SAT is at 75 mm Hg in the first weeks of life is increasing day by 1-2 mm Hg continue its growth slows.
Interpretation of blood pressure in children is based on epidemiological studies have established its guidelines for different age groups. Unlike adults, for children there is no single blood pressure that is treated as normal. The upper limit of normal pressure taken overall, 90-94 percent of its distribution in children of this age group and sex (Table. 1). Blood pressure equal to the 95th or exceeds its interest is considered hypertension.

The level of blood pressure depends not only on the age and sex of the child or young person, but also on its growth, the higher the growth, the higher the blood pressure. Most children with elevated blood pressure with mild hypertension. If the pressure is significantly increased, indicating the nature of secondary hypertension. The prevalence of secondary hypertension in children is 5 to 10% of cases improve AT the same way as adults.

To facilitate the application of these regulations should remember that children under the age of ten should alert the physician pressure exceeding 110/70 mm Hg, and after ten years - higher than 120/80 mmHg. In such cases, refer to the tables that define pressure ratios for children, and if the pressure is higher than normal, to resort to a detailed examination and treatment. About 70% of secondary hypertension in children are caused by diseases of kidney parenchyma, 10% - vascular diseases. Diagnostic studies to identify the causes of hypertension in children-the same as in adults.

The goal of treatment orphaned reducing blood pressure to normal for the age, sex and height values of blood pressure. Immediate treatment should begin when the child has severe hypertension or if the background or mild to moderate hypertension are diabetes, kidney disease or target organ damage. In all other cases, treatment begins with non-drug measures (weight reduction, limiting salt intake, exercise). Uncomplicated hypertension is not a reason to restrict physical activity. Children should be encouraged dynamic exercise (without competition). Isometric (static) load should be minimized, and in severe hypertension and hypertrophy of the left shlunochka- presence banned. If non-pharmacological treatment has no effect, prescribe medicines. For the treatment of children apply the same classes of drugs as for the treatment of adults: β-blockers, diuretics, calcium channel blockers, ACE inhibitors, a1-blockers and angiotensin II receptor blockers. Doses should be individualized, taking into account body weight. Children should limit early puberty purpose of beta-blockers because of their effect on the central regulatory structures involved in processes of puberty. Most children and teenagers using ACE inhibitors and calcium channel blockers. The use of ACE inhibitors in children rarely causes side effects (cough, rash or neutropenia), this group of drugs, in addition to antihypertensive inherent even nefroprotektorny effect, which leads to their use in children with diabetes. However, using ACE inhibitors should be mindful of their impact on the processes of proliferation, maturation and hypertrophy of collagen,
Monitoring the progress of arterial hypertension in children and adolescents show that high blood pressure can be stored in them as adults, with the highest predictive value to stabilize hypertension are heredity, obesity and left ventricular hypertrophy, when it can not be established, to talk about essential (idiopathic) Background hypertension is associated with a high prevalence of it, low awareness of the existence of this disease and insufficient effectiveness of treatment. The frequency of hypertension. children in the first two years of life is 2-3%, the older it grows, reaching school-age children 8-10%. Primary or essential, hypertension (hypertension) in children is relatively rare. However, according to many researchers, this option hypertension. it begins to develop in childhood, especially in children from families with hereditary predisposition to hypertension. Most neurogenic hypertension in adolescents - a manifestation neurocirculatory dystonia. Blood pressure is gradually increased with age. In newborns it is about 75/40 mm Hg. c., boys - 100/65 and adults - 140/90 mm Hg. c. On the recommendation of the experts say in hypertension when blood pressure in the brachial artery in repeated measurements in terms of its basic exchange lasted than age-sex-zrostovu norm. There systolic hypertension, due to an increase in cardiac output or arterial stiffness and diastolic, which occurs due to increased vascular resistance blood flow at the level of arterioles. Systolic and diastolic hypertension usually combined, but may exist separately.

Hypertension in children and adolescents is common especially (among students 12-13 years of 10-14%). Maybe primary and secondary (usually kidney). To identify patients with hypertension is important to regularly measure blood pressure to all children during checkups in child care centers and clinics. Children with high blood pressure should be under medical supervision (in clinics).

Assessment of blood pressure. With targeted and mass measurement of blood pressure can be made protsentylnyy analysis: the pressure distributed to 100% scale; believe that top level, corresponding to 95 percentile points to the risk of hypertension and the level of over 99% suggests hypertension. Primary hypertension in children with hypertension often seen as an initial step that could lead to hypertension.

Great value in the event of primary hypertension providing genetic predisposition (it belongs to the multifactorial diseases with polygenic inheritance). To diagnose especially important to have hypertension parents (mostly mother). The implementation of disease is equally significant and adverse environmental impacts, conflict and stress in the family, alcoholism, active and passive smoking, lack of exercise. Some important are personal characteristics (increased anxiety, reduced tolerance to impact others). Increased body weight is a risk factor. Revealed correlative links between hypertension and atmospheric pressure, air temperature, relative humidity.

The clinical picture.
The main complaint of sick children - a headache. Children wake of the "severe" head upright headache expire and then amplified by the end of lessons. Localized on both sides. Possible crisis - severe headache attacks against the backdrop of increase (significantly more than normal) blood pressure.

Diagnosis.Already in the early stages of the disease set (using conjunctival biomicroscopy) significant microcirculatory disorders, spastic phenomena in the
department of blood, increasing twisting and increased caliber venuljarnom
department. With the progression of the disease is increasing spastic phenomena.
This pattern confirms rheographic study. Blood pressure in children with primary
hypertension is characterized mainly moderate increase in systolic pressure, its great
lability, increased middle and lateral pressure. As the disease progresses and
increases diastolic pressure. At follow-up blood pressure sometimes changes in the
direction of normalization, while it can occasionally grow again, sometimes high
blood pressure stabilizing or progressing. Typical of central hemodynamics: with
about equal frequency in the early stages of the disease occurs hyperkinetic variant
(disproportionately increased output and reduced peripheral resistance) and
hypokinetic (reduced output and increased peripheral resistance adequately). ECG
changes reflect enhanced sympathetic effects (tachycardia, decreased T wave in leads
II, aVF, Vs), metabolic changes (increase in T wave in leads II, V2-C), as well as
signs of hyperfunction (and in later stages and hypertrophy) of the left ventricle.
Typical phase shift diastole and systole. metabolic changes (increase in T wave in
leads II, V2-C), as well as signs of hyperfunction (and in later stages and
hypertrophy) of the left ventricle. Typical phase shift diastole and systole. metabolic
changes (increase in T wave in leads II, V2-C), as well as signs of hyperfunction (and
in later stages and hypertrophy) of the left ventricle. Typical phase shift diastole and
systole.

A variety of causes of high blood pressure is often confusing to a novice doctor,
forcing him to rush the patient and expose a large number of different studies to
confirm or exclude SAG. Since the vast majority of patients discover the specific
cause of hypertension difficult (or not possible), in its future work and the doctor
does not try to understand everything, and puts patients amorphous diagnosis
"hypertension" and "hypertension" the pattern.

In order to avoid these extremes in their practice, it is necessary to leave the
established statistical fact: massive population survey showed that about 95% of
people with high blood pressure suffer from hypertension (EH), and only about 5% -
hypertension is a symptom other diseases. Thus, the diagnosis of GC can be set only
by the exclusion of all other forms of possible SAG. Otherwise, some patients
received SAG fundamentally wrong, prolonged and unsuccessful treatment, losing
the chance for radical treatment.

Having found when examining the patient's high blood pressure, we may only
talk about "AH syndrome", referring to its numerous reasons.

Classification of SAG.

First place in the structure of SAG take nephrogenic (renal) hypertension (3-4%),
which in turn are divided into 3 groups:

1. parenchymal renal disease (glomerulonephritis, pyelonephritis, polycystic,
nephropathy various origins, amyloidosis, tumors, tuberculosis, kidney, etc.);
2. vasorenal (renovascular) hypertension (0.5-1%), due to various reasons (renal
artery atherosclerosis, thromboembolism, dysplasia, aneurysm, creases, compression
of the renal artery Nephroptosis etc.)
3. SAG-related disorders urodynamic (urine outflow), the so-called "urological
AG" (urolithiasis, congenital kidney, hydronephrosis, BPH, reflux nephropathy, etc.);
Keep in mind that the malignant form of hypertension SAG occur in 10 - 20 times
more than in GB and almost all of them Nephrogenic.
Less common endocrine hypertension (0.1 - 0.3%) due to pheochromocytoma, primary aldosteronism (Conn's syndrome), a disease or syndrome, Cushing's, acromegaly, hyperthyroidism, etc. Some diseases of the heart, aorta, and large vessels accompanied by hemodynamic (cardiovascular) AG as a result of change of the systemic blood flow (atherosclerosis of the aorta, aortic coarctation, aortic insufficiency valves, complete atrioventricular block, congestive heart failure, etc.). Most of hypertension characteristically isolated or claim elevation increase in systolic blood pressure.

Disease (damage) of the brain and/or spinal cord is causing neurogenic (tsentrohenniy) SAG (injury, subarachnoid hemorrhage, brain tumors, abscesses, encephalitis, meningitis, diencephalic syndrome, etc.). In addition to the above SAG there are special forms of hypertension: the abuse food salt products containing tyramine (aged cheeses, some brands of red wine, pickled herring, beans, chocolate), especially in patients taking inhibitors of the enzyme oxidase (MAO); as a result of taking certain medicines or substances (corticosteroids, cyclosporine, ergotamine, oral contraceptives, sympathomimetics, some drugs, alcohol).

The sharp increase in blood pressure observed in patients after heart surgery (coronary artery bypass surgery, aortic valve implantation, transplantation of the heart). SAG above list is far from complete, but of greatest practical interest in the work of medical practitioners. "Classic" versions course SAG rare, more common forms are combined, so differential diagnosis difficult because many of the same symptoms, no pathognomonic symptoms and subjective errors.

**Scheme of the examination of patients.**

In our country, proposed and successfully tested a long-term practice two-step system of inspection of patients: I stage - is binding studies easily feasible in outpatient therapeutic departments of hospitals, diagnostic centers. And not all patients must carry them in full, the older the patient - the smaller the amount of research that younger - the more likely SAG.

In phase II, all studies conducted strictly on the testimony (after a joint survey on the I stage) in specialized centers or offices, using invasive techniques, radioisotope studies, selective angiography, computed tomography, etc. Thus, the I stage of a detailed questioning of the patient, analysis of all complaints, a detailed history of life and disease. Then, a review of a patient with multiple (5.6 times) the measurement of blood pressure in both arms and legs, palpation and auscultation of the heart and major arteries; performed instrumental examination, ECG, ultrasound of the heart, the aorta and its branches, kidneys and renal arteries; X-rays of the chest, if necessary - viewing a picture or renal excretory urography; rheography brain, heart, limbs, Ophthalmoscopes (ocular fundus).

**A guideline on hypertension in children**

1. Blood pressure should be assessed each time the survey of children 3 years and starshe.2. It is necessary to fix the rise of blood pressure during at least three separate visits (90 percentile on age, height and gender) to establish a diagnosis of hypertension.

3. Patients with a primary diagnosis of hypertension should be a comprehensive assessment of cardiovascular risk factors (lipid profile, fasting blood glucose, body mass index).
4. pharmacological treatments (eg, weight loss, dietary changes, exercise) should be first-line therapy in patients with stage I hypertension.

5. Pharmacological treatment should be initiated in patients with stage 2 hypertension, symptomatic hypertension when present end organ damage (left ventricular hypertrophy, retinopathy, proteinuria), and stage I hypertension when blood pressure does not respond to lifestyle changes.

### The causes of childhood hypertension by age group (in frequency)

<table>
<thead>
<tr>
<th>Age</th>
<th>Causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>One to 6 years</td>
<td>Renal parenchymal disease, renal disease; endocrine causes of hypertension; coarctation of the aorta; hypertension</td>
</tr>
<tr>
<td>From six to 12 years</td>
<td>renal parenchymal disease; hypertension, renal disease; endocrine causes coarctation of the aorta; iatrogenic disease</td>
</tr>
<tr>
<td>From 12 to 18 years</td>
<td>Hypertension; iatrogenic disease, renal parenchymal disease, renal disease; endocrine causes aortic coarctation</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Family history</th>
<th>Possible causes and / or associations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease, such as myocardial infarction, stroke</td>
<td>Primary hypertension</td>
</tr>
<tr>
<td>Deafness</td>
<td>Congenital kidney disease or family</td>
</tr>
<tr>
<td>dyslipidemia</td>
<td>Primary hypertension</td>
</tr>
<tr>
<td>Endocrine problems (such as diabetes, thyroid, adrenals)</td>
<td>Family endocrinopathy</td>
</tr>
<tr>
<td>Hypertension</td>
<td>Primary hypertension</td>
</tr>
<tr>
<td>kidney disease</td>
<td>Congenital kidney disease or family</td>
</tr>
<tr>
<td>sleep apnea</td>
<td>Primary hypertension</td>
</tr>
<tr>
<td>Children's history</td>
<td></td>
</tr>
<tr>
<td>Chest pain</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Sweating (abnormal)</td>
<td></td>
</tr>
<tr>
<td>Shortness of breath on exertion</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Edema</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Enuresis</td>
<td>Renovascular renal disease</td>
</tr>
<tr>
<td>deficit growth</td>
<td>endocrinopathies</td>
</tr>
<tr>
<td>Heat or cold intolerance</td>
<td>endocrinopathies</td>
</tr>
<tr>
<td>Heart palpitations</td>
<td>Cardiovascular disease</td>
</tr>
<tr>
<td>Headache</td>
<td>Primary hypertension</td>
</tr>
<tr>
<td>Hematuria</td>
<td>Renovascular renal disease</td>
</tr>
<tr>
<td>Joint pain or swelling</td>
<td>Rheumatic disorders</td>
</tr>
<tr>
<td>myalgia</td>
<td>Rheumatic disorders</td>
</tr>
<tr>
<td>Hypovolemia newborns / shock</td>
<td>Renovascular renal disease</td>
</tr>
<tr>
<td>Periodic eruptions</td>
<td>Rheumatic disorders</td>
</tr>
<tr>
<td>Snoring and other sleep problems</td>
<td>Primary hypertension</td>
</tr>
<tr>
<td>Catheterization umbilical artery</td>
<td>Renovascular renal disease</td>
</tr>
<tr>
<td>Urinary tract infections (periodically)</td>
<td>Renovascular renal disease</td>
</tr>
<tr>
<td>Weight or changes in appetite</td>
<td>endocrinopathies</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>------------------</td>
</tr>
</tbody>
</table>

**These physical examination, which may indicate a secondary cause of hypertension**

<table>
<thead>
<tr>
<th>Physical examination</th>
<th>search for possible causes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal noise</td>
<td>renal artery stenosis</td>
</tr>
<tr>
<td>Pelvic masses</td>
<td>polycystic kidney disease; hydronephrosis / obstructive kidney damage; neuroblastoma; Wilms tumor</td>
</tr>
<tr>
<td>Acne</td>
<td>Cushing's syndrome</td>
</tr>
<tr>
<td>Adenotonsilyarna hypertrophy</td>
<td>Sleep disorders associated with hypertension</td>
</tr>
<tr>
<td>Reduced perfusion of the lower extremities</td>
<td>coarctation of aorta</td>
</tr>
<tr>
<td>Sweating</td>
<td>pheochromocytoma</td>
</tr>
<tr>
<td>Flushing</td>
<td>pheochromocytoma</td>
</tr>
<tr>
<td>The delay in growth</td>
<td>Chronic renal failure</td>
</tr>
<tr>
<td>Hirsutism</td>
<td>Cushing's syndrome</td>
</tr>
<tr>
<td>Swelling of joints</td>
<td>Systemic lupus erythematous</td>
</tr>
<tr>
<td>Rash</td>
<td>Systemic lupus erythematous</td>
</tr>
<tr>
<td>moon face</td>
<td>Cushing's syndrome</td>
</tr>
<tr>
<td>Blemish</td>
<td>coarctation of aorta</td>
</tr>
<tr>
<td>Muscle weakness</td>
<td>hyperaldosteronism</td>
</tr>
<tr>
<td>Obesity (General)</td>
<td>Association pervynnoyuhipertoniyyeyu</td>
</tr>
<tr>
<td>Obesity (face, neck or torso)</td>
<td>Cushing's syndrome</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Hyperthyroidism; pheochromocytoma; neuroblastoma</td>
</tr>
<tr>
<td>Tymomehaliya</td>
<td>hyperthyroidism</td>
</tr>
</tbody>
</table>

### The list of laboratory tests for hypertension in children

<table>
<thead>
<tr>
<th>Prychyna testing</th>
<th>Test</th>
<th>Goal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Determine the cause</td>
<td>Zach platelets, Electrolytes, urea, creatinine, calcium, phosphorus, uric acid, kidney ultrasound, ASDs tank sat urine</td>
<td>Exclude anemia in CKD background, Delete pyelonephritis, HN, kalkuloz, Delete dysmorfiyu renal anomalies, sclerosis, Exclude infection, hematuria, proteinuria</td>
</tr>
<tr>
<td>Identify related illness</td>
<td>Screening for drugs, Fasting lipid profile, glucose, insulin, polysomnography</td>
<td>Identify Drug-induced hypertension, Hyperlipidemia, metabolic syndrome, diabetes, Sleep disorders that are associated with hypertension</td>
</tr>
<tr>
<td>Identify target organ damage</td>
<td>ultrasound of the heart</td>
<td>Identify ventricular hypertrophy, Identify retinal vascular changes</td>
</tr>
<tr>
<td>Additional tests (if indicated)</td>
<td>Survey of fundus</td>
<td>Identify CKD</td>
</tr>
<tr>
<td>--------------------------------</td>
<td>---------------------------------------------------------------------------------</td>
<td>-------------------------------------------------</td>
</tr>
<tr>
<td></td>
<td>The protein and creatinine in a daily urine creatinine clearance</td>
<td>renovascular disease</td>
</tr>
<tr>
<td></td>
<td>Scanning kidney, MRI, CT, Doppler, arteriography</td>
<td>Exclude hypertension from white coat</td>
</tr>
<tr>
<td></td>
<td>Ambulatory blood pressure monitoring</td>
<td>Hyperthyroidism, adrenal dysfunction</td>
</tr>
<tr>
<td></td>
<td>Levels of hormones (thyroid, adrenal)</td>
<td>Diseases associated with mineralocorticoids</td>
</tr>
<tr>
<td></td>
<td>The level of plasma renin</td>
<td>Delete catecholamine-associated hypertension</td>
</tr>
<tr>
<td></td>
<td>Plasma and urine Catecholamines</td>
<td></td>
</tr>
</tbody>
</table>

**Diagnostic algorithm for hypertension in children.**

Blood pressure is gradually increased with age. In newborns it is about 75/40 mm Hg. c., boys - 100/65 and adults - 140/90 mm Hg. c. The recommendation of experts of the World Health Organization, the upper limit of normal blood pressure is 160/95 mm Hg. c. About hypertension say in cases where the blood pressure in the brachial artery in repeated measurements in terms of its basic exchange lasted exceeds the specified value. There systolic hypertension, due to an increase in cardiac output or arterial stiffness and diastolic, which occurs due to increased vascular resistance blood flow at the level of arterioles. Systolic and diastolic hypertension usually combined, but may exist separately. When the cause of hypertension can not figure out called symptomatic hypertension when it can not be established, to talk about essential (idiopathic) hypertension, or hypertension. Given the height of the blood pressure and the nature of the disease all of hypertension are divided into benign and malignant. Hypertensive heart disease is a benign course now the most common type of hypertension.

Differential diagnosis. Putting history, should pay attention to symptoms and signs that distinguish this sickness of the above-described typical course of hypertension. Often the cause of hypertension can not figure out even when questioning the patient. Particular attention should be paid to renal illness or past existence of signs that naturally occur in the kidney. Some diseases with great regularity found in the same family. Message patient hypertension disease of his relatives polycystic kidney, adrenal gland tumors, diabetes should be regarded as an indirect indication of its possible link hypertension to any congenital anomalies of the kidney or any hereditary disease.

**Topic:** Immunization of children's infectious diseases.
Important role in dilnosti pediatrician takes preventive pediatric infectious disease prevention through vaccinations. Every pediatrician should be aware that active immunization of the child is the most effective tool in the prevention of infectious diseases. Organization of vaccination requires a clear cooperation between pediatric and epidemiological services, health education full of seven "her.

The epidemic situation in Ukraine infections. Against which the specific means of prevention, zalyayetsya complex. Every year, tuberculosis, diphtheria, pertussis, tetanus, polio, measles, mumps, rubella, hepatitis B sick 70-90 thousand. People. Of these, 70% - children. Due to insufficient immune stratum of the population there are periodic and seasonal rises in the incidence of these infections.

The objective of immunization have control immune response to prevent disease in individuals and populations.

Ways immunization.
Active - stimulating the production of its own antibodies.
Passive - the introduction of ready-made antibodies.

Characteristics of drug-induced.
1. Vaccines goals include killing bacteria (pertussis, typhoid, cholera) or inaktyvovani viruses (influenza, polio Salk vaccine).
2. toxoid containing inaktyvovany pathogen toxin (diphtheria, tetanus).
3. Vaccines with live attenuated viruses (cow, mumps etc.).
4. Vaccines containing live microorganisms are cross-reactive (BCG).
5. Chemical vaccine fractions of dead microorganisms (pneumococcal, meningococcal).
6. genetically engineered recombinant, chemically synthesized (hepatitis B, influenza).
7. Associates (comprising of several vaccines).

The composition of vaccines.
1. Active or immunizing antigens.
2. Liquid foundation.
3. preservatives, stabilizers, antibiotics.
4. Aids.

Ways of introducing vaccines.
1. Intramuscular (DPT, ADP, ADP-M, rabies, meningococcal B).
2. Subcutaneous (cow, mumps, rubella, meningococcal A + C).
3. intradermal (BCG).
4. Skin (plague, tulyaremichna, brutselozna).
5. Because of the mouth (polio).
6. intranasally (inaktyvovani influenza).

vaccination process- a change homeostasis, resulting in the body in response to the drug-induced and include a set of complex reactions, which include antibody production, adaptation and post-vaccination reactions post-vaccination complications.
induced reactions occur in response to the vaccine, characterized by the appearance of manifest clinical symptoms characteristic of this type of vaccine are cyclical course, short-term, without causing serious disorders of the organism.

Post-vaccination complications - all abnormal phenomena that occur after vaccination and not characteristic of normal vaccination process, but their obvious connection with the vaccination conducted:

1) Unusual vaccination reactions and complications caused by the vaccine itself ("true");
2) joining intercurrent infection in the post-vaccination period;
3) and worsening of chronic primary manifestations of latent diseases.

RULES preventive vaccinations

Routine vaccination is postponed until the end of the acute manifestations of the disease and exacerbation of chronic diseases and conducted immediately after recovery, or in remission.

In case of contact with infectious patients prophylactic vaccination is not contraindicated.

It is unacceptable to combine in a single day vaccination against tuberculosis with other parenteral injections and manipulations.

BCG vaccination and conducting Mantoux test should not be conducted within 4 weeks after measles vaccine or measles.

After OPV vaccinations offered to limit injections, parenteral intervention, planned operations for 2 weeks. Children receiving immunosuppressive therapy with cytostatics, corticosteroids (more than 1 mg / kg / day for prednisone) for more than 14 days, you can vaccinate 1 month after discontinuation of these drugs.

Vaccination against measles, mumps and rubella after administration of immunoglobulin possible within the time specified in the instructions to the antibody, but not earlier than 3 months. After emergency prevention of tetanus neonatal BCG vaccination is carried by a common scheme.

If the interval between vaccination against measles, mumps, rubella and the introduction of immunoglobulin therapeutic and prophylactic purposes for at least 14 days, vaccinated against these infections should be repeated.

Vaccination of children burdened with history.

1. Children with allergic burdened history:
clarify allergic history;
selection of the optimal time of vaccination;
lengthening the interval between administration of the vaccine;
appointment antihistamines 2-3 days before vaccination and 5-10 days after her;
hypoallergenic diet.

2. Children with neurological disorders:
vaccination is carried out in a period of stable remission after neurological examination against the background of anticonvulsants, sedatives;
fever with antipyretics destination for children who have a history of febrile convulsions;
acute NEUROINFECTION vaccination is carried out after steady recovery (in 3-6 months after onset of the disease).

3. **Children often suffer from:**
   imparted 2-4 weeks after recovery from the last episode of infection;
   recommended to vaccination in the warmer months of strict isolation for 3-5 days.

4. **Children with long subfebrile:**
   vaccination is not contraindicated (in satisfactory condition, normal blood and urine).

5. **Children with immunodeficiency states:**
   IDS is contraindicated during the initial introduction of live vaccines (BCG, polio, measles, mumps);
   secondary IDS transferred due to infectious disease is not a contraindication (conduct vaccinations 2-4 weeks after recovery);
   the treatment of corticosteroid vaccination is carried out in 3 months after corticosteroid therapy;
   the treatment with cytostatics, radiotherapy - after 6 months after its completion;
   tymomehaliya not a contraindication to vaccination.

**Passive immunization is shown:**

1. Children with inadequate synthesis of antibodies as a result of congenital or acquired defects in cell lymphocytes.
2. In the absence of a vaccine against infection when the only way to protect the input is finished antibodies.
3. If necessary immediate disease prevention for epidpokazaniyam (contact with sick measles, prophylaxis of rabies, tetanus).
4. To neutralize toxin antigen-specific antitoxic antibodies.
5. With the purpose of treatment early in the disease (with diphtheria, botulism, tetanus).