MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Departments of Pediatrics №2

CONFIRMED by

Vice-rector for research and educational work

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METHODOLOGICAL RECOMMENDATIONS ON PRACTICAL CLASSES FOR STUDENTS

International Medical Faculty, course 6

Educational discipline "**PEDIATRICS**"

Approved
at the meeting of the department of Pediatrics №2
Protocol No. 11 dated 28/08/2022

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1. **Topic №**16

   Differential diagnosis of cyanosis, shortness of breath, cardiomegaly in congenital and acquired heart defects in children. Leading clinical symptoms and syndromes of diseases of the cardiovascular system in children accompanied by cardiomegaly (congenital and acquired heart defects). Differential diagnosis of cyanosis, shortness of breath, cardiomegaly in congenital and acquired heart defects in children. Data of laboratory and instrumental studies in congenital and acquired heart defects in children. Differential diagnosis of cyanosis, shortness of breath, cardiomegaly in congenital and acquired heart defects in children. Differential diagnosis of cyanosis, shortness of breath, cardiomegaly in congenital and acquired heart defects in children. Patient management tactics for congenital and acquired heart defects in children. Treatment and prevention of chronic heart failure. Medical supervision.

2. **Relevance of the topic:**

Any pathological enlargement of the heart is called cardiomegaly. The reasons for this increase may be: expansion of one or more chambers of the heart, hypertrophy or infiltration of the myocardium, pericardial effusion or ventricular aneurysm. Cardiomegaly can be detected already on physical examination, more often on chest x-ray. Cardiomegaly is the result of a chronic process, therefore, a complete examination of the patient is required to identify the disease that led to an increase in the size of the heart, as well as to assess the physiological consequences of cardiomegaly itself,

Cardiomegaly can be roughly divided into 2 groups:

1. associated with heart disease
2. not associated with heart damage.

Among the cardiac causes of cardiomegaly, there are 3 main groups of diseases:

1.Congenital heart defects

2Acquired heart defects

3. Non-rheumatic carditis.

3. Objectives of the course:

3.1. General goals: To get acquainted with the modern definition of the concept of carditis, their etiology, clinical signs, to be able to diagnose in children, to draw up a plan of therapeutic and preventive measures.

3.2. Educational goals: to get acquainted with the contribution of domestic and foreign scientists to the study of the problems of inflammatory heart diseases, to compare the percentage of morbidity in different regions of Ukraine, to remove factors that contribute to the development of diseases, to determine the need for prevention of carditis in children.

3.3. Specific goals:

- *toknow:*
- etiology, pathogenesis and non-inflammatory heart disease;
- features of collecting anamnesis in children of different age groups with cardiomegaly syndrome;
- clinical variants of the course and complications of myocarditis, endocarditis, pericarditis in children.
- classification of inflammatory heart diseases
- methods of diagnostics and differential diagnosis of inflammatory heart diseases,

accompanied by cardiomegaly;

- the main clinical manifestations of inflammatory heart diseases, accompanied by

cardiomegaly;

- ECG, EchoCG and X-ray changes in inflammatory heart diseases;
- tactics of patient treatment with myocarditis, endocarditis, pericarditis in children.
- provision of emergency care for acute cardiovascular failure.

3.4. *Based on theoretical knowledge on the topic*:

- *master the techniques / be able to* /:
- to collect anamnesis and conduct a physical examination of a patient with cardiomegaly syndrome;
- to evaluate the results of paraclinical studies (ECG, EchoCG, X-ray)
- tomake and formulate a diagnosis (cause of cardiomegaly)

- toassess the severity of the disease;
- toappoint a plan of examination and treatment

4.Materials for classroom self-study (interdisciplinary integration)

№	Disciplines	To know	Be able to do
1	**2**	**3**	**4**
1.	Normal anatomy, physiology	Anatomical and physiological features of the cardiovascular system in children of different age groups	Use knowledge to adequate assessment of clinical parameters
2.	Biochemistry	Biochemical blood test parameters	Assess biochemical blood tests
3.	Pathoanatomy	The main pathological changes in the cardiovascular system	Know the stages and timing of the development of inflammatory and non-inflammatory changes for timely and adequate therapy
4.	Pathophysiology Microbiology	Parameters of laboratory studies of the cardiovascular system	Evaluate immunological, biochemical and microbiological research
5.	Propedeutics of childhood diseases	Research methodology and semiotics of diseases of the cardiovascular system. ECG technique,EchoKG research	Conduct an objective review of the patient (examination, palpation, percussion, auscultation), evaluate the received results, evaluate the results of ECG, EchoCG studies
6.	Faculty Pediatrics	Clinical-anamnestic and laboratory-instrumental complex of signs of heart disease in children of different ages	Identify signs of cardiopathy in children of different ages
7.	Therapy	Clinical-anamnestic and laboratory-instrumental complex of signs of cardiopathy in adults and principles of treatment	Identify the features of cardiopathies in children, predict further complications
8.	Radiation diagnostics of the cardiovascular system	Indications and methods of radiation diagnostics of diseases	Evaluate radiographs and ultrasound diagnostic protocols

5. Lesson content

Cardiomegaly (KMG) is an increase in the size of the heart due to its hypertrophy and dilatation or accumulation of products by metabolic disorders, or the development of neoplastic processes. Common signs of cardiomegaly:

• rhythm and conduction disturbances

• physical data (expansion of the borders of the heart, displacement of the pulsation of the apical impulse, etc.)

• specific signs are determined by the disease that led to KMG.

The main reasons for KMG:

1. Physiological (sports, pregnancy)

2. Extracardiac causes - "apparent cardiomegaly": kyphoscoliosis, straight back syndrome, fatty deposits in the epicardium, formations in the mediastinum
3. IHD, HD
4. Toxic cardiomyopathies
5. Endocrine cardiomyopathies: diabetes mellitus, thyrotoxicosis, acromegaly, pheochromocytoma
6. Heart rhythm disturbances: AF

Heart defects

Cardiomyopathies

1. Carditis
2. Conditions with high cardiac output: anemia, arteriovenous fistulas
3. Tumors of the heart

Diseases accompanied by cardiomegaly syndrome can be grouped as follows:

A. Non-communicable heart disease:

1. Congenital heart defects
2. Tumors of the heart
3. Acquired heart defects
4. Cardiomyopathy

B. Infectious heart diseases:

1. Non-rheumatic carditis
2. Pericarditis
3. Infective endocarditis

B. Diseases of the neuromuscular system:

1. Friedreich's ataxia
2. Muscular dystrophy (Duchenne disease)

D. Congenital metabolic disorders

1. Glycogenosis
2. Mucopolysaccharidosis

D. Amyloidosis

Cardiomegaly can be seen first when:

1.routine physical examination (atrial examination, palpation, percussion, auscultation)
2. using the X-ray method (for example, fluorography)
3. performing ultrasound **examination of the heart (echocardiography)**

When a patient has cardiomegaly, it is necessary to determine a number of important questions:

1. Isn't cardiomegaly apparent (epicardial fat, changes in the mediastinal organs, chest deformities)?
2. What parts of the heart are enlarged (chambers of the heart, vessels departing from it, pericardium)?
3. How pronounced is the increase?
4. What caused the increase (dilatation, hypertrophy or infiltration of the myocardium, aneurysm of the heart, aorta or pulmonary artery, accumulation of fluid in the pericardial cavity, its tumor)?
5. Are these morphological changes in the heart (increase) accompanied by dysfunctions of the heart (excitation, conduction, contractility, relaxation)?
6. Does the patient have, clinical syndrome of heart failure along with myocardial dysfunction?
7. What disease (nosological form) underlies cardiomegaly? (How to formulate a clinical diagnosis?)

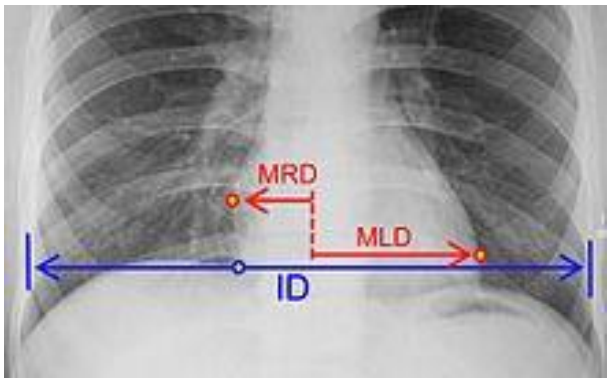
Mandatory questions to ask a patient with cardiomegaly:

1. Have you ever had a heart murmur or rheumatism?
2. Have you ever had high blood pressure?
3. Do you drink alcohol? How much?

4. Have you recently had a viral infection or flu, were there any complications?
5. Have you recently been pregnant? Were there any complications, including shortness of breath, swelling, palpitations?
6. Do you or any of your relatives have diabetes mellitus?
7. Have you had heart attacks or chest pains?
8. What medications are you taking?
9. Do you get tired quickly? Do you experience shortness of breath on exertion? Do you wake up at night due to shortness of breath, cough and have to sit up?
10. Do you notice swelling in your legs by the end of the day?

Methods for examining a patient with cardiomegaly:
1. Radiography (determination of CTE)
2. Electrocardiography
3. Echocardiography
4. Clinical blood test
5. Biochemical blood tests
6. Coronary angiography
7. Endomyocardial biopsy of the myocardium (and possibly other tissues)

Radiography can determine the overall size of the heart, the cardio-thoracic index, the position of the heart and its configuration. X-rays sometimes give false positive results. This situation is especially common in children and adolescents, because until now the concept of the norm in this group of patients varies widely. In addition, the technique of performing the study, the position of the patient is very important.



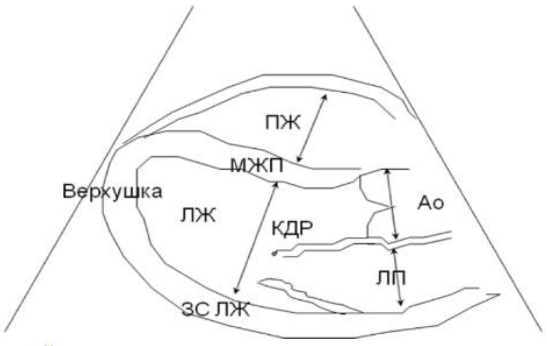
Cardio-thoracic index (CTI) is the ratio of the transverse size of the heart to the internal transverse size of the chest in its widest part
CTI = (MRD + MLD)/IDx100%
MRD = greatest perpendicular diameter from the midline to the right border of the heart
MLD = greatest perpendicular diameter from the midline to the left border of the heart
ID = inner diameter of the chest at the level of the right hemidiaphragm
Normally CTI in adults <50%, in children – 55-58%
Stage 1 cardiomegaly - an increase in CTIon5%
Stage 2 cardiomegaly - an increase in CTI on 10%
Stage 3 cardiomegaly - an increase in CTE on more than 10%

Possibilities of ECG for differential diagnosis of cardiomegaly:

Electrocardiographic diagnosis	Probable disease
Left ventricular and / or left atrial hypertrophy	arterial hypertension, mitral regurgitation, aortic defects, dilated cardiomyopathy of various nature
Right ventricular and / or right atrial hypertrophy	pulmonary hypertension, chronic cor pulmonale, VSD, ASD

Ventricular repolarization disorders	ischemic heart disease, other diseases with systolic load on the myocardium
Pathological Q waves	Q-myocardial infarction and postinfarction cardiosclerosis. Hypertrophic cardiomyopathy.
Low voltage ECG	hypothyroidism, amyloidosis, hydropericardium
Bradycardia, AV block	hypothyroidism
Tachysystolic arrhythmias	thyrotoxicosis

Echocardiography
Normal sizes of heart cavities: end-diastolic size (EDD) up to 5.5 cm; pancreatic size up to 3.0 cm; LA diameter up to 4.0 cm; aortic diameter up to 3.7 cm



Echocardiographic analysis		
LV systolic function	**Global contractility rates**	**LV diastolic function**
1. Phenomena of local contractility disorders 2. Dyssynchrony (violation of the sequence of coverage by contraction of the ventricle); 3. Hypokinesia (local decrease in systolic shortening of the myocardium); 4. Akinesia (local absence of myocardial contractions); 5. Dyskinesia (local paradoxical systolic dilatation, protrusion of the myocardium).	1. Ejection fraction (EF,%) = EDV-ESV / EDV×100%. 2. Normal EF> 55 (60)% 3. Stroke volume (SV, ml) = EDV-ESV 4. Fraction of shortening (degree of shortening of the short LV axis) in%.	1. Transmitral blood flow 2. Ratios of the maximum velocities of the first phase (E) and the second phase (A) of filling the ventricle *. 3. The time of isovolumetric relaxation of the left ventricle (VIVR) is normally 60-70 ms, the violation is evidenced by both> 70 and <60 ms 4. The speed of movement of the myocardial wall in diastole, determined by tissue Doppler cardiography ** * In patients with sinus rhythm ** In patients with a different (non-sinus) heart rhythm

Laboratory tests for differential diagnosis of cardiomegaly

Tests	**Identified disorders and diseases**

Clinical blood test, ESR	Anemia, erythrocytosis, inflammation
Biochemical blood tests: plasma glucose total cholesterol, lipid profile plasma creatinine serum iron Proteinogram (Ig light chains)	Diabetes Atherosclerosis Renal failure Hemochromatosis Amyloidosis (AL) (+ Bence Jones protein in urine)
Thyroid hormones	Thyrotoxicosis, hypothyroidism
Rheumatoid factor, antinuclear antibodies, LE cells	Collagenoses
Antistreptococcal antibody titer	Rheumatism
Sowing blood	Infective endocarditis
Tests for detecting viral infection (antibodies in paired sera, PCR)	Viral myocarditis

Endomyocardial myocardial biopsy. Indications for EMB:
1. Heart failure <2 weeks in duration with normal or dilated LV and hemodynamic impairment
2. Heart failure lasting> 3 months with LV dilatation and new ventricular arrhythmias, grade II – III AV block, or no effect of standard treatment within 1–2 weeks (giant cell or necrotizing eosinophilic myocarditis?)
3. Heart failure associated with DCM of any duration, with an allergic reaction and / or eosinophilia;
4. HF with suspected anthracycline CMP;
5.HF with restrictive CMF of unknown origin
6. Suspected cardiac tumor (except for typical myxoma)
7. Cardiomyopathy of unknown origin in children
8. Heart failure lasting> 2 weeks with LV dilatation without new ventricular arrhythmias, grade I – III AV block and a good response to standard treatment within 1–2 weeks
9. Heart failure associated with heart changes of the type of hypertrophic CMP of unknown origin, to exclude infiltrative myocardial diseases
10. Suspicion of arrhythmogenic pancreatic dysplasia
11. Ventricular arrhythmia of unknown origin

The treatment of patients with cardiomegaly involves a combination of medication and medical / surgical procedures:
A. Drug therapy:
1. Diuretics.
2. ACE inhibitors: lower blood pressure and improve the pumping ability of the heart.
3. Angiotensin receptor blockers (ARBs).
4. Beta blockers: lower blood pressure and improve heart function.
5. Digoxin: to improve the pumping function of the heart and reduce the need for hospitalization for heart failure.
6. Anticoagulants: to reduce the risk of thrombosis.
7. Antiarrhythmic drugs.
Б. Medical devices for regulating the heartbeat
B. Surgical procedures:
1. Valvular surgery: restoration, replacement.

2. Coronary artery bypass grafting.
3. Left Ventricular Assist: (LVAD): This implantable mechanical pump when a patient is awaiting a heart transplant or, if the patient is not a candidate for a heart transplant, as a long-term treatment for heart failure.
4. Heart transplant.

CONGENITAL HEART DISEASES.

Congenital heart disease is congenital changes in the structures of the heart (valves, septa, walls, outgoing vessels) that disrupt the movement of blood inside the heart or in the circulation.

Among the causes of CHD are:

1. Genetic -7-8% (syndrome of Patau, Edwards, Down, Shereshevsky-Turner, Kleinfelter, Holt-Oram, Lawrence-Moon-Barde-Biedl, Marfan, Kartagener)
   - Chromosomal mutations 5%
   - Point mutations 2-3%
2. Environmental -1-2%
   - Physical (radiation, vibration)
   - Chemical (phenols, varnishes, alcohol, tobacco smoking, drug addiction, taking teratogenic medications)
   - Biological (age of parents, TORCH infections: rubella virus - Greg's triad: cataract, deafness, PDA, ASD, ALA; diabetes mellitus, phenylketonuria, SLE in the mother)
   - Combined (polygenic - multifactorial) - 92-93%

Pathogenesis: CHD is formed as a result of impaired embryogenesis at 2-8 weeks of gestation.

Table 1. Classification of congenital heart defects (K. Marder, 1957)

№	The nature of the hemodynamic disorder	CHD without cyanosis	CHD with cyanosis
1	CHD with enrichment of the pulmonary circulation	- Patent ductus arteriosus - Ventricular septal defect - Atrioventricular communication - Abnormal drainage of pulmonary veins	- Transposition of great vessels - Common arterial trunk - Left ventricular hypoplasia syndrome
2	CHD with depletion of the pulmonary circulation	- Isolated stenosis of the pulmonary artery	- Tetrad of Fallot - Transposition of great vessels with pulmonary artery stenosis - Right ventricular hypoplasia syndrome - Tricuspid atresia - Ebstein's anomaly
3	CHD with depletion of the systemic circulation	- Isolated aortic stenosis	-----
4	CHD without hemodynamic disturbance	- Dextrocardia - Tolochinov-Roger disease	-----

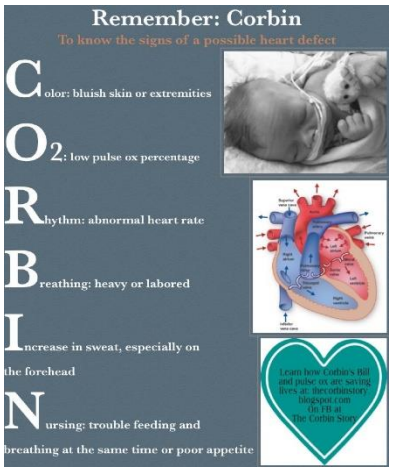
Anatomical characteristic	Features of hemodynamics

I. Isolated heart defects, a combination of a defect with abnormal drainage of pulmonary veins or stenosis, left antrioventricular foramen, observatory increased blood flow to the system of the pulmonary circulation	
1. Patent ductus arteriosus	Hypervolemia of the pulmonary circulation in combination with mitral and tricuspid insufficiency
2. Defect of the aorto-pulmonary septum	Hypervolemia of the pulmonary circulation
3. Anomalies in the development of the interatrial septum, the confluence of the pulmonary veins and atrioventricular openings in the region of the septa of the heart:	Due to the discharge of arterial blood in only one direction
а) atrial septal defects	Hypervolemia of the pulmonary circulation
б) abnormal drainage of the pulmonary veins; partial; full	Hypervolemia of the pulmonary circulation and mixing of venous blood with arterial
в) open atrioventricular canal; incomplete form; full form	Hypervolemia of the pulmonary circulation in combination with mitral regurgitation
4. Defects of the interventricular septum	Hypervolemia of the pulmonary circulation due to the discharge of arterial blood in only one direction
II. Isolated heart defects with obstruction of the outflow of blood from the ventricles	
Pulmonary artery stenosis: a) valve b) infundibular c) stem	Obstruction of the ejection of blood from the right ventricle
Aortic stenosis: a) valve b) subvalve c) supravalve d) hypoplasia of the aorta	Obstruction of the ejection of blood from the left ventricle
III. Combination of pulmonary artery stenosis with other malformations	
1.With an atrial septal defect (Fallot's triad): a) non-cyanotic form b) cyanotic form	Hypervolemia of the pulmonary circulation Discharge of venous blood into the left atrium
1.With a ventricular septal defect	Hypervolemia of the pulmonary circulation
1.With an open atrioventricular canal: a) non-cyanotic form b) cyanotic form	Hypervolemia of the pulmonary circulation Discharge of venous blood into the left side of the heart
III. Combined heart defects with anomalies of great vessels and a normal ventricular position.	
Tetralogy of Fallot: a) cyanotic form	With hypervolemia of the pulmonary circulation and increased minute volume in the large circle
b) non-cianotic form	With hypervolemia of the pulmonary circulation or balanced discharge
1. Complete transposition of great vessels	Hypervolemia of the pulmonary circulation by mixing venous blood with arterial
2. Transposition of great vessels with pulmonary artery stenosis	Hypervolemia of the pulmonary circulation and mixing of venous blood with arterial
3. Corrected transposition of the aorta and pulmonary artery	Violation of hemodynamics, depending on the presence of concomitant defects

4. Common arterial trunk	Mixing of venous blood with arterial, with hypervolemia of the pulmonary circulation
5. Discharge of the aorta and pulmonary artery from the right and left ventricles	Mixing of venous blood with arterial
IV. Combined heart defects with stenosis or atresia of the atrioventricular holes: 1. Tricuspid stenosis 2. Tricuspid atresia 3. Mitral atresia	The same
V. Combined heart defects with normal discharge of great vessels and a common ventricle	The same
VI. Various combinations of vices of the fourth, fifth and sixth groups	The same
VII. Insufficient valves: 1. Insufficiency of the pulmonary valve 2. Insufficiency of the aortic valve 3. Insufficiency of the mitral valve	Volume overload of the corresponding ventricle of the heart. In the stage of compensation, an increase in the stroke volume.
VIII. Anomaly in the development of tricuspid valve leaflets (Ebstein's anomaly)	Decreased right ventricular stroke volume with atrial overload
IX. Heart location anomaly	Violation of hemodynamics, depending on the characteristics of the heart defect
X. Disturbances in the development of the myocardium, conducting system and vessels of the pulmonary circulation.	The same
XI. Coronary artery anomaly	
1. Anomaly of the passage of the coronary arteries from the aorta	No circulatory disorders
2. Discharge of the coronary arteries from the pulmonary artery	Oxygen deficiency of the myocardium
3. Fistula between coronary arteries and heart chambers	Violation of blood circulation depending on the form of the defect
XII. Anomalies of the aortic arch and its branches	
1. Coarctation of the aorta: a) infantile type b) adult type c) coarctation of the thoracic aorta d) coarctation of the abdominal aorta	Hypertension in the vessels proximal to the coarctation and hypotension in the vessels located distal to it
1. Double aortic arch	
2. Vascular ring	
XIII. Other vices	

NB! Assuming the presence of CHD in a child is always a difficult diagnostic task in a limited time. To facilitate the task of early detection of CHD, you can use the CORBIN acronym: Color of skin, O2, Rhythm, Breathing, Increase in sweat, Nursing:

1. Skin color - cyanosis
2. O2 - low saturation
3. Change in heart rate
4. Respiratory disorders
5. Increased sweating, especially in the forehead
6. Problems with feeding, decreased appetite



CHD syndromes:

1. Heart failure syndrome: In infants, symptoms and signs of heart failure include:

- Tachycardia
- Tachypnea
- Shortness of breath when feeding;
- Diaphoresis, especially when feeding;
- Anxiety, irritability, fatigue when feeding, during outdoor play;
- Hepatomegaly
- Most babies do not have dilated neck veins, but they sometimes have periorbital edema. Symptoms in older children with heart failure are similar to those in adults.

Classification of HF in infants based on treatment (A.S.Sharykin, 2000)

NK degree	**Clinical symptoms**
1	**There is moderate tachycardia and shortness of breath (up to 120% of the norm) at rest or during exertion; liver up is to +3 cm. The size of the heart may not be increased. With the help of drug therapy, it is possible to achieve compensation of indicators**
2	**Tachycardia and shortness of breath are 125-150% of the norm at rest; liver is more than +3 cm, swelling of the feet and face, anxiety, difficulty feeding, intermittent congestive wheezing in the lungs. There is dilatation of the ventricular cavities in instrumental examination. Drug therapy reduces symptoms to grade 1**
3	**Tachycardia and shortness of breath are 160% or more, the liver is enlarged, dense, peripheral edema, ascites, constant congestive wheezing in the lungs. The child is lethargic, inhibited. The size of the heart is significantly increased. Drug therapy is ineffective, the need for mechanical ventilation. Resolving the issue of urgent operative correction of the defect.**
4	**Arterial hypotension, decompensated acidosis, peripheral spasm (weak capillary filling, cold extremities, pallor), depression of the central nervous system, oliguria. Perhaps the development of bradycardia is less than 80 beats / min.**

1. 1. The syndrome of respiratory disorders: shortness of breath, frequent colds, recurrent diseases of the lower respiratory tract.
2. Syndrome of chronic systemic hypoxia:
    - cyanosis: central - bluish color of the lips and tongue and / or nail beds (saturation <85%); perioral and acrocyanosis (cyanosis of the hands and feet) - without cyanosis of the lips or nail bed, are more often caused by peripheral vasoconstriction rather than hypoxemia, and are common, normal in newborns;
    - lag in growth and development, aggravated by shortness of breath when feeding, malnutrition, increased metabolic needs in heart failure, frequent respiratory tract infections;
    - symptoms of drumsticks and watch glasses.

3. Cardiac syndrome:
    - Pain in the heart / chest area: Infants may present with unexplained significant irritability (during or after feeding), and may be caused by an abnormal discharge of the left coronary artery from the pulmonary artery.
    - heartbeat
    - nterruptions in the work of the heart,
    - swelling and pulsation of the vessels of the neck
    - chest deformity
    - changes in blood pressure and characteristics of the peripheral pulse
    - changes in the characteristics of the apical impulse with hypertrophy / dilatation of the left ventricle
    - systolic / diastolic "cat's purr" with stenosis,
    - expansion of the boundaries of the heart, respectively, to the expanded sections, established percussion, either radiographically, or electrocardiographically,
    - the appearance of noises characteristic of each defect and a change in the auscultatory melody of the heart:
        - ✓ left-to-right shunt and obstructive lesions - usually systolic murmur
        - ✓ increased current through the pulmonary or aortic valve - increasing-decreasing mesosystolic murmur (systolic ejection)
        - ✓ regurgitation flow through the ventricular valve or through the defect of the interventricular septum - holosystolic (pansystolic) murmur, obscures S1 as its intensity increases
        - ✓ patent ductus arteriosus usually produces murmurs that are a constant 2-tone murmur, with a more pronounced sound during systole (triggered by higher pressure) than during diastole, not interrupted S2
        - ✓ abnormal 2nd tone (S2-single or sharply split), systolic click, gallop rhythm or irregular rhythm.
3. **3. Circulatory shock in newborns** may be the first manifestation of some anomalies (syndrome of hypoplasia of the left heart, critical stenosis of the aorta, interrupted aortic arch, coarctation of the aorta): extremely severe manifestations, cold extremities, weakening of the pulse, low blood pressure, as well as a reduced response to incentives.

4. **Syncope** (abnormal discharge of the coronary artery or inherited rhythm disturbance syndromes): often without warning symptoms, against the background of physical exertion (older school age athletes are often affected).

Basic principles of CHD diagnostics:

1. Antenatal: a detailed collection of anamnesis in a pregnant woman, identification of risk factors for the birth of a child with developmental abnormalities, prenatal ultrasound screening at the

prescribed time of pregnancy, if a malformation is suspected - targeted ultrasound of the fetus using an expert class apparatus, fetal ECHO-CS, karyotyping
fetus.

2. Postnatal: detailed collection of complaints, physical examination, laboratory diagnostics (clinical blood test, biochemical blood test, CBS), instrumental diagnostics (pulse oximetry: measurement of preductal and postductal saturation is measurement of saturation on the right arm and any leg, preferably simultaneously; measurement of blood pressure : on the right arm and any leg; ECG: position of the electrical axis of the heart, overload of the heart, coronary changes, arrhythmias; chest X-ray, CT, MRI, angio-, ventriculography; ECHO-KG: defect detailing, determination of treatment tactics).

General approaches to the treatment of patients with CHD:

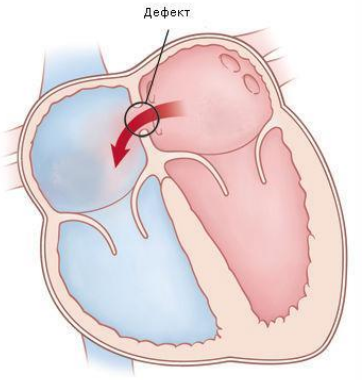
1. Conservative therapy for heart failure: diuretics, ACE inhibitors, beta-blockers, digoxin, salt restriction, oxygen therapy, prostaglandin E1):

   - if critical CHD is suspected / when the diagnosis is confirmed: intravenous infusion of prostaglandin E1 at a dose of 0.05-0.1 μg / kg / min
   - *oxygen therapy*: ↓ hypoxemia and relieves respiratory distress syndrome in heart failure, if possible, the fraction of inhaled oxygen (Fio2) should be kept at <40% to minimize the risk of damage to the lung epithelium. Mechanical ventilation may be necessary in severe condition; in other cases, supplemental oxygen should be prescribed judiciously or even delayed (vasoconstrictive effect on patent ductus arteriosus, with left-to-right shunts or obstructive left heart defects, exacerbation of excess pulmonary circulation)
   - *diuretics*: furosemide as an initial bolus of 1 mg / kg intravenously or 1-3 mg / kg orally every 8-24 hours, titrate the dose based on the volume of urine generated; potassium-sparing diuretics (spironolactone 1 mg / kg orally 1-2 times / day, titrated to 2 mg / kg / dose if necessary)
   - *ACE inhibitors* (captopril 0.1-0.3 mg / kg orally 3 times / day)
   - *beta blockers* (carvedilol, metoprolol)
   - **inotropic drugs**: dopamine or dobutamine can maintain blood pressure, but ↑ heart rate and afterload, myocardial oxygen consumption.
   - Digoxin is used less frequently than in the past, but may play a role in children with heart failure who have significant left-to-right shunts and in some postoperative patients with CHD
   - *Nutrition*: restricting salt intake, dietary modifications may be necessary depending on the specific disorder and manifestation:

   ✓ with critical defects, especially with obstructive lesions of the left heart, it may be recommended to abstain from feeding in order to minimize the risk of developing necrotizing enterocolitis.
   ✓ for shunts from left to right, it is recommended to use foods with a high calorie content;
   ✓ Sometimes tube feeding is required to maintain growth.

1. Surgical recovery or transcatheter intervention. Transcatheter procedures:
- balloon atrioseptostomy - palliative treatment of severe cyanosis in newborns with transposition of the main arteries;
- balloon dilatation of severe aortic valve stenosis or pulmonary valve stenosis;
- closure of cardiac shunts (more often atrial septal defect and patent ductus arteriosus).

Atrial septum defect (ASD)



The basis of hemodynamic disturbances is the discharge of blood from the LA to the PN and the enrichment of the MCC. Often manifests itself only as an asymptomatic heart murmur. HF symptoms usually do not appear during the first 10 years of life.
Usually, during the first months of life, hemodynamic compensation and regression of the clinical picture occur.

Typical symptoms:

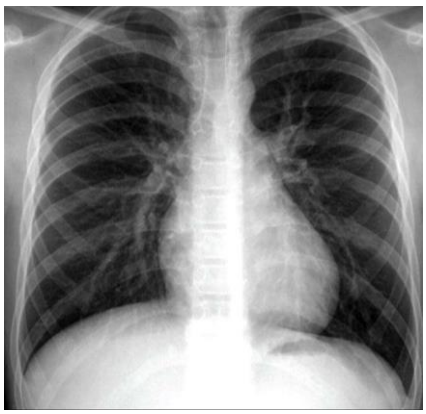
1. Persistent pallor, graceful physique, lag in physical development.
2. Rapid fatigue, dizziness, fainting, shortness of breath on exertion
3. Susceptibility to colds.
4. Cerebral or systemic thromboembolic events (stroke): passage of microemboli from the venous bed through ASD (paradoxical embolism), often associated with arrhythmias.

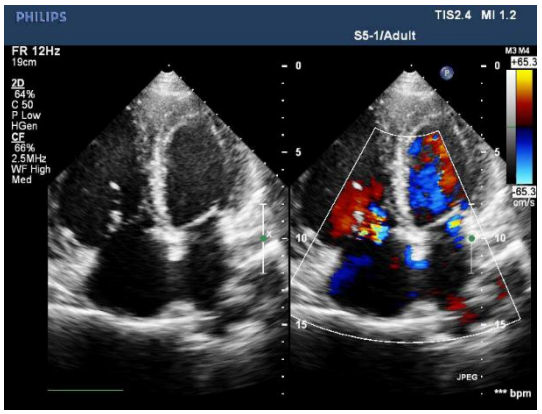
Rarely, Eisenmenger syndrome develops if ASD has not been diagnosed and has been present without treatment for decades.

Objectively:

• the borders of the heart are increased to the right and upward due to the atria
• gentle systolic murmur with the epicenter above the LA (it is better to listen in the patient's supine position)
• strengthening of the I tone, II tone over the aircraft is strengthened and often split

The diagnosis of ASD is suggested by cardiac examination, chest x-ray, and ECG, and is confirmed by two-dimensional echocardiography with color flow and Doppler examination.





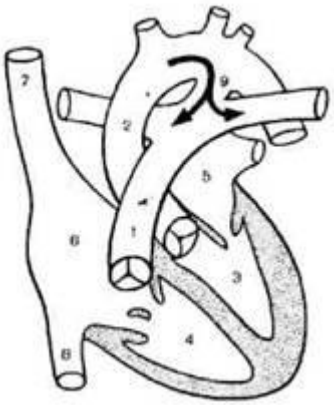
ECG: signs of overload of the right heart with hypertrophy of the right ventricle and atrium, incomplete or complete blockade of the right bundle branch

Treatment

1. Observation and periodic echocardiography (usually about every 3-5 years). Most small (<3 mm), centrally located ASDs are closed spontaneously; many defects 3–8 mm in size will spontaneously are closed by 3 years. These defects are likely to represent a stretching of the foramen ovale, and not a true secundum ASD.

2. Transcatheter closure or surgery. ASD ostium primum and venous sinus are not spontaneously closed. Moderately large PMDs (echocardiographic evidence of right ventricular volume overload) should usually be closed at 2–6 years of age. Earlier recovery may be considered for children with chronic lung disease. Transcatheter closure using a variety of commercial devices (Amplatzer® or Gore HELEX® septal occluders) is preferred when appropriate anatomical features such as adequate septal tissue margins and distance from vital structures (eg, aortic root, pulmonary veins, tricuspid ring) are presented. Otherwise (defects of the venous sinus and ostium primum), surgical intervention

is indicated. If ASD is corrected in childhood, perioperative mortality approaches zero and long-term survival approaches that in the general population.



OPEN AORTIC DUCT (PDA) ismaintaining the fetal connection (ductus arteriosus) between the aorta and the pulmonary artery after birth.

Enrichment of PC.

• Left-right shunt.

• Physiological consequences depend on the size of the ducts. A small duct rarely causes symptoms.

• A large duct causes a strong left-to-right shunt.

• Over time, a large shunt leads to an increase in the left heart, pulmonary hypertension and increased pulmonary vascular resistance, causing Eisenmenger syndrome.

Clinical picture:

1. Depends on the size of the patent ductus arteriosus and gestational age at the time of delivery.
2. Infants and young children with small PDA are usually asymptomatic;
3. Signs of HF may be present in infants with large PDA (developmental delay, poor appetite, rapid breathing, shortness of breath when feeding, tachycardia).
4. Premature babies: respiratory failure, apnea, deterioration of mechanical requirements for ventilation or other serious complications (necrotizing enterocolitis, acute renal failure).
5. HF occurs earlier in premature babies.
6. A large duct shunt in a premature baby is often a major cause of severe lung disease.

Objectively:

1.Systolic tremor of the anterior chest wall to the left of the sternum

2.Extension of the boundaries of the heart

3. Dullness of percussion sound in II MR to the left of the sternum (Gergardt's zone).

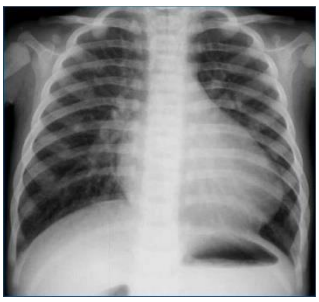
4. Most children have normal 1st and 2nd heart sounds and peripheral pulse.

5. Constant "machine-like" systolic-diastolic Gibson murmur is better heard in the upper part of the left edge of the sternum in II MR.

6. Full-term infants with significant blood discharge through the PDA have a filled peripheral pulse with a wide pulse pressure.

7.Premature babies with a significant shunt have a fast pulse and hyperdynamic precordial space. Heart murmurs occur in the pulmonary region; murmur can be continuous systolic with a short diastolic component or only systolic, depending on the pressure in the pulmonary artery. Some babies do not have an audible heart murmur.

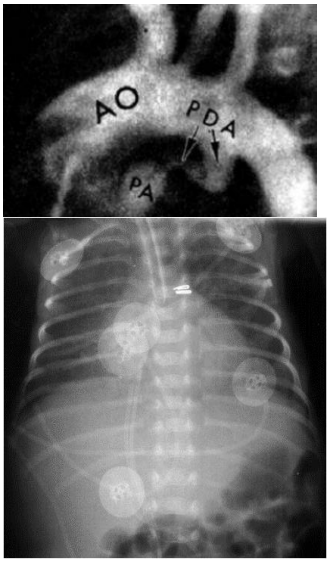
Diagnostics



Diagnosis is by clinical examination, supported by chest x-ray and ECG, and by two-dimensional color flow echocardiography and Doppler imaging.

Echocardiography provides important information about the hemodynamic significance of a PDA by evaluating a number of parameters:

1. The size of the PDA (often compared to the size of the left pulmonary artery)
2. Flow rates in the PDA
3. The presence of an increase in the left heart
4. Presence of diastolic reverse flow in the descending aorta
5. Presence of diastolic antegrade flow in the left pulmonary artery.



Treatment

1. Supportive drug therapy:
    - fluid restriction
    - diuretics (usually thiazide)
    - maintaining hematocrit ≥ 35
    - ensuring a neutral ambient temperature
    - use of positive end-expiratory pressure (PEEP) to improve gas exchange.
    - in premature infants with clinical manifestations - therapy with cyclooxygenase (COX) inhibitors (indomethacin, ibuprofen lysine). Three intravenous indomethacin doses should be given every 12-24 hours, depending on urine output; if diuresis is <0.6 ml / kg / h, the drug is stopped. An alternative is ibuprofen lysine at a dose of 10 mg / kg by mouth, followed by 2 doses of 5 mg / kg at 24-hour intervals.PDA requires surgical correctionif fluid restriction and / or COX inhibitors have not been successful.
    - COX inhibitors are usually ineffectivein term infants.

2. Transcatheter closure or surgery:

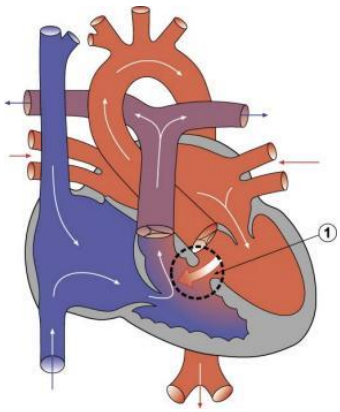
Transcatheter closure has become the preferred treatment for children with PDA> 1 yr. Various catheter occlusion devices (coils, septal duct occluder) are available.

Prevention of PDA:

1.Antenatal corticosteroid therapy - prevention of RDS

2. Respiratory therapy
    - PEEP not less than 5 mm H2O
    - Shorter inspiratory duration
    - Hyperoxia leads to a decrease in the drug
    - Adequate oxygenation and the absence of episodes of hypoxia, prevention of apnea, "mild" respiratory therapy techniques are important: NCPAP, early extubation, INSURE (intubation-surfactant-extubation)
3. Limiting the volume of injected fluid 130 - 150 ml / kg per day
4. Daily monitoring of weight and urine output
5. Nursing in conditions of high air humidity

Interventricular septum defect (VSD)isan opening in the interventricular septum leading to communication between the ventricles. Large defects result in significant left-to-right shunt and cause shortness of breath during feeding and low growth rates during infancy.



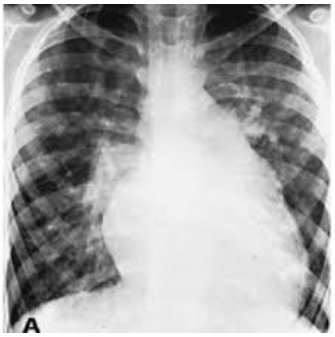
Classification

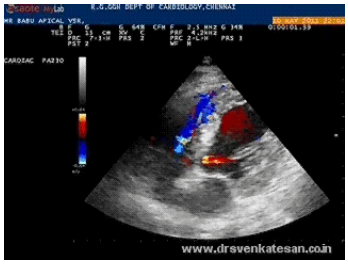
1. Perimembranous (also called conoventricular);
2. Trabecular muscular;
3. Subpulmonary outflow tract defects (supra-crestal, conoseptal, double committed subaortic)

4. Defects of the bringing tract (atrioventricular septum, similar to the atrioventricular canal)

Clinical manifestations

1. Symptoms depend on the size of the defect and the size of the left-to-right shunt. Children with mild VSD are usually asymptomatic, grow, and develop normally. Symptoms of heart failure appear at the age of 4–6 weeks, when pulmonary vascular resistance decreases in children with a large defect.
2. Frequent lower respiratory tract infections may occur.
3. Untreated patients may develop symptoms of Eisenmenger syndrome.
4. Small VSDs usually produce high, short systolic murmurs.
5. Moderate and large VSDs cause a systolic murmur, intense, over the entire region of the heart, is carried out on the right side of the chest and on the back with punctum maximum in IV MR to the left of the sternum, "girdle murmur", the appearance of 3 tones is accompanied by tremor of the chest.
6.The systolic murmur is often attenuated with large defects that equalize the pressure in the left and right ventricles





The diagnosis of VSD is suggested by clinical examination, confirmed by chest x-ray and ECG, and established based on echocardiographic findings.
Chest X-ray: cardiomegaly, increased pulmonary vascular pattern.
ECG: Right ventricular hypertrophy or combined ventricular hypertrophy and sometimes left atrial enlargement.
ECGs and chest x-rays are usually normal for small VSDs.

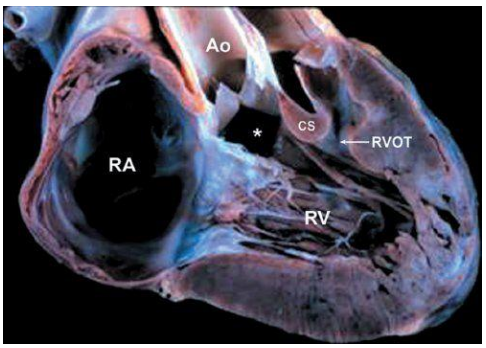
Treatment

1. Drug therapy for heart failure (diuretics, digoxin, ACE inhibitors)
2. Surgical intervention. Small VSDs, especially those of the muscle layer, often close spontaneously during the first few years of life. A small defect that remains open does not require medical or surgical treatment. Larger defects are less likely to close spontaneously.

TETRADA OF FALLOT

Multicomponent UPU:

1. Stenosis of the pancreatic outlet
2. VSD
3. Dextraposition of the aorta
4. Hypertrophy of the pancreas myocardium



The pathophysiology depends on the degree of right ventricular outflow obstruction.
Moderate obstruction can result in a clear left-to-right shunt through the ventricular septal defect; severe obstruction causes a right-to-left shunt, resulting in low systemic arterial saturation (cyanosis) that does not respond to supplemental oxygen.

Dyspnea-cyanotic attacks. Some children with uncorrected tetralogy of Fallot, most often from several months to 2 years of age, may experience sudden attacks of severe cyanosis and hypoxia, which can be fatal. A seizure can be caused by any event that slightly decreases oxygen saturation

(eg, crying, defecation), or dramatically decreases systemic vascular resistance (eg, play, kicking on awakening), or the sudden onset of tachycardia or hypovolemia.

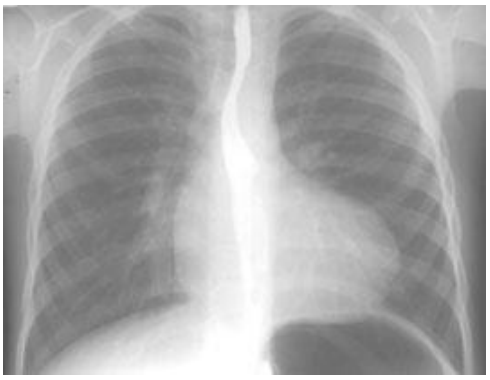
Clinical manifestations

1. Severe cyanosis and dyspnea during feeding (severe obstruction of the outflow tract of the right ventricle)
2. Poor weight gain.
3. Auscultation: coarse ejection murmur in the left middle and upper edges of the sternum. Ventricular septal defect is usually asymptomatic because it is extensive and lacks a pressure gradient. II heart sound (S2), as a rule, is single, since the pulmonary component is markedly reduced. Right ventricular tremors and systolic chest vibration may be present.

Hypercyanotic seizures:

1. can be triggered by activity
2. hyperpnea (quick and deep breaths)
3. Irritability, prolonged crying
4. increased cyanosis
5. reduction of intensity or disappearance of heart murmurs
6. Most often occur in young children
7. The peak incidence occurs at 2–4 months.
8. Severe seizures can lead to lethargy, seizures, and sometimes death.
9. While playing, some toddlers may occasionally squat in a position that increases systemic vascular resistance and aortic pressure, which decreases right-to-left ventricular shunting and therefore increases arterial oxygenation.

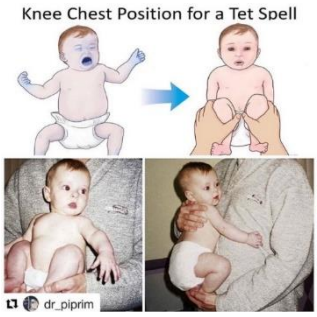
Diagnosis of tetrad of Fallot is suggested by history and clinical examination, supported by chest X-ray and ECG, and established by two-dimensional color flow echocardiography and Doppler examination.

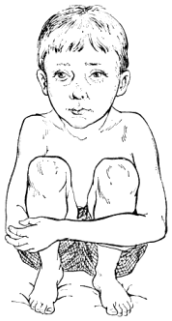


A chest X-ray reveals a wooden shoe heart with a concave main pulmonary artery segment and reduced pulmonary vascular pattern. The right aortic arch is present in 25%.
ECG shows right ventricular hypertrophy, and may also show right atrial hypertrophy.

Treatment

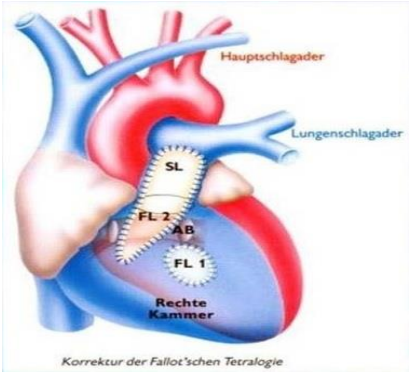
1. Injection of prostaglandin E1 is for newborns with severe symptoms.
2. With hypercyanotic attacks:
   • immediate intervention
   • take a knee-chest position. For infants, immediately press your knees to your chest (older children usually squat spontaneously and do not develop dyspnea
   • provide peace
   • oxygen supply,
   • intravenous fluids to increase blood volume
   • Medication includes morphine, phenylephrine, and beta-blockers (propranolol or esmolol).
   • systemic blood pressure can be increased with ketamine 0.5-3 mg / kg intravenously or 2-3 mg / kg intramuscularly (ketamine also has a beneficial sedative effect).





• in the presence of metabolic acidosis - sodium bicarbonate, intravenous injection in the amount of 1 mEq / kg
• Tracheal intubation, extracorporeal membrane oxygenation (ECMO), or urgent surgery may be needed.
• propranolol orally at a dose of 0.25 to 1 mg / kg every 6 hours (before surgery) may prevent relapses
• most experts believe that even one significant attack indicates the need for surgical correction

Radical treatment: elimination of the defect of the interventricular septum by applying a patch, expanding the outlet of the right ventricle by resecting the muscles, pulmonary valvuloplasty and, if necessary, enlarging the patch of the pulmonary trunk. If there is significant hypoplasia of the pulmonary valve annulus, a transannular patch is placed. Surgery is usually performed selectively at 2–6 months of age, but it can be performed at any time if symptoms are present or if there is severe right ventricular outflow tract obstruction.

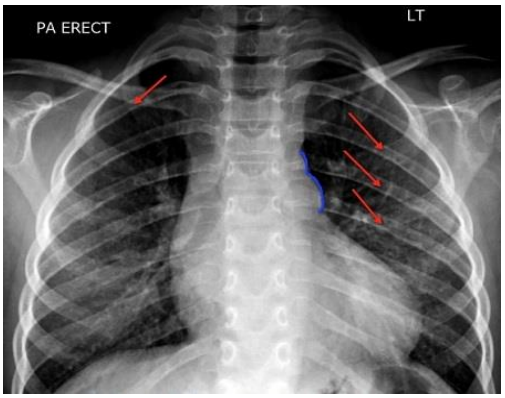


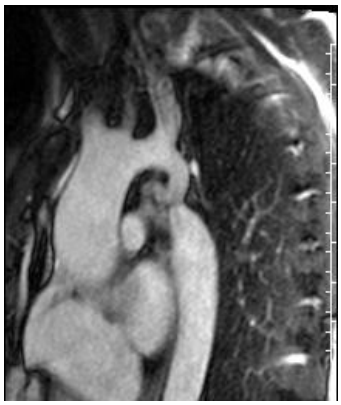
Korrektur der Fallot'schen Tetralogie

AORTIC COARCTION islocalized narrowing of the aortic lumen, leading to hypertension of the upper extremities, left ventricular hypertrophy and insufficient perfusion of the abdominal and lower extremities.
Symptoms:
1.depend on the severity of the anomaly
2.headache
3.chest pain
4.lameness
5.cold limbs
6.fatigue
7. intermittent claudication.
If coarctation is significant, circulatory shock with renal failure (oliguria or anuria) and metabolic acidosis may develop in the first 7–10 days of life and mimic the effects of other systemic diseases such as sepsis.
Less severe coarctation may be asymptomatic during infancy. Mild symptoms may appear as the child grows older. Hypertension in the upper extremities is often present, but heart failure rarely develops after the neonatal period. Rarely, an intracerebral aneurysm ruptures as a result of subarachnoid or intracerebral hemorrhage.

Typical symptoms on physical examination:
1. solid pulse
2. hypertension in the upper limbs
3. decrease or delay in the pulse of the femoral artery
4. BP gradient with low or non-measurable BP in the lower extremities
5. fulminant heart failure and shock may develop
6. Soft noise can be heard outside the coarctation site
7. expansion of intercostal collateral arteries can lead to continuous murmurs in the intercostal spaces.

Diagnosis is by echocardiography, CT, or MR angiography.





X-ray:

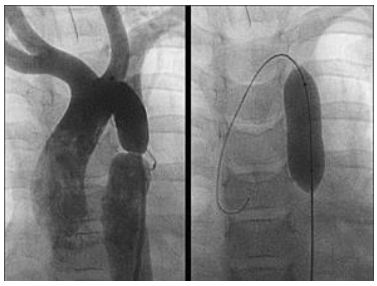
1. narrowing in the form of a number 3 in the upper left shadow of the mediastinum.
2. the heart is of normal size, except in cases of heart failure.
3. dilated intercostal collateral arteries can erode the 3rd to 8th ribs, resulting in the formation of notches on the ribs.

ECG:

1.hypertrophy of the left ventricle
2. can be normal.
3. right ventricular hypertrophyis in newborns and young children.

Treatment

1. Immediate Prostaglandin E1 is for Symptomatic Newborns
2. Beta-blockers (ACE inhibitors are contraindicated)is with arterial hypertension. After correcting coarctation, hypertension may persist or develop years after recovery, and may be treated with beta blockers, ACE inhibitors, angiotensin II receptor blockers, or calcium channel blockers
3. Surgical correction or balloon angioplasty (sometimes with stent placement). The choice of a surgical method depends on the anatomy and preference of the center.
4. Control before correction is for children with very mild coarctation and no signs of hypoperfusion of the lower body



ACQUIRED HEART DISEASES are acquired morphological changes in the valve apparatus, leading to a violation of its function and hemodynamics.

Most often they arise as a result of previous rheumatism, infective endocarditis, systemic diseases of the connective tissue, etc.

AHD	Hemodynamics	Clinical picture	Instrumental data
Insufficiency of the mitral valve	Retrograde blood flow during LV systole toward the atrium Increased blood supply to the left atrium and left ventricle. Due to the dilatation and hypertrophy of these parts of the heart, the required amount of blood enters the aorta, and part of it returns back to the left atrium.	Usually children do not complain Heart hump The apical impulse is enhanced, slightly shifted to the left. The border of relative dullness is extended to the left. Attenuation of 1 tone at the apex Strengthening 2 tones on the pulmonary artery Systolic murmur at the apex and at the 5th point, is carried out to the base of the heart and to the left axillary region (less often on the back). The noise increases when the child is on the left side	ECG - signs of left atrial and LV hypertrophy. EchoCG: the valve leaflets are not closed during systole, an increase in the size of the LA and LV Doppler echocardiography turbulent blood flow in the LA.
Mitral stenosis *primary* (latent or subacute current rheumatism) *secondary* (against the background of mitral valve insufficiency)	Increased pressure in the LA LA hypertrophy Acceleration of blood flow in the LV due to a narrowed mitral foramen. With severe stenosis, pulmonary hypertension develops	Dyspnea Fast fatiguability Pallor in combination with a peculiar color of the face - cyanotic blush of the cheeks and lips - "facies mitralis" With pronounced dilatation of the LA and pulmonary artery, compression of the recurrent nerve and hoarseness of the voice (Ortner's syndrome). Orthopedic and nocturnal attacks of shortness of breath occur with pulmonary hypertension and right ventricular failure The apical impulse is weakened, "cat's purring" is palpable. Loud and short ("clapping") tone and diastolic murmur at the apex of the heart. Accent 2 tone on the pulmonary artery. Atrial fibrillation	ECG - the P wave is enlarged, then it becomes bimodal. The radiograph shows an increase in LA. EchoCG: unidirectional movement of the anterior and posterior valve cusps forward, decrease in the rate of diastolic valve closure, expansion of the right ventricular cavity. Narrowing of the mitral foramen
Combined mitral valve disease	dominance of clinical and instrumental signs of one type of defect, usually mitral insufficiency in combination with less pronounced manifestations of mitral stenosis.		
Aortic valve insufficiency (rheumatism, infective	Part of the blood during diastole returns from the aorta to the left ventricle, as a result of which the muscle fibers of the left	Dyspnea Palpitations Often pain in the region of the heart. Pallor	ECG - signs of LV diastolic overload The aortic configuration with an accentuated waist is on the roentgenogram.

endocarditis, myxomatous degeneration of the valves, hereditary connective tissue diseases)	ventricle stretch and hypertrophy.	Increased pulsation of the carotid arteries (carotid dance) The pulse is fast and high, less often - the appearance of a capillary pulse. BP is a decrease in the minimum with a normal or moderately increased maximum. The apical impulse is enhanced and displaced outward and downward. The borders of the heart are expanded to the left. Weakening of 1 tone is at the apex of the heart Diastolic murmur (quiet, gentle, pouring) ison the basis, in 3-4 intercostal space to the left of the sternum, it is better heard in the patient's standing position, when the body is tilted forward. Flint's diastolic murmur is heard at the apex of the heart (the reverse flow of blood from the aorta raises the mitral valve leaflets lowered into the left ventricle).	Echocardiography - the leaflets of the aortic valve do not close. Vegetation on the valves can be detected, indirect signs of the defect are LV dilation and hyperkinesis of the valves. Doppler examination can detect aortic insufficiency.
Aortic stenosis: rheumatism (almost always accompanied by mitral valve lesions), isolated calcification of the aortic valves, infective endocarditis with massive vegetations	Narrowing of the LV outflow tract and increased load on the LV	15-20% of patients is with aortic stenosis die suddenly. There are no complaints in the compensation phase. Systolic tremor at the base of the heart. In the 2nd intercostal space on the right and at Botkin's point, a systolic murmur (scraping, blowing) is performed on the carotid arteries. 2 tone on the aorta is weakened. Pulse is small, slow, systolic pressure is reduced.	ECG - signs of LV hypertrophy. EchoCG - with severe stenosis, the valves are located as a single conglomerate of the echo signal, an increase in the LV is detected. Doppler echocardiography - the pressure gradient between the aorta and the left ventricle.
Mitral valve prolapse (MVP): primary (idiopathic) secondary (congenital or acquired diseases)	Flexion of the mitral leaflets into the LA cavity during LV systole	The "auscultatory" and "mute" forms of MVP are distinguished. Children with MVP do not show any special complaints Usually discovered by accident. Sometimes there is pain in the heart, dizziness, fainting, often there is a decrease in blood pressure. Mesosystolic click at the apex of the heart, less often in the second or fourth intercostal space to the left of the sternum	ECG: normal variant / reduced, biphasic T waves. EchoCG: prolapse of one or both leaflets. Changes are not detected with a "mute" PMK on the ECG and radiographs, it is recognized only by echocardiography. The size of the heart is normal on chest x-ray. in primary MVP without regurgitation

		May be associated with late systolic murmur. The intensity of the noise varies in time and duration in the same patient Noise and clicking are heard better when the patient is standing.	

6.Materials for the methodological support of the lesson

6.1. Control materials for the preparatory stage of the lesson (tests, tasks)

Tests

1. A 12-year-old girl was admitted to the cardiology department with manifestations of carditis. Two weeks ago she suffered a lacunar sore throat. What is the most likely etiological factor for carditis in this case?
* A. streptococcus
B. Staphylococcus
C. Klebsiella
D. Pneumococcus
E. Proteus

2. A 5-year-old child was discharged after treatment for rheumatism with grade I activity. Anti-relapse treatment in the coming years provides for the introduction of Bicillin-5. What is the dose of Bicillin-5?
* A. 750,000 from 2 times a month
B. 1,500,000 from 1 time per month
C. 750,000 from 1 time per month
D. 600000 from 2 times a month
E. 600000 from 1 time per month

3. A nine-year-old boy suffered from scarlet fever 2 weeks ago. There is general weakness, pallor of the skin, an increase in body temperature up to 38 ° C, pain in the heart, shortness of breath for three days. At auscultation of the heart, weakened tones, systolic murmur above the apex, bifurcation of the 1st tone were noted. ECG: lengthening of the QT interval, PQ (0.22 s), decrease in the amplitude of the T wave, single extrasystoles. What disease will the local pediatrician suspect?
* A. rheumatic myocarditis
B. Cardiomyopathy
C. Neurocirculatory dysfunction
D. Pneumonia
E. congenital heart disease

4. A 12-year-old boy is under dispensary supervision by a cardioreheumatologist with a diagnosis of rheumatism, active phase, and rheumatic heart disease with mitral valve lesions. How long a secondary Bicillin drug prophylaxis of rheumatism should be carried out?
* A. for life
B. 3 years
C. 1 year
D. 18 yearsold
E. 25 years old

5. The child is 10 years old. She is inpatient treatment in the cardiology department for 10 days due to the active phase of rheumatism. Bed mode. What load should be assigned for the Shalkovim functional test?
* A. Transition from a horizontal position to a sitting position 5 times
B. 10 deep squats in 20 seconds.
C. 20 deepsquatsin 30 seconds
D. Climb 20 steps
E. Climb 30 Steps

6. A leading place is given to one of the following factors in the etiology of rheumatism:
A. viruses
B. mycoplasma infection
C. staphylococcus
* D. Streptococcus group A
E. bacterial-viral associations

7. What indicators of ESR characterizes the II degree of activity of the rheumatic process:
A. up to 20 mm / h
B. within the age norm
* C. 20-30 mm / h
D. more than 30 mm / h
E. more than 50 mm / h

8. The main diagnostic criterion for rheumatism:
A. polyserosite
B. myofibrosis
C. subfebriletemperature
* D. chorea
E. Arthralgia

9. The condition of a 7-year-old child with rheumatic heart disease has significantly worsened. Diagnosed with effusion pericarditis. How has this child's heart rate changed?
A. thread-like
B. arrhythmic
C.slow-motion
D. voltages
* E. Paradoxical

Tasks

<table>
<tr><td colspan="2">Task 1. The patient is 12 years old. Complains of an increase in body temperature within 37.5-38.5 0C, pain in the knee joints and their swelling. History of frequent sore throat disease. Objectively revealed swelling and sharp soreness, hyperemia of the skin of the knee and ankle joints. An annular, pale pink rash was found on the skin of the abdomen. The left border of the heart is 1.5 cm. Outward from the left midclavicular line, the tones are muffled, a rough systolic murmur above the apex, tachycardia, blood pressure is 90/50 mm Hg, heart rate is 110 in 1 min.</td></tr>
<tr><td>Exercise:
• Formulate a nosological diagnosis
• List the main criteria for this disease
• What tissue of the heart is affected in this patient?
• Formulate a clinical diagnosis
• Prescribe etiotropic therapy.</td><td>Sample answer:
1.Rheumatism.
2.Carditis, polyarthritis, chorea, erythema annulus, rheumatic nodules.
3. The defeat of mainly 2 membranes of the heart: myocardium (displacement of the borders of the heart), endocardium (rough systolic murmur).
4. Rheumatism 1, active phase, activity II degree, endomyocarditis, polyarthritis, erythema annular, acute course, NK I.
5.In / m injected penicillin at 600 thousand units -2000 thousand units per day (4 doses) for 2 weeks (100 thousand units per 1 kg of body weight), then in / m bicillin-5. Macrolides are shown in case of intolerance to drugs of the penicillin series.</td></tr>
</table>

<table>
<tr><td colspan="2">Task 2. Patient is 13 years old. He had complaints of irritability, rapid fatigability, involuntary contraction of facial muscles, changes in handwriting. A month before, he had had a sore throat. Objectively muscle hypotonia, hyperreflexia, expansion of the boundaries of the heart, muffled heart sounds, mild systolic murmur at the apex, tachycardiawere revealed.</td></tr>
<tr><td>Exercise:
• Indicate the cause of the neurological disorders
• What symptoms can be found in the patient?
• Formulate a clinical diagnosis
• What options for the course of this disease do you know?
• Give prophylaxis with bicillin.</td><td>Sample answer.
1. The child has developed subcortical encephalitis - "small" chorea.
2. Symptoms of Czerny, "tongue-eyes", Gordon. Increased tendon reflexes.
3.Rheumatism II, active phase, activity II degree, myocarditis, chorea minor, acute course, NK I.
4. Acute, subacute, protracted, constantly recurrent, latent.
5. Bitsilin prophylaxis: children who have had rheumatism without carditis - up to 18 years old, with carditis - up to 25 years old, with the prevailing wadi - all their lives.</td></tr>
<tr><td colspan="2">Task 3. After treatment in a hospital and a sanatorium, a ten-year-old girl, who suffered a primary rheumatic attack, proceeding with polyarthritis, erythema on the skin against the background of chorea was discharged. An increase in the content of acute phase proteins and a high titer of anti-streptococcal antibodies were observed in the blood. Currently, there are no signs of activity of the rheumatic process.</td></tr>
<tr><td>Exercise:
• Formulate a clinical diagnosis
• Prescribe chorea therapy
• What effect do quinoline drugs have?
• List the main links of therapy for patients with rheumatism
• What is the optimal bicilin therapy regimen for this patient?</td><td>Sample answer.
1. Rheumatism II, inactive phase, NC 0.
2.Nonsteroidal and anti-inflammatory drugs (diclofenac 3.0-3.5 mg / kg / day, indomethacin 2.5-3 mg / kg / day)
- bromine preparations (1% sodium bromide solution)
-seduxen
- vit. B1, B6-course 15-20 injections
-physiotherapy
- electrosleep, bromine
- electrophoresis on the collar zone.
-balneotherapy (pine baths).
3. Delagil, Plaquenil - drugs of the quinoline series have anti-inflammatory and antiproliferative effects.</td></tr>
<tr><td colspan="2">Task 4. 13 years old patient is under dispensary observation for: rheumatism II, active phase, activity II century, endocarditis, polyarthritis, subacute course, NK 0.</td></tr>
<tr><td>Exercise:
• How to organize the daily routine for patients with rheumatism in the hospital?
• Alternative antibiotic therapy in case of intolerance of the penicillin series
• Will make a differential diagnosis between rheumatic arthritis and reactive arthritis
• List the features of the course of rheumatism in children
• Indicate for how long it is necessary to prescribe bicillin prophylaxis for this patient?</td><td>Sample answer.
1. Strict bed rest, bed rest, semi-bed rest, training regimen
2. Prescribing antibiotics from the macrolide group, for example, erythromycin.
3. In rheumatism, mainly medium and large joints are affected, articular pain syndrome is unstable, the nature of pain is migratory, joint deformity is absent, muscle atrophy is absent, there are changes in the heart and a connection with streptococcal infection, symmetry of lesions.
Reactive arthritis is associated with intestinal infection, asymmetric, arising against the background of diarrhea, heart damage is not typical.
4. There is reducing the severity of carditis. There is mostly moderate and minimal activity of the inflammatory process. There is minimum diagnostic value of erythema erythema and rheumatic nodules. Significant improvement in the prognosis of the disease. Decrease in the frequency of formation of heart defects.</td></tr>
</table>

	5. Bitsilinotherapy must be carried out year-round until patients reach the age of 25.
Task 5. A 10-year-old child was admitted to the clinic with complaints of shortness of breath, acrocyanosis. Objectively: lags behind in physical development, low nutrition. BH - 30 / min. The apical impulse of the heart is shifted. The size of the heart is within normal limits. A loud clapping 2 tone is heard. ECG extended two-humped PII, III, V5, V6. There are signs of venous and arterial pulmonary hypertension, an increase in the left atrium on the Rö-gram.	
Exercise: • Formulate a nosological diagnosis • What kind of heart defect did this patient develop? • What type of heart failure develops with this defect? • Specify the type and extent of surgical intervention for this pathology • In which variant of the course of rheumatism, the heart defect is formed asymptomatically?	Sample answer: 1 Rheumatism 2. Mitral stenosis. 3. Right ventricular type. 4.Comisurotomy of the cusps of the mitral valve. 5.Latent current.
Problem 6. A 13-year-old boy complains of frequent fainting and dizziness, palpitations, shortness of breath on exertion, cardialgia. Objectively: pulse rate - 68 / min., Decreased filling. The apical impulse is displaced. The borders of the heart are expanded by 0.5 cm to the left. On palpation in the second intercostal space on the right - systolic tremor. A coarse systolic murmur is heard with a weakening of the II tone in the second intercostal space on the right, produced on the carotid artery. ECG shows left ventricular myocardial hypertrophy. PCG shows high-frequency rhomboid systolic murmur on the aorta, associated with I tone, II tone is reduced.	
Exercise: • Form a nosological diagnosis • What kind of heart defect did this patient develop? • What type of heart failure develops with this defect? • What is the degree of circulatory failure in this patient? • What groups of drugs are used for the pathogenetic therapy of rheumatism?	Sample answer: 1.Rheumatism. 2. Stenosis of the aortic orifice. 3. Left ventricular type. 4.1 degree 5.a) NSAIDs (aspirin, indomethacin, diclofinac, ibuprofen). b.) Steroidal anti-inflammatory drugs (prednisolone, triamcinolone, dexamethasone). c.) Quinolone drugs (delagil, gelaquinyl).
Task 7. A 14-year-old patient complains of pain in the region of the heart, shortness of breath, fever up to 38.5 ° C. 15 days later she suffered a sore throat. Objectively: the patient's condition is serious. The skin is pale, but clean. Breathing - 28 / min. On the left, above the lungs, behind the lower third of the scapula, weakened breathing, immediately there is a shortening of the percussion sound. Between the hearts are widened in diameter, the tones are weakened. The pulse is paradoxical. A pericardial rubbing noise is heard over the entire region of the heart. On the ECGthere is a decrease in the voltage of the QRS complexes, a rise above the isoline of the ST segment, deformation of the T wave. The liver is enlarged by 3 cm, painful. ESR is 42 mm / h, ASL-O is1260 ED, CRB is ++++.	
Exercise:	Sample answer: 1.Rheumatism.

• Establish a nosological diagnosis • What is the evidence of the presence of a paradoxical pulse in this patient? • Determine the degree of circulatory failure in this patient • Formulate a clinical diagnosis • Prescribe treatment	2. The presence of pancarditis. 3.2 degree. 4. Rheumatism 1, active phase, activity 3 tbsp., Pancarditis, acute course, NK II. 5. a.) Strict bed rest. b.) Diet with restriction of table salt and liquids. Potassium-rich foods are prescribed.
Task 8. A 13-year-old patient, upon entering the clinic, complained of severe weakness, pain in the limbs, knee and ankle joints, palpitations, ring-shaped rash on the trunk. She fell ill 3 weeks later, when after hypothermia with "weakness, runny nose, sore throat, subfebrile condition. She did not take any medications. Objectively: the radial-wrist and knee joints were swollen, painful. The pulse is 96 / min. Heart: the left border is shifted by 0.5 cm outward from the midclavicular line, tones are muffled, systolic murmur is over the entire surface Leukocytes are 15 X 10^3 / l, ESR is 55 mm / hour, CRP is +++.	
Exercise: • Establish a nosological diagnosis • Indicate the norms of cardiac dullness at this age • Make a differential diagnosis with infectious-allergic polyarthritis • Form a clinical diagnosis • Prescribe drugs to normalize the metabolism of the heart muscle and other tissues.	Sample answer: 1.Rheumatism. 2. Apical impulse is along the midclavicular line 0.5-1 cm inside from it in the fifth intercostal space. Absolute dullness of the heart. The upper limit is 4 ribs. The left edge is between the left midclavicular and parasternal lines. The right edge is closer to the parasternal line. Relative dullness of the heart. Top is 3 rib. Left is along the midclavicular line. Right is in the middle between the right parasternal and the right edge of the sternum. 3. No carditis, chorea and other major signs of rheumatism. 4. Rheumatism 1, active phase, activity of the 3rd degree, Carditis, polyarthritis, annular erythema, acute course, NK II. 5. For the normalization of metabolic disorders in the heart muscle and other tissues are used: panangin, asparkam, riboxin, vitamins gr. V.

6.2.<u>The information necessary for the formation of knowledge and skills can be found in the textbooks:</u>

basic:

1. Volosovets O.P, Snisar V.I. Recommendations for cardiopulmonary resuscitation in children. Methodical manual. Dnepropetrovsk: ART-PRESS, 2015. 48 p.
2. D 362 State form of medicines. Issue ten. Kyiv, 2018 https://moz.gov.ua/uploads/1/5052-dn_20180510_868_dod_2.pdf
3. Differential diagnosis of the most common diseases of childhood. Textbook / ed. V.M. Dudnyk, 1st edition. Vinnytsia: Nilan Ltd., 2017. 560 p.
4. Karen J. Markdante, Robert M. Kligman. Fundamentals of Pediatrics according to Nelson: translation of the 8th English. edition: in 2 volumes. Volume 1. Kyiv: VSV "Medicine", 2019. XIV, 378 p.
5. Karen J. Markdante, Robert M. Kligman. Fundamentals of pediatrics according to Nelson: translation of the 8th English. edition: in 2 volumes. Volume 2. Kyiv: VSV "Medicine", 2019. XIV, 426 p.6.

6. Kryuchko T.A, Abaturov A.E, Kushnereva T.V Pediatrics: textbook (University IV level. A); under ed. AND. Крючко, A.E. Abaturov. Kiev: VSI "Medicine", 2020. 224 p.
7. Emergencies in pediatric practice: Textbook. way. for students. med. ZVO, interns. - 2nd type. Recommended by the Ministry of Education and Science, Recommended by the Academic Council of NMU. O.O. Bogomolets / Marushko Y.V, Chef G.G etc. Kyiv: VSV "Medicine", 2020. 440 p.
8. Pediatrics: a national textbook: in 2 volumes / Ed. prof. Berezhnogo V.V Kyiv, 2013. Vol.1. Kyiv, 2013. 1040 p.
9. Pediatrics: a national textbook: in 2 volumes / Ed. prof. Berezhnogo V.V Kyiv, 2013. Vol.2. Kyiv, 2013. 1024 p.
10. Pediatrics: a textbook for students. higher education institutions IV level accred. / for ed. prof. O.V Severe. View. 5th, ed. and add. Vinnytsia: Nova Kniga, 2018. 1152 p .: ill.
11. Maidannyk V.G, Yemchynska E.A. Clinical guidelines for the diagnosis and treatment of community-acquired pneumonia in children from the standpoint of evidence-based medicine. - K., 2014.- 43 p. http://pediatrics.kiev.ua/library/metod/5.pdf

additional:
1. Nyankovsky S.L, Babik I.V. Features of asthenic syndrome and autonomic disorders in children with community-acquired pneumonia. Child Health.-№3 (63) 2015-.S 16-21.
2. Pediatric immunology: textbook. for doctors-interns, doctors-cadets higher. med. est. (Ph.D.) postgraduate. education, as well as for students., higher education teachers. med. textbook est. IV level of accreditation / ed. prof. L.I. Chernyshova, A.P. Volokha. - K.:Medicine, 2013. - 719 c.
1. Pediatric cardiology. - Yu.M. Belozerov. - M. - MEDpress-inform. - 2004.- S. 155-160.
2. Ultrasound semiotics and diagnostics in pediatric cardiology. - Yu.M. Belozerov, V.V. Bolbikov. - M. - MEDpress. - 2001.- S. 45-56.
3. Congenital heart defects. - N.A. Belokon, V.P. Podzolkov. - M. - Medicine. - 1991. - S. 85-90.
4. Selected issues of pediatric cardio-rheumatology. - Kiev, Kharkov. - 2006.- S. 102-107.
5. Pediatrics (pediatric cardiology and nephrology). - Odessa. - 2014. - pp. 78-103, 131-146.
6. Montero J.V, Nieto E.M, Vallejo I.R, et al: Intranasal midazolam for the emergency management of hypercyanotic spells in tetralogy of Fallot. Pediatr Emerg Care 31(4): 269–271, 2015.
7. Tsze D.S, Vitberg Y.M, Berezow J, et al: Treatment of tetralogy of Fallot hypoxic spell with intranasal fentanyl. Pediatrics 134(1): e266–e269, 2014.
8. Sandoval J.P, Chaturvedi R.R, Benson L, et al: Right ventricular outflow tract stenting in tetralogy of Fallot infants with risk factors for early primary repair. Circ Cardiovasc Interv 9(12): pii: e003979, 2016.
9.Materna-Kiryluk A, Wiśniewska K, Badura-Stronka M, et al: Parental age as a risk factor for isolated congenital malformations in a Polish population. Paediatr Perinat Epidemiol 23(1):29-40, 2009. doi: 10.1111/j.1365-3016. 2008.00979.x.
10. Russell M.W, Chung W.K, Kaltman J.R, Miller T.A: Advances in the understanding of the genetic determinants of congenital heart disease and their impact on clinical outcomes. J Am Heart Assoc 7(6):e006906, 2018. doi:10.1161/JAHA.117.006906.
11. Oster M.E, Kelleman M, McCracken C, et al: Association of digoxin with interstage mortality: Results from the Pediatric Heart Network Single Ventricle Reconstruction Trial Public Use Dataset. J Am Heart Assoc 5(1): e002566., 2016.
12. Bolin E.H, Lang S.M, Tang X, et al: Propranolol versus digoxin in the neonate for supraventricular tachycardia (from the Pediatric Health Information System). AmJCardiol 119(10): 1605–1610, 2017.

7. Materials for self-control of the quality of training.

A. *Questions for self-control*
1. Definition of the concept of cardiomegaly
2. Cardio-thoracic index
3. Difdiagnosis of diseases accompanied by cardiomegaly.

3. The main clinical manifestations of congenital heart defects
4. The main diagnostic criteria for non-rheumatic carditis in children.
5. Differential diagnosis of rheumatic and non-rheumatic heart disease.
6. Manifestations of cardiac forms of autonomic dysfunction in children.
7. Basic approaches to the treatment of rheumatism in children.
8. Peculiarities of treatment of non-rheumatic carditis in children.
9. Heart failure in children. Clinical manifestations.
10. Principles of providing emergency care in heart failure.

B. Tests for self-control with reference standards:
1. What is the maximum daily dose of aspirin:
A. 1.0 g
B. 1.5 g
C. 2.0 g
D. 2.5g
* E. 3.0 g

2. At what disease does the “quail rhythm” appear:
A. mitral insufficiency
* B. mitral stenosis
C. Aortic insufficiency
D. mitral valve prolapse
E. aortic stenosis

3. At what disease the diastolic descending murmur is heard:
A. mitral insufficiency
B. mitral stenosis
* C. aortic insufficiency
D. mitral valve prolapse
E. aortic stenosis

4. At what disease is the pansystolic murmur heard:
* A. mitral insufficiency
B. mitral stenosis
C. Aortic insufficiency
D. mitral valve prolapse
E. aortic stenosis

5. A decrease in the amplitude of the I tone on the PCG is noted when:
* A. mitral insufficiency
B. mitral stenosis
C. Aortic insufficiency
D. mitral valve prolapse
E. aortic stenosis

6. Mitral insufficiency is characterized by:
A. displacement of the left border of the heart
B. Levogram according to ECG data
C systolic murmur arising simultaneously with I tone
D. maximum volume of systolic murmur at the apex of the heart

* E. all of the above

7. A 6-year-old boy complains of constant pain in the region of the heart. Percussion - between the hearts without changes, the heart sounds are sonorous, along the left edge of the sternum, a limited, unstable noise is heard, reminiscent of the crunch of snow. On the ECGthere is a biphasic T wave, the S-T interval is concordantly shifted.
A. Non-rheumatic carditis
B. Rheumatism
* C. pericarditis
D. Dry pleurisy.
E. Myocardial infarction.

8. A 9-year-old girl, after having had a sore throat 2 weeks ago, suddenly had a rise in body temperature to 38˚C, pains in the knee and elbow joints, which were volatile, general weakness, lethargy, and deterioration in appetite were noted. On auscultation, there was some muffling of tones. What disease can be suspected in a child?
A. Juvenile rheumatoid arthritis
* B. rheumatic myocarditis
C. Tonsilogenic cardiomyopathy
D. Rheumatoid arthritis
E. infectious-allergic myocarditis

9. A 12-year-old girl complains of weakness, fatigue, pain in the joints and heart area. History of frequent tonsillitis. On examination, the boundaries of the heart were not expanded, the tones were sonorous, and there was a functional systolic murmur. Single right ventricular extrasystoles, a decrease in T, positive changes in the potassium-obzidan test are on the ECG. What is the most likely diagnosis?
A. Non-rheumatic carditis
* B. tonsillogenic cardiomyopathy
C rheumatic heart disease
D. NDC
E. Infective endocarditis

10. A 12-year-old child was hospitalized for rheumatism 1, active phase, activity of the 3rd degree, Carditis, chorea, arthritis, acute course, H0. After 2 months there are complaints of pain in the heart and joints, ESR is 30 mm / h. What course of rheumatism can you think of?
A. Ostry
B. Podostry
C. Latent
* D. Continuous-relapsing
E. lingering-flabby

11. Cabbage, oat and buckwheat porridge, cheese, butter, baked potatoes, raisins, prunes, tavern were introduced into the diet of a 9-year-old child. For what disease is it advisable?
A. Peptic ulcer
B. Acute pneumonia
C. Dysmetobolic nephropathy with oxaluria
* D. rheumatic heart disease
E. Bronchial asthma

B. *Tasks for self-control with answers:*

<table>
<tr><td colspan="2">Task 1. A 12-year-old patient has a history of frequent tonsillitis and scarlet fever. After the next lacunar sore throat, pain in the joints increased, which were also recorded earlier, and shortness of breath appeared on the way up the stairs. Objectively: pallor of the skin, enlarged and painful lymph nodes, tonsils are hypertrophied, loosened and scarred. The apical impulse is weakened, the left border of the heart is displaced by more than 1 cm to the left of the left midclavicular line. The upper limit is in the second intercostal space. At the apex of the heart and at Botkin's point, a clear systolic murmur with a blowing shade is heard, which decreases significantly in an upright position. Pulse - 96 / min. Leukocytes are 6.6 X 109 / l, e-1, n-2, s-43, l-52, m 2, ESR is 32 mm.h., ASL-O is 1000 U, CRPis ++.</td></tr>
<tr><td>Exercise:
• Establish a nosological diagnosis
• What are the relative criteria of this disease manifested in the patient?
• Form a clinical diagnosis
• Prescribe non-drug therapy
• Indicate the main directions of primary prevention of this disease.</td><td>Sample answer:
1.Rheumatism.
2.SOE is 32 mm / h, ASLO is 1000 U, SRB is ++.
3.Rheumatism 1, active phase, activity 2nd st., Carditis, subacute course, NK I
4. Mode, diet, laser magnetotherapy, peloid therapy.
5. After a streptococcal infection, the child should be under the supervision of a local doctor for 1 month.
-Systematic hardening
- physical education
-compliance with the sleep schedule
- sufficient stay in the fresh air
- full food</td></tr>
<tr><td colspan="2">Task 2. An 11-year-old girl was admitted to the clinic with complaints of inattention, irritability, muscle weakness, violent movements of the trunk, limbs and facial muscles, changes in handwriting and movements. During sleep, hyperkinesis disappear, and it intensifies against the background of emotional arousal. Revealed minor changes in the cardiovascular system (systolic murmur at the apex of the heart, between the heartthere are no changes). Increased ESR is up to 18 mm / h., Neutrophilic leukocytosis is 9.5 X 109 / l.</td></tr>
<tr><td>Exercise:
• Establish a nosological diagnosis
• List the diagnostic criteria, confirming the presence of streptococcal infection in the body
• How many levels of activity of the inflammatory process are accrued in this disease?
• Form a clinical diagnosis
• Give anticonvulsant therapy.</td><td>Sample answer:
1.Rheumatism.
2.Increased titer of anti-streptococcal antibodies (ASL-O, etc.)
- identification of group A streptococcus
- recently transferred scarlet fever
3. III stage of activity.
4. Rheumatism 1, active phase, activity 1 degree, Minor chorea, subacute course.
5 Seduxen</td></tr>
<tr><td colspan="2">Task 3. A 10-year-old girl was hospitalized for a month and a half with a diagnosis of rheumatism II, active phase, activity II degree, endomyocarditis, polyarthritis, acute course, NK II. Despite the ongoing therapy, the child developed a heart defect - mitral valve insufficiency.</td></tr>
<tr><td>Exercise:
• What pathomorphological phase of the rheumatic process is most likely in the girl now?
• Determine the timing of bicillin prophylaxis in this case
• List the auscultatory signs and name the place of the maximum loudness of the noise in this defect.</td><td>Sample answer:
1. 4 stage sclerosis (hyalinosis).
2. Round-the-clock - for life.
3. At the apex of the heart - p. Max systolic murmur, attenuation of 1 tone.
4. Stenosis of the mitral valve.
5. Replacing the valve.</td></tr>
</table>

• For what acquired heart disease is P mitrale observed on the ECG? • Assign the amount of cardiac care for mitral regurgitation	
Problem 4. A 12-year-old child was diagnosed with rheumatic heart disease six weeks after suffering a sore throat. The activity of the process corresponds to the II degree. The anti-inflammatory therapy was intended, which made it possible to stop laboratory signs of activity by the end of the third month of treatment, at the same time aortic valve insufficiency was diagnosed.	
Exercise: • Determine the nature of the course of rheumatism in this case • List the auscultatory signs and name the place of the maximum loudness of the noise at this wadi • What are the extracardiac signs of aortic valve insufficiency? • Determine the amount of cardiac care for aortic valve insufficiency. • What are the principles of treatment of children with rheumatism?	Sample answer: 1. Subacute course of rheumatism. 2. Diastolic murmur on the left edge of the sternum 3-4 intercostal space, weakening of 2 tones on the right in the 2nd intercostal space. 3. Feeling of pulsation in the head, extremities, pulsation of carotids, capillary pulse, Musset's symptom, Landolfi's sign - constriction and dilation of the pupil. 4. Valve replacement 5.The fundamentals of treatment of children with rheumatism combine the principle of complex staged therapy

8. Materials for classroom self-study:

8.1. The list of educational practical tasks that must be completed during the practical lesson:

1. Work at the patient's bedside.
2. Make a clinical diagnosis.
3. Prescribe treatment.
4. Outline preventive measures.

9.Guidance materials for mastering professional skills:

9.1. Methodology for performing work, stages of implementation.

1. Collect complaints, anamnesis of life and illness.
2. To assess the data of anamnesis, life and illness, risk factors contributing to the development of the disease.
3. Conduct a clinical examination of CVS in patients with myocarditis, pericarditis and IE.
4. Make a plan for additional survey methods.
5. Evaluate the results of laboratory and instrumental data.
6. Formulate a clinical diagnosis.
7. Provide emergency care when needed and determine the following therapeutic measures.

10. Materials for self-control of mastering knowledge, abilities, skills provided for by this work.

10.1. Tests:

1. What antibiotics should be used for repeated prophylaxis of rheumatism:
   A. tetracyclines
   B. lincomycin
   C. cephalosporins
* D. penicillin group
   E. aminoglycosides

2. What is the doctor's tactics for finding primary active rheumatism:
   A. outpatient examination

B. outpatient treatment
* C. hospitalization
D. registration of dispensary registration
E. prescribing sanatorium treatment

3. Specify the dose of bicillin-5 for secondary prevention of rheumatism in schoolchildren:
A. 500.000
B. 750.000
C. 1000.000
* D. 1500.000
E. 2000.000

4. Specify the dose of prednisolone for complex therapy with II degree of activity of the rheumatic process:
A. 0.1 mg / kg
B. 0.5 mg / kg
* C. 1.0 mg / kg
D. 1.5 mg / kg
E. 2.0 mg / kg