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DIAGNOSTIC CRITERIA, TREATMENT, PREVENTION, DISEASE OF DISEASES ACCOMPANIED BY CARDIOMYGALY SYNDROME AND HEART MURMURS

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Tutorial

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The textbook covers the main criteria for diagnosis, differential diagnosis, prevention, medical examination of cardiovascular diseases; the manual also includes control tasks, test questions, specially developed algorithms that allow you to effectively organize independent work of students. The textbook is intended for students of medical institutes and. corresponds to the 6th year program.

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INTRODUCTION

Diseases accompanied by cardiomygaly syndrome and heart murmurs are among the most common and often lead to disability. The future of the country depends on the health of children. Only a healthy generation with the harmonious development of physical and spiritual forces is capable of solving issues of further development and strengthening of society, ensuring its prosperity. Protecting children's health is not only a medical, but also a social problem. In our country and other countries, children are provided with favorable conditions for physical development and a comprehensive education, and an environment has been created that is conducive to the moral education of the younger generation in the spirit of humanism, hard work and patriotism. Protecting the health of children and mothers in Uzbekistan has become and is the focus of attention of the government of the Republic of Uzbekistan. The care of the younger generation for the family, the protection of motherhood and childhood, is enshrined in the Constitution of Uzbekistan and begins even before the birth of the child. From year to year, the Government increases allocations for health care and social activities aimed at improving the health of the child population. The preventive focus is becoming increasingly important: the material and technical base of the joint venture is being improved, the pediatric areas are being strengthened by the emergency nursery, offices of specialized specialists are being organized, and medical examinations of children in the areas are regularly carried out. The textbook is written in full compliance with the pediatrics program for students of higher educational institutions. The textbook is intended for 6th year students for early diagnosis of diseases accompanied by heart murmur syndromes and cardiomegaly.

Chapter - 1. Basic characteristics of heart murmurs and cardiomegaly

Cardiomegaly is a significant increase in the size of the heart due to: its hypertrophy and dilatation; accumulation of products of impaired metabolism in it; development of neoplastic processes.

Signs of cardiomegaly General: Physical X-ray EchoCG signs Specific - determined by the underlying disease

General signs of cardiomegaly

1. Physical: expansion of the boundaries of the heart; muffled or dull tones; weakening of the 1st tone at the apex; the appearance of a protodiastolic or presystolic gallop rhythm (3rd and 4th sounds); the appearance of sounds of relative mitral and tricuspid insufficiency; less commonly, diastolic murmur of relative pulmonary artery insufficiency (Graham-Steele); diastolic murmur of functional mitral stenosis (Flint)

2. X-ray: an increase in the transverse size of the cardiac shadow to 15.5 cm or more in men and up to 14.5 cm or more in women; increase in cardiothoracic index (the ratio of the transverse size of the heart shadow to the internal transverse size of the chest) more than 50%

3. Echocardiographic: enlargement of the heart chambers (left ventricular EDV index more than 55 ml/m² according to Simpson, left atrium index - more than 21 ml/m² according to Simpson, diastolic size of the right ventricle more than 20 mm); an increase (not always) in the mass of the left ventricle (in men more than 183 g, in women more than 141 g) or the left ventricular myocardial mass index (in men more than 94 g/m², in women more than 89 g/m²).

Classification

1. Arterial hypertension:

1.1. Hypertonic disease

1.2. Secondary hypertension

2. Coronary heart disease

2.1. Atherosclerotic cardiosclerosis

2.2. Heart aneurysm

2.3 Bernheim syndrome

3. Myocarditis

3.1. Myocarditis rheumatic

3.2. Viral myocarditis

3.3. Rickettsial, bacterial, spirochetal myocarditis

3.4. Fungal and parasitic myocarditis

3.5. Non-infectious allergic and toxic-allergic myocarditis

3.6. Idiopathic Abramov-Friendler myocarditis

3.7. Myocardial cardiosclerosis

4. Heart defects

4.1. Congenital

4.2. Purchased

5. Primary cardiomyopathies

5.1. Dilated or congestive cardiomyopathies

5.2. Hypertrophic obstructive and non-obstructive cardiomyopathies.

5.3. Restrictive cardiomyopathies.

6. Alcoholic myocardial dystrophy

7. Pulmonary heart

8. Endocrinopathies

8.1. Myocardial dystrophy in diabetes mellitus

8.2. Myocardial dystrophy in thyrotoxicosis

8.3. Myocardial dystrophy in hypothyroidism

9. Metabolic diseases

9.1. Hemochromatosis

9.2. Glycogenoses

9.3. Mucopolysaccharidoses

9.4. Lipoidosis (Fabry disease)

9.5. Cardiac amyloidosis

10. Heart tumors

10.1. Myxoma

10.2. Rhabdomyoma

10.3. Malignant heart tumors

11. Blood diseases

11.1. Anemia

11.2. Leukemia

12. Pericardial diseases

12.1. Acute and chronic effusion pericarditis

12.2. Non-inflammatory effusions in the pericardial cavity

12.3. Constrictive pericarditis

12.4. Pericardial tumors

Differential diagnosis of cardiomegaly

To identify “bull heart” syndrome (as cardiomegaly is informally called), cardiologists use the following diagnostic methods:

- palpation of the cardiac region, listening to the heart;
- CT scan;
- echocardiography;
- Ultrasound of the heart;
- ECG;
- chest x-ray;
- blood analysis;
- biopsy (tissue sampling from the inner surface of the heart ventricles) – extremely rare.

Differential diagnosis of cardiomegaly depending on the etiological factor: acquired cardiomegaly caused by an infectious lesion of the heart. This type of cardiomegaly is observed much more often, due to more frequent contacts of children with infections. In this regard, the role of rheumatic disease is most significant. Rheumatic myocarditis is characterized by allergic inflammatory changes in the interstitium, which in childhood acquire a clearly expressed exudative character, as a result of which cardiomegaly is often noted, resulting from relaxation of the heart. During auscultation, slow heart activity is heard, the first sound at the apex subsides and a gentle systolic murmur appears. Pericardial friction requires special attention because it may initially be gentle or resemble a third heart sound. Due to the special variability of the inflammatory process, cardiomegaly can reach large sizes in a short time and disappear in a short time - “accordion heart”. The electrocardiogram often shows the presence of atrioventricular block I and II degrees

Of the parasitic diseases of the heart, we most often encounter cardiac echinococcus, which is usually localized in the wall of the atrium. An hydatid cyst can rupture into the pericardium and cause pericardial effusion, accompanied by or without anaphylactic manifestations. More often, however, significant amounts of fluid may accumulate in the pericardium as a result of irritation.

Congenital heart defects are a common cause of enlarged heart size in infants, as abnormalities begin to affect its size usually after birth. At this age, cardiomegaly caused by heart defects incompatible with life does not

occur, as well as defects that affect its size very slowly and late. For example, with congenital pulmonary artery stenosis, which is an independent anomaly or combined with other developmental defects, the heart appears to be of normal size or Annals of Mechnikov Institute, N 2, 2013 80 www.imiamn.org.ua /journal.htm slightly enlarged, since hypertrophy is insignificant and affects only the right ventricle, i.e. the anteroposterior diameter of the heart increases, which is difficult to detect

However, especially large heart sizes are observed in infancy with Ebstein's disease. The syndrome is often an accidental discovery due to good endurance and the absence of obvious functional disorders before the onset of decompensation. An X-ray examination reveals a huge rounded heart, the shadow of which is very reminiscent of the shadow of exudative pericarditis. The electrocardiogram shows a large P-pulmonale in most leads, conduction disturbances, and often complete or partial right bundle branch block. There is no evidence of right ventricular hypertrophy, especially in the first few months of a child’s life.

Cardiomegaly of metabolic origin. With cardiac glycogenosis or Pompe disease (cardiomegalia glycogenica congenita), there is a general increase in the size of the heart due mainly to the left ventricle. The disease is characterized by impaired glycogenolysis, with glycogen accumulating in the heart muscle, striated muscles, skeleton, liver and kidneys. In most cases, the clinical picture of the disease manifests itself in the first few months after birth with a picture of isolated primary cardiomegaly, in more rare cases with a picture of already identified dyspnea and cyanosis. Subsequently, signs of left ventricular failure are detected. X-rays reveal an increase in predominantly the left ventricle. Diagnosis is made by muscle biopsy, which reveals glycogen deposition in the muscle fibers.

Early cardiomegaly was diagnosed solely based on examination of the patient - percussion, percussion of the borders of the heart. Then X-rays came to the doctor’s aid; with this study, one can quite accurately determine the size of the heart and even the condition of its various parts. But most accurately, ultrasound examination confirms or excludes cardiomegaly, which allows you to measure the thickness of the walls of the heart and the diameter of its cavities. Most often, cardiomegaly occurs in hypertension, when, under the influence of high pressure, thickening of

the walls of the myocardium occurs, and then expansion of its cavities. Often, cardiomegaly is a consequence of congenital or acquired defects of the heart or large vessels extending from it. From all of the above, it becomes quite obvious that there are always good reasons for the development of this condition, and in order to figure out which of them led to an enlarged heart, you need to evaluate the complaints of the child (mother), the medical history and all the data obtained during the research. Speaking about the diagnosis of cardiomegaly, it must be said that radiography sometimes gives false positive results: it “attributes” this pathology to those patients who actually do not have it. This situation is especially common in children and adolescents, because until now the concept of normal in this group of patients varies widely. However, you should not neglect X-ray data (including fluorography) or the doctor’s assumptions. In such situations, it is much easier to perform an ultrasound of the heart and finally figure out what’s what. After all, timely diagnosis is extremely important for this condition, which will allow treatment to begin before serious problems arise.

Treatment of cardiomegaly

Treatment of cardiomegaly (with the exception of bull's heart syndrome in newborns) begins with eliminating the causes that caused the disease. Both drug therapy and surgical methods can be used here. For example, for arterial hypertension, medications reduce blood pressure, and for kidney disease, they remove excess fluid from the body. If cardiomegaly is caused by damage to the heart valves, a cardiac surgeon will surgically restore them.

In situations where the cause of the pathology is drugs or alcohol, the patient’s condition will improve only if he gives up the bad habit. Treatment results directly depend on the stage and causes. Some patients are able to recover completely, while others have to take medication for the rest of their lives. In severe cases, a heart transplant is performed.

Risk group for the development of cardiomegaly

The greatest risk of developing cardiomegaly is in patients with:

arterial hypertension;

congenital heart disease;

• have had a heart attack;

• having a hereditary predisposition (someone in the family has already suffered from cardiomegaly).

Why is cardiomegaly dangerous?

An enlarged heart can cause serious health problems.

Among them:

• formation of blood clots;

• heart rhythm disturbance;

• cardiac arrest.

Individuals who have an enlarged left ventricle of the heart are at greater risk.

Prevention of cardiomegaly

Since cardiomegaly (with the exception of the congenital form) is not an independent disease, prevention is aimed at preventing the development of provoking diseases. It is important to exclude risk factors that lead to increased blood pressure, coronary heart disease, and lung problems. It is also necessary to give up smoking, drugs, and alcoholic beverages.

Prevention of congenital cardiomegaly includes:

• registration before 12 weeks of pregnancy;

• compliance with medical recommendations;

•rejection of bad habits;

• eating only healthy foods.

Main characteristics of heart murmurs

Noises have important diagnostic value for the differential diagnosis of various pathological conditions of the body. A murmur is any sound coming from the heart that is not a tone and lasts more than 0.1 second.

Murmurs over the heart area are divided into:

 Intracardiac (valvular and non-valvular)

- Physiological (systolic, above the pulmonary artery);
- Functional (mainly systolic, except Graham-Still noise - diastolic),
- Organic (both systolic and diastolic)

 Extracardiac

- Pericardial friction rub
- Pleuropericardial murmurs

Physical basis of the occurrence of heart murmurs.

For noise to occur, the occurrence of turbulent blood flows and/or vibration of individual parts of the heart is necessary: chords, valves, papillary muscles, etc. Typically, turbulent flows occur:

1. When the blood flow paths, inflow and outflow paths are narrowed.

2. With non-physiological expansion of blood flow paths.

3. In the event of (congenital or acquired) non-physiological anastomosis (holes) with a difference (gradient) in blood pressure.

4. When the rheological properties of blood change: a decrease in viscosity, fluidity, etc., as happens with anemia. In this case, turbulent flows can form under other normal hemodynamic parameters.

1. When the blood flow paths, inflow and outflow paths are narrowed (passing through a narrow opening, the blood swirls and makes noise):

Stenosis of the aortic orifice, pulmonary artery (systolic ejection murmurs),

Mitral or tricuspid stenosis (diastolic ejection murmurs).

Similar mechanisms of noise formation occur with valve insufficiency (regurgitation noises - both systolic and diastolic).

2. The occurrence of pathological expansions and aneurysms along the blood flow.

Blood entering such a cavity will swirl, causing noise.

3. In the event of non-physiological anastomosis (holes) with a difference (gradient) in blood pressure - ASD, VSD, PDA, etc.

4. Other causes of noise:

1. Formation of noise as a result of sound vibrations of the chords, valve or its valves, papillary muscles. Such reasons account for 3-5% of all reasons.

2. Changes in the endocardium, changes in its smoothness. 0.2-1.0% of all causes of noise formation.

Heart murmur analysis scheme:

1. Determine the ratio of noise to the phases of cardiac activity

2. Determine the strength of the noise

3. Determine the ratio of noise to heart sounds

4. Determine the shape of the noise.

5. Determine the best place to listen to the noise

6. Determine noise irradiation

7. Determine the influence of breathing phases and load on the nature of noise

8. Determine, if necessary, the dynamics of noise over time: days, weeks, months, years.

Noise characteristics

1. Determine the ratio of noise to the phases of cardiac activity:

- Systolic murmur
- Diastolic murmur

2. Determine the strength of the noise:

- noise that is heard only in silence and intense attention is weak (quiet);
- a noise that is heard immediately, without straining attention, but is significantly weaker than heart sounds, is a noise of medium strength;
- a noise that is equal to or exceeds the strength of heart sounds, “amazing”, is strong;
- the noise that “hits the ear”, forces you to loosen the pressure on the phonendoscope, is very strong.

3. Determine the ratio of noise to heart sounds. There are noises that: deform the heart sounds, merge with them, and noises that do not deform the tones, that is, heard separately from the tones.

4. Determine the shape of the noise.

Noise shape:

- decreasing: starts from a tone and decreases in audibility to another tone;

growing: arises in the depths of the phase and grows towards the tone, merging with it;

- diamond-shaped: appears after a tone, reaches a maximum in the middle of the phase and decreases again towards another tone;
- ribbon-shaped: noise occupies the entire phase from one tone to another.

5. Determine the place of best auscultation of the murmur by sequentially auscultating the entire area of the heart.

6. Determine the irradiation of the noise: whether the noise is carried beyond the contour of the heart or not, the location of the noise.

7. Determine the influence of breathing phases, load, functional tests on the nature of the noise.

8. Determine, if necessary, the dynamics of noise over time: days, weeks, months, years. Physiological noises

With the ideal adaptation of the heart and blood vessels of most healthy people to the function of blood circulation, significant turbulent blood flows do not arise during their work and, therefore, heart murmurs do not occur.

In childhood, some (1-10%) may experience temporary imbalances during growth, and most often there is a relative narrowness of the pulmonary artery. During this period of life, a systolic murmur can be heard above the pulmonary artery (3rd point of auscultation): a weak or medium-volume murmur does not deform heart sounds, fusiform, and is not carried out.

At the same time, no signs of pathology of the heart and blood vessels are determined. By 3-5 years this noise usually disappears.

Functional noise

Functional murmurs include heart murmurs resulting from:

changes in heart function (with adrenergic dysregulation - thyrotoxicosis, neurocirculatory dystonia), rheological properties of blood (anemia).

Peculiarities:

1. The murmur in such cases is associated with the acceleration of blood flow through the unchanged heart.

2. Such people have no changes in the valvular apparatus of the heart, no changes in the inflow and outflow pathways, no pathological anastomosis.

Some cardiologists classify as functional murmurs that occur with relative valve insufficiency due to:

- damage to the heart muscle (myocarditis)
- its expansion (hypertrophy and dilatation).

Functional murmurs also include some variants of mitral valve prolapse, in which there is no significant blood regurgitation.

The most important differences in functional noise (FN):

Only systolic murmurs can be functional;

FS weak or medium volume;
FS do not deform heart sounds;
FS occupy, as a rule, less than half of the systole;
FS are not carried out beyond the contour of the heart;
FS are labile and rarely progress;
and most importantly:
there are no signs of damage to the heart valve apparatus,
there are no signs of narrowing of the inflow and outflow pathways,
there are no signs of non-physiological anastomosis.

Organic noises.

Organic noises are divided into:
regurgitation sounds;
ejection noises;
murmurs due to congenital or acquired pathological anastomosis

A regurgitant murmur is the sound of abnormal blood flow caused by valve insufficiency.

They can be:

1) systolic, associated with insufficiency of the atrioventricular valve, left or right

2) diastolic, associated with insufficiency of the semilunar valve of the aorta or pulmonary artery

Ejection murmurs are caused by stenosis of the blood outflow tract, that is, they are associated with stenosis of the orifices:

1) systolic ejection murmurs – stenosis of the aortic or pulmonary artery

2) diastolic murmurs - stenosis of the atrioventricular orifices (mitral and tricuspid)

Noises of pathological anastomosis.

Pathological anastomosis occurs with some congenital heart defects:
atrial septal defect (ASD),
ventricular septal defect (VSD),
patent ductus arteriosus (PDA), etc.

Differential diagnosis

Organic

There is organic (inflammation, dystrophy, atherosclerosis) damage to the heart muscle and valve apparatus

Have a maximum noise point
Timbre – rough, rumbling, scratching, blowing
Permanent
Have conductivity (through blood flow, muscle)
Do not disappear under the influence of treatment
Accompanied by “cat purring”
Accompanied by palpation and percussion signs of damage to the heart valves
There is a position in which the noise is better heard
A) systolic - lying down B) diastolic - standing
Increases under load
Inorganic
There are no organic heart lesions
Do not have a maximum point
Soft, blowing
Unstable, labile
No conductivity
Weaken or disappear
Not accompanied
There are no other signs of heart damage
No special position
When loaded, weaken (or disappear)

Signs of noise	Organic noise	Functional noise
Relation to the phases of the cardiac cycle	Systolic and diastolic	Systolic
Listening point	Depending on the affected valve	Often - on the pulmonary artery and apex
Age of patients	In young and old	More often in young people (children, teenagers)
Character	Rude	Delicate, soft
Volume	Loud	Quiet

Duration	Long lasting	Short
Irradiation	Carried out beyond the heart	Not carried out beyond the heart area
Variability depending on body position, physical activity	Doesn't disappear	May disappear or intensify
Other signs of the defect	Changes in the boundaries of the heart, heart sounds and other signs of defect, EchoCG	None

Chapter - 2. Congenital heart defects
Vitiurn cordis congenitum

CONGENITAL HEART DEVICE is a pathological condition characterized by certain defects in the development of the heart and great vessels, resulting from exposure to various harmful factors on the embryo and fetus.

Congenital heart defects (CHD) occupy one of the leading places among congenital malformations. The frequency of congenital heart disease in all countries of the world, including Russia, reaches from 2.4 to 14.2 per 1000 newborns, however, taking into account the much higher ante- and intrapartum mortality, among live births the number of patients with various variants of congenital heart disease is 0.7-1. 2‰. Timely diagnosis of this pathology and appropriate surgical correction are extremely important. If timely treatment is not carried out, then about 55% of children with congenital heart disease die in the 1st year of life, and before 5 years of age - 85%. Knowledge of the clinic and diagnostic methods allows you to timely identify congenital heart disease and avoid complications and early disability

According to WHO, among all newborns, children with heart defects make up about 1%; 5-6 children per 100,000 people die from congenital heart defects.

Scheme of the pathogenesis of congenital heart defects

Predisposing factors
Rubella, influenza, herpes simplex, ARVI, chronic diseases, occupational hazards, use of medications, alcoholism, smoking, previous abortions, unfavorable pregnancy, hereditary predisposition suffered during pregnancy
Disturbance of embryogenesis during the 2nd and 6th weeks of pregnancy
Animal hemodynamics
The emergence of clinical-functional signs of one or another anatomical variant of spanking

Patent ductus arteriosus (PDA) is the presence of an abnormal vascular connection in which blood from the aorta flows into the pulmonary artery.

Epidemiology. The incidence of PDA is 0.14-0.3/1000 live births. Among all congenital heart diseases, PDA occurs in 6-7% of cases. PDA predominantly affects girls. In 5-10% of cases, PDA is combined with other congenital heart defects. In some cases, it is considered as a compensating defect (with tetralogy of Fallot, pulmonary stenosis, preductal coarctation of the aorta, aortic atresia); in other cases, as a defect that increases hemodynamic disturbances: with postductal coarctation of the aorta, VSD, ASD.

Anatomical entity

the arterial (Botallov) duct (AP) is open,

connecting the aorta to the pulmonary artery.

Hemodynamics in non-unionization of the AP:

- part of the oxygenated blood from the aorta enters the pulmonary artery through the PDA, then into the lungs;

- repeated circulation of additional volumes of blood in the lungs leads to overflow of their vascular bed;

- excess blood flowing from the lungs into the left atrium and left ventricle causes their hypertrophy;

- a reduced amount of blood enters the systemic circulation.

I. Diagnostic Criteria

Anamnestic:

- the presence of congenital or acquired lesions of the heart and blood vessels in the genealogical history;

- maternal diseases during pregnancy (measles rubella, measles, chicken pox, toxoplasmosis), especially in the first trimester;

- exposure of a pregnant woman to radioactive radiation, toxic and chemical factors; diet violations during pregnancy toxicosis, leading to high-quality starvation and polyhypovitaminosis;

- unfavorable obstetric history (abortions, miscarriages);

- early or late pregnancy; complicated course of labor;

- large difference in the age of parents, consanguineous marriages; attacks of asphyxia in the neonatal period;

-delayed physical development;

- frequent bronchopulmonary diseases of the child.

II. Clinical:

- fast fatiguability; shortness of breath on exertion;

- pallor of the skin, cyanosis of the lower half of the body when screaming, straining, disappearing immediately after stopping the load, persistent cyanosis of the skin and mucous membranes during the reverse discharge of blood (from the pulmonary artery to the aorta), due to the development of pulmonary hypertension;

- pulsation of the vessels of the neck, high, fast, galloping pulse, cardiac hump, increased cardiac impulse, systole-diastolic trembling at the base of the heart;

- displacement of the borders of the heart to the left, expansion of the borders of the vascular bundle;

- emphasis and splitting of the second tone on the pulmonary artery;

- rough, “machine-like”, continuous, systole-diastolic noise with maximum sound in the 2nd intercostal space to the left of the sternum (above the pulmonary artery), carried into the interscapular space and to the vessels of the neck;

- symptoms of circulatory disorders of the left ventricular type are possible;

- Blood pressure - increased systolic pressure, decreased diastolic (sometimes to 0), large pulse amplitude.

III. Paraclinical:

1. Instrumental and graphic:

a) FCG - high-amplitude, diamond-shaped systole-diastolic murmur in the 2nd intercostal space to the left of the sternum, increased amplitude of the second tone;

b) ECG - signs of overload of the left ventricle, later - of both ventricles (with moderate pulmonary hypertension); with a decrease in arteriovenous blood discharge, hypertrophy of the right ventricle is more pronounced;

c) ECHO CG - an increase in the size of the cavities of the left atrium and left ventricle, an increase in the amplitude of movement of the mitral valve.

2. X-ray:

- increased vascular pattern of the lungs (hypervolemia);

- an increase in the size of the heart due to hypertrophy of the left ventricle and left atrium; with the development of pulmonary hypertension, the right ventricle also increases;

- bulging of the pulmonary artery arch, expansion of the ascending aorta, pulsation of the roots of the lungs.

Differential diagnosis

Performed with aortopulmonary septal defect; fistulas of the coronary arteries; aortic insufficiency combined with a ventricular septal defect.

Treatment. Specific conservative therapy is possible only in premature infants, by intravenous administration of three doses per 48 hours of a prostaglandin synthesis inhibitor (indomethacin at a dose of 0.2; 0.1; 0.1 mg/kg). If there is no effect after 24 hours, an additional three-time administration of the drug at a dose of 0.1 mg/kg with an interval of 24 hours is possible. The effectiveness of treatment is 70 - 80%. The indication for surgical treatment is the presence of a PDA. In young children, they resort to clipping the vessel. In older children, the vessel is

ligated. The optimal timing of the operation is from 6-12 months to 3-5 years. Postoperative mortality is less than 1%. Elimination of the defect is also carried out using the endovascular method - using special spirals (with a duct diameter of up to 3 mm); for larger holes (up to 6 mm), several spirals or special occluders are used.

Complications of PDA. Long-term overload of the pulmonary circulation leads to the formation of pulmonary hypertension, which contributes to the formation of hypertrophy of the right ventricle and right atrium, change of the shunt to right-to-left, arterial hypoxemia, chronic right ventricular or total heart failure. With small ducts, bacterial endocarditis may develop. Rarely does a complication develop such as a PDA aneurysm with its rupture, thrombosis or infection. Postoperative complications: hemorrhage, vascular damage, injury to the recurrent laryngeal and phrenic nerves, infective endocarditis, congestive heart failure. Possible complications of transcatheter duct occlusion: residual shunt, migration of the coil, hemolysis and thrombosis of the femoral vessels through which the catheter was passed

Forecast. After surgical correction of the defect, in the absence of complications, the development of such children is normal. Premature babies with a large PDA often develop bronchopulmonary dysplasia in a short time.

Atrial septal defect

Epidemiology. The incidence of ASD is 0.1-0.53/1000 newborns. Female patients predominate (2:1). Among all congenital heart defects, ASD is detected in 10-12%. Depending on the nature and degree of underdevelopment of the primary and secondary interatrial septa and endocardial ridges, primary, secondary defects and the complete absence of the interatrial septum (single common atrium, three-chamber heart) are distinguished.

Etiology. Primary ASD occurs due to underdevelopment of the primary interatrial septum and preservation of the primary communication between the atria; this is, as a rule, a large defect (1/3-1/2 part of the septum), which is localized in the lower part of the septum. This defect is most often combined with Down syndrome (trisomy 21). Secondary ASD occurs as a result of underdevelopment of the secondary

interatrial septum and in most cases is located in the center of the interatrial septum. This defect is often combined with valvular stenosis of the pulmonary artery. Isolated secondary ASD is often inherited in an autosomal dominant manner. Combinations of primary and secondary ASDs are possible. In some cases, a single atrium is formed. The patent foramen ovale (PFO) is an interatrial communication through which, during intrauterine development, blood from the inferior vena cava is sent directly to the left atrium. At birth, the pressure in the left atrium is higher than in the right, the valve of the oval window is pressed against the oval window and its physiological closure occurs. By the end of the first month of life, LLC with underdevelopment of the oval valve or its defect persists in 7-3%, by the year in 2%. LLC is found in 15% of adult patients.

Anatomical entity

- one or more holes in the interatrial septum.

Hemodynamics in ASD:

- discharge of arterial blood from the left atrium through the defect between the atrial septum to the right;
- volume overload of the right atrium and right ventricle;
- volume overload of the vessels of the pulmonary circulation;
- reduction in the amount of blood in the systemic circulation.

Diagnostic criteria

I. Anamnestic:

the presence of congenital or acquired lesions of the heart and blood vessels in the genealogical history; maternal diseases during pregnancy (measles rubella, measles, chicken pox, toxoplasmosis), especially in the first trimester; exposure of pregnant women to radioactive radiation, toxic and chemical factors; diet violations during pregnancy toxicosis, leading to high-quality starvation and polyhypovitaminosis; unfavorable obstetric history (abortions, miscarriages); early or late pregnancy; complicated course of labor; large difference in the age of parents, consanguineous marriages; attacks of asphyxia in the neonatal period; delayed physical development; frequent bronchopulmonary diseases of the child.

II. Clinical:

- fatigue, shortness of breath, palpitations during physical activity;

- pallor of the skin and visible mucous membranes;

- shift of the borders of relative cardiac dullness to the left: in the 1st-2nd intercostal space due to the expansion of the vascular bundle, in the 2nd intercostal space also due to swelling of the cone and trunk of the pulmonary artery and in the 3rd-4th intercostal space due to hypertrophy and dilatation of the right sections hearts;

- accent and bifurcation of the second tone above the pulmonary artery, moderate systolic murmur, which is well carried to the left in the axillary region and to the angle of the scapula with the maximum sounding point in the 2-3 intercostal space on the left at the sternum.

III. Paraclinical:

1.Instrumental and graphic:

a) FCG - systolic murmur of diamond-shaped or fusiform shape, medium or small amplitude with a maximum sounding point in the 2-3 intercostal space on the left at the sternum; splitting and increase in the amplitude of the second tone above the pulmonary artery;

b) ECG - predominance of electrical activity of the right ventricle; incomplete blockade of the right bundle branch; increase, sharpening of the P wave in leads II and III;

c) ECHO CG - increase in the size of the cavity of the right ventricle; paradoxical movement of the interventricular septum

2. X-ray:

- strengthening of the pulmonary arterial pattern;
- increased pulsation of the roots of the lungs;
- increase in the shadow of the heart due to the right parts;
- bulging of the pulmonary artery arch;
- upward displacement of the right cardiovasal angle, reduction and poor differentiation of the aortic shadow.

Differential diagnosis:

ASD should be differentiated from pulmonary artery stenosis, PDA, high pulmonary hypertension, or a defect

interventricular septum.

Treatment. Indications for surgical correction of the defect: heart failure, retardation in physical development, pulmonary diseases. The hemodynamic indication for surgery is a ratio of pulmonary to systemic

blood flow of more than 2:1. If conservative drug therapy is effective, surgery can be postponed until 3-5 years of age (maximum 8-10 years). Secondary defects are closed by suturing; Primary ASDs are closed with a patch of autopericardium or synthetic tissue using thoracotomy and artificial circulation. Endovascular repair of the defect using occluders is possible only with a secondary ASD up to 25-40 mm in size, around which there is a septal border up to 10 mm wide.

Complications of ASD appear at the sclerotic stage of pulmonary hypertension in the form of Eisenmenger syndrome (pulmonary hypertension, with dilation of the pulmonary artery trunk and a change in shunt from right to left, with the appearance of constant cyanosis). Characteristic of this condition is a combination of a dilated pulmonary artery trunk and the formation of a relative functional stenosis of the pulmonary valve against the background of an increased stroke volume of the right ventricle. In the later stages of ASD with Eisenmenger syndrome, dystrophy and sclerosis of the right ventricular myocardium occurs, which leads to the development of first right ventricular and then total heart failure. Complications after surgery: acute heart failure, cardiac arrhythmias, infective endocarditis, residual shunt. Possible complications with catheter occlusion of a defect: perforation of the vessel wall, vessel occlusion, incomplete closure of the defect, infective endocarditis.

Ventricular septal defect (VSD)

Epidemiology. VSD occurs in 1.5–3.5 cases per 1000 full-term newborns and in 4.5–7 cases in premature newborns. Among congenital heart disease, the incidence of VSD is 15-20%. Peremembranous (membranous part of the septum) defects account for approximately 80% of all VSDs. Muscle defects account for 5-20% of isolated VSDs. Etiology. VSD has a multifactorial origin. Genetic risk factors: high frequency of cardiac anomalies in the family history of the parents. Maternal risk factors: diabetes, phenylketonuria, maternal alcoholism. The mechanism of VSD formation has not been sufficiently studied. It is believed that the peremembranous defect occurs due to impaired fusion of the heart parts due to a transient circulatory disorder in the developing septum; muscle defects are a consequence of cell death in the septum

Anatomical entity

 There is communication between the right and left chambers of the heart at the level of the ventricles.

Hemodynamics in VSD:

- discharge of blood during systole from the left ventricle to the right;
- volume overload of the right and left parts of the heart;
- volume overload of the pulmonary circulation.

Diagnostic criteria

I. Anamnestic:

the presence of congenital or acquired lesions of the heart and blood vessels in the genealogical history; maternal diseases during pregnancy (measles rubella, measles, chicken pox, toxoplasmosis), especially in the first trimester; exposure of pregnant women to radioactive radiation, toxic and chemical factors; diet violations during pregnancy toxicosis, leading to high-quality starvation and polyhypovitaminosis; unfavorable obstetric history (abortions, miscarriages); early or late pregnancy; complicated course of labor; large difference in the age of parents, consanguineous marriages; attacks of asphyxia in the neonatal period; delayed physical development; frequent bronchopulmonary diseases of the child.

II. Clinical:

- fatigue, shortness of breath during physical activity;
- cardiac hump, increased cardiac impulse, systolic tremors in the 3-5 intercostal spaces on the left at the edge of the sternum;
- displacement of the boundaries of relative cardiac dullness in both directions;
- The first tone at the apex is poorly heard, as it is “covered” by a systolic murmur, the accent of the second tone is over the pulmonary artery, a rough systolic murmur over the heart area, with maximum sound in the 3rd and 4th intercostal space at the left edge of the sternum, intensifying to the xiphoid process, not carried out on large vessels of the neck;
- with large defects, symptoms of heart failure appear early (mainly left ventricular, often total).

III. Paraclinical:

1. Instrumental and graphic:

- FCG - high-amplitude systolic murmur, maximum severity in the 3-4 intercostal space on the left at the sternum; increase in the amplitude of the second tone over the pulmonary artery;

- ECG - signs of overload of the left parts of the heart, and later of the right; signs of hypertrophy of the right sections as pulmonary hypertension develops;

- ECHO KG - enlargement of the cavities of the left atrium and left ventricle; disappearance of the echo-interventricular septum,

2. X-ray:

- strengthening of the pulmonary pattern,
- increase in heart size due to the left and right sections
- bulging of the pulmonary artery arch.

Differential diagnosis:

VSD must be differentiated from ASD, small CAC, moderate right ventricular outflow tract stenosis, moderate mitral valve insufficiency, moderate aortic valve stenosis.

Treatment. Treatment of circulatory failure is carried out according to general principles. Indications for surgical correction of the defect are heart failure, delayed physical development, and repeated respiratory infections. In these cases, the operation is performed starting from the first six months of life. In children older than one year, surgical intervention is indicated when the ratio of pulmonary and systemic blood flow is 2:1 or more. For large defects without heart failure, but with increased pulmonary arterial pressure according to Doppler ultrasound, radical surgery is necessary as early as possible before the age of 1 year. Plastic surgery is performed using a xenopericardium patch. In recent years, for muscular, especially multiple, and in some cases for perimembranous defects, the technique of transcatheter closure of VSD using an Amplatzer occluder has been used. Palliative surgery to narrow the pulmonary artery is performed only in the case of concomitant defects and anomalies that complicate radical correction of the VSD.

Complications. With medium and large IVS defects, hypervolemia of the pulmonary circulation is accompanied by compensatory spasm of pulmonary vessels and increased pressure in the pulmonary artery. Long-term pulmonary hypertension and hypervolemia of the pulmonary

circulation leads to morphological changes in the wall of pulmonary arterioles, small and medium-sized vessels. Chronic arteritis, fibrosis and sclerosis of the walls of blood vessels and their desolation develop. In the final sclerotic stage of pulmonary hypertension, the pulmonary artery trunk dilates; the pressure in the pulmonary artery can exceed 60-70% of the systemic blood pressure. The pressure in the right ventricle increases and becomes greater than in the left. The discharge of blood through the defect becomes cross, and then right-left, Eisenmenger syndrome develops - a complication of the terminal phase of a medium-sized and large-sized VSD in the perimembranous part.

It is characterized by: - severe pulmonary hypertension with high pulmonary resistance;

- venoarterial discharge of blood through the defect ("shunt change") with the appearance of cyanosis;

- expansion of the pulmonary artery trunk and desolation of the peripheral vessels of the pulmonary circulation;

- depletion of the peripheral pulmonary pattern on a radiograph of the chest organs in a direct projection ("amputation syndrome"), bulging of the pulmonary artery arch;

- Clinically – disappearance of systolic murmur and systolic tremor in the third and fourth intercostal spaces to the left of the sternum; the appearance of a diastolic murmur of pulmonary valve insufficiency (Graham–Still murmur);

- emphasis of the second tone on the pulmonary artery is a sign of high pulmonary hypertension. Signs of chronic hypoxia appear - deformation of the phalanges of the fingers ("drumsticks") and nails ("watch glasses"). With small defects in the muscular part of the IVS, blood discharge into the right ventricle is small and hemodynamic overload of the pulmonary circulation does not occur. Complications after surgery include atrioventricular block, bundle branch block. Tricuspid valve insufficiency, recanalization of the defect, residual pulmonary hypertension, and infective endocarditis are also possible. Prevention. For minor surgical procedures (tooth extraction, adenotomy, tonsillectomy) in non-operated patients and after palliative and radical correction of the defect, prophylaxis is carried out

Isolated pulmonary valve stenosis

Anatomical essence of ISKLS

 an obstacle to the flow of blood at the level of the pulmonary valve.

Hemodynamics in ISCL:

 an obstacle to the ejection of blood from the right ventricle;

 increased pressure in the right ventricle;

 change in the structure of the cardiac cycle towards lengthening the ejection period;

 systolic pressure gradient between the right ventricle and the pulmonary artery (systolic pressure in the pulmonary artery in most cases is within normal limits or slightly reduced);

 hypertrophy of the right ventricular myocardium and increased diastolic pressure;

 increased systolic pressure in the right atrium;

 hypertrophy and dilatation of the right atrium;

 often dilatation of the oval window and shunt from the right atrium to the left and the development of cyanosis.

Diagnostic criteria

I. Anamnestic:

the presence of congenital or acquired lesions of the heart and blood vessels in the genealogical history; maternal diseases during pregnancy (measles rubella, measles, chicken pox, toxoplasmosis), especially in the first trimester; exposure of pregnant women to radioactive radiation, toxic and chemical factors; diet violations during pregnancy toxicosis, leading to high-quality starvation and polyhypovitaminosis; unfavorable obstetric history (abortions, miscarriages); early or late pregnancy; complicated course of labor; large difference in the age of parents, consanguineous marriages; attacks of asphyxia in the neonatal period; delayed physical development; frequent bronchopulmonary diseases of the child.

II. Clinical:

 shortness of breath during physical exertion, and in severe cases, even at rest, pain in the heart region caused by a deficiency of coronary circulation;

 with an open oval window - cyanosis of the lips, bulging of the chest in the area of the heart ("heart hump");

 swelling and pulsation of the neck veins, increased cardiac impulse, systolic trembling over the heart in the projection of the pulmonary artery;

 increase in heart size, mainly to the right;

 strengthening of the first tone (except for patients with pronounced stenosis or right ventricular failure), the second tone over the pulmonary artery is not audible or is sharply weakened, the systolic murmur is well audible over the pulmonary artery, radiates towards the left clavicle, is well audible in interscapular space, in some patients a gentle diastolic murmur is detected, indicating insufficiency of the pulmonary valves with deep deformation of the leaflets;

 A/D - not changed.

II. Paraclinical:

1. Instrumental and graphic:

a) on FCG a diamond-shaped systolic murmur of large amplitude is recorded with a maximum in the 2nd intercostal space on the left at the sternum. The noise begins some time after the first tone

in the second half of systole, the second sound is split, and its pulmonary component is weakened.

b) The ECG reflects the degree of overload and hypertrophy of the right heart. A downward shift of the ST interval and a negative T wave in the right precordial leads indicate an extreme degree of overload. Right atrium overload.

c) ECHO CG - reveals valve stenosis and details its anatomical structure.

2. X-ray:

 the shadow of the heart is enlarged: hypertrophy and dilatation of the right ventricle and right atrium, outward displacement of the left border of the heart (while the apex remains localized in the 4-5 intercostal spaces). In the area of the cardiac waist, a pronounced recess is formed, due to the narrowness of the pulmonary artery trunk. The heart takes on the shape of a “Dutch shoe.” Lengthening and protrusion of the arch of the pulmonary artery. The vascular pattern is normal;

 expansion of the pulmonary artery trunk in combination with a normal or depleted pulmonary pattern is a characteristic radiological sign of isolated pulmonary artery stenosis, predominantly valvular.

Differential diagnosis.

In typical cases, isolated pulmonary artery stenosis can be diagnosed without much difficulty using conventional clinical methods. The defect must be differentiated from isolated stenosis of the outlet tract, ASD, tetralogy of Fallot and other complex defects, the complex of which includes pulmonary artery stenosis.

Tetralogy of Fallot

Anatomical essence - stenosis of the outflow tract of the right ventricle, large VSD, dextroposition of the aorta, hypertrophy of the right ventricular myocardium.

Hemodynamics:

 from the right ventricle, blood flows into the narrowed pulmonary artery and the aorta “sitting astride” on the interventricular septum;

 blood enters the aorta from the left (arterial) and right (venous) ventricles. As a result of the limited flow of blood into the pulmonary circulation and its significant discharge from the right ventricle into the aorta, cyanosis develops;

 overload of the right ventricle occurs. The development of right ventricular hypertrophy is especially influenced by its adaptation to pressure in the aorta;

 compensatory collateral circulation gradually arises between the systemic circle and the lungs, which is carried out mainly through the dilated arteries of the bronchi, chest wall, pleura, pericardium, esophagus and diaphragm;

 Over time, polycythemia develops (erythrocytes 8*1012/l, and hemoglobin up to 250 g/l).

Diagnostic criteria

I.Anamnestic

the presence of congenital or acquired lesions of the heart and blood vessels in the genealogical history; maternal diseases during pregnancy (measles rubella, measles, chicken pox, toxoplasmosis), especially in the first trimester; exposure of pregnant women to radioactive radiation, toxic

and chemical factors; diet violations during pregnancy toxicosis, leading to high-quality starvation and polyhypovitaminosis; unfavorable obstetric history (abortions, miscarriages); early or late pregnancy; complicated course of labor; large difference in the age of parents, consanguineous marriages; attacks of asphyxia in the neonatal period; delayed physical development; frequent bronchopulmonary diseases of the child.

II. Clinical:

 cyanosis, which is observed from the first months of life, appears more often by the age of one year and later, intensifying with physical activity, emotional stress, crying, screaming, etc.; extreme weakness after a certain physical activity (often squatting or lying with legs brought to the stomach);

 dyspnea-cyanotic attacks (hypoxemic): cyanosis, shortness of breath sharply intensify, tachycardia develops, anxiety, weakness, sometimes patients lose consciousness. The duration of attacks ranges from several minutes to 10-12 hours. Possible cerebrovascular accidents;

 retardation in physical development. Thickening and change in the shape of the nails (“watch glasses”) and nail phalanges (“drum sticks”), expansion of the skin capillary networks in the veins and in the forehead, epigastric pulsation;

 the borders of the heart are somewhat expanded;

 weakened II tone over the pulmonary artery. Systolic murmur to the left of the sternum in the 2-3 intercostal spaces, the intensity of which depends on the degree of narrowing of the outflow tract of the right ventricle and pulmonary arteries; symptoms of circulatory disorders are rare;

A/D is normal or slightly decreased

II. Paraclinical:

1. Blood test - compensatory polycythemia.

2. Instrumental and graphic:

a) FCG: II tone is bifurcated, a pronounced click of the closing of the arterial valves. A diamond-shaped murmur, occupying the entire systole

and decreasing towards the end of the last, is recorded along the left edge of the sternum;

b) ECG: deviation of the electrical axis to the right, hypertrophy of the right ventricle. There are no symptoms of pronounced overload and stretching of the right ventricle. The P wave in most patients is enlarged in standard leads, conduction slowdown in the myocardium of the right ventricle is detected;

c) ECHO CG: right position and expansion of the base of the aorta and the cavity of the right ventricle, reduction in the size of the left ventricle and left atrium, hypertrophy of the right ventricle, interruption of ECHO signals in the interventricular septum, narrowing of the outflow tract of the right ventricle.

3. X-ray:

 a slight increase in the size of the heart, hypertrophy of the walls of the right ventricle and a moderate expansion of its cavity, retraction in the area of the pulmonary artery, the heart resembles a “shoe” shape, a moderate displacement of the heart to the left;

 the pulmonary pattern is not clearly expressed, in some patients you observe * heaviness of the roots of the lungs, in children with well-developed collaterals the vascular pattern of the lungs is quite pronounced, and sometimes it is strengthened.

Differential diagnosis

should be carried out with transposition of large vessels.

Coarctation of the aorta (CA)

The anatomical essence of the coronary artery is a congenital segmental narrowing of the aorta, located in the area of its isthmus.

Hemodynamics:

 above the narrowing, blood pressure increases;
 the left ventricle experiences pressure overload during operation;
 below the narrowing A/D is reduced;
 blood supply is provided by collaterals.

Diagnostic criteria.

Clinical:

 decreased appetite, retarded growth and body weight, anxiety, symptoms of cardiopulmonary failure resistant to treatment;

 absence or weakening of the pulse in the femoral arteries;

 increase in heart size due to left ventricular hypertrophy;

 murmurs are uncharacteristic and unreliable symptoms. The most commonly heard systolic murmur is in the left subclavian fossa and interscapular region;

 A/D is decreased in the lower extremities compared to those in the upper extremities.

II. Paraclinical:

1. Instrumental and graphic:

a) ECG - signs of left ventricular hypertrophy;

b) ECHO CG - left ventricular myocardial hypertrophy.

2. X-ray:

-an increase in the size of the heart, expansion and increased pulsation of the ascending part of the aortic arch. Usuration of ribs. Strengthening of the pulmonary vascular pattern. Bulging of the pulmonary artery arch.

Differential diagnosis

should be performed with fibroelastosis.

Diagnosis standard:

congenital heart disease, coarctation of the aorta, relative compensation phase, NK.

Atrioventricular communication - open atrioventricular canal (AVC) - CHD, the components of which are a primary atrial septal defect, cleft cusps of the mitral and tricuspid valves, a ventricular septal defect. The defect can be partial or complete. With complete AVK, both septal defects and a single atrioventricular valve are identified. With partial AVK, there is a primary ASD with separated openings for the mitral and tricuspid valves, which are formed at the same level (normally, the tricuspid valve is displaced to the apex of the heart). AV valves, in this type of defect, are attached to the upper edge of the interventricular septum. The splitting of the mitral valve leaflet can be in the form of a small diastasis or reach its base. Additional chords attached to the interventricular septum often extend from the edges of the cleft, and abnormalities of the papillary muscles are also possible.

Epidemiology. The incidence of VKA among all congenital heart disease is 3-4%, and 6% among critical congenital heart disease. In 60-70% of children with VVC, the full form of the defect is detected, 50% of these patients have Down syndrome.

Hemodynamics. With partial VKA, hemodynamic disturbances are determined by regurgitation on the mitral valve and shunting of blood through the atrial septal defect. In most cases, there is a large shunt of blood from left to right, hypervolemia of the pulmonary circulation and volume overload of both ventricles. During ventricular systole, blood backflows from the left ventricle into the left atrium due to mitral valve insufficiency, and then from the left atrium to the right atrium through the ASD. Moderate pulmonary hypertension can be a consequence of both constant hypervolemia in the pulmonary circulation and venous stagnation with small ASD sizes, leading to increased pressure in the left atrium. High pulmonary hypertension develops in the later stages of the disease. In the full form of VKA, hemodynamic disturbances are determined by insufficiency of the atrioventricular valves and shunting of blood through defects of the interatrial and interventricular septa. With this form of the defect, the load on the heart occurs already in utero and can lead to heart failure and fetal hydrops. Due to the large left-right shunt of blood, which again enters the pulmonary artery, volume overload develops in the right and left parts of the heart. The pressure in the right and left ventricles early becomes equal, which leads to the early development of high pulmonary hypertension. Clinic. In the partial form of VKA, clinical symptoms are determined primarily by the degree of mitral regurgitation. With its small size, the defect proceeds like a simple ASD, but manifests itself at an earlier stage.

Diagnostic criteria

I. Clinically

-With significant valve regurgitation, already in the neonatal period, signs of severe heart failure may appear, ARI may occur, with a tendency to a protracted course; children from the first months of life are lagging behind in physical development

-immediately after the birth of the child, rapid fatigue during feeding, sweating, and signs of heart failure increase

-children lag behind in physical development, they experience repeated acute respiratory infections and pneumonia.

- during physical examination, a left-sided cardiac hump, systolic vibration in the fourth intercostal space and/or above the apex of the heart is determined.

- Two murmurs are heard on auscultation: the murmur of relative stenosis of the pulmonary artery in the second intercostal space to the left of the sternum (expulsion murmur) and the systolic murmur of mitral insufficiency at the apex and along the left edge of the sternum.

- in the full form of VKA, the main deterioration occurs at the end of the first month of life, when the resistance of the pulmonary vessels decreases and pulmonary blood flow increases.

– heart sounds are amplified, with the development of high pulmonary hypertension, an accent of the second tone is heard over the pulmonary artery, along the left edge of the sternum there is a rough systolic murmur (ventricular septal defect), a systolic murmur of valvular insufficiency, conducted to the left axillary region. However, the murmur of mitral valve insufficiency may be obscured by the murmur of a VSD. High pulmonary hypertension (Eisenmenger syndrome) develops by the end of the first year of life, and at the end of the second year irreversible changes occur in the blood vessels of the lungs.

II.Paraclinical

1.Instrumental-graphic

An ECG with a partial form of AVK reveals a deviation of the electrical axis to the left, including in newborns. The levogram is most often combined with signs of right ventricular overload. In case of significant mitral valve insufficiency, signs of overload of the left atrium and left ventricle begin to predominate. In the full form of AVK, a deviation of the electrical axis of the heart to the left is detected in combination with severe overloads of both ventricles and the left atrium

2. X-ray - with partial form of AVK, the pulmonary pattern varies; It is characterized by its intensification, both in the arterial and venous beds.

The shape of the heart is different. Severe mitral regurgitation is accompanied by enlargement of the left atrium and left ventricle. With a small degree, signs of enlargement of the right atrium predominate. In the full form of VKA, the pulmonary pattern is significantly enhanced along the arterial bed, and less so along the venous bed. Signs of high pulmonary hypertension may be detected, the shadow of the heart is increased in size due to all the chambers of the heart.

Differential diagnosis of an incomplete form of VKA is carried out with congenital mitral insufficiency, with a complete form of VKA.

The causes of death in VKA are severe heart failure, high pulmonary hypertension, and fatal arrhythmias.

Treatment.

Drug treatment for VKA is aimed at relieving signs of heart failure with the inclusion of digoxin in the treatment regimen, and, if necessary, ACE inhibitors and diuretics. Elective surgical intervention for incomplete VKA is indicated at the age of 1-2 years. In cases of severe mitral insufficiency or common atrium, surgical correction is performed earlier. The correction consists of plastic surgery of the atrial septal defect and reconstruction of the split cusps of the mitral and tricuspid valves.

Surgical treatment for the full form of VKA is possible only at an early age (usually in the first months of life), before the development of Eisenmenger syndrome. The essence of the radical operation is that the common atrioventricular canal is divided into the mitral and tricuspid parts, and the septal defects are closed with one or two patches. Complications. In severe forms of the defect, there is a high probability of rapid development of heart failure, cardiac arrhythmias, and infective endocarditis.

The prognosis for VKA remains unsatisfactory. In the natural course of the defect, up to 27% of patients die during the first six months of life, and 40% die before the end of the first year of life. Over the next four years, another 10% die. In isolated cases, patients live to adulthood, but are severely disabled. After an adequately performed radical operation, with complete AVK in early childhood, repeated operations to reconstruct the atrioventricular valves are required in subsequent years of life. The prognosis is determined by the size of the ASD and the degree of mitral

regurgitation. The prognosis after radical surgical correction for incomplete VKA is satisfactory; patient survival after 20 years is 94%. Residual mitral regurgitation does not affect the prognosis; in rare cases, repeated valve surgery or valve replacement is required. Postoperative mortality in the complete form of VKA is 50%. Appendix 2

Congenital heart defects (Marder, supplemented by K.F. Shiryaeva, 1965)

Hymodynamic disturbance	**Without cyanosis**	**With cyanosis**	**Phase Currents**
Enrichment of the pulmonary circulation	Patent ductus arteriosus, atrial septal defect, ventricular septal defect, atrioventricular communication	Eisenmenger complex, transposition of the great vessels, common truncus arteriosus, left-wing syndrome	Primary adaptation phase Relative compensation phase 3.Terminal phase
Depletion of pulmonary circulation	Isolated pulmonary stenosis	Fallot's disease, tricuspid atresia, complete transposition of vessels with pulmonary artery stenosis, common truncus arteriosus (false), Ebstein's disease	
Depletion of the systemic circulation	Isolated arterial stenosis. Coarctation of the aorta.		
Without hemodynamic disturbances	Dextrocardia, vascular position anomaly, Tolochinov-Roger disease, vascular ring		

The adaptation phase is determined by the time of adaptation of the cardiovascular system and the body as a whole to the conditions of inadequate hemodynamics. Cardiac activity in this phase is characterized by unstable balance and, against the background of increasing physical activity during the child's growth, acute respiratory viral and other infections, circulatory insufficiency (decompensation), which is usually called primary, easily develops.

The most common complications during this phase of the disease are:

1. Circulatory failure (early or primary) I, IIA, IIB, III degrees, most often of mixed right and left ventricular type.
2. Dystrophy (hypoplasia, hypotrophy).
3. Hypostatic pneumonia (heart defects of groups **I** and **III**).
4. Septic endocarditis.
5. Tendency to intercurrent diseases.
6. Early hypertension of the pulmonary circulation (heart defects of group I).
7. Hypoxemic crises (group II heart defects).
8. Collaptoid conditions (group II heart defects),
9. Arrhythmias (extrasystole, paroxysmal tachycardia), complete or partial atrioventricular block.

The adaptation phase continues for 2-3 months in the uncomplicated course, and up to 1.5-2 years in the presence of complications.

The phase of relative compensation occurs due to the inclusion of all compensatory mechanisms of the body, including myocardial hypertrophy. It is characterized by the stability of cardiac activity, which determines satisfactory physical development, resistance to infections, and the ability to implement an age-appropriate motor and household regimen.

Complications of the relative compensation phase:

1. Septic endocarditis.
2. Hypertension of the pulmonary circulation.
3. Relative hypochromic anemia (group II heart defects).

4. Thrombosis of blood vessels in the brain and other organs (group II heart defects).

5. Pulmonary tuberculosis (group II heart defects).

6. Hypoxemic arthritis (group II heart defects).

7. Hemorrhagic syndrome of the vasculitis type (group II heart defects).

8. Hypertension syndrome (headaches, fainting, nosebleeds (group III heart defects).

9. Angina syndrome (group III heart defects).

The duration of the relative compensation phase ranges from several months to 20-30 years. The listed complications usually accelerate the onset of the next stage of the disease.

The terminal phase is caused by the appearance of pronounced dystrophic changes in the myocardium, which are the basis for the development of chronic (late, secondary) circulatory failure.

Circulatory failure is the main complication of the terminal phase of the disease and, depending on the nature of the heart defect, it can be left-, right-, or mixed-type, I, IA, 11B, and III degree of severity. During this phase, any of the complications listed above may develop.

Diagnostic program for detecting congenital heart disease

Minimum

 Collection and analysis of the child’s biological and genealogical history.

 Assessment of anthropometric data.

 Cardiomegaly.

 Organic nature of noise.

 Signs of poor circulation.

-Change A/D.

-FKG

-ECG.

Maximum

 ECHO CT.

 X-ray examination of the heart in 3 projections.

Scheme of clinical observation and rehabilitation of children with congenital heart defects

Frequency of inspection by specialists	When examining, pay attention to:	Additional Research	Main ways of recovery	Duration Observations	Preventive vaccinations
Pediatrician, cardio-rheumatologist in phase I - for severe cases, 1 time per month; in phase II - 2 times a year; in phase 111 - 2-3 times a year. ENT doctor, dentist - 2 times a year. Other specialists - according to indications	Lack of weight gain, fatigue, shortness of breath, cyanosis, pastosity, edema, liver enlargement, dyspnea-cyanotic attacks, heartbeat, pulse, blood pressure, heart size, sound quality of tones, dynamics of heart murmurs, frequency and course of bronchopulmonary diseases, increase in temperature	In phase I: FECG, blood test, urine test - once every 2-3 months, x-ray of the heart - 1 time in 1-1.5 g. In phase II: FECG, general blood and urine test - 2 once a year, x-ray of the heart - once every 2-3 years. In phase III: blood test, urine test, FECG - 3-4 times a year and according to indications	Sanitation of hearth lesions infections. Careful treatment of intercurrent diseases. Agents that improve metabolic processes in the myocardium 1-2 times a year. Diet therapy, cardiac glycosides, vasodilators, diuretics, symptomatic drugs - according to indications depending on the stage of NK. Antiarrhythmic drugs - according to indications. Stay in the fresh air for at least 3-4 hours a day. Sanatorium, local treatment in phase II.	All life. After radical surgery -1-2 years	In phases I and III - contraindicated. In phase II, in the absence of complications and fainting conditions and 2 years after radical surgery, they are indicated with preliminary preparation.

TEST QUESTIONS FOR THE TOPIC "CONGENITAL HEART DEFECTS"

Situational task 1

A 6 month old child was born from the first pregnancy. At the 10th week of pregnancy, the woman suffered from the flu and there was a threat of miscarriage. The child was born at term, weighing 3100 g, length 52 cm, and cried immediately. He was attached to the breast on the first day and sucked sluggishly. Weight gain by month is 400-500 g. Natural feeding. He suffered an acute respiratory infection at 2 and 5 months, and bronchiolitis at 3 months.

Genealogical history: mom has chronic tonsillitis, dad is healthy; on the maternal side: my grandmother has coronary heart disease, my 8-year-old brother has congenital heart disease; on my father's side: my sister had rheumatic heart disease, my grandfather died of stomach cancer, my grandmother had hypertension.

On examination the condition is moderate. The child is lethargic, restless, the skin is icy, and when crying, cyanosis of the nasolabial triangle appears. Reduced subcutaneous fat layer on the limbs and torso. The auricles have an abnormal shape, arachnodactyly. The number of respirations is 40 per minute, puerile breathing, isolated moist, fine, scattered bubbling rales, and on percussion there is a pulmonary sound. The apical impulse in the 4th-5th intercostal space is strengthened. The boundaries of relative cardiac dullness: right - the edge of the sternum, upper - the second intercostal space on the left, left - 2 cm outward from the left midclavicular line. Heart sounds are loud, systole-diastolic murmur over the entire region of the heart with the epicenter in the second intercostal space on the left. from the sternum, carried to the back. The abdomen is soft, the liver is palpable 1 cm below the edge of the costal arch. Urination is free.

Studies were carried out: FCG and x-ray of the heart in 3 projections.

Note the unfavorable factors of the perinatal and postnatal periods.

1. Assess the pedigree.
2. Make a preliminary diagnosis.

3. Indicate the changes on the FCG and X-ray that are characteristic of the suspected pathology.

4. Determine tactics for further observation.

Situational task 2

A 1-year-old child weighing 10 kg was diagnosed with congenital heart disease, ventricular septal defect, primary adaptation phase. Complication - NC IIB.

1. Prescribe drug treatment indicating the dose of drugs.

2. List your recommendations for monitoring the effectiveness of treatment.

Tests

1. All signs are typical for congenital heart disease, except:

1 Dyspnea from birth of inspiratory nature.

1. Normal weight of the child at birth.

2. Small weight gain of the child in the first year of life.

3. Persistent systolic heart murmur.

4. Tendency to respiratory infections.

2. All signs are typical for a patent ductus arteriosus, except:

1. Frequent bronchopulmonary diseases.

2. Strengthening of the first tone at the apex of the heart.

3. Strengthening and splitting of the second tone in the pulmonary artery.

4.Systole-diastolic murmur with an epicenter in the second intercostal space on the left.

5.Expansion of the boundaries of the heart.

3. All signs are typical for a ventricular septal defect except:

1. Recurrent bronchitis, repeated pneumonia

2. Weakening of the first tone at the apex of the heart.

3. Emphasis of the second tone on the pulmonary artery.

4. Systolic trembling in the III-IV intercostal space to the left of the sternum.

5. Pansystolic murmur with an epicenter in the IV intercostal space to the left of the sternum.

4. Specify the leading symptom of aortic coarctation:

1. Asthenic constitution.
2. Sinus tachycardia.
3. Emphasis of the second tone on the pulmonary artery,
4. Absence of pulse in the femoral artery.
5. Lability of blood pressure.

5. Tetralogy of Fallot includes developmental anomalies, except:

1. Aortic stenosis.
2. Ventricular septal defect.
3. Dextraposition of the aorta.
4. Pulmonary aortic stenosis.
5. Right ventricular hypertrophy.

6. For tetralogy of Fallot in the primary adaptation phase, all complications are typical, except:

1. Dyspnea-cyanotic attacks.
2. Frequent bronchopulmonary diseases.
3. Polycythemia.
4. Relative anemia.
5. Delay in psychomotor development.

7. All symptoms are typical for mitral stenosis, except:

1.Acrocyanosis.
2. Shortness of breath.
3. Weakening of the first heart sound.
4. Clapping 1st tone.
5. Diastolic murmur with presystolic amplification.

8. All symptoms are typical for aortic valve insufficiency except:

1 1. The pulse is soft, weakly filled and tense.
1. Pulse is high, falling quickly.
2. Capillary pulse.
3. The phenomenon of infinite tone.
5. Systolic murmur with an epicenter in the 2nd intercostal space to the right of the sternum.

9. Solve the issue of immunoprophylaxis for a child with ASD in the phase of relative compensation:

1. Free from vaccinations.
2. Conduct vaccinations according to general rules,
3. According to an individual schedule.
4. According to epidemiological indications.

Chapter- 3 CARDITIS Carditis

Early congenital carditis

Early congenital carditis occurs in the early fetal period (4-7th month of intrauterine development); its obligatory morphological substrate is fibroelastosis (FE) or elastofibrosis (EF) of the endo- and myocardium.

The diagnosis of congenital carditis is considered reliable if symptoms of cardiac pathology are detected in utero or in the first days of life, probable - if they occur in the first months of the child's life without a previous intercurrent disease or with anamnestic data on the mother's illness during pregnancy.

DIAGNOSTIC CRITERIA

I.Anamnestic:

concentration of cardiovascular diseases in a given family: rheumatism, carditis, congenital heart disease, idiopathic cardiomyopathies, sudden death at a young age, etc.; maternal illnesses during pregnancy; exacerbation of chronic infectious diseases in the mother during pregnancy (rheumatism, pyelonephritis, etc.); decrease in the child's body weight at birth (in 25-30% of patients).

The first symptoms of the disease appear in the first half of life, in children with post-myocardial elastofibrosis - at 6-18 months of life.

I. Clinical:

1**. Extra cardiac:** unmotivated poor weight gain, retardation in physical development, delayed development of static functions, pallor, lethargy, sweating, aphonia, uncontrollable attacks of anxiety.

2. **Cardiac:** moderate cyanosis of the mucous membranes, fingertips; left-sided cardiac hump; the apex beat is weakened or not detected; displacement of the borders of cardiac dullness predominantly to the left; muffled or dull heart sounds; the murmur is often absent or associated with mitral valve insufficiency; tachycardia resistant to treatment. Arrhythmias are rarely observed, only with post-myocardial elastofibrosis. Cardiovascular insufficiency (the most constant sign of carditis), usually total, but with a predominance of the left ventricular: tachycardia, shortness of breath, various wet and dry wheezing, liver enlargement, swelling or pastyness of the legs and feet (signs and stages of heart failure accuracy) (see table).

III Paraclinical:

1. Laboratory:

ESR, leukocytes, serum protein fractions, ASL and AST titers are normal or slightly altered.

2. Instrumental and graphic:

ECG: normal position of the electrical axis of the heart, high voltage of the teeth, rigid frequent rhythm (without arrhythmias and conduction disturbances), left ventricular myocardial hypertrophy with signs of ischemia of its subendocardial parts, narrow QRS complexes, II-III degree blockade or blockade may rarely occur legs and branches of the His bundle, ST interval shift.

The ECG picture with congenital EF of the endomyocardium differs from the ECG with PE: the voltage of the QRS complex remains high, the heart rate and rhythm rigidity decrease with age and against the background of therapy, which indicates a more favorable prognosis; conduction disturbances are common.

3. X-ray:

- the pulmonary pattern is normal or slightly enhanced along the venous bed, atelectasis of the lower lobe of the left lung. Spherical or oval shape of the heart, enlargement of the cavities of the heart with pronounced dilatation of the left ventricle, and to a lesser extent the right.

Signs and stages of heart failure in non-rheumatic carditis in children (N.A. Belokon, 1984)

Stages	Left ventricular failure	Right ventricular failure
I	Heart failure is absent at rest or appears after exercise in the form of tachycardia or shortness of breath	
II_A	Number of heartbeats and respirations per minute. increased by 15-30 and 30-50%, respectively, relative to the norm	The liver protrudes 2-3 cm from under the costal arch
II_B	Number of heartbeats and respirations per minute. increased by 30-50 and 50-70%, respectively, relative to the norm, possible acrocyanosis, obsessive cough, moist intervesicular rales in the lungs	The liver protrudes 3-5 cm from under the costal arch, pastiness, swelling of the neck veins
III	Number of heartbeats and respirations per minute. increased by 50-60 and 70-100%, respectively, relative to the norm, clinical picture of pre-edema and pulmonary edema	Hepatomegaly, edematous syndrome (swelling of the face, legs, hydrothorax, hydropericardium, ascites)

Differential diagnostic ECG signs for congenital EF, post-myocardial EF

Signs	FE	EF
1. Electrical axis of the heart	Normal	Normal or deviated to the left
2.Rhythm and conduction disturbances	Rarely	Often
3. General increase in the voltage of the QRS complex	Much	Moderately
4.Hypertrophy of the left myocardium Ventricle	Always	Always
5.Hypertrophy of the right ventricular myocardium	Rarely	Often
Q wave enlargement	Rarely	Moderate in II, III, aVF, V_5 leads
Negative T wave in leads I, II, III, V5, V6	Often	Happens, but not often
8.Smoothed T wave in leads I, III, V5, V6	Rarely	Often
9.Positive T wave in leads V 1-3	Often	Rarely
10. Downward shift of the ST segment in V5, V6	Often, significantly	Maybe, but more often the T is shifted upward
11. ECG dynamics	Absent, positive negative, less often	Often positive or absent

Differential diagnosis
should be performed with congenital heart disease and acquired carditis.
Diagnosis standard:
early congenital carditis (fibroelastosis), acute course, severe total heart failure stage II B.

Working classification. non-rheumatic carditis in children (N.A. Belokon, 1984)

Period of occurrence of the disease	**Congenital (antenatal) - early and late. Acquired**
Etiological factor	Viral, viral-bacterial, bacterial, parasitic, fungal, yersinia allergic (drug, serum post-vaccination), idiopathic
Form (according to the predominant localization of the process) flow Severity of carditis Form and degree of heart failure	Carditis. Damage to the conduction system of the heart Acute – up to 3 months Acute – up to 18 months Chronic - more than 18 months (recurrent, primary chronic) stagnant, hypertrophic, restrictive options Light, Medium, Heavy Left ventricular I, IIa, IIb, III degree I, IIa, IIb, III degree total
Complication outcomes	Cardiosclerosis, myocardial hypertrophy, rhythm and conduction disturbances, pulmonary hypertension, damage to the valvular apparatus, thromboembolic syndrome, constrictive myocarditis

LATE CONGENITAL CARDITIS

Late congenital carditis is considered to be carditis that occurs in the fetus in the last trimester of pregnancy. The diagnosis is considered reliable if:

a) symptoms of cardiac pathology are detected in utero or in the first days of life;

b) there are no symptoms of fibroelastosis or elastofibrosis.

DIAGNOSTIC CRITERIA

1. Clinical:

Extracardiac: normal (as a rule) birth weight, intrauterine malnutrition is less common; fatigue during breastfeeding; lag in physical development after 3-5 months of life; delay in the development of static functions; frequent respiratory diseases; sweating; changes in the nervous system in the form of an attack of sudden anxiety, shortness of breath, tachycardia, sometimes with loss of consciousness, convulsions; sometimes hoarseness of voice; noisy (stridor) breathing.

2. Cardiac: shortness of breath that has existed since birth, tachycardia or bradycardia; pallor, cyanosis of the mucous membranes and fingertips, especially at the height of heart failure; symptoms of cardiovascular insufficiency; often enlarged spleen; edema syndrome; strengthened rising, displaced downward apical impulse; heart sounds are quite loud; there may be a systolic murmur; rhythm disturbance (characteristic sign).

II. Paraclinical:

1. Instrumental and graphic:

ECG: electrical potentials of the left ventricle, rhythm and conduction disturbances predominate. Shift of the ST segment below the isoline.

2. X-ray:

Normal or trapezoidal heart shape. An increase in the cardiac shadow due to dilatation of all cavities, especially the left ones, a decrease in the amplitude of systole-diastolic oscillations along the contour of the left ventricle.

3. Laboratory:

There are no changes in peripheral blood, rheumatic tests are negative.

Differential diagnosis

should be carried out with congenital heart disease, early congenital carditis, acquired carditis, myocardial dystrophy.

Diagnosis standard:

late congenital carditis (viral), subacute course of moderate severity, heart failure II A total.

Chapter - 4. Acquired carditis diagnostic criteria

I.Anamnestic:

maternal illnesses during pregnancy (especially ARVI), industrial hazards, long-term use of certain medications, alcohol abuse; the first signs of the disease appear against the background of acute respiratory viral infection or 1-2 weeks after it; previous sensitization of the child's body (repeated infectious diseases); the presence of constitutional anomalies (atopic, lymphatic, uric acid diathesis); non-compliance with vaccination rules.

II. Clinical:

1. **Extracardiac:** decreased appetite; delayed or poor weight gain; weakness, sweating, fatigue; irritability, attacks of excitement, sometimes loss of consciousness, convulsions, hemiparesis, restlessness and moaning at night; nausea and vomiting; pallor with a grayish tint to the skin; obsessive cough, worsening with changes in body position.

2. **Cardiac:** heart failure, first left ventricular, and then total; cyanosis of the nasolabial triangle, acrocyanosis; disturbance, rhythm, conductivity; pulse of reduced or satisfactory filling; the apex beat is weakly resistant or undetectable at all; the boundaries of relative cardiac dullness are shifted; muffledness or deafness of the first tone, accent of the second tone over the pulmonary artery; systolic murmur of a functional nature or relative mitral valve insufficiency; A/D may be normal or tend to be hypotensive.

III. Paraclinical:

1. Laboratory:

The results of laboratory tests are not very informative.

2. Instrumental and graphic:

ECG: deviation of the electrical axis of the heart to the right. Reducing the voltage of the QRS complex teeth. Various rhythm and conduction disorders. Changes in the ST segment (displacement below the isoline) and T wave.

3. X-ray:

- pulmonary venous congestion, enlarged heart shadow, dilatation of the left ventricle.

Differential diagnosis

should be carried out with congenital heart disease, congenital carditis, myocardial dystrophy.

Diagnosis standard:

acquired carditis, viral-bacterial, severe, acute course, heart failure II B.

DIAGNOSTIC PROGRAM FOR DETECTING CARDITIS IN CHILDREN

Minimum

- Collection and analysis of biological and genealogical history.
- Assessment of anthropometric data (lag in physical development).
- Identifying signs of heart failure.
- Shifting the boundaries of relative cardiac dullness.
- Changes in heart tones (deafness of the 1st tone, accent of the 2nd tone).
- Presence of systolic murmur.
- ECG, FCG.

Maximum

- ECHO CT.
- X-ray examination of the heart.

ALGORITHM FOR TREATMENT MEASURES FOR CIRCULATORY INSUFFICIENCY

Organizational events	Therapeutic measures	Performance monitoring
	1. Antibacterial and anti-inflammatory drugs, glucocorticosteroids according to indications 2. Cardiac glycosides, depending on the stage of NC in a saturation dose per 1 kg of weight digoxin H II A = 0.03 – 0.05 mg HIIB = 0.075 mg HIII = 0.1 mg	Normalization of clinical and functional state: reduction of changes in the heart, increase in diuresis, disappearance of edema, reduction in liver size. Blood test, PKG, normalization of pulse, disappearance of shortness of breath
1. Clarification of the cause of the disease	3.Selection of saturation rate: fast - 1-3 days medium - 3-5 days slow - 7-10 days	
2. Organization of a regimen and exercise therapy corresponding to the stage of circulatory failure, control of diuresis 3.Organization of rational mechanically and chemically gentle nutrition 4.Organization of monitoring the	4. Upon receipt of a therapeutic effect - transfer to a maintenance dose equal to 1/5 -1/6 dose of nasa 1/6 saturation dose	
	5. Potassium preparations (panangin, asparkam, potassium chloride, etc.) b. Diuretics (Lasix, hypothiazide,	

effectiveness of therapy and identifying side effects of glucocoroids and cardiac glycosides	veroshpiron, etc.) 7. Agents that improve metabolic processes in the myocardium (cocarboxylase, riboxin, ATP, vitamins B5, B-is, Bi2, B6) 8 Peripheral vasodilators (nitrates, hydralazine, phentolamine, sodium nitroprusside, prazosin, etc.). 9. Diet therapy (see below)	

PRINCIPLES OF DIET THERAPY FOR CIRCULATORY INSUFFICIENCY

1. Compliance with a diet according to the stage of NK, taking into account the nature of the underlying disease, the state of the gastrointestinal tract, kidneys, and electrolyte balance.
2. The approach is individual, taking into account the child's wishes and product tolerance.
3. Food must contain vitamins and microelements in sufficient quantities:
- include potassium-rich foods in your diet: potatoes, apricots, dried apricots, prunes, cottage cheese, milk, etc.;
- exclude meat and fish broths, fatty foods, smoked foods, coffee, strong tea, chocolate;
- limit foods that cause fermentation during flatulence: legumes, cabbage, black bread;
- if you are prone to constipation - yogurt, kefir, honey, apricot, beet juice.
4. Increase the number of meals to 4-5 times.
5. For NK I - salt in food in the usual amount, eliminate excess salt;
NK IIA - limit salt to 2-3 g/day, "hepatic" table;
NK IIB-III - "achloride" table: salt in food no more than 1 - 1.5 g, protein reduced to 40 g / day for 3-7 days.
Fasting days - 1-2 times a week until swelling disappears.
Curd and milk day
Cottage cheese - 200.0 -250.0

Milk - 400.0 -500.0
Dried fruit compote -200.0
Fruit day:
Apples (or baked potatoes) – 600.0-800.0
Raisins (dried apricots, prunes) - 150.0 -200.0g
Fruit juice - 200.0 g
6.Water regime depends on the stage of NK
for NKIIA - according to diuresis of the previous day
for NC IIB – III – no more than 600 – 800 ml/day
7. At an early age - reduce the amount of food per feeding, increase the frequency of feedings.
For NC – fractional feeding with expressed breast milk.

Scheme for the treatment of heart failure with glycosides in young children (according to M.P. Chernova)

Stage of heart failure	Cardiac glycoside saturation dose	
	Digitalis	Digoxin
II_A	30 mg/kg for 2 days (2-3 times a day)	0.05 mg/kg in 3 doses every 8 hours (1/2 + 1/4 + 1/4)
II_B	45 mg/kg for 2-3 days (3 times a day)	0.075 mg/kg in 3 divided doses every 8 hours (1/2 + 1/4+ 1/4)
II_B-III_A	60-75 mg/kg for 3 days (3 times a day)	0.1(mg/kg in | 3 divided doses

Note: In case of stage I circulatory failure, preference is given to digoxin; treatment is carried out with maintenance doses without first saturating the body with the drug.

SCHEME OF DISPENSARY OBSERVATION AND REHABILITATION OF CHILDREN AT RISK OF

CIRCULATORY FAILURE (CI) AND WHO HAVE EXPERIENCED CI

Inspection Frequency Specialists	When examining, pay attention to:	Additional Research	Main ways of recovery	Duration of observation	Preventive vaccinations
Threatened by NC: children with congenital heart defects, myocardial fibroselastosis, myocardiopathy in the phase of primary adaptation, relative compensation, who have had non-rheumatic carditis, who are in the inactive phase of the process,					
Pediatrician in the 1st year of life - 2 times a month. Cardio-rheumatologist - once every 2-3 months. Pediatrician-cardio-rheumatologist, ENT doctor, dentist - 2 times a year	Emotional tone, appetite, condition of the subcutaneous fat layer, skin and mucous membrane color, blood pressure, pulse and respiratory rate, presence or absence of peripheral edema, dynamics of changes in the heart, liver condition, diuresis, hematological parameters	General blood test, biochemical test, urine test - once every 6 months. Functional stress tests, FECG - 2 times a year	Proper general hygiene regime, aeration, physical therapy. Diega, enriched with vitamins, is high in calories. Courses of drugs that improve metabolic processes in the myocardium - 1-2 times a year: co-carboxylase, riboxin, ATP, vitamins C, B5, B15, B12, B6, Tsanangin (asparkam), potassium orotate, etc.	For congenital heart disease - after radical surgery after 1.5 years in the absence of signs of complicatio ns.	

THERAPEUTIC DOSES OF CARDIAC GLYCOSIDES FOR YOUNG CHILDREN

Age	Strophant hin mg/kg	Digoxin mg/kg	Isolanide mg/kg	Digitoxin mg/kg	Digitalis mg/kg
Early	0,03	0,075	0,075	0,035	35

TEST TASKS ON THE TOPIC OF CARDITAS

Situational task

The 11-month-old child grew and developed according to his age. On the tenth day after suffering from acute respiratory viral infection, his condition worsened, shortness of breath appeared, and he became lethargic and pale. On examination, the pulse is weak, tachycardia. Borders of the heart: right - along the right parasternal line, left - 3 cm to the left of the midclavicular line. The tones are muted. A short systolic murmur with a gallop rhythm at the apex. There is an abundance of fine-bubbly moist rales in the lungs. The liver protrudes 3 cm from under the costal margin.

The ECG shows a decrease in the voltage of the QRS complex, a violation of atrioventricular and intraventricular conduction, an increase in systolic value, and flattening of the T wave.

Make a diagnosis:

1. Pneumonia.
2. Carditis.
3. Bronchiolitis.
4. Pleurisy.

To clarify the diagnosis you need:

1. History of prenatal development.
2. Information about heredity.
3. Echocardiography and phenocardiography.
4. X-ray of the chest organs.
5. All of the above.

Tests

1. Total cyanosis of the skin and mucous lips in a newborn, persisting for more than 3 hours, can be caused by all of the following conditions, except:

1. Pulmonary pathology.

2. Encephalopathy.

3. Carditis.

4. Congenital heart disease.

2. It is not typical for acquired carditis:

1. Thickening of the nail phalanges of the hands and feet in the form of “drumsticks”.

2. Changing nails in the form of “watch glasses”.

3. Persistent cyanosis.

SCHEME OF DISPENSARY OBSERVATION AND REHABILITATION OF CHILDREN WITH CARDITIS

Frequency of inspections by specialists	Upon examination attention to:	Additional Research	Main ways of recovery	Duration of observation	Preventive vaccinations
After discharge from the hospital, once a month for 3 months, once a quarter for 6-9 months, then once every 6 months. - pediatrician, cardio-rheumatologist, ENT doctor, dentist; when treating with aminocholine drugs - an ophthalmologist once every 3-6 months, other specialists - according to indications	Frequency of intercurrent diseases, fatigue, temperature, signs of circulatory insufficiency, heart size, volume of tones, noises, their dynamics; adequacy of response to physical activity	1. General blood test - once every 3 months, then 2 times a year 2. Blood test for C-reactive protein, protein fractions, sialic acids - 2 times a year 3. General urine test 2 times a year FECG - once every 3 months, then - 2 times every 6 months. X-ray of the heart in 3 projections, bicycle ergometry Functional test according to Shalkoff	Sanitation of hearth lesions infections. Treatment of intercurrent diseases. In the presence of foci of chronic infections - seasonal bicillin prophylaxis for 1-3 years. Seasonal prophylaxis for 4 weeks 2 times a year with non-steroidal drugs in half the dose in combination with cardiotrophic drugs. For protracted and chronic course of carditis - 4-aminoquinoline drugs for 1-2 years	At least 3 years, in case of exacerbation, protracted course of the process - at least 5 years, in case of chronic course - transfer from the age of 15 under the supervision of a doctor in the adolescent office	Contraindicated for a year, further observation period - individually

Chapter -5. Acquired heart defects

Heart disease is an organic lesion of the heart valves, its septa, large vessels and myocardium, which leads to dysfunction of the heart, stagnation of blood in the veins, tissues and organs. There are congenital and acquired heart defects. Acquired heart defects are anomalies and defects of the heart valves, its openings or partitions between chambers, and vessels extending from it, which lead to disruption of intracardiac and systemic hemodynamics and, as a consequence, to the development of acute or chronic circulatory failure. There is no unified classification of acquired heart defects; therefore, for individual heart defects, it is advisable to use the most common and reasonable classifications that take into account the characteristics of the clinical condition, the degree of hemodynamic disorders and indications for surgical treatment.

If, as a result of a pathological process, deformation of the valve tissue occurs and the hole through which blood enters the next part of the heart narrows, then such a defect is called stenosis. Deformation can lead to the heart valves not closing due to changes in shape, shortening them as a result of scarring of the affected tissues; this defect is called insufficiency. Valve insufficiency can be functional, resulting from stretching of the chambers of the heart; the area of the unchanged valve is not enough to close the enlarged hole - the leaflets sag (prolapse).

Causes of acquired heart defects

In 90% of cases in adults and children, acquired defects are a consequence of acute rheumatic fever (rheumatism). Also, the cause of defects can be bacterial endocarditis (damage to the inner lining of the heart due to pathogens entering the blood - sepsis, and their settling on the valves).

In other cases, rare causes in adults are autoimmune diseases (rheumatoid arthritis, systemic scleroderma, etc.), atherosclerosis, coronary heart disease, myocardial infarction, especially with the formation of an extensive post-infarction scar.

The mitral valve is affected more often than the aortic valve. Pathologies of the tricuspid valve and pulmonary valve are less common.

Methods for diagnosing heart defects

In order to establish a diagnosis of heart disease, an anamnesis is collected, the presence of diseases that could lead to deformation of the heart valve is revealed: rheumatic diseases, infectious, inflammatory processes, autoimmune diseases, injuries.

• The patient must be examined to identify the presence of shortness of breath, cyanosis, edema, and pulsation of peripheral veins. Using percussion, the boundaries of the heart are identified and sounds and murmurs in the heart are listened to. The size of the liver and spleen is determined.

• The main method for diagnosing valve pathology is **echocardiography**, which allows you to identify the defect, determine the area of the opening between the atrium and the ventricle, the size of the valves, cardiac fraction, and pressure in the pulmonary artery. More accurate information about the condition of the valves can be obtained by performing **transesophageal echocardiography.**

• Electrocardiography is also used in diagnostics, which allows one to assess the presence of atrial and ventricular hypertrophy and identify signs of overload of the heart. 24-hour Holter ECG monitoring allows identifying abnormalities

• Highly informative methods for diagnosing heart defects are cardiac MRI or cardiac MSCT. Computed tomography scans provide precise and numerous sections, which can be used to accurately diagnose the defect and its type.

• Laboratory tests play an important role in diagnosis, including urine and blood tests, determination of blood sugar, cholesterol levels, and rheumatoid tests. Laboratory tests allow us to identify the cause of the disease, which plays an important role in subsequent treatment and patient behavior.

Classification

Many classifications of acquired defects have been proposed.

• By etiology: rheumatic, atherosclerotic, as a result of bacterial endocarditis, syphilitic, etc.

• according to the severity of the defect, which determines the degree of disturbance of intracardiac hemodynamics: a defect without a

significant effect on intracardiac hemodynamics, moderate and severe severity

• according to the state of general hemodynamics: compensated, subcompensated and decompensated defects

By location of heart damage

Monovalvular defects (one valve is affected)

Mitral valve

Aortic defect

Tricuspid defect

Combined defects (two or more valves are affected)

Two-valve defects

Mitral-aortic disease

Aortic-mitral valve disease

Mitral-tricuspid defect

Aortic-tricuspid disease

Three-valve defects

Aortic-mitral-tricuspid disease

Mitral-aortic-tricuspid disease

According to functional form

Simple vices

Stenosis

Failure

Combined defects - the presence of stenosis and insufficiency of several valves.

Combined - the presence of stenosis and insufficiency on one valve.

Types of defects

Affected valve	Stenosis	Failure
Aortic	Aortic valve stenosis	Aortic valve insufficiency
Mitral	Mitral valve stenosis	mitral valve insufficiency
Tricuspid	Tricuspid valve stenosis	Tricuspid valve insufficiency
Pulmonary valve	Pulmonary valve stenosis	Pulmonary valve insufficiency

Mitral valve insufficiency (mitral regurgitation) is a heart defect characterized by incomplete closure of its valves during left ventricular systole, leading to reverse blood flow (regurgitation) from the left ventricle into the left atrium.

Etiology. The most common cause of organic damage to the mitral valve is rheumatism, much less often – infective endocarditis, atherosclerosis; diffuse connective tissue diseases - systemic lupus erythematosus, systemic scleroderma, dermatomyositis; Cases of defects of traumatic origin have been described.

Pathogenesis. Heart disease is formed as a result of rheumatic endocarditis or valvulitis with the development of fibrosis and deformation of the valves. Rigidity and wrinkling of the valve flaps are formed, leading to incomplete closure of the valve edges. Subsequently, thickening and shortening of the tendon threads and scar formation of the mitral ring occur. In infective endocarditis, pathomorphological changes are characterized by perforation of the valve leaflets or rupture of the chordae tendineae. Atherosclerosis leads to sclerotic changes in the mitral valve and its insufficiency due to thickening and shortening of the leaflets.

Hemodynamic disturbances in mitral valve insufficiency are caused by incomplete closure of the valves during systole, which leads to reverse flow of blood (regurgitation) from the left ventricle into the left atrium. Regurgitation of up to 5 ml of blood with each contraction has virtually no effect on general and intracardiac hemodynamics, up to 10 ml is considered minor, more than 10 ml is considered moderate, and 25–30 ml is considered a severe degree of mitral regurgitation. The volume of blood regurgitation depends on the degree of damage to the valve apparatus and the condition of the heart muscle. There is more blood in the left atrium (the volume normally coming from the pulmonary circulation and the regurgitant volume). During presystole, excess blood enters the left ventricle, causing its dilation and hypertrophy. Violation of intracardiac hemodynamics with mitral insufficiency The main compensatory mechanisms for mitral valve insufficiency: hypertrophy of the left atrium, left ventricle and increase in stroke volume of the left ventricle. With depletion of the left atrium myocardium and a decrease in its compensatory mechanism, the pressure in its cavity increases and retrogradely spreads to the pulmonary veins, which leads to passive (venous) pulmonary hypertension. An increase in pressure in the pulmonary artery is accompanied by hyperfunction, and then the development of hypertrophy of the right ventricular myocardium. With an increase in congestion in the pulmonary circulation, damage to the right ventricle progresses, the contractility of its myocardium decreases, and signs of congestion in the systemic circulation develop.

Clinical manifestations of mitral valve insufficiency.

Subjectively - in the compensation stage there are no complaints; with a decrease in the contractile function of the left ventricle and an increase in pressure in the pulmonary circulation, complaints appear about:

shortness of breath, initially during exercise, and then at rest, palpitations

pain in the heart area of an ischemic nature (due to delayed development of coronary collaterals with myocardial hypertrophy) dry cough

As the symptoms of right ventricular failure increase, swelling in the legs and pain in the right hypochondrium appear (due to enlargement of the liver and stretching of its capsule).

Objectively:

examination: acrocyanosis, swelling of the jugular veins

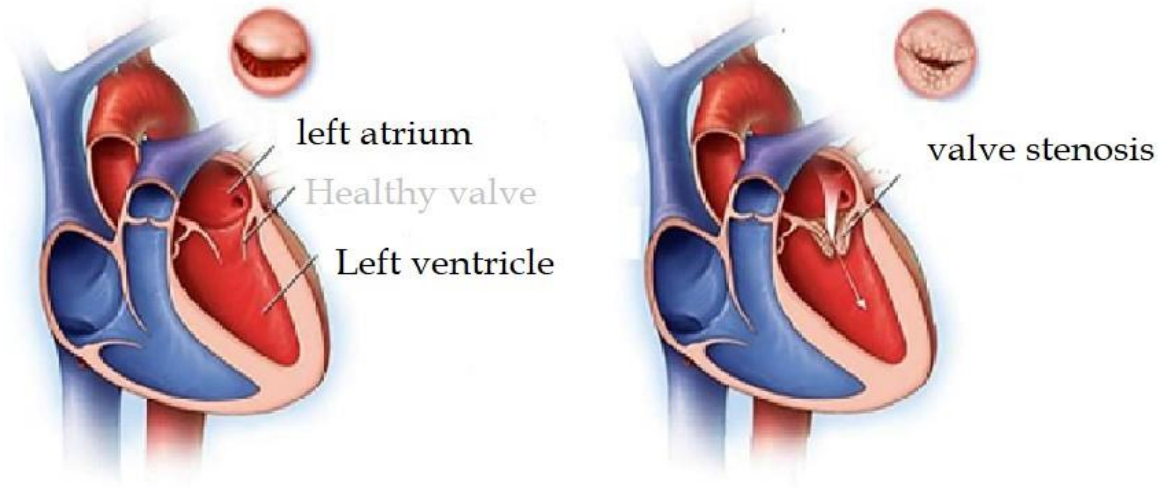
palpation: increased diffuse apical impulse, shifted to the left and often down in the 5th or 6th intercostal space; cardiac hump (with hypertrophy and dilatation of the right ventricle)

percussion: expansion of the relative dullness of the heart, first to the left, up, and then to the right.

Auscultation: the first sound at the apex of the heart is weakened or absent (since there is no "period of closed valves"); systolic murmur at the apex, spreading along the V and VI ribs to the axilla and to Botkin's point and decreasing at the end of systole (arises due to the passage of blood from the left ventricle into the left atrium through a narrow gap between the valve leaflets); emphasis of the second tone over the pulmonary artery and its splitting (due to the rapid slamming of the valves with increasing pressure). Pulse and blood pressure characteristics: no characteristic changes.

X-ray examination: enlargement of the left atrium in the form of smoothness of the contour of the heart or protrusion of its third arch (mitral configuration of the heart); in the left lateral projection – displacement of the contrasted esophagus along a large radius arc by the enlarged left atrium; increased pulmonary pattern (stagnation in the pulmonary circulation).

MITRAL STENOSIS. Mitral stenosis is a narrowing of the left atrioventricular orifice, leading to disruption of the diastolic flow of blood from the left atrium to the left ventricle. There are two possible types of pathological changes leading to narrowing of the left atrioventricular orifice: damage mainly to the valve leaflets in the form of fibrous thickening with the formation of a slit-like mitral orifice (like a "button loop"); damage to the tendon threads in the form of their fusion and shortening (like a "fish mouth"), which leads to impaired mobility of the valve.



Etiology. The main cause of mitral stenosis is rheumatic endocarditis. Very rarely, mitral stenosis occurs due to infective endocarditis.

Pathogenesis Normally in humans, the area of the left atrioventricular orifice is 4–6 cm2. When it is reduced by half, signs of intracardiac hemodynamic disturbances appear, and an area of 1 cm2 is critical. A decrease in the area of the mitral orifice impedes the outflow of blood from the left atrium to the left ventricle (the "first barrier" of intracardiac hemodynamic disturbances) leading to increased pressure in the left atrium and a retrograde increase in pressure in the pulmonary veins and capillaries. When the pressure gradient in the left atrium and pulmonary veins is disturbed, Kitaev's "protective vasoconstrictor reflex" occurs, caused by stimulation of baroreceptors 10 and manifested by a reflex narrowing of the pulmonary arterioles and paradise, manifested by a reflex narrowing of the pulmonary arterioles and the development of active (arterial) pulmonary hypertension. The main compensatory mechanisms for mitral orifice stenosis: left atrial hypertrophy, Kitaev reflex. Functional narrowing of the pulmonary arteries over time leads to the development of smooth muscle proliferation, diffuse sclerosis and narrowing of the pulmonary artery lumen (the "second barrier" to intracardiac hemodynamic disturbances) and the development of passive pulmonary hypertension. An increasing load on the right ventricle leads to its hypertrophy and subsequent dystrophic changes, and an increase in

diastolic pressure leads to progressive dilatation of its cavity and the formation of relative insufficiency of the tricuspid valve, hypertrophy of the right atrium, a decrease in the contractility of the right parts of the heart and the appearance of congestion in the systemic circulation

Clinical manifestations of mitral stenosis

Subjectively - complaints about:

• shortness of breath, first during physical exposure, then during other periods; sharply raise the pressure in a small circle of blood circulation, this may lead to heart failure.

• cough is dry or in the form of a mixture of sputum

• hoarseness (Ortner's sign)

• hemoptysis (in the sputum of solid siderophages - “cells of heart defects”)

• pain in the heart area, palpitations, interruptions; frequently developing atrial fibrillation

• weakness, increased fatigue (as characterized by fixation of cardiac output - lack of an adequate increase in cardiac output in physical conditions).

Objectively:

look: against the background of pale skin, a sharply defined purple “mitral” blush of the cheeks with cyanosis of the lip and tip of the nose (facies mitralis) is revealed; increased epigastric pulsation of the right ventricle (“heartbeat”); absence or weakening of the apex impulse (since the left ventricle is not enlarged in volume and is displaced by the hypertrophied right ventricle)

palpation: when palpating the apex of the heart, especially after physical activity in the position on the left side while exhaling - diastolic tremors (“cat purring” - due to changes in the blood as it passes through the narrowed mitral orifice); symptom of Nesterov’s two hammers - if you place your hand with the palm on the top, with a finger on the area of the II intercostal space to the left of the sternum, then the flapping I is toned by the palm as the first “hammer”, the accented II is toned by the fingers as the second “hammer”; heart hump.

percussion: with the boundaries of the relative dullness of the heart moved upward (due to the counter of the left atrium hypertrophy) and to the right (due to the expansion of the right ventricle), with this boundary, more absolute cardiac dullness is observed than relative (since due to the counter of the expansion of the right ventricle of the heart, spreading the edges lungs, presses against the chest wall of its right enlarged half).

Auscultation: increased (popping) 1st sound above the apex of the heart (in diastole, the left ventricle is not filled with blood sufficiently and contracts rapidly); additional III sound at the apex (the sound of the mitral valve opening; associated with a sharp movement of the mitral valve trunk at the beginning of diastole); I tone + II tone + click of the opening of the mitral valve - a three-part rhythm at the apex of the heart - the “quail” rhythm; Diastolic murmur at the apex of the heart, occurring during different periods of diastole:

protodiastolic murmur - occurring at the beginning of diastole, associated with the movement of blood through a narrowed opening due to the difference in pressure in the atrium and ventricle; decreasing, low rumbling timbre (palpation equivalent - “cat purring”), heard in a limited space, better after physical activity, on the lower side, on exhalation, not performed.

presystolic murmur - occurring at the end of diastole due to active atrial systole; has an increasing character, disappears with atrial fibrillation

characteristics of the pulse and blood pressure: different pulse (Popov's symptom) - the pulse on the left radial artery is weaker than on the right due to compression of the left subclavian artery and the dilated left atrium; Blood pressure is reduced due to cardiac output.

ECG - confirms hypertrophy of the left atrium and right ventricle: hypertrophy of the left atrium: P-mitrale wave in leads I, aVL, V4–V6; right ventricular hypertrophy: deviation of the EOS to the right in combination in the left precordial leads with depression of the S-T interval and a negative T wave in leads avF, III; high R wave in the right chest leads and deep S wave in the left chest leads; 14 depression of the S-T interval and negative T wave in the right precordial leads in case of

progression of pulmonary hypertension. EchoCG: paradoxical movement of the posterior mitral valve leaflet relative to the anterior one; expansion of the cavity of the left atrium (36 mm, normal 17 mm); the cavity of the left ventricle is not changed

An X-ray examination of the heart reveals an enlargement of the left atrium, and calcification of the mitral valve is an important X-ray sign of the defect.

Complications with mitral stenosis occur more often than with other heart defects. Heart rhythm disturbances in the form of atrial fibrillation (more than 50%), hemoptysis (10–20%), thromboembolic complications (15–45%), pulmonary edema.

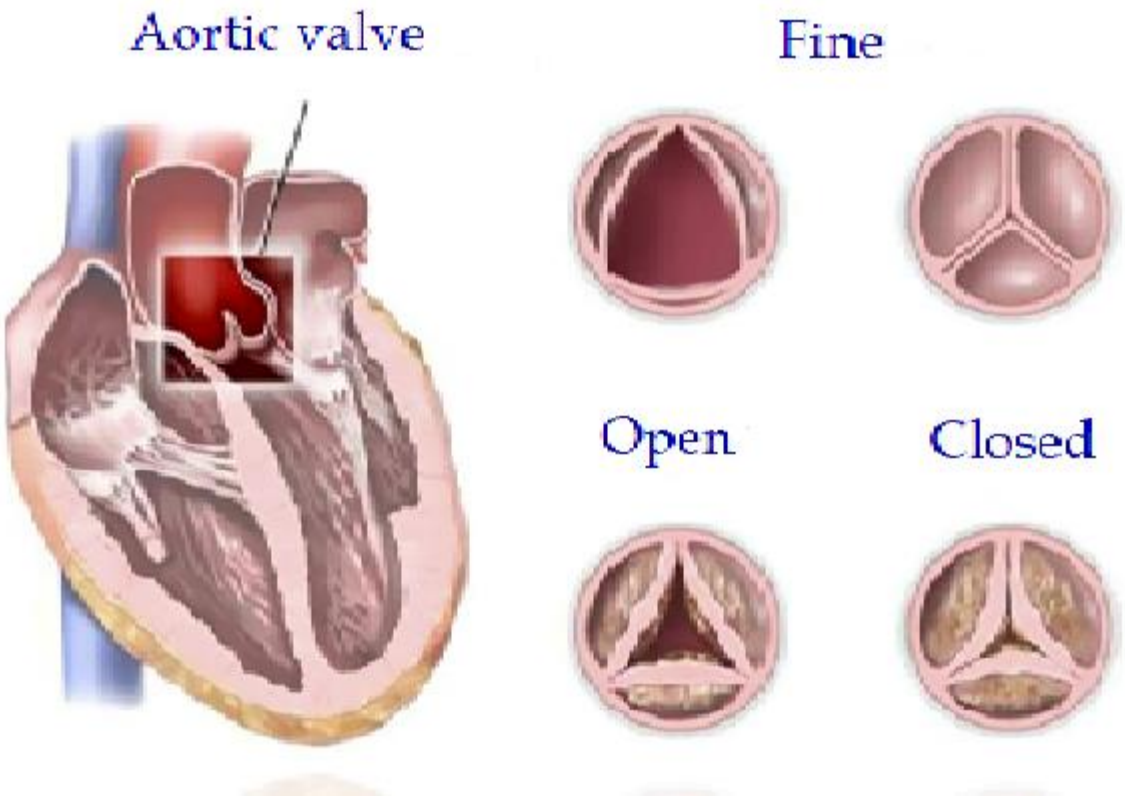
3.3. Stenosis of the aortic mouth (aortic stenosis) is a narrowing of the outflow tract of the left ventricle in the area of the aortic valve, leading to difficulty in the outflow of blood from the left ventricle to the aorta and a sharp increase in the pressure gradient between them.

Etiology. Aortic stenosis can be congenital or acquired due to rheumatism, atherosclerosis, or infective endocarditis. Aortic stenosis is detected in 20–25% of people suffering from heart defects, and in men it occurs 3–4 times more often than in women. Causes of acquired aortic stenosis: rheumatic damage to the valve leaflets (the most common cause is fusion, hardening and rigidity of the valve leaflets); atherosclerosis of the aorta, accompanied by severe sclerosis, degenerative processes, calcification and stiffness of the leaflets and fibrous valve ring; infective endocarditis; rheumatoid arthritis, systemic lupus erythematosus, etc.; primary degenerative changes in the valves followed by their calcification (“idiopathic calcified stenosis of the aortic mouth”); congenital valvular stenosis of the aortic mouth with the formation of a bicuspid aortic valve.

Pathogenesis. Normally, the area of the aortic opening is 3 cm2. With moderate stenosis, no noticeable circulatory disturbances occur. If the area of the aortic opening decreases to 0.5 cm2 or less, hemodynamic disturbances occur: the outflow of blood from the left ventricle to the aorta becomes difficult. With aortic stenosis, the systolic pressure gradient between the left ventricular cavity and the aorta increases significantly (usually exceeding 20 mm Hg, and sometimes reaching 100 mm Hg).

Pressure load increases the function of the left ventricle, causing hypertrophy of its myocardium depending on the degree of narrowing of the aortic opening: reducing it by half (1.5 cm2) is accompanied by severe hemodynamic disturbances, and up to 0.5 cm2 - severe hemodynamic disturbances. End-diastolic pressure remains normal for a long time or slightly increases (up to 10–12 mm Hg) due to impaired relaxation of the hypertrophied left ventricle.

Main hemodynamic changes: concentric hypertrophy of the left ventricle; diastolic dysfunction; fixed stroke volume; coronary perfusion disorders; cardiac decompensation. Narrowing of the aortic mouth and obstruction of blood outflow from the left ventricle lead to a significant increase in the systolic pressure gradient (up to 50 mm Hg or more), an increase in afterload and the development of concentric hypertrophy of the left ventricular myocardium without dilatation of its cavity. For a long time (up to 15–20 years), the defect remains fully compensated. This is an important compensatory mechanism for aortic stenosis. Severe hypertrophy of the left ventricular myocardium is accompanied by an increase in cardiac pressure due to inhibition of active myocardial relaxation and the development of diastolic dysfunction. An increase in diastolic load on the left atrium leads to its hypertrophy - the second compensatory mechanism of aortic stenosis. In the later stages of the defect, cardiac decompensation develops: the contractility of the hypertrophied myocardium of the left ventricle decreases, stroke volume and ejection fraction decrease, its myogenic dilatation develops, the increase in cardiac pressure and systolic dysfunction of the left ventricle. An increase in pressure in the left atrium and veins of the pulmonary circulation is accompanied by the development of left ventricular failure (LVF). In patients with severe left ventricular dysfunction with significant dilatation of the left ventricle, relative mitral valve insufficiency develops ("mitralization" of aortic disease), which further aggravates the signs of blood stagnation in the lungs. If death does not occur within 2–3 years from the onset of LVAD, compensatory hypertrophy of the right ventricle develops, and then its failure (Fig. 4). Rice. 4. Violation of intracardiac hemodynamics in aortic stenosis



Clinical picture of aortic stenosis

The appearance of complaints in patients begins when the area of the aortic mouth decreases by more than half. When complaints appear, this indicates an advanced process, a high degree of stenosis and a high pressure gradient between the left ventricle and the aorta. In this case, treatment should be discussed taking into account surgical correction of the defect.

Subjectively - it is asymptomatic for a long time, the main complaints appear when the aortic opening is narrowed by 2/3 of the norm (less than 0.75 cm2):

compressive pain behind the sternum during physical activity (decreased coronary circulation).

dizziness, fainting (deterioration of cerebral circulation);

Subsequently, with a decrease in the contractile function of the left ventricle, the following appear: attacks of cardiac asthma; shortness of breath at rest; increased fatigue (due to the lack of an adequate increase in cardiac output during physical activity). When congestion occurs in the systemic circulation, patients complain of: swelling of the lower

extremities; pain in the right hypochondrium (associated with liver enlargement and capsule stretching).

Objectively:

examination: pallor of the skin (due to spasm of skin vessels as a reaction to low cardiac output), acrocyanosis (with decompensation), swelling of the lower extremities, swelling of the neck veins, pronounced apical impulse.

palpation: a diffuse "high" resistant apical impulse is palpated, displaced downwards (VI intercostal space) and to the left (to the anterior axillary line); at the Botkin point and especially in the second intercostal space to the right of the sternum (above the aorta), systolic trembling ("cat purring") is often detected, resulting from the turbulence of the blood as it passes through the narrowed aortic opening.

percussion: expansion of the left border of the relative dullness of the heart, an increase in the size of the diameter of the heart (aortic configuration of the heart).

auscultation: the first sound is weakened (due to the slow contraction of the left ventricle and prolongation of systole), the second sound is weakened above the aorta (and if the fused leaflets of the aortic valve are immobilized, it can completely disappear); a rough, increasing-decreasing systolic murmur with an epicenter above the aorta, carried out on the carotid arteries, intensifies when listening to the patient on the right side with breath-holding during exhalation.

characteristics of pulse and blood pressure: pulse is small, slow and rare (blood passes into the aorta slowly and in smaller quantities); systolic blood pressure is reduced, diastolic blood pressure is normal or increased, pulse pressure is reduced.

-X-ray examination: characteristic signs in the form of left ventricular hypertrophy, poststenotic dilatation of the ascending aorta and calcification of the aortic valve.

-ECG indicates left ventricular hypertrophy (increased amplitude of the RV5.6 wave, SV1.2 wave, depression of the S-T interval in leads I, aVL, V4.5) and systolic overload of the left ventricle (negative T wave in

leads V4-6, I, aVL), which is a prognostically unfavorable sign, precedes left ventricular failure.

Complication. Heart failure acts as a clinical symptom and complication of aortic stenosis. It can manifest itself as attacks of angina pectoris and even the development of myocardial infarction. Conduction disturbances are manifested by left bundle branch block. Sudden death is possible with the development of ventricular fibrillation. Results of additional examination

AORTIC INSUFFICIENCY (REGURGITATION) Insufficiency of the aortic valves (aortic insufficiency) is the absence of complete closure of the leaflets of the semilunar valves of the aorta in diastole and the occurrence of reverse blood flow (regurgitation) from the aorta into the cavity of the left ventricle.

Etiology. Among the causes of aortic insufficiency, the leading place is occupied by rheumatism (80%), less often – infective endocarditis, syphilis, atherosclerosis, and diffuse connective tissue diseases. The cause of the defect can be a congenital pathology - Marfan syndrome, as well as a combination of the defect with other congenital defects - coarctation of the aorta, subaortic stenosis, and extremely rarely - traumatic rupture of the leaflet.

Pathogenesis. Violation of intracardiac hemodynamics with aortic valve insufficiency is manifested by regurgitation of blood from the aorta into the left ventricle in diastole. The amount of returned blood is determined by the valve defect, the pressure gradient in the aorta and left ventricle, and the duration of diastole. In cases of severe failure, up to 60% of the systolic blood volume may return to the left ventricle. Compensatory mechanisms. The additional volume of blood entering the left ventricle during diastole is accompanied by its stretching and increased contraction (Frank-Starling law), and hyperfunction of the left ventricle is accompanied by the development of hypertrophy of its myocardium and an increase in systolic output. Tachycardia reduces the volume of regurgitation by shortening diastole, maintaining the minute

blood volume at the proper level. The peripheral compensatory mechanism is a reflex change in arterial tone: during systole, a decrease in total peripheral resistance promotes blood flow, and during diastole its increase maintains sufficient blood pressure. Due to low diastolic pressure in the aorta, the blood supply to the sharply hypertrophied myocardium of the left ventricle decreases. Constant overload of the left ventricle leads to depletion of its reserve capacity, a decrease in its contractility and the development of myogenic dilatation. Severe dilatation of the cavity leads to stretching of the fibrous ring of the left atrioventricular orifice and the formation of relative mitral valve insufficiency - the stage of "mitralization" of aortic disease. As a result of increased pressure in the left atrium and in the pulmonary circulation, the load on the right ventricle increases, which over time leads to the development of right ventricular failure

Clinical picture of aortic insufficiency

Subjectively - in the stage of compensation of the defect, the general state of health is satisfactory, only sometimes patients feel palpitations (due to compensatory tachycardia) and pulsation behind the sternum (due to the movement of an increased volume of blood from the left ventricle to the aorta and back), with decompensation complaints of: pain in areas of the heart with angina pectoris, poorly or not responding to nitroglycerin (caused by relative coronary insufficiency due to myocardial hypertrophy, deterioration of blood supply to the coronary arteries with low diastolic pressure in the aorta and due to compression of the subendocardial layers by excess blood volume)

dizziness, tendency to faint (associated with malnutrition of the brain), shortness of breath, first during physical activity, and then at rest (appears with a decrease in the contractile function of the left ventricle) swelling, heaviness and pain in the right hypochondrium (with the development of right ventricular failure)

Objectively:

examination: pallor of the skin (caused by low blood supply to the arterial system during diastole); pulsation of peripheral arteries - carotid ("carotid dance"), subclavian, brachial, temporal; rhythmic shaking of the head, synchronous with the pulse (Musset's symptom); a rhythmic change in the color of the nail bed and lips with light pressure with a transparent glass on the mucous membrane of the lips, at the end of the nail, the so-called capillary pulse (Quincke's symptom).

palpation: the apical impulse is determined in the sixth, sometimes in the seventh intercostal space, outward from the midclavicular line, diffuse, intensified, lifting, dome-shaped.

percussion: the border of relative cardiac dullness is shifted down and to the left, the diameter of the heart and the width of the vascular bundle are increased (aortic configuration).

Auscultation: the first sound at the apex of the heart is weakened (the mitral valve leaflets close with less amplitude due to the left ventricle being overfilled with blood, the pressure in the cavity of the left ventricle increases slowly in the absence of periods of closed valves); The second tone on the aorta in case of rheumatic disease is weakened, in case of syphilitic and atherosclerotic disease it is sonorous, sometimes intensified and even with a metallic tint;

noises:

organic murmur - a soft, blowing protodiastolic murmur over the aorta, conducted to the apex of the heart; with rheumatic disease, this noise is not rough, it is better heard at the Botkin-Erb point, with syphilitic disease - the noise is rougher, better heard in the second intercostal space on the right

functional murmurs: systolic murmur at the apex (relative mitral valve insufficiency with large dilatation of the left ventricle); diastolic, presystolic Flint murmur (reverse blood flow during diastole from the aorta to the ventricle occurs with considerable force and pushes back the mitral valve leaflet, which creates functional stenosis of the mitral orifice, and during diastole an obstacle is created to the blood flow from the left atrium to the ventricle).

On the femoral artery, a double Traube sound is heard (as a result of vibrations of the vessel wall during systole and during diastole) and a double Vinogradov-Durozier murmur (the first stenotic murmur is caused by blood flow through a vessel narrowed by a stethoscope; the second is due to the acceleration of reverse blood flow towards the heart during diastole time).

characteristics of pulse and blood pressure: pulse is fast, high, large (due to high pulse pressure and increased volume of blood entering the aorta during systole); systolic blood pressure increases, diastolic blood pressure decreases, and pulse pressure increases.

1. X-ray examination: expansion and lengthening of the contour of the left ventricle; the angle between the aortic arch and the left ventricular arch is well defined, deep, the heart takes on a "sitting duck" configuration. the ascending aorta is dilated, its pulsation is deep and rare; sometimes a "yoke" symptom is noted.

2. ECG. Deviation of EOS to the left, signs of left ventricular hypertrophy, rhythm and conduction disturbances (branch block and its branches, AV block, extrasystole). EchoCG. Echocardiography reveals direct signs of aortic insufficiency: diastolic vibration of the anterior mitral valve leaflet, non-closure of the aortic valve leaflets in diastole (a relatively rare sign) - and indirect signs: dilatation of the cavity and hypertrophy of the left ventricular myocardium.

Complications of aortic insufficiency appear as manifestations of the defect at its different stages: angina pectoris, heart failure manifests itself quite early, heart rhythm and conduction are disturbed, and occurs at the stage of failure of both ventricles.

Tricuspid valve insufficiency

Insuficientia valvulae tricuspidalis.

There are organic and relative insufficiency of the tricuspid valve. With organic failure, there are morphological changes in the papillary muscles of the chordae tendineae.

The causes of the development of organic tricuspid insufficiency are: acute rheumatic fever (often in drug addicts); traumatic valve damage (extremely rare); congenital defect (tricuspid insufficiency is combined

with dystopia of the valve into the cavity of the RV, sometimes there is an atrial septal defect - Ebstein's disease). Relative insufficiency of the tricuspid valve develops in most cases secondary to combined mitral-aortic or mitral defects in the stage of their decompensation. Sometimes it can develop in patients with cor pulmonale (wrinkling, shortening)

PATHOGENESIS AND CHANGES IN HEMODYNAMICS

During systole, as a result of incomplete closure of the tricuspid valve leaflets, reverse flow of blood occurs from the pancreatic cavity into the RA. The severity of the defect is determined by the magnitude of regurgitation. The RA receives blood from the pancreas, vena cava and coronary sinus, which leads to its overflow and then to dilatation. Thus, compensation for this defect occurs due to increased work of the right ventricle, which leads to earlier decompensation of the defect like right ventricular failure. A decrease in the contractility of the pancreas leads to a decrease in the volume of blood entering the pulmonary artery, which reduces congestion in the pulmonary vessels.

The main signs of tricuspid valve insufficiency: 1. Puffiness and cyanosis of the face. 2. Sharp swelling and pulsation of the veins of the neck. 3. Palpation reveals pulsation of the liver. 4. Percussion reveals a shift of the border of relative cardiac dullness to the right. 5. Auscultatory changes: − weakening of the first tone at the base of the xiphoid process on the right; - weakening of the second tone over the pulmonary trunk; - systolic murmur, which is heard at the base of the xiphoid process on the right and in the III, IV intercostal space to the right of the sternum; ECG - in leads aVF, III, II, the amplitude of the P wave increases, the P wave is sharpened. With RA dilatation, there is an increase in the time of internal deviation of the positive phase of the P wave in lead V1 (more than 0.04 s). The ECG will reveal the complex complex - right ventricular dilatation syndrome: - low amplitude of the QRS complex in the chest leads; - in leads V1 (V2) rSR - 90 - Rivero's symptom 6. The ECG revealed the heart... During EchoCG, regurgitation of blood is detected through the right at-Corvallo.

TRICUSPIDAL STENOSIS

Etiology

The most common causes of acquired tricuspid valve disease are rheumatism (more than 90%) and carcinoid syndrome (10–50%). Other causes include trauma, tumors of the right atrium, and infective endocarditis (in injection drug users).

Pathological physiology With stenosis of the tricuspid valve opening, the movement of blood from the right atrium to the right ventricle occurs in the presence of a pressure gradient between them, which increases with inspiration, or during exercise and decreases with exhalation. Compensation of blood circulation within certain limits occurs due to expansion and hypertrophy of the right atrium. Subsequently, the relatively weak right atrium rapidly dilates when the mean right atrium pressure exceeds 10 mm Hg. Art., stagnation develops in the systemic circulation with the involvement of the abdominal organs. The early development of venous congestion in the systemic circulation, characteristic of tricuspid valve orifice stenosis, contrasts with the absence of orthopnea and pulmonary congestion.

Clinical picture and diagnosis Patients do not show any particular complaints; shortness of breath often occurs. On examination, swollen jugular veins are noted - one of the first signs of tricuspid stenosis. In most cases, presystolic pulsation of the neck veins is clearly expressed. Venous pressure is sharply increased, arterial hypotension is noted.

Auscultation reveals a characteristic low-amplitude diastolic murmur, most intense at the end of diastole. On the ECG, the characteristic signs of the defect are hypertrophy of the right atrium (high P wave in leads II and III and aVF) with mild RV hypertrophy; nonspecific changes in the final part of the ventricular complex (flattening, inversion of the T wave, decrease in the ST segment) in the precordial leads are possible. Atrial fibrillation is often observed. **X-ray examination** reveals an increase in the size of the right atrium. An echoCG examination reveals a typical picture of orifice stenosis with the presence of a diastolic pressure gradient between the RV and the right

atrium. There are no gradations of the severity of the defect. The average pressure gradient is more than 5 mmHg. Art. is considered clinically significant; if the orifice area is <2.0 cm2, severe tricuspid stenosis is diagnosed. The morphology of the valve and subvalvular apparatus can be determined, as well as the degree of concomitant tricuspid regurgitation. The leaflets are often thickened with reduced mobility due to the inflammatory process along the commissures. During catheterization of the right heart, cardiac output is reduced, pressure in the RV and PA is normal or reduced. The main hemodynamic sign of the defect is the diastolic pressure gradient between the RV and the right atrium.

Treatment There are no specific conservative methods of therapy; developing HF is treated using generally accepted methods. Treatment is aimed at reducing congestion in the systemic circulation and increasing pancreatic filling. The effectiveness of diuretics is limited. It is necessary to prevent the occurrence of infective endocarditis. Surgical treatment is carried out under conditions of artificial circulation, using plastic surgery and valve replacement. Tricuspid valve replacement is performed only in cases of gross changes in the leaflets and subvalvular structures that cause severe stenosis. Preference is given to biological prostheses over mechanical ones due to the high risk of thrombosis. Prognosis The period of compensation for the defect is usually short. Atrial fibrillation occurs early, further disrupting hemodynamics. Tricuspid stenosis is considered one of the most unfavorable acquired heart defects

 Combined defects and combination of pathological conditions

The most common combination is mitral stenosis and mitral insufficiency. With this pathological combination, cyanosis and shortness of breath are noted already in the early stages. Aortic disease is characterized by stenosis and valve insufficiency at the same time, and usually has mild signs of both conditions.

With combined defects, several valves are affected, and each of them can have either isolated pathologies or a combination of them.

Prevention and prognosis for PPS

There are no preventive measures that would protect one hundred percent from acquired heart disease. But there are a number of measures that will reduce the risk of developing heart defects. This means the following:

• timely treatment of infections caused by streptococcus (in particular tonsillitis);

• bicillin prophylaxis in the event of a rheumatic attack;

• taking antibiotics before surgical and dental procedures if there is a risk of infective endocarditis;

• prevention of syphilis, sepsis, rheumatism: sanitation of infectious foci, proper nutrition, work and rest schedule;

•rejection of bad habits;

• presence of moderate physical activity, accessible physical exercises;

• hardening.

The prognosis for the life and ability to work of people with heart defects depends on the general condition, fitness of the person, and physical endurance. If there are no symptoms of decompensation, a person can live and work as usual. If circulatory failure develops, work should either be lightened or stopped; sanatorium treatment at specialized resorts is indicated.

It is necessary to be observed by a cardiologist in order to monitor the dynamics of the process and, as the disease progresses, to promptly determine the indications for cardiac surgical treatment of heart disease.

Treatment of acquired heart defects can be conservative or surgical

Conservative treatment is effective only in the early stages of the development of heart disease and requires mandatory follow-up by a cardiologist.

PPS should be treated surgically when:

• Heart failure progresses.

• Pathological changes in the valve significantly affect hemodynamics.

• Conservative therapy does not have the desired effect.

• And there are fears of serious complications.

Types of operations for heart defects

Open heart surgery is performed under artificial circulation through a median sternotomy. Median sternotomy creates optimal working conditions for a cardiac surgeon - performing the necessary surgical interventions for various pathologies and connecting a heart-lung machine. The soft tissue incision is approximately equal in length to the length of the sternum (about 20 cm), and the sternum is dissected along its entire length.

The main two types of operations that are currently used for PPS are reconstruction of the affected valves (plastic) or their prosthetics.

Valve-sparing surgery

Performed to eliminate the cause of valve dysfunction.

If the valves do not close (valve insufficiency), then the cardiac surgeon during the operation achieves normalization of the closure of the valve leaflets, performing resection of the valve leaflets, annuloplasty, commissural plastic surgery, and chord replacement. If there is valve stenosis, then the sections of the valves that have fused due to a pathological process are separated and an open commissurotomy is performed.

Valve replacement surgery

If it is impossible to perform plastic surgery, when there are no conditions for this, valve replacement operations for prosthetic heart valves are performed. In case of intervention on the mitral valve, replacement is performed with full or partial preservation of the anterior or posterior valve leaflets, and if impossible, without their preservation.

In valve replacement surgeries, prostheses are used.

• Prosthetics can be made from animal or human tissue. Such prostheses are called biological. Its main advantage is that the patient does not need to take anticoagulant drugs during subsequent years of life, and their main disadvantage is their limited service life (10-15 years).

• Prostheses consisting entirely of mechanical elements (titanium and pyrolytic carbon) are called mechanical. They are very reliable and can

serve trouble-free for many years, without replacement, but after such an operation the patient must always take anticoagulants for life, this is a negative aspect of using a mechanical prosthesis.

Minimally invasive surgeries

Modern surgery, thanks to the creation of new instruments, has the opportunity to modify surgical approaches to the heart, which leads to operations becoming minimally traumatic for the patient.

The point of such operations is that access to the heart is carried out through small incisions in the skin. During minimally invasive operations on the mitral valve, a right-sided lateral minithoracotomy is performed, with a skin incision of no more than 5 cm, this allows one to completely avoid dissecting the sternum and provides convenient access to the heart. To improve visualization, endoscopic video support with multiple magnification is used. With minimally invasive access to the aortic valve, the skin incision is approximately half as large (incision length 8 cm), and the sternum is incised lengthwise in its upper part. The advantage of this method is that the uncut portion of the sternum provides greater stability after surgery, as well as a better cosmetic effect by reducing the size of the suture.

Endovascular operations - transcatheter aortic valve replacement (TAVI).

Methods of transcatheter aortic valve implantation:

• The entire operation is carried out through a blood vessel (femoral or subclavian artery). The meaning of the procedure is to puncture the femoral or subclavian artery with a guide catheter and deliver the stent valve against the blood flow to the site of its implantation (aortic root).

• Through the aorta. The essence of the method is to dissect the sternum over a short distance (ministernotomy) and puncture the aortic wall in the ascending section and implantation of a stent valve into the aortic root. The method is used when it is impossible to deliver the valve through the femoral and subclavian arteries, as well as when there is a pronounced bend in the arterial arch.

• Through the apex of the heart. The meaning of the procedure is to make a small incision in the fifth intercostal space on the left

(minithoracotomy), puncture the apex of the heart with a guide catheter and install a stent valve. Once the new valve is implanted, the catheter is removed. The new valve starts working immediately.

There are two types of stent valves:

• Self-expanding stent-valve expands to the desired size after removing the restrictive sleeve sleeve from it.

• Balloon-dilatable stent valve, which expands to the desired size when the balloon is inflated; After final installation of the stent valve, the balloon is deflated and removed.

To determine whether TAVI surgery is appropriate, the patient must undergo a series of tests, including an ECG, echocardiography, computed tomography (CT) scan, and angiography.

Currently, the TAVI procedure is increasingly used not only for aortic stenosis, but also for aortic insufficiency, as well as their combination. In addition, TAVI surgery is used for dysfunction of the biological prosthetic aortic valve.

TAVI surgery is performed under general anesthesia and requires a multidisciplinary approach. The procedure is performed by a specialized team, which includes an interventional cardiologist, cardiac surgeon, anesthesiologist, and radiologist.

The presence of a stent valve is not an indication for the patient to take the indirect anticoagulant Warfarin (in the absence of other indications).

TEST TASKS

Situational tasks

Task No. 1

The child is 6 months old. Complaints upon admission were lethargy, poor weight gain, shortness of breath and excessive sweating. These complaints are almost from birth.

From the anamnesis: from 1st pregnancy, in the 7th week of which the woman suffered from influenza. The birth was urgent, birth weight 3000 g, height 51 cm. Attached to the breast on the first day, suckled sluggishly, rested often. Weight gain by month is 300-400 g. At the age of 2 and 5 months he suffered from acute respiratory viral infection, and at 3 months he suffered from obstructive bronchitis.

Genealogical history: mother has chronic decompensated tonsillitis, father is healthy. On the maternal side: my uncle has congenital heart disease, my grandmother has coronary artery disease. On the paternal side: my grandmother has hypertension.

Objectively: the condition is of moderate severity. The child is lethargic, pale skin, high humidity, and when crying there is cyanosis of the nasolabial triangle. The subcutaneous fat layer is poorly developed. There are stigmas of disembryogenesis - abnormal ears, syndactyly of the 4th and 5th toes. The number of breaths per minute is 40, puerile breathing in the lungs, isolated moist rales. The chest is deformed, there is a bulge in the heart area to the left of the sternum. The apical impulse is strengthened and diffuse. The borders of the heart are expanded to the left. A rough systole-diastolic murmur is heard over the entire area of the heart. P.max in the 2nd intercostal space on the left, carried out into the axillary region and onto the back. The tones are rhythmic and loud. The abdomen is soft, painless, liver + 2.5-3 cm from under the rib, sensitive to palpation. Urination is free. The child underwent: ECG, Doppler-EchoCS.

Exercise:

•Note the unfavorable factors of the pre- and postnatal periods.

•Assess the pedigree.

•Make a preliminary diagnosis.

•Indicate changes in ECG and Doppler-EchoCS characteristic of preliminary diagnosis.

•Further tactics of patient management.

Task No. 2

• The child is 2 years old. The mother complains of constant shortness of breath, which sharply increases with the slightest physical exertion, cyanosis of the skin, and delayed physical and motor development.

Similar complaints have been a concern since 3.5-4 months. Repeatedly

was examined in a hospital and received conservative treatment. Over the past 2 months, the child began to experience attacks of a sharp deterioration in his condition, for which he was hospitalized. In the morning, after taking blood for clinical analysis, the child suddenly became restless, moaning, crying, shortness of breath and cyanosis sharply increased. Lies on his side with his legs brought to his stomach.

Auscultation – loud heart sounds, tachycardia. Rude

The systolic murmur in the 2nd intercostal space on the left, which was always present, ceased to be heard. After 2-3 minutes, shortness of breath began to decrease, and then the intensity of cyanosis. A rough systolic murmur over the pulmonary artery began to be heard again. After 10 minutes, the state became the same as before the attack, but remained lethargic.

Exercise:

1. What diagnosis does the child have?
2. What condition did he develop?
3. What causes the development of this condition?
4. How can you explain the patient’s posture during an attack?
5. What explains the indicated auscultatory pattern during seizure?
6. How can you prevent the development of such conditions?

Task No. 3

A boy, 8 years old, was admitted to the cardiology department with

complaints of weakness, fever, excessive sweating, pain in the legs. I was sick for about four weeks, had a sore throat, was treated at home, my condition improved and attended school for a week. But weakness increased, loss of appetite, the temperature began to rise to 38.5 - 39 ° C, its drop was accompanied by profuse sweating. The child lost weight. The local doctor listened to the heart murmur and sent him to the hospital. An objective examination of the child revealed that the boy was malnourished. The skin is clear, with an earthy-gray tint, “shadows” under the eyes, isolated petechial rashes on the legs, near the collarbones. Arthralgia in the left knee and right elbow joints without any visible changes. The borders of the heart are expanded 1 cm to the left. The tones are rhythmic, muffled, tachycardia. In the second intercostal space on the right, a soft, “flowing” diastolic murmur is heard along the sternum. The liver and spleen are not enlarged. Physiological effects are unremarkable.

• The examination revealed: blood test Hb 80 g/l, er.-3.3x1012/l, CP 1.0, L-22.4x109/l, p/i-10, s-65, l-13, m-12, ESR-26 mm/hour. SRP ++, RF – negative, seromucoids-0.6; Ig A 3.0 g/l, Ig M 0.99, G 18.9; No LE cells were detected. CEC 95. Hemoculture - viridans streptococcus, sensitive to penicillin, oxacillin, streptomycin, was inoculated.

• On the ECG - manifestations of hypoxia against the background of pronounced metabolic

violations.

• EchoCG – hyperechogenicity of the aortic valve leaflets, on the coronary leaflet

vegetation is determined to be 1.5-2 mm.

Question

1.What is your clinical diagnosis?

2.Principles of treatment of infectious diseases

endocarditis.

Standards of answers to situational problems

Task No. 1.

1. Viral infection suffered in the 1st trimester of pregnancy

infection, sluggish sucking, small weight gain by month,

frequent bronchopulmonary diseases.

2. Heredity is burdened with cardiovascular pathology

in 2 and 3 generations, incl. according to the UPS.

3. Diagnosis: congenital heart disease with enrichment of the pulmonary circulation,

pale type – open aorterial duct, primary phase

adaptation, CHF II degrees a-b. BEN II degree.

4. ECG - levogram, LA and LV hypertrophy with increasing pulmonary hypertension - rightogram, RA and RV hypertrophy. Doppler-EchoCS - enlargement of the left cavities of the heart, then the right, systolic flow in the pulmonary artery, increase in MPAP (average pressure in the PA)

above 30 mmHg.

5. Before examining the child (except for general clinical tests), an X-ray of the chest organs (direct and left lateral images) with contrast of the esophagus should be performed in order to assess the condition of the vessels of the ICC, the size of the cavities of the heart, identifying

systolic or diastolic overload. Finally, a consultation with a cardiac surgeon.

6. Diagnosis of congenital heart disease: PDA in a child after 6 months is

indication for surgical treatment. But in this case, the child has CHF - degree IIa-b, which has not yet been treated conservatively.

Therefore, it is advisable to carry out a full range of therapeutic treatment (select a maintenance dose of digoxin, ACE inhibitors (Capoten), give diuretics (furosemide + veroshpiron), cardiotrophics (Elcar or Mildronate)). After relieving the symptoms of CHF, improving the condition, increasing body weight, surgical

correction of the defect. If conservative therapy is not effective, surgery for health reasons at any age.

Task No. 2.

1. Diagnosis of congenital heart disease with depletion of the pulmonary circulation, with cyanosis -

Tetralogy of Fallot.

2. The child developed a dyspnea-cyanotic attack.

3. The attack develops as a result of a spasm of the outflow tract of the pancreas, which

happens reflexively. Provoking factors can be physical activity, psychogenic effects, fever, etc.

4. Mechanical compression of the abdominal aorta leads to an increase in peripheral resistance in the vessels of the systemic circle, therefore, discharge into the aorta decreases and blood flow in the pulmonary circle increases.

5. During an attack, the stenotic pulmonary artery, the outflow tract of the pancreas, spasms and this turns off the blood flow in the pulmonary circulation.

Hence the disappearance of stenotic noise during an attack and its reappearance at the end of the attack.

6. In order to prevent attacks, a b-adrenergic blocker is prescribed - anaprilin at a dose of 0.5-1 mg/kg/day in 3 doses for 3-6 months, with gradual

cancellation.

Task No. 3

1.Primary infective endocarditis, infectious-toxic phase, stage II activity, myocarditis, aortic valve valvulitis type

insufficiency, acute course, HF degree I.

2.A) Parenteral administration of high doses of antibiotics

B) The choice of a bactericidal antibiotic, since the pathogen inside the vegetation is protected from immune factors, a/b with bacteriostatic

action is not enough to eliminate it. Beta-lactam a/b (penicillins and cephalosporins, or vancomycin) are more often used. If the causative agent is viridans streptococcus or enterococcus, then gentamicin must be added.

C) Doses of a/b should be high and exceed the minimum bactericidal concentration.

D) With any a/b therapy regimen, the duration of treatment should be at least 6 weeks.

Clinical case No. 1

A mother and her 7-year-old daughter Madina came to see you. Mom says that for the last two weeks the girl has been complaining of palpitations, a feeling of a sinking heart, rapid fatigue, poor appetite, and abdominal pain.

Question 1. What possible reasons can you name for this girl's condition?

Complaints may be associated with myocarditis, congenital heart disease, heart failure, anemia, and helminthic infestation.

Question 2. What from the anamnesis interests you?

How did the mother's pregnancy proceed, namely: infections suffered during pregnancy - influenza, rubella, etc., especially in the 1st trimester (she was not sick with anything), did she take any medications (no), did she abuse alcohol during pregnancy (no), smokes (no), does the girl's mother have chronic diseases – SLE, diabetes mellitus (no).

How was the birth? (Fine). Birth weight (2800), full term. She screamed immediately. Attached to the breast immediately after birth. She took the breast and sucked actively. Vaccinations? (I received all the necessary vaccinations at the maternity hospital)

How did the girl develop? Was there any weight loss? (Mom says that Madina was not plump. But she did not fall behind in weight either). Vaccinations by age? (Yes)

Do you often get sick or catch a cold? Past illnesses? (like all children, they catch a cold or the flu 1-2 times a year).

Has Madina been ill with anything in the last 2 months? (Yes. The last time I had the flu very badly. I had a high fever for a long time. I had to take antibiotics. It's been a month since we seem to have recovered from the flu, but Madina is still lethargic, doesn't eat well, and gets tired).

Question 3. What will you look for during a clinical examination?

VVP at rest: pulse (150), respiration (32-34), blood pressure (90/60), temperature (37.4)

Weight – (25 kg).

Other signs of external examination: weight loss (no), cyanosis (no), pallor of the skin and mucous membranes (yes), drumsticks (no), swelling in the legs (no), visible cervical pulsation (no). L/s are not palpable.

Inspection, palpation, percussion, auscultation of the chest: cardiac hump (no), visible displacement or expansion of the area of the apical impulse (yes, not pronounced), systolic tremors (no). Percussion: (the left border of relative dullness is 1.5 cm outward from the midclavicular line, i.e. expanded). Auscultation: (unpronounced systolic murmur in the IV intercostal space, muffled heart sounds, tachycardia)

Examination of the abdominal area: liver (painless, at the edge of the costal arch), spleen (not palpable).

Question 4. What did you suspect? And your tactics?

Acute non-rheumatic carditis, possibly of viral origin. Madina should be referred to a cardiologist at the Central Children's Hospital to confirm the diagnosis and possibly resolve the issue of inpatient treatment.

Question 5. What necessary laboratory and instrumental studies can you do in your SP/SVP/SSP before sending the girl for a consultation with a cardiologist at the Central Children's Hospital?

General blood test, general urine test, general stool test and stool test for worm eggs and Giardia cysts, C-reactive protein, ECG.

Clinical case No. 2

You are visiting a mother and her 9-month-old son Abduvahid. They came for the next medical examination and vaccination. Abduvahid has been registered as “D” with a diagnosis of Non-rheumatic carditis since the age of 3 months. Weight at the time of the visit – 8800 g, height 71 cm. At rest: RR 32/min and pulse 130/min. At 3 months: weight – 6000 g, height – 53 cm. At 6 months: weight – 7500 g, height – 68 cm. From the anamnesis: exclusively breastfed for up to 6 months. Now he receives complementary foods in the form of porridge, vegetable puree, meat, etc. In the maternity hospital, Abduvahid received HBV1, OPV0 and BCG1. Then, due to myocarditis, Abduvahid was given a medical exemption from vaccinations for an indefinite period. At the time of examination: the

skin is clean, there is no cyanosis. Auscultation: no noise, heart sounds are clear and rhythmic. There is vesicular breathing in the lungs, no wheezing. The abdomen is soft, b/w. The liver is at the edge of the costal arch, palpated b/w. Stool and urination are normal. KBC – Hb – 115 g/l, ESR – 5 mm/h, leukocytes – 4.5x10. ECG – heart rate 130 per minute, sinus rhythm, regular. Chest X-ray – unremarkable. Psychomotor development: sits down, says “dad”, “mom”, expresses desires, takes objects with two fingers.

Question 1. Does the child have symptoms of decompensation/heart failure of non-rheumatic carditis? (No). If yes, which ones? (no symptoms of decompensation)

Question 2. Will you vaccinate your child again? (Yes)

Question 3. What vaccination(s) is Abduvahid subject to? ((penta-1(DPT-1,HBV-2,HIB.-1) OPV-1, Rota-1 (oral rotavirus) PNEUMO-1))

Question 4. What could be a contraindication for Abduvahid to be vaccinated? (symptoms of decompensation - heart failure or exacerbation of myocarditis). Question 5. List the symptoms of exacerbation of myocarditis, which would be a contraindication to routine immunization in Abduvahid.

(Fever, ↑pulse/or heart rate, ↑RR - shortness of breath, cyanosis, wheezing in the lower parts of the lungs, enlarged liver, lack of weight gain, retardation in psychomotor development, moody, restless, refuses to eat. Changes in laboratory and instrumental parameters) .

Clinical case No. 3.

Azat, 13 years old, came to you for an appointment at the joint venture. He needs a medical certificate to participate in the sports section of the USU. At the time of the examination, Azat makes no complaints. Goes to school. After reviewing the outpatient card, the GP discovered that Azat suffered from rheumatic fever at the age of 11 years. Over the past two years, I have not seen a doctor or been to the hospital. According to Azat, their family lived in Russia, where he once had an echocardiogram, which indicated the diagnosis: PPS. Mitral valve insufficiency (MV) 1st degree.

Question 1. Before issuing a certificate to Azat, what should you do as a GP? (collect complaints, data from the medical history, conduct a clinical examination)

2 Question. Azat has no complaints. What data from the anamnesis are you interested in?

repeated rheumatic attacks (no)

bicillin prophylaxis - seasonal or year-round (yes, bicillin-3, 600 thousand once a week for 6 weeks) reaction to bicillin (no) sore throats - pharyngitis, tonsillitis, how often (flu only or colds 2 times) whether it was registered as “D” in the clinic, was it observed by a cardio-rheumatologist at the place of residence (yes) results of laboratory and instrumental studies (ECHOCG only)

Question 3. What will you look for during a clinical examination? (for symptoms of decompensation of NMC - symptoms of heart failure - shortness of breath, tachycardia, weight loss, fatigue; it is also important to recognize the symptoms of repeated rheumatic attack - temperature, articular syndrome, chorea, rash, expansion of the borders of the heart, the appearance of a new cardiac murmur).

Question 4. During the clinical examination, you did not find any symptoms of decompensation of the urinary tract and repeated rheumatic attacks. Will you give Azat a certificate for practicing Wushu sports? (probably not. Since Azat should conduct laboratory tests - UBC, OAM, C-reactive protein; and instrumental studies - ECG, chest x-ray)

Question 5. All results of Azat’s laboratory and instrumental studies are within normal limits. What's your tactic? (I will register Azat as “D”, routinely refer him for a consultation with a cardio-rheumatologist, give recommendations for the prevention of a recurrent rheumatic attack and issue a certificate for playing Wushu sports).

Clinical case No. 4.

The GP received a call to the house of a girl named Arina, 8 years old. Complaints of fever, headaches, lethargy, and volatile joint pain for 3 days. From the anamnesis: three weeks ago Arina had follicular tonsillitis. She took azithromycin for 4 days. She felt better. Arina stopped taking

azithromycin and went to school. On examination: t0 37.6, pulse 110/min, respiratory rate 22/min. The skin is pale, the throat is clean, hyperemic. The GP saw that the knee, ankle and elbow joints were swollen, hyperemic, and hot to the touch. Movements are severely limited. Heart sounds: tachycardia, muffled. A soft, non-rough systolic murmur at the apex of the heart, the intensity of which decreases when moving to a vertical position. The left border of the heart is 1.5 cm outward from the nipple line. The abdomen is soft, b/w. Stool and urination are regular, b/w.

Question 1. What did you suspect about Arina and what should you differentiate from? (rheumatic fever. Differential diagnosis should be carried out with PPS NMC, juvenile rheumatoid arthritis, reactive arthritis, diffuse connective tissue diseases)

Question 2. What do you think is the reason for the development of rheumatic fever in Arina? (a sore throat suffered 3 weeks ago, the causative agent of which was group A streptococcus. Also, Arina did not completely finish treatment of the sore throat with an antibiotic. The course of treatment should be up to 10 days)

Question 3. List Arina's symptoms that indicate rheumatic fever? (polyarthritis/polyarthralgia - knee, ankle and elbow joints are swollen, hyperemic, hot to the touch, movements are severely limited; myocarditis - tachycardia, muffled tones, systolic murmur at the apex; connection with previous streptococcal disease).

Question 4. Cardiac symptoms indicate rheumomyocarditis (why?) or formed PPS (why)?

(Cardiac symptoms indicate rheumyocarditis. Because for the formation of PPS, at least one year is necessary after a severe rheumatic attack or repeated rheumatic attacks, when the endocardium - the valvular apparatus of the heart - is involved in the lesion).

Question 5. Of course, the GP will refer Arina to the hospital for treatment. What is the GP's further tactics after Arina's discharge from the hospital? (timely registration for "D", drawing up an observation plan together with a cardio-rheumatologist, regular monitoring of bicillin prophylaxis, aspirin intake, laboratory and instrumental studies, timely detection and treatment of bacterial pharyngitis, tonsillitis)

Clinical case No. 5.

You are a GP and you are conducting a health examination at school. During a medical examination of 12-year-old Ilyas, auscultation of the chest revealed a heart murmur. The murmur is systolic, of moderate intensity, blowing, with the sound epicenter in the V intercostal space, radiating to the axillary region. At the apex there is a weakening of the first tone. Percussion: the left border is in the 5th intercostal space 1 cm outward from the nipple line. Weight – 33 kg. Height – 143 cm. When asked about complaints, Ilyas replied that lately he has not been able to perform the required load in physical education lessons and gets tired quickly.

Question 1. Does Ilyas have functional or organic noise? (organic). List the signs confirming that Ilyas's murmur is organic (systolic murmur, moderate intensity, blowing, with the sound epicenter in the V intercostal space, radiating to the axillary region. At the apex there is a weakening of the 1st tone. Percussion expansion of the border of the heart to the left, retardation in physical development - lower weight moderate, exercise intolerance).

Question 2. What do you suspect of Ilyas having congenital heart disease (which one?) or rheumatic PPS (which one?) (Mitral valve insufficiency). What data from the anamnesis will help you differentiate PPS from congenital heart disease? (history of chronic tonsillitis, history of follicular tonsillitis, rheumatic fever, course of pregnancy in the mother, developmental features in early, preschool age, history of frequent colds)

Question 3. When collecting anamnesis, you found out that Ilyas did not suffer from sore throats and was not in the hospital for rheumatism. He does not remember that he once had the symptoms of rheumatic fever you listed. What course of rheumatic fever did you think about? Why? (about latent course, when there is no active phase of rheumatic fever and there has not been in the past, there is no rheumatic history. Heart disease is determined immediately and more often it is mitral valve insufficiency).

Question 4. What is your tactic? (register Ilyas as “D” and immediately send him for a consultation with a cardio-rheumatologist to confirm the diagnosis and draw up a joint plan for observation and year-round bicillin prophylaxis)

Question 5. What kind of drug is bicillin? What is the purpose of bicillin prophylaxis? (this is the use of long-acting penicillin preparations for the purpose of secondary prevention of rheumatism, namely suppressing streptococcal infection, reducing relapses of rheumatic fever, stopping the rheumatic process and preventing the formation of PPS)

Clinical case No. 6

A mother with a 2-year-old girl, Shokhsanam, contacted you. She has already been diagnosed with Non-rheumatic carditis. Mom is worried that her daughter has not received a single vaccination and wants her to start at least some vaccinations. Results from regular monitoring of growth and development indicate no weight gain. Upon examination, the girl looks pale, thin, walks unsteadily, does not run, has a vocabulary of no more than 4 words, and draws doodles. History of frequent respiratory tract infections, pneumonia. On examination: RR – 34 at rest, pulse 140 per minute. Auscultation: muffled heart sounds, systolic murmur in the 4th intercostal space. The liver protrudes 1.5 cm from under the edge of the costal arch. Receives digoxin prescribed by a cardiologist in a maintenance dose once a day. In the Shokhsanam family they feed food from the family table.

Question 1. The child has not yet received any vaccinations in his medical history. Will you start vaccinating Shokhsanam? If yes, why? If not, why (no, you should not start vaccinations, because Shokhsanam has contraindications to vaccinations in the form of symptoms of decompensation of non-rheumatic myocarditis - heart failure).

Question 2. What symptoms of decompensation do you notice in Shokhsanam and are they contraindications to vaccinations? (tachycardia, shortness of breath - RR is higher than the age norm, liver enlargement, takes digoxin, lags in growth and development).

Question 3. What is your tactic as a GP in this case? (mandatory referral for hospitalization to the cardiology department)

Question 4. After being discharged from the hospital, you visited Shokhsanam at home. The girl's condition is satisfactory. There is no shortness of breath. Pulse is age appropriate. The liver is at the edge of the costal arch. Shokhsanam gained 800 grams in weight. What will you include in your child's follow-up plan? (monthly monitoring of the child's physical development, symptoms of heart failure - RR, pulse, weight gain, monitoring of medication intake - cardiac glycosides, their side effects. Once every 2 months - monitoring of blood flow levels, urine, ECG, consultation with a cardiologist as planned, and in case of deterioration of the condition. Prevention of backendocarditis).

Question 5. Provide training to the mother on how to care for Shokhsanam. (adequate, balanced nutrition - including bread or baked goods, vegetables, fresh seasonal fruits, milk and dairy products in the form of milk porridge - rice, buckwheat, oatmeal with the addition of butter; katyk, cottage cheese. Be sure to include in the diet in sufficient quantities foods rich in protein - meat, fish, eggs, legumes. Adhering to a daily routine - sleep, limiting physical activity for now, walking in the fresh air in a stroller, taking medications regularly. Maintaining hygiene rules when preparing and storing food, promptly consulting a doctor if you have a cold or infectious diseases.

Clinical case No. 7.

Lena is 4 years old. She was diagnosed with non-rheumatic carditis, which developed as a result of severe rubella. Lena has been registered as "D" for the last year and a half. Lately, Lena's mother has been noticing difficulty in her daughter's nasal breathing and slight cyanosis of the nasolabial triangle. She is worried that this is due to the deterioration of her condition due to myocarditis. Objectively: Lena's condition is satisfactory. RR 30/min, pulse 110. Vesicular breathing in the lungs. Auscultation: heart sounds are clear and rhythmic. There is no noise. The limits of relative dullness within her age. The abdomen is soft, painless,

the liver is at the edge of the costal arch. Urination and stool are normal. Weight 14 kg.

Question 1. Does Lena have symptoms of circulatory failure associated with non-rheumatic myocarditis? (Lena has no symptoms of NK).

Question 2. What do you think is the reason for Lena's difficulty in nasal breathing and slight cyanosis of the nasolabial triangle? (Perhaps Lena has adenoids, polyps or other problems with her nose)

Question 3. What are your tactics? (refer for consultation to an ENT to rule out adenoids or other nasal problems)

Question 4. The ENT doctor diagnosed Lena's adenoids and recommended removing them. What measures will you take before removing the adenoids, taking into account her non-rheumatic carditis? (prevention of bacterial endocarditis, namely: check general analysis, blood and urine cultures, ECG. Then prescribe amoxicillin - 50 mg/kg one hour before the procedure and again 8 hours after the first administration).

Clinical case No. 8

The month of June. Start of summer holidays. You are visiting a mother and her daughter Clara, aged 12. Her daughter needs a health certificate to go to the swimming pool. They contacted you because the cardio-rheumatologist at your clinic is studying and then going on vacation. For the last two years, Clara has been registered as "D" after suffering from rheumatic fever. When collecting anamnesis, it turned out that there were no complaints. I feel good. There were no recurrent rheumatic attacks. Receives year-round bicillin prophylaxis. She studies very well at school, attends physical education classes and does the same workload as her classmates. Periodically 1-2 times a year ARVI, colds. ZhDA – registered. The last (3 months ago) Hb test was 100. General examination: GI tract – BP 100/70, RR 20/min, Ps 90/min. Weight 45 kg. Height 155 cm. Dental caries was found in the oral cavity. Clinical examination: the skin is clean, visible mucous membranes are pink. Vesicular breathing in the lungs. Heart tones are clear and rhythmic. In the third intercostal space on the left, a systolic murmur is heard,

unpronounced, short, and with physical activity its intensity weakens. Does not radiate to other points of auscultation. Percussion: the left border of the heart in the 5th intercostal space 1 cm medially from the left nipple line. The belly is soft. The liver and spleen are not palpable. Regular urination and bowel movements are painless. The echocardiography report noted that there is an additional chord.

Question 1. You heard a noise in your heart. Is this murmur related to previous rheumatic fever, a developed PPS, or is it functional? And justify it. (it seems to be functional, since the heart sounds are not changed, the murmur is systolic, not diastolic, unpronounced, short, its intensity weakens with physical activity. Does not radiate to other points of auscultation).

Question 2. What objective data does Clara have that indicate that the heart murmur is not associated with rheumatic fever or developed PPS? (normal weight, height, development of the girl, absence of frequent diseases, normal blood pressure, pulse, respiratory rate, heart is not enlarged, good tolerance to physical activity. And most importantly, echocardiography data, which indicates the presence of an additional chord, which creates a murmur in the heart).

Question 3. Will you prescribe any other tests for Clara or will you give her a certificate for playing sports? If yes, which ones and why? (yes, I'll send Clara for a general blood test to check Hb and give recommendations for the treatment of IDA and dental caries. I'll do an ECG to rule out enlargement/hypertrophy of the heart).

Question 4. Complete blood count Hb 112, leukocytes - 6*109, ESR 6 mm/h. ECG – heart rate 95/min, sinus rhythm, no ECG signs of hypertrophy. Now will you give Clara a certificate to the pool or will you wait for the cardio-rheumatologist from school or vacation? (yes, I will establish and issue a certificate that Clara is healthy and can go to the pool and swim).

Clinical case No. 9.

The parents of Davron, 15 years old, came to you for an appointment. A month ago in Russia, Davron underwent surgical correction of valvular

post-rheumatic acquired heart disease. After the operation, they returned home and one of Davron's parents needed a sick leave to care for him.

Question 1. What indicators should you, as a GP, monitor for Davron, who was discharged after surgery for PPS? (t0, blood pressure, respiratory rate, pulse, pulse oximetry, weight, height, exercise tolerance, BAC, BAM, coagulogram parameters, echocardiography, x-ray, pain and heart murmur, palpitations, arrhythmias).

Question 2. What signs/symptoms of rheumatic process activity should the GP monitor for Davron? (fever, weakness, sweating, tachycardia, shortness of breath, joint pain, long-term decompensation refractory to therapy, leukocytosis, accelerated ESR, the appearance of C-reactive protein, increased sialic acid levels)

Question 3. What positive results after the operation will you notice with Davron? (weight gain, exercise tolerance will improve, symptoms of circulatory failure will decrease - shortness of breath, fatigue.).

Question 3. What postoperative complications may arise for Davron in the long term? (thromboembolism, prosthesis thrombosis, infective endocarditis, bleeding due to anticoagulant therapy)

Question 4. Does Davron need to undergo secondary prevention of rheumatism? If yes, which one? (Yes, he does. For three years, Davron needs a continuous course of bicillin prophylaxis: bicillin intramuscularly - 5 1.5 million units every month. Twice a year, usually in spring and autumn, during periods of sharp temperature fluctuations, it is necessary for 1- Prescribe a preventive course of aspirin for 1.5 months.Current prevention of rheumatism also involves a course of treatment with bicillin during acute or exacerbation of chronic infectious processes).

Clinical case No. 10.

A mother and her son Tolib, 11 years old, came to see you. Tolib has been playing football for 5 years. Mom is worried that for the last 3 weeks Tolib has looked tired and lost weight. His coach said Tolib often sits on the bench to rest during training.

Question 1. What information do you need to make a diagnosis?

• Recent illnesses in the last 2 months (no)

• Presence of chronic infectious (tuberculosis) and non-infectious diseases (asthma, diabetes) (no)

Question 2: What should you look for during a clinical examination?

• VVP at rest: pulse (130), respiration (28), blood pressure (90/60), temperature (37.2)

• Weight – 31 kg (a month ago my weight was 35). Height 145 cm

• Other signs of external examination: cyanosis (no), pallor of the skin and mucous membranes (yes), drumsticks (no), swelling in the legs (no), visible cervical pulsation (no). L/s are not palpable.

• Inspection, palpation, percussion, auscultation of the chest: cardiac hump (no), visible displacement or expansion of the area of the apical impulse (yes, not pronounced), systolic tremors (no). Percussion: (the left border of relative dullness is 1.5 cm outward from the midclavicular line, i.e. expanded). Auscultation: (no murmur, muffled heart sounds, tachycardia)

• Examination of the abdominal area: liver (painless, at the edge of the costal arch), spleen (not palpable).

Question 3. What research will you conduct for Toliba in the joint venture?

• Complete blood count (ESR 20 mm/h), complete urine test (normal), general stool test and stool test for worm eggs and Giardia cysts (roundworms), ECG (heart rate 120, sinus rhythm, tachycardia. Signs of left ventricular hypertrophy. Severe disturbances in the repolarization process in the myocardium).

Question 4. What did you suspect? (Non-rheumatic carditis - parasitic origin)

Question 5. Your tactics. (urgent hospitalization of Tolib in the cardiology department for the treatment of myocarditis - relief of symptoms of heart failure and treatment of ascariasis. All family members, except newborns, pregnant women and breastfeeding, should be treated for ascariasis with mebendazole (Vermox))

Memo for the teacher on conducting a clinical case analysis

During the analysis of a clinical case in a practical lesson, the teacher has to participate in a dialogue with the student, reading out the situational

task, sequentially or alternately asking him questions included in the structure of the situational task and informing the details presented in it.

Indicative standards of answers for the teacher are marked under the bulls (•) and/or in brackets (....)

The expression “additional information” is also intended for the instructor to provide this information to the student if the student asks for it. Additional information is provided to the student by the teacher in a timely manner, immediately after each question, if this is necessary according to the content of the situational task.

In some problems, “additional information” on anamnesis, physical examination, laboratory and instrumental data, diagnosis, treatment and prevention is indicated in parentheses next to indicative answers for the teacher.

When analyzing a clinical case, the teacher should not interrupt, comment, correct or explain anything, or ask the student leading questions. This can be done after completing the analysis of the clinical case on the basis of feedback (what was done well and correctly, and what needs to be improved), also by making a conclusion on the case under discussion and on the topic as a whole.

Test tasks

1. What place in frequency does congenital heart disease occupy among developmental anomalies of the central nervous system, gastrointestinal tract, respiratory organs, and musculoskeletal system:

a) 1
b) 2
at 3
d) 4
e) 5

2. With depletion of the systemic circulation, the following occurs:

a) Ebstein's disease
b) transposition of the great vessels
c) aortic stenosis
d) ventricular septal defect

3. With complete transposition of the great vessels, the presence compensatory defect

a) necessarily

b) not necessary

4. With tetralogy of Fallot II sound above the pulmonary artery:

a) strengthened

b) weakened

c) not changed

5. Which of the following viruses is associated with the possibility occurrence of congenital heart disease:

a) rubella

b) coxsackie

c) herpes

d) chickenpox

e) with all of the above

e) only a and b

6. The leading symptom of aortic coarctation is:

a) asthenic constitution

b) sinus tachycardia

c) fast and high pulse in the arms and legs

d) absence of pulse in the femoral artery

e) blood pressure lability

7.Which of the following is the main pathogenetic mechanism of dyspnea-cyanotic attack in tetralogy of Fallot:

a) hypoxia of the central nervous system

b) pulmonary hypervolemia

c) stagnation in the systemic circulation

d) spasm of the infundibular section of the right ventricle

e) left-right discharge into the VSD

8. In which congenital heart disease does cyanosis appear immediately after the birth of a child:

a) transposition of the great vessels

b) Ebstein's anomaly

c) pulmonary artery stenosis

d) coarctation of the aorta

9. What kind of congenital heart disease would you suspect in a child who complains of headaches when

detecting high blood pressure:

a) Tolochinov-Roger disease

b) aortic stenosis

c) pulmonary artery stenosis

d) Ebstein's disease

e) coarctation of the aorta

10. The endocardium of which valve is often affected by infective endocarditis:

a) mitral

b) aortic

c) tricuspid

d) pulmonary artery

Control questions

1.Symptoms and signs of congenital and acquired heart defects

2. Differential diagnosis of heart defects

3.Symptoms and signs of rheumatism

4. Differential diagnosis of heart failure in myocarditis, cardiomyopathies.

5. Differential diagnosis of rheumatism and non-rheumatic carditis of the heart

6.Classification of congenital heart defects?

7. What clinical signs are characteristic of restrictive cardiomyopathy?

8. What clinical signs are characteristic of hypertrophic cardiomyopathy?

9. What clinical signs are characteristic of dilatation cardiomyopathy?

10. Tell the GP’s tactics when managing patients with congenital, acquired heart defects

List of questions for written and oral control

1. Prevalence and incidence of rheumatic fever (RF) in children from the point of view of the level of development of countries. Current trends in the development and decline of rheumatic fever incidence at the end of the 20th century.

Answer: in developed countries, there has been a sharp decline in the incidence of LC since 1950 and the annual incidence reaches 282 cases per 100,000. The reasons for the decline in LC in developed countries remain unclear. Possible reasons are explained by improved living conditions, the discovery of antibiotics, and the availability of medical care. However, in developing countries, the endemic nature of the incidence of LC remains.

2. Principles for diagnosing rheumatic fever at the primary care level. Major and minor criteria for rheumatic attack.

Answer: Major criteria: carditis, polyarthritis, annular erythema, subcutaneous nodules. Minor: clinical – arthralgia, fever; laboratory and instrumental – increased ESR, C-reactive protein, ECG – prolongation of P-Q(R). Separately, the diagnosis takes into account the signs of streptococcal infection - group A streptococci in the throat and an increased level of antibodies to group A streptococcus.

3. Differential diagnosis of rheumatic fever.

Answer: differential diagnosis with infectious (reactive arthritis, viral carditis, infective endocarditis) and non-infectious diseases (rheumatoid arthritis, SLE, sickle cell anemia, malignant tumors, SLE, tics, hyperactivity syndrome)

4. List and interpretation of the results of clinical and biochemical laboratory diagnosis of rheumatic fever at the primary care level.

Answer: UAC (increased ESR, band neutrophils, leukocytes, decreased hemoglobin), TAM depending on the severity (protein, red blood cells, leukocytes), increased C-reactive protein

5. List and interpretation of the results of instrumental diagnostics (ECG and chest X-ray) of rheumatic fever at the PHC level

Answer: ECG - prolongation of P-Q(R). X-ray – expansion of the heart shadow.

6. GP tactics for suspected rheumatic fever.

Answer: If rheumatism is suspected, send to a specialist from the Central Medical Center (cardiologist or rheumatologist), who will carry out specific therapy: bed rest, antibacterial, anti-inflammatory, symptomatic (for the treatment of heart failure), sedative (for severe forms of chorea) therapy

7. Principles of prevention and medical examination of children with rheumatic fever at the primary health care level.

Answer: fighting streptococcal upper respiratory tract infections. Primary prevention - prevention of the first rheumatic attack - identification and destruction of group A streptococci. After a rheumatic attack, secondary prevention - constant antibiotic prophylaxis.

8. The most common rheumatic acquired heart defects (RAVs) in children. Reasons and timing for the formation of teaching staff.

Answer: The most common PPS are defects of the mitral (insufficiency and stenosis) and aortic valves (insufficiency and stenosis). The tricuspid and pulmonary valves in children are practically not affected. The reason is repeated multiple rheumatic attacks. The timing of the formation of PPS is 12-24 months after one or two rheumatic attacks.

9. Principles for diagnosing common PPS at the primary care level.

Answer: identify, recognize and evaluate based on: collection of complaints, medical history: history of repeated rheumatic attack. Clinical examination: shortness of breath, fatigue, palpitations, heart murmur, change in tones, increase in heart size. Depending on the severity of PPS - changes on the ECG - left ventricular hypertrophy.

10. Tactics of GPs in case of suspected PPS.

Answer: referral to a cardiologist.

11. Principles of prevention and medical examination of children with PPS at the primary health care level

Answer: fighting streptococcal upper respiratory tract infections. Primary prevention – prevention of rheumatic fever – identification and destruction of group A streptococci. Secondary prevention – constant antibiotic prophylaxis. For rheumatic heart disease, lifelong prevention,

as well as prevention of bacterial endocarditis. In the absence of rheumatic heart defects, prevention is for 5 years.

Drugs and doses. Long-acting penicillins, Bicilin-5: preschoolers - 750,000 units, schoolchildren - 1,500,000 units once a month, monthly all year round. All children in spring and autumn are prescribed salicylates - aspirin for 6 weeks at the rate of 0.1 g/year of life, no more than 1.0 g per day in 2-3 doses. If you are allergic to penicillins, you can use erythromycin, cephalosporins

12. Features of postoperative (regarding PPS) observation in primary care settings.

Answer: taking into account age, the severity of PPS, it is important: control of the gastrointestinal tract - blood pressure, pulse, respiratory rate, physical development - weight gain, weight loss, depression, complications. Prevention of backendocarditis. Regularly: OBC, OAM, ECG. Control and treatment of anemia. Visit the patient 3, 6 and 12 months after surgery.

13. Epidemiology and common causes of acquired non-rheumatic carditis in children.

Answer: the most common causes are viral infections (Coxsackie, herpes, rubella, influenza, measles, infectious mononucleosis). The true prevalence is unknown. Many cases are unrecognized. They are sporadic. There are cases of epidemic.

14. Clinical manifestations of non-rheumatic carditis depending on the age of the child.

Answer: in the early neonatal period there is an acute fulminant course. At the age of 1-2 years, less fulminant, in the form of myopericarditis. Older children and adolescents may have no symptoms.

15. Principles of diagnosing non-rheumatic carditis at the primary care level.

Answer: collection of complaints and anamnesis. Symptoms: fever, pain in the chest, muscles, joints, arrhythmia, weakness, signs of acute heart failure, severe fatigue, association with a viral infection. Physical examination findings. Conducting clinical tests (OAC, OAM, OAK and worm eggs and biochemical tests).

16. List and interpretation of the results of clinical and biochemical laboratory diagnostics of non-rheumatic carditis at the primary care level

Answer: Diagnostic tests: general blood test (increased ESR, leukocytosis), urine, acute phase tests

17. List and interpretation of the results of instrumental diagnostics (ECG and chest X-ray) of non-rheumatic carditis at the primary care level

Answer: X-ray (increase in heart size, increased pulmonary pattern), ECG (tachycardia, decreased QRS amplitude, ST-T shift, T changes, arrhythmia, blockades), X-ray - in severe myocarditis, the cardiac shadow is expanded, pulmonary edema. In CRMP: EchoCG – dilatation of the left ventricle, decreased myocardial contractility.

18. GP tactics for suspected non-rheumatic carditis

Answer: referral and treatment from a cardiologist.

19. Principles of clinical examination and prevention of non-rheumatic carditis at the primary care level.

Answer: regularity, continuity together with a cardiologist. Routine vaccination in the absence of contraindications. Control of vital signs: blood pressure, pulse, respiration, physical and mental development, temperature. Monitor heart failure symptoms, deterioration, medications and their side effects. Laboratory and instrumental studies: CBC, OAM, feces for worm eggs, ECG, chest x-ray. Referral to a specialist cardiologist and others as indicated. Prevention and control of complications – backendocarditis, heart failure. Monitoring the intake of medications and their side effects.

20. Complications and prognosis of acquired heart diseases (PPS and non-rheumatic carditis).

Answer: heart failure, rhythm and conduction disturbances, thromboembolism, sudden cardiac death. Newborns with severe symptoms have a poor prognosis. The weaker the clinical picture, the better the prognosis, until complete resolution of the disease.

1. A 17-year-old patient at the draft board complained of tinnitus, which worsens with physical activity. Blood pressure – 150/30 mm Hg. Art, a diastolic murmur was heard over the aorta. On a plain radiograph,

the shadow of the heart has an aortic configuration, and the aortic arch and left ventricle are enlarged. The pulmonary pattern is not changed. Aortic pulsation increased

1. The identified radiological changes most likely correspond to:

A. Hypertension.

D. Dextraposition of the aorta.

B. Atherosclerosis of the aorta.

E. Coarctation of the aorta.

C. Aortic insufficiency.

2. A 59-year-old patient complains of dizziness, severe shortness of breath, and chest pain during physical exertion. Objectively: pulse 70 beats/min, blood pressure – 110/90 mm Hg. Art. The apex beat is resistant. In the second intercostal space to the right of the sternum there is a rough systolic murmur, which is carried out in the first intercostal space. EchoCG – left ventricular cavity 6 cm, walls 1.8 cm. Diastolic dysfunction of the left ventricle.

What is the cause of the development of left ventricular diastolic dysfunction?

A. Aortic stenosis.

B. Hypertrophic cardiomyopathy.

C. Tricuspid valve insufficiency.

D. Constrictive pericarditis.

E. Mitral insufficiency.

3. A 35-year-old patient complains of inspiratory shortness of breath, nocturnal orthopnea, periodic hemoptysis, and interruptions in cardiac function. History of rheumatism. When listening to the heart: arrhythmic sounds, the first tone is increased at the apex, the accent of the second tone is on the pulmonary trunk, at the apex there is a diastolic murmur.

What heart defect is most likely formed in the patient?

A. Mitral valve insufficiency.

B. Aortic valve insufficiency.

C. Tricuspid valve insufficiency.

D. Mitral stenosis.

E. Stenosis of the aortic mouth.

4. A 23-year-old patient who has suffered from rheumatism since childhood, upon examination reveals pallor, swaying of the head, pulsation of the carotid arteries, and a high apical impulse. The left border of the heart is determined in the VI intercostal space 24 2 cm to the left of the midclavicular line. In the second intercostal space to the right of the sternum and at Botkin's point, a systolic murmur is heard, and immediately after the second tone - a murmur of a decreasing nature, occupying the first third of diastole. The most likely heart defect in a patient is:

A. Combined mitral disease (stenosis and insufficiency).

B. Combined defect (aortic stenosis and mitral insufficiency).

C. Combined defect (mitral stenosis and aortic insufficiency).

D. Combined defect (aortic stenosis and mitral insufficiency).

E. Combined aortic disease (stenosis and insufficiency).

5. A 42-year-old woman was admitted to the hospital with complaints of shortness of breath and palpitations with minor physical exertion. The borders of the heart are expanded upward and to the right, the first sound at the apex is intensified, and a protodiastolic murmur is heard here. The liver protrudes from under the edge of the costal arch by 5 cm, the legs are swollen.

What are the causes of heart failure?

A. Mitral stenosis.

B. Mitral insufficiency.

C. Tricuspid insufficiency.

D. Tricuspid stenosis.

E. Ventricular septal defect.

6. A 28-year-old patient complains of shortness of breath and “interruptions” in the activity of the heart. The apex beat is not displaced, the first sound at the apex is increased, an additional tone in diastole, diastolic murmur at the apex.

What disease can you think about?

A. Aortic stenosis.

B. Mitral orifice stenosis. C. Mitral valve insufficiency.

D. Insufficiency of the aortic valves.

E. Atrial septal defect.

7. During a medical examination of a woman, auscultation of the heart revealed: increased 1st sound at the apex, mitral valve opening tone, presystolic murmur, accent and splitting of the 2nd sound on the pulmonary artery.

What heart defect can be suspected?

A. Mitral stenosis.

B. Combined mitral valve disease.

C. Mitral valve insufficiency.

D. Stenosis of the aortic mouth.

E. Aortic valve insufficiency.

8. The patient complains of shortness of breath during physical activity, aching pain in the heart area, and palpitations. Auscultation: at the apex - increased 1st sound, diastolic murmur, after the 2nd tone an additional sound in diastole 25 after 0.10 s. Accent of the second tone over the pulmonary artery. Blood pressure – 120/80 mm Hg. Art., pulse – 78 beats/min.

What is your preliminary diagnosis?

A. Tricuspid valve stenosis.

B. Mitral valve insufficiency.

C. Stenosis of the aortic mouth.

D. Aortic valve insufficiency.

E. Stenosis of the left atrioventricular orifice.

9. In an 18-year-old patient who has been suffering from rheumatism for 3 years, during auscultation of the heart, a weakening of the first sound at the apex is heard, an accentuation of the second tone on the pulmonary artery and a systolic murmur at the apex, conducted to the left axillary region.

What heart damage is most likely?

A. Mitral insufficiency.

B. Tricuspid insufficiency.

C. Mitral stenosis.

D. Ventricular septal defect.

E. Stenosis of the left atrioventricular orifice.

10. A 44-year-old patient with rheumatic stenosis of the aortic mouth developed attacks of suffocation at night. Objectively: the first sound is weakened, the systolic murmur is at the apex of the heart, the accent of the second sound is over the pulmonary artery, the rough systolic murmur is over the aorta. On the radiograph: the cardiac waist is smoothed, the left border of the heart is shifted to the left by 5 cm.

What causes the appearance of systolic murmur at the apex of the heart?

A. Mitralization of aortic disease.

B. Development of stenosis of the left atrioventricular orifice.

C. Attachment of aortic insufficiency.

D. An increase in the degree of stenosis of the aortic mouth.

E. Pulmonary embolism.

11. A 40-year-old patient, who had previously suffered from tonsillitis for a long time and was not treated, was found to have a weakening of the first tone and a systolic murmur at the apex, carried out in the second intercostal space on the left and into the left axilla, with an emphasis of the second tone on the pulmonary artery.

What pathological condition are these clinical symptoms typical for?

A. Insufficiency of the semilunar valves of the aorta.

B. Mitral valve insufficiency..

C. Stenosis of the aortic mouth.

D. Tricuspid valve insufficiency.

E. Mitral stenosis.

12. A 22-year-old patient who has been suffering from rheumatism for 11 years began to complain of a feeling of pulsation in the head, rhythmic shaking of the head, and dizziness. When examining the patient, a positive Quincke's pulse was revealed, the presence of Landolfi and Musset symptoms, blood pressure - 170/40 mm Hg. Art.

What causes the patient's hemodynamic disorders?

A. Aortic valve insufficiency.

B. Aortic valve stenosis.

C. Tricuspid valve insufficiency.

D. Combined mitral heart disease.

E. Manifestation of “minor chorea”.

13. Patient, 70 years old. Complaints of suffocation, sharp pain behind the sternum radiating to the left arm. The skin is pale. The activity of the heart is rhythmic, the first sound is weakened over the apex, the second sound is weakened over the aorta; in the second intercostal space on the right there is a rough systolic murmur, carried to the vessels of the neck. ECG: left ventricular hypertrophy.

What is the most likely pathology that predetermines this picture?

A. Myocardial infarction.

D. Left-sided pleurisy.

B. Aortic stenosis.

E. Dry pericarditis.

C. Angina pectoris.

14. A 63-year-old patient complains of shortness of breath and palpitations during physical activity. The boundaries of relative cardiac dullness are expanded upward and to the left. The activity of the heart is rhythmic, the first tone is weakened, the accent of the second tone is over a. pulmonalis, a loud systolic murmur over the apex, is carried out in the fossa axillaris sinistra. ECG – levogram, P wave is 0.12 s, FCG – I tone in the form of low-amplitude fluctuations, systolic murmur merges with the I tone, but does not reach the II tone. What is the most likely pathology that predetermines this picture?

A. Mitral stenosis.

D. Hypertension.

B. Myocarditis.

E. Mitral insufficiency.

C. Aortic stenosis.

15. At an appointment in the clinic, a patient with rheumatism was found to have: the right border of the heart - 1 cm outward from the right parasternal line, the upper - the lower edge of the 1st rib, the left - 1 cm inward from the left midclavicular line. Auscultation: atrial fibrillation, increased 1st sound at the apex, accentuated 2nd tone over the pulmonary artery. Echocardiography revealed a U-shaped movement of the mitral valve leaflets.

What heart defect does this symptomatology correspond to?

A. Mitral stenosis.

B. Mitral valve prolapse.

C. Mitral valve insufficiency.

D. Stenosis of the aortic mouth.

E. Tricuspid valve insufficiency.

16. At an appointment at the clinic, the patient was found to have a diastolic murmur with an epicenter at the apex and above the projection of the aorta, weakening of the first and second sounds. The borders of the heart are expanded to the left, a high, resistant 27 apical impulse is noted, displaced in the 7th intercostal space. Blood pressure – 140/30 mm Hg. Art. Pulse – 92 beats/min, resistant, high in amplitude.

Determine the type of heart defect.

A. Insufficiency of the aortic valves.

B. Stenosis of the aortic mouth.

C. Coarctation of the aortic arch.

D. Combined mitral valve disease.

E. Non-closure of the interatrial septum.

17. A patient complains of dizziness, shortness of breath and pain during physical activity. Objectively: signs of Marfan syndrome, pale, “carotid dancing.” Auscultation: in the second intercostal space on the right at the edge of the sternum there is a diastolic murmur that extends to the apex, a Durosier murmur above the femoral artery, blood pressure - 160/50 mm Hg. Art., P – celler at altus, 90 beats/min. EchoCG shows LV diastolic size 7.0 cm.

What diagnosis can be made?

A. Aortic stenosis.

B. Mitral insufficiency.

C. Patent ductus arteriosus.

D. Aortic insufficiency.

E. Isolated systolic hypertension.

18. In a patient with rheumatism, diastolic trembling of the chest wall (“cat purring”), increased 1st sound at the apex, diastolic murmur with

presystolic intensification, a click of the opening of the mitral valve, and an accentuation of the 2nd tone over the pulmonary artery are detected.

What heart defect does the patient have?

A. Stenosis of the left atrioventricular orifice.

B. Aortic valve insufficiency.

C. Pulmonary artery stenosis.

D. Mitral valve insufficiency.

E. Patent ductus arteriosus.

19. A 35-year-old patient feels tremors throughout her body, “pulsation” in her head, and periodic syncope. The left border of the heart along the left anterior axillary line, in Botkin’s point - diastolic murmur. Blood pressure – 150/20 mm Hg. Art.

What disease is most likely?

A. Insufficiency of the aortic valves.

B. Hypertrophic cardiomyopathy.

C. Thyrotoxic heart.

D. Hypertension.

E. Mitral orifice stenosis.

20. A 75-year-old patient complains of suffocation, sharp pain behind the sternum radiating to the left arm. The skin is pale. On percussion, there is an expansion of the boundaries of relative cardiac dullness to the left. The activity of the heart is rhythmic, the 1st sound is weakened over the apex, the 2nd sound is weakened over the aorta; in the second intercostal space on the right there is a rough systolic murmur, which is carried out to the vessels of the neck. ECG: left ventricular hypertrophy. FCG: systolic rhomboid murmur over the aorta.

What is the most likely pathology that predetermines this picture?

A. Aortic stenosis.

B. Angina pectoris.

C. Myocardial infarction. .

D. Left-sided pleurisy

E. Mitral orifice stenosis

List of abbreviations.

ACG - angiocardiography
CHD – congenital heart defect
ASD – atrial septal defect
VSD – ventricular septal defect
IHD – coronary heart disease
IR - artificial blood circulation
ICS - artificial heart valve
IE – infective endocarditis
CA – coarctation of the aorta
PH – pulmonary hypertension
INR – international normalized ratio
AAV – aortic valve insufficiency
MMV - mitral valve insufficiency
NTK - tricuspid valve insufficiency
PDA – patent ductus arteriosus
OG – general hypothermia
PPS – acquired heart defect
PS - heart defect
SAV – aortic valve stenosis
MVC - mitral valve stenosis
TVC - tricuspid valve stenosis
TF – tetralogy of Fallot
VO2max – maximum oxygen consumption
FCG – phonocardiography
ECG – electrocardiography
EchoCG – echocardiography

GLOSSARY

ATRIO - a component of compound words, meaning “pertaining to the atrium”; for example, atrioventricular node, atrioventricular block, etc.

ATRIOVENTRICULAR BLOCK - a violation of the conduction of impulses from the atria to the ventricles at the level of the atrioventricular node or atrioventricular bundle and its legs (can be complete or incomplete); the cause may be acute myocardial infarction, rheumatic carditis, intoxication with digitalis drugs, quinidine, myocarditis and myocardiopathy, congenital heart defects, etc.

HEART ARRHYTHMIA - a violation of the frequency or sequence of heart contractions: acceleration (tachycardia) or slowdown (bradycardia) of the rhythm, premature contractions (extrasystole), disorganization of rhythmic activity (atrial fibrillation), etc.; may be a consequence of diseases of the heart muscle, neurosis, alcohol or nicotine intoxication, etc.

Hypertrophy (hypertrophy, hypertrophia) - An increase in the size of any tissue or organ, associated with an increase in the cells included in its composition, and not with their increased reproduction (as during normal growth of the tissue or the formation of a tumor in it). Muscles undergo this change in response to increased load.

LEFT VENTRICULAR FAILURE - heart failure caused by excessive load on the left ventricle, which leads to a decrease in the release of blood into the systemic circulation, overstretching of the left atrium and stagnation in the pulmonary circulation; observed with heart defects, myocarditis, hypertension, etc.

INFARCTION is a section of an organ or tissue that has undergone necrosis due to a sudden disruption of its blood supply.

I. LUNG - hemorrhagic I. in the lung tissue, having the shape of a triangle with a wide base facing the pleura; occurs, as a rule, when the

branches of the pulmonary artery are blocked; manifested by hemoptysis, chest pain.

I. MYOCARDIA - an acute disease characterized by the development of one or multiple changes in the myocardium, arising as a consequence of acute coronary insufficiency and manifested by a variety of clinical symptoms, depending on the nature of cardiac dysfunction and reflex reactions

LEFT VENTRICULAR FAILURE - heart failure caused by excessive load on the left ventricle, which leads to a decrease in the release of blood into the systemic circulation, overstretching of the left atrium and stagnation in the pulmonary circulation; observed with heart defects, myocarditis, hypertension, etc.

MYOCARDITIS - inflammation of the heart muscle; manifested by signs of impaired contractility, excitability and conductivity.

MYOCARDIAL DYSTROPHY is a disease associated with destructive disorders of the heart muscle (for example, as a result of infection, intoxication, etc.).

INFARCTION - necrosis of a tissue area due to cessation of blood supply; I. is most often observed in the myocardium, less often in the lungs, kidneys, spleen and other organs.

ISCHEMIC (CORONARY) HEART DISEASE is a chronic pathological process caused by insufficient blood supply to the myocardium; in the vast majority (97-98%) of cases it is a consequence of atherosclerosis of the coronary arteries of the heart.

ISCHEMIA is a decrease in blood flow to an organ.

CARDIOSCLEROSIS - damage to the muscle (myocardiosclerosis) and heart valves due to the development of scar tissue in them in the form of nests of various sizes (from microscopic to large scar lesions and fields) and extent, replacing the myocardium and (or) deforming the valves.

FUNCTIONAL STATE - an integral complex of available characteristics of those qualities and properties of the body that directly or indirectly determine human activity.

FUNCTION (functio - activity) - interaction of elements in a system, interaction and subordination of part and whole in a living organism.

The electrical activity of the heart is the result of the cyclic movement of ions in the cells and extracellular fluid.

Sinoatrial blockade is a slowdown in the conduction of impulses from the sinoatrial node to the atria.

Atrial fibrillation is an irregular contraction of groups of myofibrils with a frequency of 400-700 per minute, leading to a lack of coordinated atrial systole.

Atrial flutter is a regular contraction of groups of atrial fibrils with a frequency of 250-350 per minute.

Extrasystole is a premature contraction of the heart or its individual chambers caused by excitation outside the sinus node.

Sinus bradycardia is a slow heart rate of less than 60 beats per minute.

Sinus tachycardia – an increase in heart rate of more than 100 beats per minute (up to 160 -180).

BIBLIOGRAPHY:

Main:

1.Shabalov, N.P. Children's diseases textbook. for universities in 2 volumes / N.P. Shabalov. – 7th ed., revised. and additional – St. Petersburg: Peter, 2012

2. Pediatrics. National leadership: in 2 volumes - M.: GEOTAR-Media, 2009, volume 1−1024 pp., volume 2 – 1023 pp.

Additional:

3.Childhood diseases: textbook with computer. - disk / ed. A.A. Baranova. 2nd ed. – M., 2007 – 1008 p.

4. Classification of major diseases and syndromes in children / Bykov V.O., Dushko S.A., Filimonov Yu.A., Tkacheva N.V., Vodovozova E.V., Gubareva G.N., Kuznetsova I.G. ., Ledeneva L.N., Mironova E.V., Popova T.A., Untevsky I.I. –

4th ed., Stavropol, 2008.

3. Knyshov G.V., Bendet Ya.A. (1997) Acquired heart defects. Institute of Cardiovascular Surgery, Kyiv, 280 p.

5.Kovalenko V.N., Nesukai E.G. (2001) Non-coronary heart diseases. Practical management. Morion, Kyiv, 480 p.

5. Pediatrician's Handbook: textbook / ed. IN. Bykova, A.S. Kalmykova. – 3rd ed., revised. and additional – Rostov n/d: Phoenix, 2007. – 573 p.

6. Guide to the practical skills of a pediatrician [Text]: textbook. manual for universities / ed. IN. Bykova – ed. 3rd, stereotype. – Rostov n/d: Phoenix, 2010. – 574 pp.: table, fig.

7.Pathology of young children. Part 1: textbook / V.O. Bykov, E.V. Vodovozova, S.A. Dushko, G.N. Gubareva, I.G. Kuznetsova, E.V. Kulakova, L.N. Ledeneva, E.V. Mironova, T.A. Popova, I.A. Stremenkova, E.V. Shchetinin. – Stavropol: St. State Medical Academy Publishing House, 2011 – 173 p.:

Electronic educational resources:

Laboratory and functional studies in pediatric practice / R.R. Kildiyarova. 2012 http://www.studmedlib.ru/ru/book/ISBN9785970419588.html G. A. Samsygina. Pediatrics. Selected lectures, 2009 http://www.studmedlib.ru/ru/book/ISBN9785970410905.html

 Children's diseases / ed. R. R. Kildiyarova. 2014 http://www.studmedlib.ru/ru/book/ISBN9785970429488.html

 Federal clinical guidelines for the provision of medical care to children with chronic heart failure 2015 http://www.pediatrrussia.ru/sites/default/files/file/kr_hsn.pdf

 Federal clinical recommendations for the provision of medical care to children with congenital heart defects. 2015 http://www.pediatrrussia.ru/sites/default/files/file/kr_vps.pdf National program for optimizing feeding of children in the first year of life in the Russian Federation federation. 2008 http://www.sprjournal.ru/webasyst/published/SC/html/scripts/product_files/Nats_programma_blok_2 0_07_2011.pdf 7.

Table of content

O‘QUV ADABIYOTINING NASHR RUXSATNOMASI

O‘zbekiston Respublikasi Sog‘liqni saqlash vazirligi Toshkent pediatriya tibbiyot institutining **2024** yil “**04**” iyuldagi “**335**” -sonli buyrug‘iga asosan

E.A.Shamansurova, A.I.Ulugov, A.N.Fayziyev, N.X.Isaxanova

(muallifning familiyasi, ism-sharifi)

Tibbiyot oliygohlari talabalari uchun

(ta’lim yo‘nalishi (mutaxassisligi))

ning talabalari (o‘quvchilari) uchun tavsiya etilgan

Diagnostic criteria, treatment, prevention, disease of diseases accompanied by cardiomygaly syndrome and heart murmurs

nomli o‘quv qo‘llanma ga

(o‘quv adabiyotining nomi ya turi: darslik, o‘quv qo‘llanma)

O‘zbekiston Respublikasi Vazirlar Mahkamasi tomonidan litsenziya berilgan nashriyotlarda nashr etishga ruxsat berildi.

Rektor _____ B.T.Daminov

Ro‘yxatga olish raqami

335-256

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DIAGNOSTIC CRITERIA, TREATMENT, PREVENTION, DISEASE OF DISEASES ACCOMPANIED BY CARDIOMYGALY SYNDROME AND HEART MURMURS

Tutorial

Muharrir: F. Tishaboev
Texnik muharrir: M. Hakimov

O`zbekiston Matbuot va axborot agentligining « Voris-nashriyot» nashriyotiga
2006 yil 23 fevralda berilgan 2015881-sonli litsenziyasi
Nashriyot manzili: Toshkent shahri, Shiroq kochasi, 100-uy.

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