Theoretical mateiras for 2nd year students of the Faculty of Pharmacy for practical classes in Pathology

Topic: Violation of cardiac conduction, excitability, contractility. The main types of ECG in pathology. Pathology of the heart.Heart defects. Heart failure.

1. Purpose: To study the causes, mechanisms of development of heart failure. Consider the main clinical and morphological forms of congenital heart defects. Explore cardiomyopathies. To study the occurrence, development and manifestations of inflammatory diseases of the pericardium and their complications.

2. Requirements for the level of the student in mastering the discipline - pathology.

Theoretical aspects:

Heart failure (HF) is a violation of the ability of the heart to provide blood supply to organs and tissues in accordance with their metabolic needs.

Total peripheral vascular resistance (OPVR) is the resistance to blood flow in the arterial system.

Afterload is the force that the myocardium must develop in systole to ensure the exit of blood from the ventricles and overcome the OPSS (pressure load).

Preload - the load on the heart created by the volume of blood entering the ventricle in diastole (volume load)

Cardiac output - an indicator of the pumping function of the heart, the volume of blood ejected by the heart in one minute depends on the heart rate and stroke volume of the heart.

Stroke volume is the volume of blood ejected by the ventricle in 1 complete contraction.

Heart sounds are sounds that occur when the valves vibrate during myocardial contraction. Normally, I tone (at the beginning of systole) and II tone (at the beginning of diastole) are determined.

A heart murmur is a sound produced by turbulent blood flow in the heart. Not normally defined.

Cardiac asthma is an asthma attack caused by the development of acute left ventricular failure.

Alternation of the heart - a violation of the contractile function, a regular alternation of relatively strong with weaker heart contractions.

HEART FAILURE

Etiology

1. Reduced contractility:

a. Myocardial infarction (MI)

b. Congenital and acquired heart defects, leading to an overload of the heart cavities with pressure or volume.

c. Arterial hypertension. A decrease in myocardial contractility is characteristic of persistent severe hypertension, which quickly leads to the development of heart failure.

d. Cardiomyopathy

2. Increased afterload.

OPSS can increase significantly, making it difficult for the heart to work.

3. Changes in preload.

a. An increase in preload with normal afterload and myocardial contractility is rare. The exception is severe renal insufficiency or excessive fluid therapy, in which an increase in circulating blood volume (CBV) entails a significant increase in preload. The patient can develop all the signs of heart failure with a healthy heart.

b. Reduced preload.

Heart failure with high cardiac output. Cardiac output is several times higher than normal, but even then the blood supply to organs and tissues is not enough. An example is thyrotoxicosis, which causes a sharp acceleration of metabolism in tissues, as well as severe chronic anemia, when oxygen transport is significantly reduced.

Left ventricular failure - weakness of the myocardium of the left ventricle. The defeat of the left ventricle (for example, MI) reduces its contractility, without affecting the function of the right ventricle. Right ventricular failure

Left ventricular weakness is considered to be the most common cause of right ventricular failure. An increase in the filling pressure of the left ventricle worsens the work of the right one, which leads to its insufficiency. Right ventricular failure may develop on its own. Chronic obstructive pulmonary diseases cause an increase in vascular resistance in the small circle, which leads to an increase in the load of the right ventricle and ends in right ventricular heart failure.

Acute heart failure is a sudden violation of the contractile function of the myocardium, which leads to a sharp violation of adequate blood circulation.

Chronic heart failure. It is subdivided into the following stages (N.D. Strazhesko, V.Kh. Vasilenko).

Stage I (initial) - latent HF, manifested only during physical exertion (shortness of breath, tachycardia).

Stage II (expressed) - stagnation in the large and small circles of blood circulation is expressed at rest.

Period A - shortness of breath with minimal physical exertion, stagnation of blood in the small circle.

Period B - constant shortness of breath, at rest, stagnation in the small and large circles of blood circulation.

Stage III (final, dystrophic) - severe hemodynamic disturbances, persistent changes in metabolism and functions of all organs, pronounced stagnation in the systemic and pulmonary circulation.

Clinical manifestatons

a). ***Shortness of breath*** is the most common symptom of heart failure. Congestive plethora of the vessels of the lungs causes a feeling of lack of air. Causes: decrease in oxygenation of blood in the lungs, a decrease in their elasticity, an increase in the frequency of respiratory movements and increased fatigue of the respiratory muscles. Another cause of shortness of breath is reduced cardiac output to the peripheral vessels due to an increase in peripheral vascular resistance. In the initial stages of heart failure, shortness of breath occurs during exercise, and later on at rest.

b. ***Orthopnea*** - shortness of breath in the supine position - a sign of redistribution of blood from the veins of the abdominal cavity and lower extremities into the chest, which is accompanied by a decrease in the airiness of the lungs, an increase in hydrostatic pressure in the pulmonary capillaries, and a decrease in blood oxygenation. Shortness of breath decreases or completely disappears in a sitting or standing position.

c. ***Paroxysmal nocturnal dyspnea***. Causes: centralization of blood circulation, increased pressure in the pulmonary capillaries, interstitial pulmonary edema. Shortness of breath usually decreases 5-20 minutes after taking a vertical position, otherwise alveolar pulmonary edema develops.

d. ***Nocturia*** occurs in heart failure as a result of increased renal blood flow during sleep.

During the day, when the patient is active, the reduced cardiac output is redistributed mainly in favor of the skeletal muscles. The reaction of the kidneys - activation of the renin-angiotensin system - is similar to that in hypovolemia. At night, due to increased blood circulation in the kidneys, diuresis is restored.

Edema occurs when systemic hydrostatic venous pressure exceeds systemic oncotic venous pressure (a sign of right ventricular failure).

e. ***Anorexia*** often occurs in the later stages of heart failure, accompanied by right ventricular and liver failure.

Physical examination

a. Tachycardia occurs compensatory to maintain cardiac output in conditions of reduced stroke volume.

b. Wheezing over the lungs. An increase in the filling pressure of the left ventricle in heart failure is accompanied by the accumulation of blood in the left atrium and pulmonary veins. The increased hydrostatic pressure leads to the extravasation of plasma into the alveoli and its effervescence by the circulating air.

c. Enlargement of the heart. Circulatory failure causes expansion of the chambers of the heart and myocardial hypertrophy.

d. The third heart sound (tone III) creates an auscultatory picture of the protodiastolic gallop rhythm. A ventricular gallop rhythm is perhaps the most reliable sign of heart failure, defined physically. The appearance of the III tone is explained by the fluctuation of the walls of the left ventricle at the very beginning of its diastole under the action of high filling pressure in the left atrium.

e. Atrial gallop rhythm. In heart failure and sinus rhythm, an additional presystolic tone (fourth heart sound, sound IV) is often heard - an acoustic phenomenon of blood ejection from the left atrium into the left ventricle immediately before ventricular systole.

f. Swelling of the jugular veins.

g. Edema of the lower extremities, ascites are common symptoms of right ventricular failure; has diagnostic value when combined with an increase in the liver and swelling of the jugular veins.

Diagnostics

1. Assessment of etiological aspects.

2. Instrumental research methods.

a. ***Electrocardiography***. An ECG almost always registers altered electrical activity of the heart (signs of atrial and ventricular hypertrophy, myocardial ischemia, rhythm and conduction disturbances, ST segment changes, etc.), but these signs are nonspecific.

b. ***A chest x-ray*** can detect enlargement of the heart chambers and confirm pulmonary congestion. The method provides objective evidence of heart failure.

c. Echocardiography characterizes changes in the chambers of the heart and gives an idea of the function of the left ventricle and valves. It is especially informative for lesions of the mitral valve.

d. ***The Doppler effect*** makes it possible to determine the direction and speed of blood flow through the cavities of the heart and large vessels. The method can be used to detect abnormal direction of blood flow, which is typical for valvular regurgitation and intracardiac shunts.

The use of Doppler scanning allows you to determine the degree of valvular stenosis. These possibilities significantly improve the diagnosis of heart failure in valvular heart disease and intracardiac shunts.

e. ***Radionuclide veitriculography*** is a non-invasive quantitative method. Allows you to accurately determine the degree of dysfunction of the right and left ventricles of the heart.

e. ***Catheterization of the heart cavities*** is the most accurate (at the same time, the most dangerous invasive) method for diagnosing heart diseases, it allows you to measure intracardiac pressure, the anatomy of the cavities, valvular stenosis, determine the anatomy and patency of the coronary arteries.

CONGENITAL HEART DEFECTS IN ADULTS

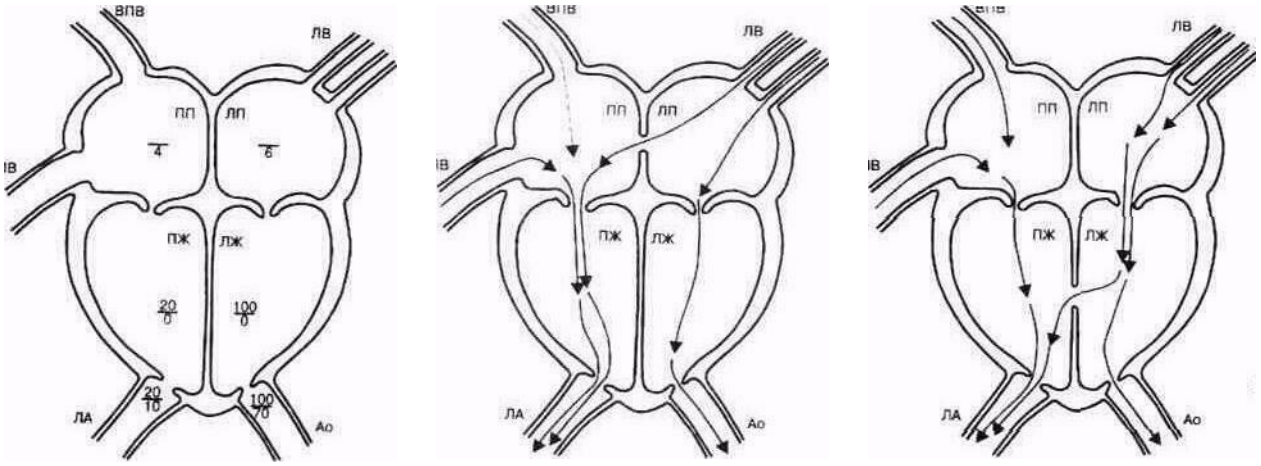
Atrial septal defect (ASD)

ASD is the constant communication of the left and right atria through a defect in the interatrial septum. In all variants of ASD, the right ventricle and pulmonary artery are usually enlarged. Defect may located in the upper part of the interatrial septum at the confluence of the superior vena cava - high secondary ASD (sinus venosus defect); the defect is often accompanied by abnormal drainage into the right atrium of one or more pulmonary veins. The central defect of the secondary septum (central secondary ASD) is located in the middle part of the interatrial septum in the region of the oval fossa.

A low secondary septal defect (low secondary ASD) is localized in the region of the orifice of the inferior vena cava. The defect of the primary interatrial septum - primary ASD - is located at the site of the formation of ridges.

Pathogenesis

Greater compliance of the right ventricular wall compared to the left, low pulmonary vascular resistance, and higher pressure in the left atrium than in the right, lead to left-to-right shunting at the atrial level and increased blood flow through the tricuspid valve and pulmonic valve. The wall of the left ventricle is thicker than the right ventricle, so it is more difficult to fill. The final result is an increase in the load on the right ventricle.



Blood circulation is normal (left), with ASD (center), with VSD (right).

Symptoms: in childhood - usually not manifested. Adults may have a long asymptomatic period. Over time, palpitations, fatigue, shortness of breath on exertion, orthopnea, and symptoms of right ventricular failure appear.

Physical signs:

1. Increased blood filling of the right ventricle prolongs the closure time of the pulmonary valve, which causes persistent splitting of the II sound over the pulmonary artery. A classic finding in ASD.

2. Noises. Moderate systolic ejection murmur over the pulmonary artery. When the discharge into the pulmonary circulation is 3 times higher than normal blood flow - diastolic murmur over the tricuspid valve.

3. Right ventricular failure - expansion of the cervical veins, edema, ascites.

Diagnostics:

1. Electrocardiography: a significant deviation of the electrical axis of the heart (EOS) to the left in primary ASD, often a deviation of the EOS to the right - in secondary ASD.

2. Radiography: enhancement of the vascular pattern.

3. Echocardiography. enlargement of the right ventricle.

4. Radionuclide study: recirculation of blood in the lungs.

5. Cardiac catheterization:

(1) It is possible to pass the probe through the defect.

(2) The pressure in the left and right atria is equal.

(3) In blood samples from the superior vena cava and the right atrium, an increased oxygen concentration (oxygenation surge) is detected due to the shunt of oxygenated blood from the left atrium to the right. This technique allows you to roughly estimate the severity of the defect.

Ventricular septal defect (VSD)

VSD is the constant communication of the left and right ventricles through a defect in the interventricular septum. VSD can be single or multiple and localized in any part of the septum; usually the defect is located in its membranous part.

Pathogenesis. In VSD, the left ventricle shunts blood into the right ventricle, which overloads both ventricles and increases pulmonary blood flow. Small defects cause a small reset. If the defect is large, then the volume and direction of the shunt directly depend on the magnitude of the resistance of both the pulmonary and systemic vascular bed. As long as pulmonary vascular resistance is below systemic resistance, shunting of blood from left to right occurs.

If pulmonary vascular resistance exceeds systemic resistance, then the direction of the shunt is reversed. With large defects, there is a tendency to develop pulmonary hypertension, while with small defects, pulmonary hemodynamics is slightly impaired. The dimensions of the left atrium and left ventricle are directly proportional to the size of the left-right shunt. Right ventricular enlargement occurs when pulmonary vascular resistance increases. Pulmonary hypertension is more common and more pronounced in VSD than in ASD. Prolonged pulmonary hypertension can lead to the development of an organic lesion (obstruction) of the pulmonary vessels and a change in the direction of the shunt.

Clinic. Most VSDs are diagnosed and corrected in childhood; VSDs are rare in adults.

Symptoms. The severity depends on the magnitude of the reset. With a small defect, there are no clinical manifestations. If the defect is large sizes, and pulmonary resistance is slightly increased (large left-right shunt), congestive heart failure and recurrent lower lobe pneumonia are possible. With large defects and high pulmonary vascular resistance (Eisenmenger's syndrome), shortness of breath at rest and during exercise, chest pain, and cyanosis may occur.

physical signs

(1) A rough systolic murmur is heard along the left edge of the sternum with its epicenter at the fourth intercostal space. Often noise accompanies systolic trembling.

(2) Aortic insufficiency.

Diagnostics

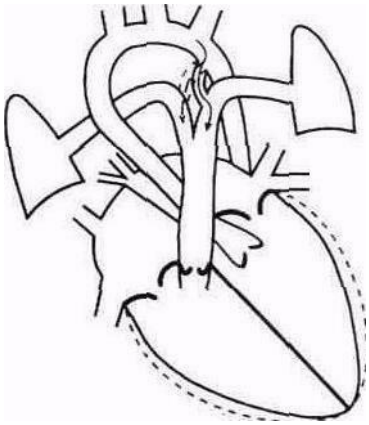
a. Electrocardiography. Signs of hypertrophy of both ventricles.

b. Chest X-ray. Determine the increase in the heart. If the shunt ratio is more than 2:1, then an increase in the vascular pattern of the lungs (shunt vascularization) becomes noticeable.

c. Echocardiography. In most cases, a septal defect and enlargement of both ventricles are found. Doppler study: abnormal flow of blood from the left ventricle to the right.

d. Radionuclide methods, as in ASD, are quite informative. e. Cardiac catheterization Left-sided ventriculogram obtained in the left anterior oblique view demonstrates the passage of contrast through the defect from the left ventricle to the right. During catheterization,

identify a jump in oxygenation, and if there is pulmonary hypertension, then quantify it.



Open ductus arteriosus (PDA).

The arterial (botallian) duct connects the pulmonary artery and the aorta; its size is different in different patients. The degree of enlargement of the left atrium and ventricle directly depends on the amount of blood shunt through duct. Enlargement of the right ventricle occurs only with pulmonary hypertension.

Pathogenesis. In PDA, part of the blood from the aorta is dumped into the pulmonary artery. Volume overload falls on the left ventricle, which pumps blood into the vessels of both the large and small circles, which eventually leads to left ventricular failure. Increased pulmonary blood flow can form pulmonary hypertension, in which pressure overload acts on the right ventricle. Shunting of blood from left to right (both in systole and diastole) is due to the difference in pressure in the aorta and pulmonary artery. The direction of blood flow through a wide PDA depends on the resistance of the pulmonary and systemic vascular bed. While pulmonary vascular resistance is lower systemic, shunting of blood occurs from left to right. If pulmonary vascular resistance exceeds systemic resistance, then a change in the direction of the shunt occurs. With small sizes of the SAR, the discharge from left to right is limited. The amount of blood shed depends on the diameter of the PDA and the magnitude of pulmonary and systemic vascular resistance. With large sizes of the duct, changes in the pulmonary vessels occur early (Eisenmenger's syndrome).

physical signs

a. Continuous systole-diastolic murmur. Systolic noise proceeds in a diastole (machine noise, noise of a top). With an increase in pulmonary vascular resistance, first the diastolic and then the systolic murmur becomes softer and shorter.

b. Pulse. The filling of the pulse depends on the amount of reset from left to right. If it is small, then the pulse is normal. With a large reset, a high frequent pulse is observed, resulting from the diastolic outflow of blood from the aorta through the duct into the pulmonary artery.

Diagnostics

a. Radiography reveals an enlarged shadow of the heart and signs of shunt vascularization. The patent ductus arteriosus may become calcified and become visible on x-ray.

b. Cardiac catheterization

(1) Usually, during probing, a probe can be passed from the pulmonary artery into the descending aorta, which confirms the diagnosis.
(2) Oximetry quantifies the degree of shunting.
(3) Aortography. The contrast passes from the aorta through the PDA to the pulmonary artery.

Coarctation of the aorta.

The narrowing of the aorta is almost always located at the confluence of the ductus arteriosus with the aortic arch, immediately after the left subclavian artery leaves. The narrowing can capture a short section of the aorta, but it can also be extended.

Pathogenesis. Coarctation of the aorta is often complicated by arterial hypertension in the brachiocephalic vessels.

a. In severe cases, blood flow is limited distal to the stenosis, and extensive collaterals support the blood supply.

b. Although renal function is usually preserved in coarctation of the aorta, renal blood flow is subnormal.

c. There is evidence of elevated renin levels and activation of the renin-angiotensin system in adult patients with aortic coarctation, which helps to explain the cause of hypertension.

Clinic. If heart failure does not occur in childhood due to pressure overload, then the defect may not be detected until hypertension appears in an adult.

Symptoms. Patients may complain of headaches, intermittent claudication, fatigue.
physical signs
(1) BP is elevated in the arms, while pulses in the lower extremities may be absent, confirming the pressure gradient at the site of coarctation.
(2) Appearance. Attention is drawn to the good development of the upper body with underdevelopment of the lower part.
(3) A mid-systole murmur is heard on the back. With significant stenosis, the noise becomes prolonged. Noises can also be heard over the entire chest due to the developed collateral circulation.

Diagnostics
a. Electrocardiography. Left ventricular hypertrophy.
b. A chest x-ray shows an enlarged heart. With dilatation of the aorta proximal and distal to coarctation, a peculiar configuration of the aorta in the form of the figure "Z" is often formed. With dilated intercostal arteries, uzuras appear on the edges of the ribs.
c. Cardiac catheterization measures the pressure gradient at the site of coarctation, and aortography visualizes the defect.

Complications. Hypertension, infective endocarditis, thoracic aortic dissection, and ruptured cerebral aneurysms. Hypertension may persist even after surgical correction of aortic coarctation.

Ebstein anomaly

Pathogenesis. With Ebstein's anomaly, the tricuspid valve is located so low that part of it is located directly in the cavity of the right ventricle. There is atrialization of the right ventricle, leading to a decrease in the volume of the chamber. Tricuspid insufficiency develops. In 75% of patients, an atrial septal defect is also

detected.
Clinic
Symptoms. A wide range of clinical manifestations from an asymptomatic course to conditions with severe cyanosis (depending on the degree of tricuspid insufficiency and the presence of an atrial septal defect).
(1) Symptoms of right ventricular failure are common.
physical signs
(1) Heart sounds. In most cases, T1 and T2 are split, and since T3 and T4 are often present, a three- or four-term gallop rhythm is heard.
(2) Noise. On the edge of the sternum, a systolic murmur of tricuspid insufficiency is heard, sometimes accompanied by palpable trembling.

Diagnostics
a. Electrocardiography. You can find a short R-R interval, delta wave, giant P waves, right bundle branch block.

b. Echocardiography. Delayed closing of the tricuspid valve compared to the mitral valve, as well as displacement of the tricuspid valve down and to the left.

c. Cardiac catheterization. The identification of the same pressure in the right atrium and the atrialized part of the right ventricle is considered pathognomonic.

Eisenmenger syndrome

1. ***Pathogenesis.*** In Eisenmenger's syndrome (which can occur with any intracardiac shunt), the shunt changes from left to right to right to left. Reversion occurs due to irreversible damage to the vessels of the lungs and an increase in resistance in them. These changes lead to a decrease in relaxation of the right heart, an increase in pressure in it, and the development of a right-to-left shunt. Cyanosis is a consequence of a right-to-left shunt.

Clinic
a. Cyanosis may be permanent or appear after exercise. If a PDA is present, the upper torso may be pink and the lower torso may be cyanotic.
b. Angina. Patients with Eisenmenger's syndrome may suffer from exercise-induced chest pain even in the absence of changes in the coronary vessels. The cause of pain may be a decrease in myocardial oxygenation and increased tension in the walls of the right ventricle.
c. Heart failure. Shortness of breath on exertion, ascites, and edema are often observed.

Diagnostics
but. Electrocardiography. Always determine right ventricular hypertrophy.
b. Echocardiography. After injection of isotonic saline with micro-air bubbles, right-to-left shunting of blood in an ASD or VSD can be detected, or a shunt can be detected using a Doppler study.
in. Blood study. In patients with Eisenmenger's syndrome, polycythemia is often observed. The level of Hb can exceed 200 g / l.
d Cardiac catheterization. The pressure in the right ventricle is markedly increased. Oximetry quantifies the degree of shunting. Pure oxygen delivered through a mask does not affect arterial oxygenation.

CARDIOMYOPATHY

Dilated cardiomyopathy

Dilated cardiomyopathy (DCM) is interstitial and perivascular fibrosis of the heart muscle with dilation of all cavities, cardiomegaly, and a progressive decrease in contractile function. Increasing heart failure, arrhythmias and thromboembolism causes death in 50% of patients after 2.5 years from the onset of advanced clinical symptoms of the disease.

Etiology. The cause of most cases of DCM is unknown. Conditions that are important for the development of DCMP.
a Chronic alcoholism is the most common cause of reversible DCM.
b Toxic substances (cobalt, tin, lead, high doses of catecholamines, doxorubicin treatment) can lead to myocardial damage and DCM.
c. Endocrine disorders (thyrotoxicosis, hypothyroidism, acromegaly) are considered as possible causes of DCM. Successful treatment of thyrotoxicosis and hypothyroidism can lead to the regression of DCM symptoms.
d. Metabolic disorders (eg, hypophosphatemia, hypocalcemia, and thiamine deficiency) can cause DCM.
e. Hemoglobinopathies (sickle cell anemia and thalassemia).

Symptoms of DKMP consist of symptoms of right and left ventricular failure.
(1) Typically, symptoms of left ventricular failure precede symptoms of right ventricular dysfunction.
(2) Chest pain may occur without coronary artery occlusion. The cause of pain is considered to be an increase in the oxygen demand of a large mass of myocardium.

The physical signs of DCM are the same as those of heart failure.
A systolic murmur of mitral regurgitation may be heard.
Mitral insufficiency occurs due to an increase in the left ventricle and stretching of the papillary muscles.

Diagnostics
a. ECG. Left ventricular hypertrophy and nonspecific changes in the ST segment and T wave. Often determine the blockade of the left leg of the His bundle.
b. Chest X-ray. Cardiomegaly and signs of pulmonary congestion.
c. Echocardiography. Enlarged and poorly reduced left and right ventricles, as well as enlargement of the atria.
d. Cardiac catheterization is usually not indicated, but in some cases it is necessary for differential diagnosis (aortic stenosis, coronary artery disease).

Hypertrophic cardiomyopathy (HCM)

HCM is accompanied by significant hypertrophy of the interventricular septum. The hypertrophied septum, together with the anterior leaflet of the mitral valve, creates an obstacle to ejection from the left ventricle.

Etiology. In most cases, HCM is inherited, but sporadic cases also occur.

Pathogenesis
a. Mechanism of obstruction

(1) A hypertrophied interventricular septum approaches the anterior leaflet of the mitral valve and blocks the ejection of blood from the left ventricle.
(2) During systole, when blood passes through the narrow area between the septum and the anterior leaflet of the mitral valve, a zone of low pressure is created (Bernoulli effect). There is a displacement of the anterior leaflet of the mitral valve to the septum and narrowing of the outflow tract of the left ventricle.
(3) The contraction of the septum, and hence its thickening, during systole is very small, so it is the anterior leaflet of the mitral valve that plays the main role in the development of obstruction.

b. The degree of obstruction of the outflow tract is individual and may vary from time to time in each individual patient. Conditions that change the size of the left ventricle change the obstruction.
c. Outflow tract obstruction can cause secondary ventricular wall hypertrophy, but the hypertrophied septum will always be thicker than any of the 3 free ventricular walls.

Symptoms
(1) *Angina pectoris.* Patients with HCM often complain of chest pain.
a In most cases, pain is atypical, may occur at rest, and is not always associated with exercise.
b The cause of the pain is not well understood, but patients with HCM have reduced coronary blood flow,

suggesting myocardial ischemia.
(2) *Fainting*
Syncope often occurs in patients with HCM after exercise as a result of a decrease in the size of the left ventricle and increased obstruction. After exercise, afterload decreases due to peripheral vasodilation. The preload decreases, as the activity of the muscles of the lower extremities is reduced, whose contractions contribute to the return of blood to the heart. Arrhythmias, which are common in HCM, can also be a cause of syncope.
(3) *Heart failure*. Dyspnea on exertion, orthopnea, and cardiac asthma are common in patients with HCM. Systolic function is usually normal or elevated.
a Symptoms of heart failure do not occur as a result of systolic dysfunction, but as a consequence of impaired diastolic compliance of the myocardium. Thickened myocardial walls require increased filling pressure for adequate diastolic stretch. This leads to stagnation of blood in the lungs. In the later stages of the disease, the resulting systolic dysfunction exacerbates heart failure.

physical signs
On the left side of the sternum, a systolic ejection murmur is heard. Unlike the murmur in aortic stenosis, it is rarely performed on the vessels of the neck.

Diagnostics

a. *Electrocardiography*. In most cases, left ventricular hypertrophy, nonspecific changes in ST and T, as well as an increase in the left atrium.

b. *Echocardiography* allows diagnosis in most patients.
(1) In asymmetric septal hypertrophy without obstruction, the ratio of septal thickness to ventricular wall thickness is 1:3 or more.
(2) In the obstructive form of the disease, the systolic displacement of the anterior leaflet of the mitral valve, the systolic trembling of the leaflets of the aortic valve and its early closure, corresponding to the peaks and domes on the carotid pulse record, are determined.

c. *Cardiac catheterization.* Carried out to patients before surgical treatment to determine the degree of obstruction. An indication for catheterization is also insufficient information content of echocardiography in a particular patient.

Restrictive cardiomyopathy

With restrictive cardiomyopathy, the structure of the myocardium changes so significantly that it loses its ability to diastolic stretch. The inability of the myocardium to stretch, by limiting the filling of the left ventricle, reduces the stroke volume and increases the filling pressure of the left ventricle.

Etiology. Infiltrative myocardial diseases: amyloidosis, hemochromatosis, idiopathic eosinophilia, carcinoid syndrome, sarcoidosis, endocardial fibroelastosis,

Pathogenesis. Systolic function is usually normal at the onset of the disease, but severe changes in the myocardium lead to a loss of diastolic stretch. The pressure in the left ventricle is higher than normal (regardless of its diastolic volume).
Increased filling pressure leads to the development of congestion in the lungs. With the progression of infiltrative processes, systolic function is also inhibited.
Symptoms of left and right ventricular heart failure are observed in patients with restrictive cardiomyopathy (the symptoms of right ventricular failure are more pronounced).

Physical signs include those found in left and right ventricular failure.

Diagnostics
a. ECG. Reduced voltage of the QRS complex, nonspecific changes in the ST interval and T wave.
b. Chest X-ray. Signs of pulmonary congestion may be associated with normal heart size. Even in the later stages of the disease, when contractility is significantly impaired, the difficult filling of the myocardium

prevents cardiodilatation.
c. echocardiography

(1) Thickening of the walls of the left and right ventricles. The combination of thickening of the wall of the left ventricle and a decrease in the voltage of the ventricular complex on the ECG is characteristic of restrictive cardiomyopathy.
(2) The dimensions of the cavities of the left and right ventricles are usually not changed, while the left and right atria are enlarged.
(3) In amyloidosis, the myocardium is more contrast on the echogram than usual. d. Cardiac catheterization. It is often difficult to distinguish between restrictive cardiomyopathy and constrictive pericarditis on probing. A deep and even image of the diastolic part of the pressure curve in the right and left ventricles is observed in both diseases. In restrictive cardiomyopathy, pressure in the left and right atria and filling pressures in the left and right ventricles are usually not the same (unlike in constrictive pericarditis). Endomyocardial biopsy during catheterization helps to distinguish between these diseases.

PERICARDIAL DISEASES
Acute pericarditis

Etiology

a. Myocardial infarction. Aseptic pericarditis may develop in the first 24 hours of transmural myocardial infarction due to irritation of the pericardium by the adjacent infarct zone. Another type of pericarditis in myocardial infarction - Dressler's syndrome - occurs in the period from the end of the 1st week to several months after a heart attack.

b. Viral infection. The etiology of most cases of acute pericarditis cannot be established. Pericarditis often complicates acute respiratory viral infections.

c. collagenoses. Acute pericarditis may be a manifestation of rheumatoid arthritis, systemic lupus erythematosus, and systemic scleroderma.

d. Bacterial pericarditis. Tuberculosis, streptococcal infections, staphylococci, and a complication of bacterial endocarditis can cause acute pericarditis.

e. Medications. Some commonly used drugs (procainamide, isoniazid, and hydralazine) can cause acute pericarditis.
f. Oncology. Pericarditis can develop secondarily due to tumor metastasis. Lung and breast carcinomas are the most common primary cancer sites.
gl. Uremia in severe chronic renal failure may be the cause of pericarditis.

Symptoms. The most common symptom of pericarditis is chest pain on inspiration.
(1) The pain is localized on the left side and is relieved by sitting and leaning forward.

(2) Sometimes the pain may resemble that of a myocardial infarction and radiate to the neck and arm.
physical signs. The classic is the pericardial friction rub, scratching, auscultated in systole and diastole. Atrial contractions may constitute the third component of the murmur.

Diagnostics
a. Objective examination. A pericardial friction rub confirms the diagnosis of pericarditis.
b. Electrocardiography. Inflammation of the pericardium causes ST segment elevation in various leads. Unlike myocardial infarction, there are no reciprocal changes in the ST segment on the ECG.
c. Echocardiography often reveals fluid effusion into the pericardial cavity.

Exudative (effusion) pericarditis

Pathogenesis. In acute pericarditis, inflammation is often accompanied by leakage of fluid into the pericardial cavity. If fluid builds up slowly, the pericardium has time to stretch, but if fluid builds up quickly, it compresses the heart and prevents it from filling. The condition is defined as cardiac tamponade.

Symptoms. Minor pericardial effusion usually remains asymptomatic.
physical signs. The fluid that accumulates in the pericardium forms a kind of "cushion" around the heart.

(1) There is no visible pulsation in the precordial region, palpation of the apex beat becomes difficult, and heart sounds become muffled.

(2) An effusion may prevent the formation of a pericardial friction rub, but in some cases it is present (even with a significant amount of fluid in the pericardium).

Diagnostics
A. Electrocardiography. Determine the decrease in voltage and electrical alternation of the heart.

B. X-ray of the chest. As fluid accumulates in the pericardial cavity, the shadow of the heart increases. The silhouette of a heart with a liquid level is typical. Significantly enlarged heart without signs of stagnation is considered characteristic of exudative pericarditis.

C. Echocardiography. The echo-free space between the two sheets of the pericardium serves as a diagnostic criterion for exudative pericarditis.

F. Puncture of the pericardium. Pericardiocentesis allows you to determine the fluid in the pericardial cavity, and the study of fluid - the etiology of the disease.
It is necessary to study the cytological composition of the fluid, count and differentiate cells, conduct bacteriological studies for the presence of microorganisms, determine the protein content and LDH activity. After centrifugation, the pellet is examined for atypical cells. For differential diagnosis with rheumatic diseases examine the resulting fluid for antinuclear antibodies and LE cells. Hemorrhagic exudate (typical of cancer and tuberculosis) may be the result of an accidental puncture of the ventricular wall with a needle
Differentiation is facilitated by the following sign: blood from the ventricle coagulates, but not from the exudate.

Cardiac tamponade

Cardiac tamponade is a life-threatening condition, accompanied by the rapid accumulation of large volumes of fluid in the pericardial cavity with the development of severe compression of the organ.

a. The heart cannot fill adequately, and a decrease in filling leads to a decrease in cardiac output.

b. External pressure from the fluid in the pericardium is evenly transmitted to all chambers of the heart. Since the external pressure is usually higher than the normal filling pressure, the pressure within the pericardium, in the right and left ventricles and atria, equalizes in diastole.

Symptoms. Complaints of fatigue, shortness of breath and orthopnea.
physical signs

(1) *Paradoxical pulse*. A slight drop in systolic blood pressure during inspiration is normal, but with cardiac tamponade, 95% of patients experience a drop in systolic blood pressure by 10 mm Hg. and more. This means a decrease in stroke volume during inspiration, which occurs for the following reasons:

a Septum displacement. Inspiratory negative chest pressure increases right ventricular filling by increasing venous return. There is an increase in the right ventricle and displacement of the interventricular septum to the left, reducing the size of the chamber and the ejection from the left ventricle.

b Stretching of the pericardium is a consequence of its downward displacement during inspiration, which increases the compression of the organ and reduces cardiac output.

c Right ventricular enlargement. Increased filling of the right ventricle during inspiration causes its expansion; it occupies a larger volume in the pericardial cavity, which prevents ejection from the left ventricle.

d The increase in the volume of the vascular bed of the lungs, which occurs during inspiration, increases its capacity and makes it difficult to fill the left atrium.

(2) *The filling of the jugular veins* reflects the pressure in the pericardial cavity and in the right atrium.

(3) *A decrease in pulse pressure* occurs as a result of a decrease in the stroke volume of the left ventricle (hence, systolic pressure) and tachycardia, which, by impeding diastolic outflow, maintains normal diastolic blood pressure.

Diagnostics.
Swelling of the jugular veins and a paradoxical pulse in a patient with a decrease in cardiac output confirm the diagnosis of cardiac tamponade.
Cardiac enlargement on x-ray and echocardiographic determination of fluid in the pericardium, combined with clinical symptoms, make the diagnosis reliable.
The certainty of the diagnosis can increase with cardiac catheterization: the same pressure in the right and left atria.

Constrictive pericarditis

Constrictive pericarditis is a diffuse thickening of the walls of the pericardium as a result of previous inflammation, leading to compression of the heart, a decrease in the extensibility of its cavities with a limitation of cardiac output and an increase in filling pressure.

Etiology. Almost all causes of acute pericarditis can lead to constrictive pericarditis.

Symptoms. Shortness of breath on exertion due to limited cardiac output. Orthopnea occurs in 50% of patients; cardiac asthma is an exception.
The symptoms of right ventricular failure predominate.

physical signs

(1) Swelling of the jugular veins occurs due to an increase in systemic venous pressure.

(2) The carotid pulse is normal, but there may be decreased filling due to low stroke volume.

(3) Heart sounds are muffled. In the early phase of diastole, a pericardial murmur may be heard.

(4) Signs of systemic venous hypertension.

Diagnostics

A. Electrocardiography. Decrease in the amplitude of the teeth in the limb leads. Frequent atrial arrhythmias.
B. Radiographically in 50% of patients, pericardial calcification is detected in the form of a radiopaque ring around the heart in lateral projections.
C. Echocardiography often reveals thickening of the pericardium, but this method is not very informative in the diagnosis of constrictive pericarditis.

GENERAL SCHEME OF ECG DECODING.

1. Heart rate and conduction analysis:
- assessment of the regularity of heart contractions
- calculation of heart rate
- determination of the source of excitation
- evaluation of conduction functions.

2. Determining the position of the electrical axis of the heart (EOS).

3. Analysis of the atrial P wave.

4. Analysis of the ventricular QRST complex:
- analysis of the QRS complex
-analysis of the S-T segment
- T wave analysis
- analysis of the Q-T interval

5. ECG conclusion.

HEART RATE AND CONDUCTIVITY ANALYSIS

Heart rate regularity is assessed by comparing the duration of R-R intervals between successive cardiac cycles, heart rate is calculated based on the R-R interval, using the formula HR=60/R-R duration. To determine the source of excitation, first of all, it is necessary to find the P wave, it is most clearly visible in the II standard lead. Irregularity, inversion, absence of a P wave indicates a non-sinus origin of the heart rhythm. Conductivity is assessed by sequential analysis of each ECG complex, paying special attention to the regularity of the complexes, the P-Q interval, the shape of the QRS complex.

Heart rhythm disturbances can be primary (independent) and secondary (as a complication of the underlying disease).
By pathogenesis: organic and functional.

The most common forms of arrhythmias are divided into groups depending on the leading pathogenetic factor leading to rhythm disturbance.

1. Arrhythmias due to a violation of the automatism of the sinus node:
-sinus tachycardia
- sinus bradycardia
- sinus arrhythmia (respiratory)

2. Arrhythmias due to impaired excitability:
- extrasystole
- paroxysmal tachycardia (supraventricular and ventricular)

3. Arrhythmias due to conduction disorders - blockade:
- sinoauricular
- intra-atrial
-atrioventricular (complete transverse)
- intraventricular (legs of the bundle of His or their branches).

4. Arrhythmias due to impaired excitability and conduction:
- atrial fibrillation

- atrial flutter
- flutter and flicker (fibrillation) of the ventricles.

ARRHYTHMIAS DUE TO DISTURBANCE OF THE SINUS NODE

Sinus tachycardia is the simplest, but most common form of arrhythmias, in which more than 90 impulses per minute occur in the sinus node, sometimes reaching 150-160. Under physiological conditions, it can occur when eating, physical and emotional

tension. In pathological conditions, it is noted with neuroses, fever, cardiac insufficiency, anemia, thyrotoxicosis, during pain.
It is caused by a direct effect on the sinus node of biological substances that increase its excitability, or by a change in the tone of the autonomic nervous system.
Patients complain of a sensation of palpitations, in pathological conditions it may be accompanied by shortness of breath. Objectively, there is an increase in heart rate, increased heart sounds.
A P wave is recorded on the ECG, indicating the occurrence of an impulse in the sinus node, the T-P interval is shortened, and T can be superimposed on P. The QRS complex does not change.

Sinus bradcardia - due to a decrease in the excitability of the sinus node, which, first of all, depends on the strengthening of the influence of the parasympathetic nervous system on the heart or the decrease in the influence of the sympathetic. Heart rate decreases to 50-40 per minute. Under physiological conditions, it can be in well-trained athletes. In pathological conditions, it is noted with an increase in intracranial pressure, myxedema, typhoid fever, jaundice, starvation, poisoning.
Patients are worried about dizziness, short-term loss of consciousness due to cerebral ischemia is possible. Objectively: a rare pulse, there may be a weakening of the sonority of tones.
On the ECG, the T-P interval increases, reflecting the lengthening of the electrical diastole of the heart. Atrial and ventricular complexes are not changed.

Sinus arrhythmia - a changing heart rate depending on the phases of breathing, due to fluctuations in the tone of the vagus nerve. On inhalation, the heart rate increases, on exhalation it slows down. It is observed in children and young men, in those recovering from infectious diseases, in some diseases of the central nervous system.
There are no complaints. Objectively: an increase in heart rate and pulse on inspiration and a decrease on expiration. Holding the breath eliminates this type of arrhythmia.

On the ECG, the duration of the R-R intervals changes with normal duration and shape of the teeth.

An extraordinary contraction of the heart is due to an additional (heterotopic or ectopic) impulse that can occur in any part of the conduction system of the excited myocardium. In practically healthy people, extrasystole occurs as a result of overexcitation of parts of the conduction system of the heart due to the influence of the extracardiac nervous system, with the abuse of smoking, strong tea, and coffee. In pathological conditions, it can be observed in inflammatory, dystrophic, sclerotic lesions of the myocardium, occur reflexively in diseases of the abdominal organs, accompany hormonal disorders and electrolyte metabolism disorders.

Depending on the location of the ectopic focus of excitation, there are: supraventricular (supraventricular), which are divided into atrial and atrioventricular, and ventricular.

If there are several ectopic foci, then the extrasystole is called polytopic.

If extrasystoles alternate with normal impulses in a certain order, then such extrasystole is called allorhythmia (bigeminia, trigeminia, quadrigeminia, etc.).

Complaints about the sensation of interruptions in the region of the heart or stop ("fading"), followed by a strong blow.

Extrasystoles are relatively easy to catch when palpating the pulse - an extraordinary, smaller in content, contraction, after which the next pulse wave follows through an unusually long interval (the so-called compensatory pause).

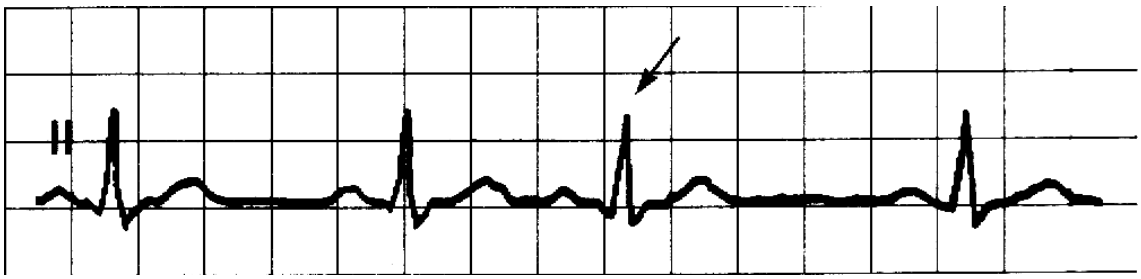
On the ECG, all extrasystoles are characterized by premature appearance of the cardiac complex, lengthening of the pause between the extrasystolic and normal contractions.

With atrial extrasystoles, an impulse occurs in any part of the atria. On the ECG, the P wave remains in front of the ventricular complex, but its height may change. The P-Q interval is shortened, the QRS complex is not changed.

With atrioventricular extrasystoles, an impulse occurs in the AV node, which consists of three parts. If the extrasystole comes from the upper part of the node, then the P wave is negative, because. the impulse propagates retrogradely from the AV node to the sinus node. The P-Q interval is shortened, the QRST complex has the correct shape.

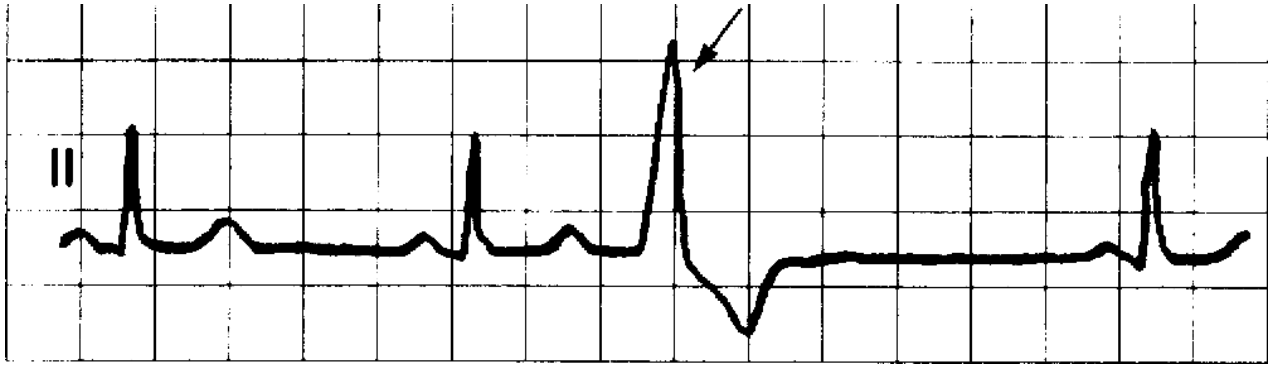
With an extrasystole from the middle part of the AV node, the P wave is absent, because excitement comes to ventricles and atria almost simultaneously and the P wave merges with the ventricular complex, the QRST complex does not change. With extrasystoles from the lower part, the P wave is negative and is located after the initial part of the QRS complex, because excitation reaches the ventricles faster than the atria. The P-Q interval is absent, the QRS complex is of the usual form.

Arrow indicates supraventricular extrasystole



Ventricular extrasystoles - are characterized by the occurrence of impulses at the level of the intraventricular conduction system, i.e. below the AV node. Therefore, the following is noted on the ECG: the P wave is absent, the QRST complex is deformed, broadened, of high amplitude, the T wave is directed back in relation to the QRS, and the S-T interval directly passes into the T wave. Right ventricular and left ventricular extrasystoles differ. With a right ventricular extrasystole, an extrasystolic complex with a high R wave is recorded in the first standard lead, and with a deep S wave in the third standard lead. A high R wave in the third standard lead and a deep S in the first standard lead are characteristic of the left ventricular extrasystole.

Arrow indicates ventricular extrasystole

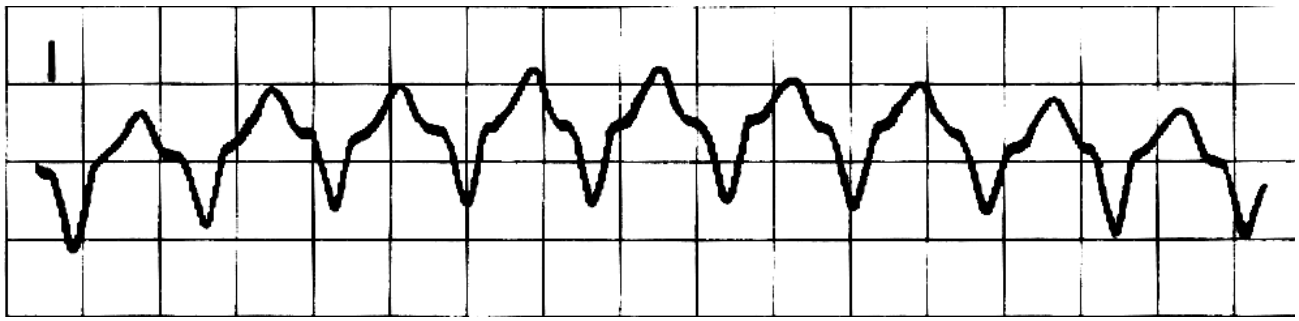


Paroxysmal tachycardia - characterized by a sudden increase in heart rate up to 160-220 per minute and above. Such a sharp increase in heart contractions occurs due to the appearance of a long chain of extrasystoles. This is due to the fact that with a high activity of the ectopic focus, it can become pacemaker.

There are supraventricular and ventricular forms of paroxysmal tachycardia.

Symptoms: sudden palpitations, tightness in the chest, discomfort (sometimes pain) in the region of the heart, shortness of breath, weakness, nausea, pallor of the skin, with a prolonged attack - cyanosis. Swelling and pulsation of the jugular veins, since with an increase in the rhythm, atrial contraction begins earlier than the ventricular systole ends. In this case, blood from the atrium is expelled back into the veins, causing a pulsation.

On the ECG with a supraventricular form, there is a frequent rhythm (160 per minute or more), the P wave can be registered or hidden in the QRS complex, the shape of the ventricular complex does not chang



In the ventricular form, the QRS complex is deformed and widened.

ARRHYTHMIAS DUE TO CONDUCTION DISORDERS - BLOCK

Conductivity is the ability of the heart muscle to conduct an impulse from its place of origin (under normal conditions, from the sinus node) to the working muscles of the heart. The conduction of the impulse is slow in the AV node, which ensures the contraction of the ventricles after completion of the atrial contraction. Due to this, during atrial contraction, blood fills the still relaxed ventricles.

Pacemakers:

- Sinus node - located in the wall of the right atrium above its ear at the confluence of the superior vena cava. Normally, it is the center of automatism (first-order pacemaker). Here impulses are generated with a frequency of 70-90 per minute

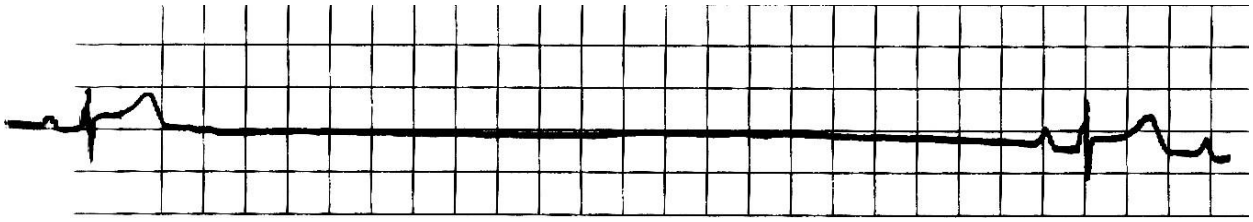
- Atrioventricular node - located in the thickness of the interventricular septum on the right at the border of the atria and ventricles. Can be pacemaker II order in case of damage to the sinus node, the impulses will occur at a frequency of 40-50 per minute.

- Bundle of His - lies in the upper part of the interventricular septum, in the thickness of the ventricles is divided into Purkinje fibers. In case of disturbances in the overlying parts of the conducting system, it can become a third-order pacemaker. The bundle of His generates impulses at a frequency of 30-40 per minute, and Purkinje fibers - 15-20 per minute.

Violation of conduction leads to blockades.
Sinoauricular blockade is characterized by a delay in the sinus node of the impulse, which then does not spread to the atria.

There are no complaints. Objectively: periodic loss of heart contractions and pulse beat.

On the ECG against the background of the correct sinus rhythm periodically - loss of the cardiac complex.



Atrioventricular block is a conduction disorder in which the passage of an impulse through the AV node is inhibited. The severity is divided into three degrees.

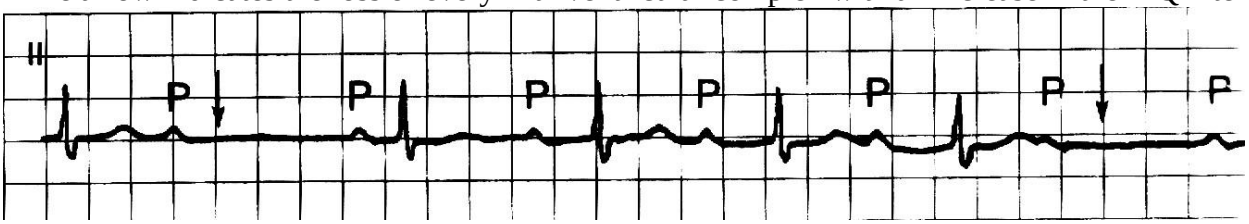
The first - is detected only on the ECG by lengthening the P-Q interval. Without clinical manifestations.

The second degree can be expressed in two types.

Complaints: "fading" or cardiac arrest, slight dizziness. Objectively: on palpation of the pulse, there is a periodic loss of a pulse beat.

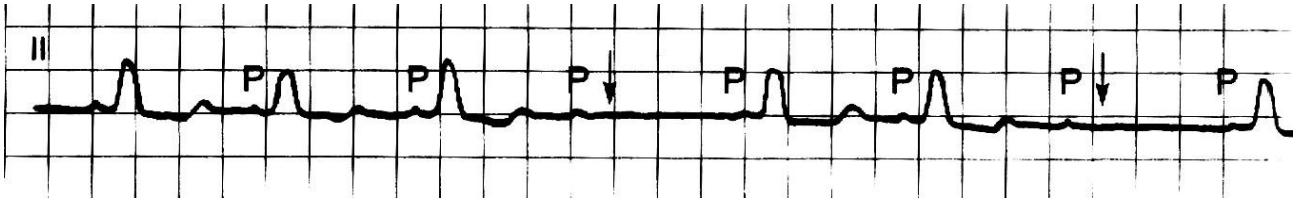
The first type (Mobitz-1) is characterized by an increasing slowdown in AV conduction up to complete blocking of the impulse, which is manifested by a loss of heart contraction. On the ECG, a P wave is recorded, and the P-Q interval lengthens with each complex, which ends with the loss of one ventricular complex (Samoilov-Wenckenbach periods).

The arrow indicates the loss of every fifth ventricular complex with an increase in the P-Q interval.



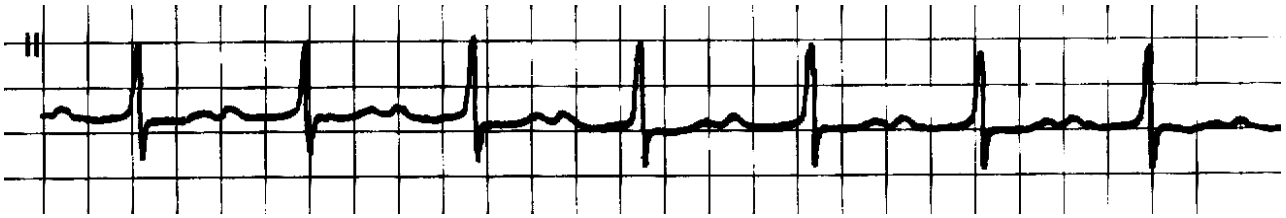
The second type (Mobitz-2) - prolongation of the P-Q interval before the prolapse of the ventricular complex is not observed. Fallout of the complex can be both regular and irregular.
The arrows indicate the loss of first the fourth and then the third QRS complex with an unchanged P-Q

interval



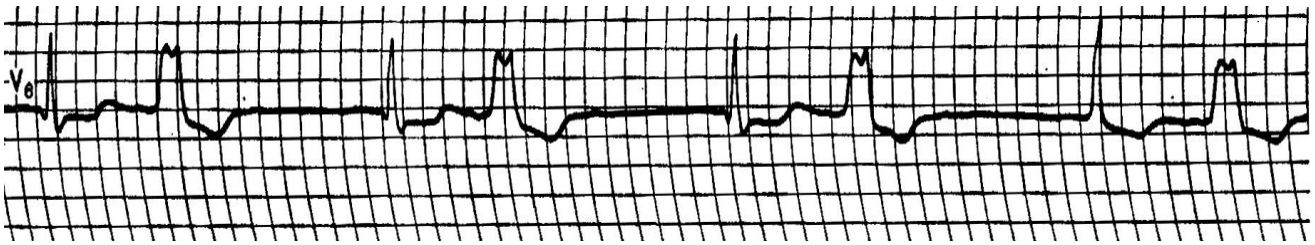
Third degree (complete AV block) - atrial impulses are not conducted to the ventricles, ventricular excitation has its own rhythm, and the atria have their own Morgagni-Adams-Stokes symptom.

Causes of AV blockade.
1. Acute ischemia - myocardial infarction.
2. Infections: rheumatism, infective endocarditis, diphtheria.
3. Medicines.
4. Parasympathicotonia (athlete's heart).
Complaints: periodic sudden loss of consciousness. With a duration of more than 15-20 seconds, muscle (epileptiform) cramps occur. Breathing is deep, the skin turns pale sharply. The pulse is rare or not defined. If automatism is not restored for a long time, a lethal outcome is possible.



Intraventricular block: more common as bundle branch block. They can be partial (the branch of the leg is blocked), complete (the entire leg is blocked), isolated (blockade of the left or right leg), combined (blockade of the branches of the left and right legs).
There are no complaints. On auscultation, splitting or bifurcation of tones due to asynchronization in the activity of the ventricles.

With blockade of the right leg, excitation spreads to the right ventricle from the left. As a result, there is a high and wide R wave in the right chest leads. With blockades of the left leg, the excitation of the left ventricle occurs after the excitation of the right ventricle. In the right chest leads, there is a small R and a wide, deep S. In the left chest leads, the R wave is widened and notched. Usually, the appearance of a bundle branch block reflects an exacerbation of the underlying heart disease.



3. Lesson plan.

To study the micro-, macropreparations. Describe macropreparations according to the scheme:

1. ***Macropreparation "Defect of the heart".*** Pay attention to the size of the heart, the thickness of the wall of the atria and ventricles, the volume of the papillary and trabecular muscles; cavity sizes; the consistency and color of the myocardium; length, thickness and transparency of chordal filaments; the shape of the opening of the affected valve; thickness, transparency, shape and fusion of the leaflets of the affected valve.
2. Macropreparation "Fibrinous pericarditis (hairy heart)". Pay attention to the thickness of the pericardium, the color and type of overlays, the connection with the underlying tissues.

Draw micropreparations in an album and mark pathological changes.

1. ***Micropreparation "Myocarditis in diphtheria"*** (staining with hematoxylin and eosin). Pay attention to the localization and cellular composition of the inflammatory infiltrate, the state of the cytoplasm and nuclei of cardiomyocytes.
2. ***Micropreparation "Fibrinous pericarditis"*** (staining with hematoxylin and eosin). Pay attention to the localization, structural features of the exudate, the state of the cells that make up the pericardial tissue.

Solve the following situational problems and write the answers in a notebook.

Case 1
In patient M., aged 56, with signs of heart failure, the examination revealed:
1. Stenosis of the left atrioventricular orifice.
2. Expansion of the left atrium.
3. Stagnation in the pulmonary circulation.
4. Violations of the function of the right ventricle.
5. Stagnation in the systemic circulation.
6. Oxygen starvation of the circulatory type.
7. Shortness of breath.

Determine the main link in the pathogenesis of this pathology, the elimination of which will cause the elimination of all the above disorders.

Case 2
Patient B., 56 years old, was taken by ambulance to the intensive care unit of the hospital with complaints of severe pressing pain in the chest, lasting about three hours and not relieved after repeated administration of nitroglycerin. On examination: a state of moderate severity; physique of hypersthenic type; there is hyperemia of the face, acrocyanosis. On auscultation: fine bubbling rales in the lungs, respiratory rate -20 per minute; heart sounds are muffled, rhythmic, pulse - 80 per minute.
BP - 90/65 mm Hg. Art. Blood test: Hb - 196 g / l, erythrocytes - 6.0x1012 / l, leukocytes - 9.0x109 / l, platelets - 450.0x109 / l, ESR - 15 mm / hour.
Prothrombin index - 120% (norm - up to 105%). In the bone marrow biopsy, the picture is characteristic of erythremia.

1. What pathological processes have developed in the patient?
2. What are the possible causes that caused each of these pathological processes? What is their relationship?
3. What are the mechanisms of heart damage in this patient?
4. Is there a pathogenetic relationship between cardiopathy and polycythemia? If yes, what is it?

Case 3

Patient H., 30 years old, intravenous drug addict; complains of weakness, fever during the last 5 days, increased fatigue, severe sweating. On examination: low nutrition, body temperature 39.30C, skin of the color "coffee with milk" with multiple petechial hemorrhages, especially pronounced on the skin of the forearm, there are traces of intravenous injections with signs of an inflammatory reaction. Auscultation of the heart marked systolic murmur. Blood culture analysis revealed Staphylococcus aureus in the blood. Hb - 106 g / l, erythrocytes - 3.7x1012 / l, leukocytes - 11.0x109 / l, platelets - 200.0x109 / l, ESR 30 mm / h.

1. What pathological processes have developed in the patient?

2. What are the possible causes that caused each of these pathological processes? What is their relationship?
3. What are the mechanisms of heart damage in this patient?

Case 4

A 38-year-old man died of chronic cardiovascular insufficiency. An autopsy revealed an enlarged flabby heart weighing 630 g, with a uniform thickening of the walls up to 2.7 cm. The cavities of the heart are reduced. Valves and coronary arteries of the heart are unchanged.
Microscopically, the myocardium consists of hypertrophied, chaotically located cardiomyocytes with jagged nuclei surrounded by a light rim. There is diffuse sclerosis of the stroma with a disordered arrangement of collagen fibers.

1. What pathological process has developed in this patient?
2. Based on the available data, explain the pathogenesis of changes in the patient's heart.
Task 5

Case 5

Microscopic examination of the heart of a 46-year-old man who died of acute heart failure revealed a diffuse inflammatory infiltrate consisting of lymphocytes, plasma cells, isolated eosinophils, macrophages, and giant cells surrounding small foci of cardiomyocyte necrosis. Conclusion:
1. What pathological process has developed in this patient?
2. Based on the available data, explain the pathogenesis of changes in the patient's heart.

Case 5

In a 40-year-old man, weakness and arrhythmia persisted for 2 months after suffering from influenza, and heart failure increased, from which he died. Histologically, the myocardium revealed diffuse lymphomacrophage infiltration with an admixture of eosinophils, dystrophy and small foci of necrosis of cardiomyocytes, edema, vascular congestion, and diapedetic hemorrhages.
1. What pathological process has developed in this patient?
2. Based on the available data, explain the pathogenesis of changes in the patient's heart.

List of recommended literature:
Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

2. Additional literature:

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