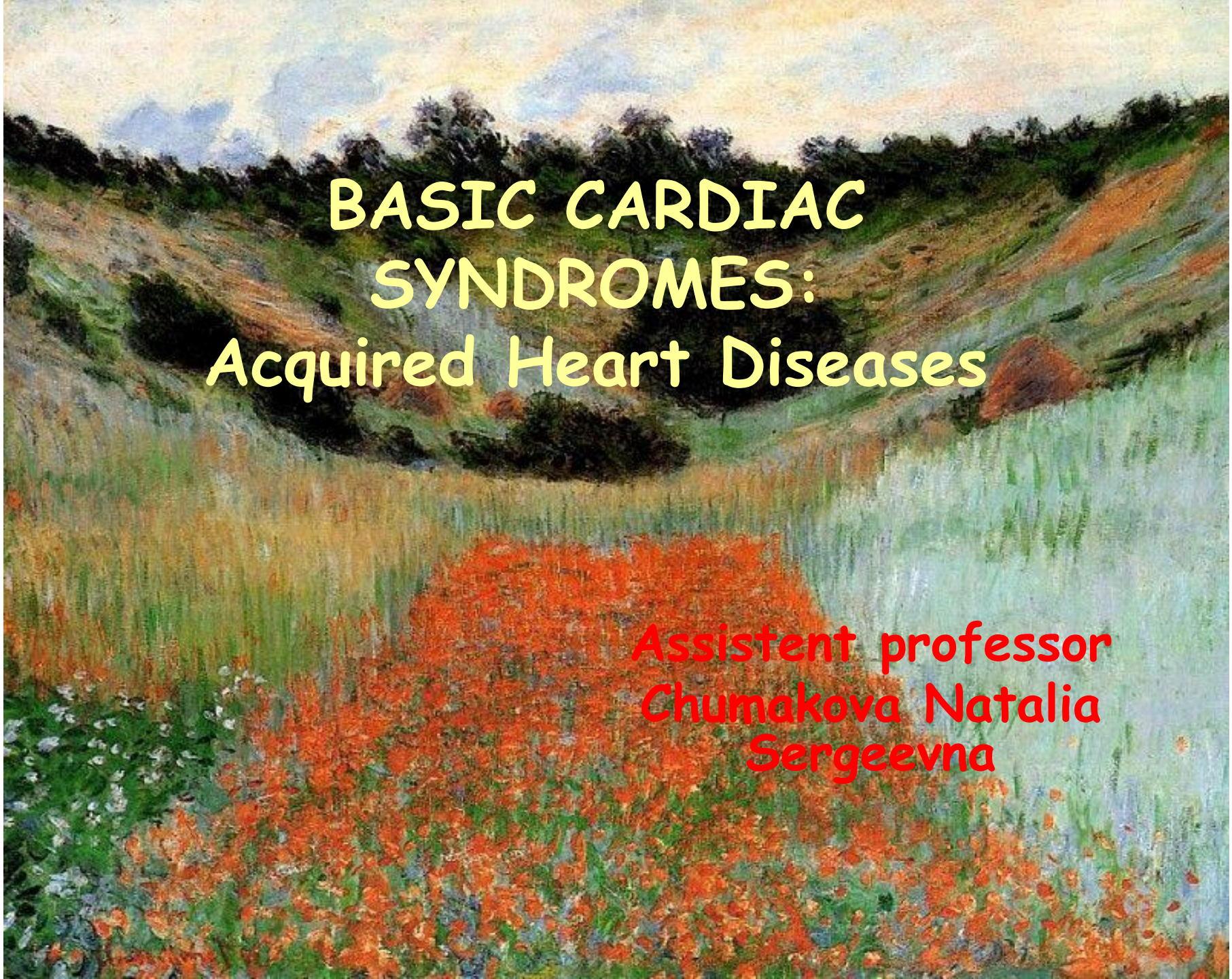


BASIC CARDIAC SYNDROMES: Acquired Heart Diseases

Assistent professor
Chumakova Natalia
Sergeevna

ACUTE RHEUMATIC FEVER

ACUTE RHEUMATIC FEVER

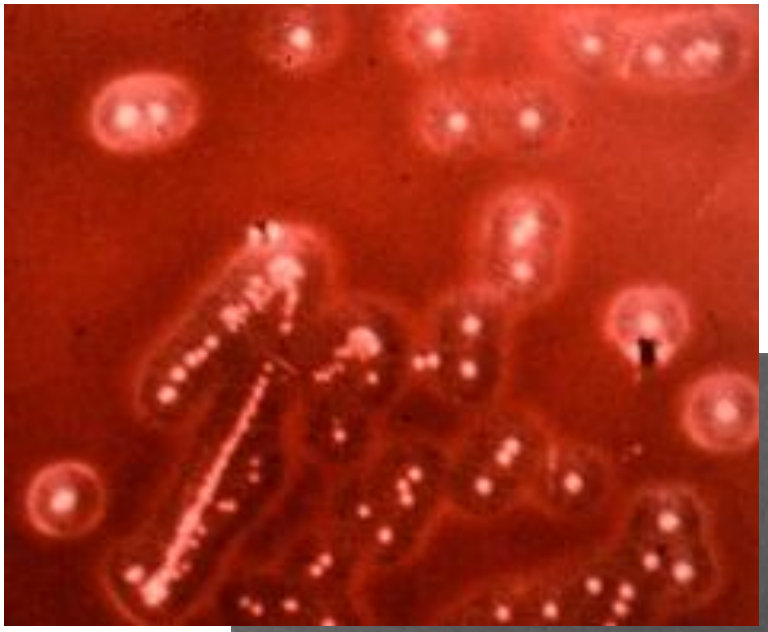
- **Definition:** Acute rheumatic fever (rheumatism) is a general infectious and allergic disease in which connective
- tissues, mainly of the cardiovascular system, are affected by inflammation;
- joints, serous membranes, internal organs, and the central nervous system are often involved.

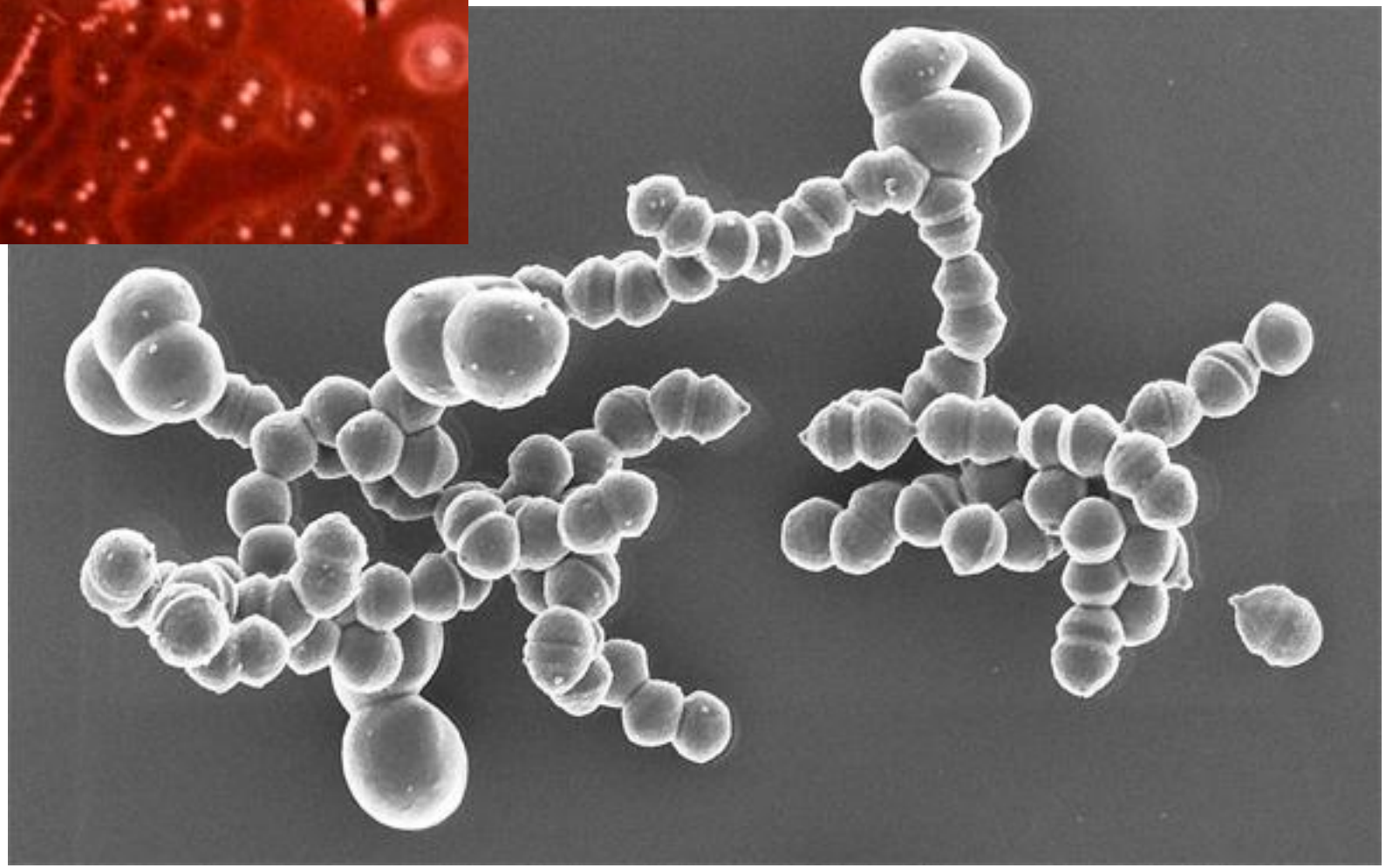
ACUTE RHEUMATIC FEVER

- Rheumatism is a collagenous disease, i.e. a disease characterized by a systemic and progressive derangement of connective tissue.
- Rheumatism was classified as an independent disease with typical affections not only of the points but also mainly have the heart in 1835 by a French clinicist Bouillaud and in 1836 by the Russian physician Sokolsky. Until that time rheumatism had been considered a disease of joints.

Etiology

beta-hemolytic streptococci





Pathogenesis

- Pathogenesis of acute rheumatic fever is complicated
- and includes three important phases:
- (1) acute, mainly oro-pharyngeal,infection by the group A streptococcus;
- (2) toxic effect of streptococcal extracellular products on the host connective tissues
- (3) an abnormal or dysfunctional immune response to one or more somatic or extracellular antigens produced by all (or perhaps only by some) group A streptococci

- At the present time, the development of the disease is described as follows. Most persons affected by streptococcus develop stable immunity.
- This immunity does not develop in 2-3 per cent of the affected subjects due to weakness of their defense mechanisms and they become sensitized by the streptococcus antigen. In these conditions the infection re-enters the body to cause a hyperergic response in connective tissues; clinical signs of the disease thus develop. Autoimmune processes are very important in the onset of rheumatism.

- The affected connective tissue acquires antigenic properties; auto-antigens (secondary antigens) cause formation of aggressive autoantibodies. They affect not only the connective tissue that has already been
- affected by the primary antigen but also intact tissue to aggravate the pathology. Re-infection, cooling, and overstrain promote formation of new auto-antigens and auto-antibodies to strengthen the pathological reaction of the upset immunity and to provide conditions for the recurring progressive course of the disease.

ревматизм

Pathological anatomy

- Four phases of derangement of connective tissue are differentiated in rheumatism:
- (1) mucoid swelling;
- (2) fibrinoid changes;
- (3) granulomatosis;
- and (4) sclerosis

Classification

- 1. Acute rheumatic fever
- 2. Rheumatic disease of heart (Rheumatic disease of heart – with defect or vice of heart, valvular heart disease).

Classification of acute rheumatic fever

1. Clinical variants:

- acute rheumatic fever,
- recurrent acute rheumatic fever.

2. Clinical manifestations:

1) basic clinical manifestations - carditis, arthritis, chorea, rheumatic nodules, and erythema annulare (erythema marginatum);
2) additional clinical manifestations – arthralgia, (poly)serositis, abdominal syndrome.

3. Degree of activity: 1) minimal activity, 2) moderate activity, 3) maximal (high) activity.

4. Clinical outcome: - recovery; - chronic rheumatic disease of heart: without heart valvular defect; heart valvular defect. 5. Functional class of chronic heart failure according to NYHA (or degree of chronic heart insufficiency).

Clinical picture of acute rheumatic fever

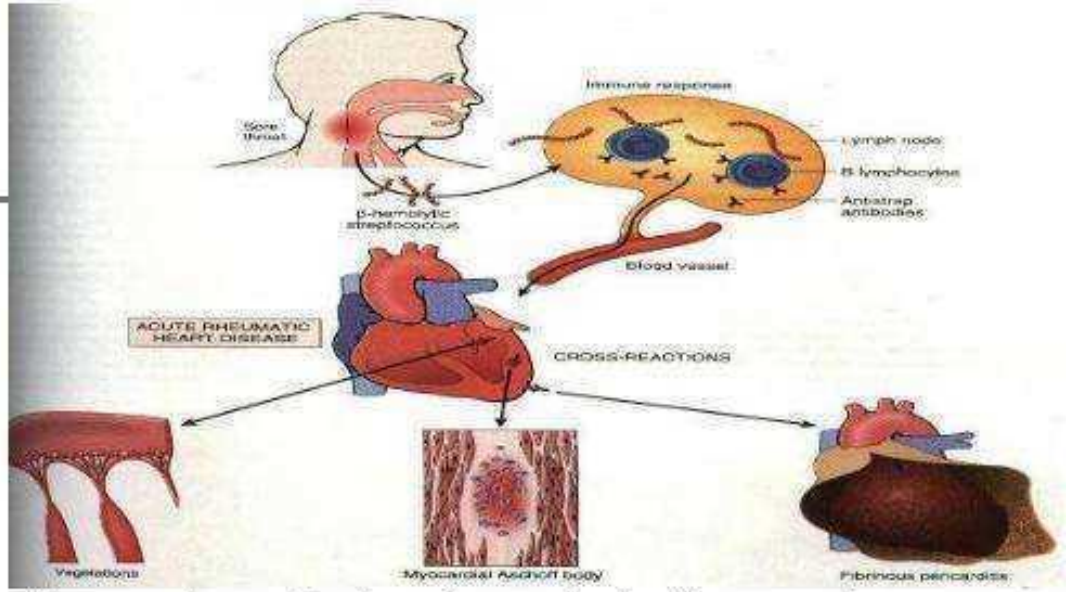
- Acute rheumatic fever affects children, adolescents, and young adults (between ages 4 and 20). The disease develops in 2-6 weeks after acute streptococcal pharyngeal infection (tonsillitis, pharyngitis or scarlet fever). The typical onset of the disease is subfebrile (less frequently febrile) temperature, weakness, and sweating during 1-3 weeks. Simultaneously (or several days later) patient feels pain in the joints, palpitation and intermissions in the work of the heart, the feeling of heaviness or pain in the heart, and dyspnea.

- The major manifestations of the acute rheumatic fever are migratory polyarthritis, chorea, carditis, subcutaneous nodules, and erythema marginatum. These can appear alone or in combination and produce many clinical patterns. Cutaneous and subcutaneous features are uncommon and almost never occur alone, usually developing in a patient who already has arthritis, chorea, or carditis.

Rheumatic carditis

- The rheumatic carditis is a pancarditis involving myocarditis (~100%), and endocarditis (~60%), pericarditis (~20%).

Cardiovascular form of rheumatism

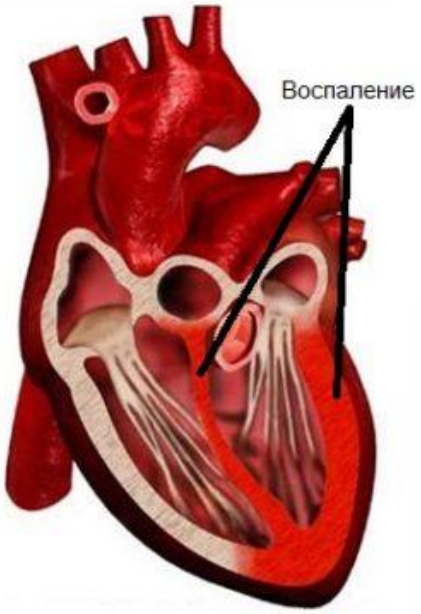


- **Endocarditis:** *valvular, chordal, atparietal; acute diffuse, acute warty, fibroplastic, relapsing warty*
- **Myocarditis**: *granulomatic (nodular productive), diffuse interstitial exudative, focal interstitial exudative.*
- **Pericarditis**: *serous, serofibrinous, fibrinous.*
- **Vasculitis**: *capillaritis, arteriolaritis, arteritis.*

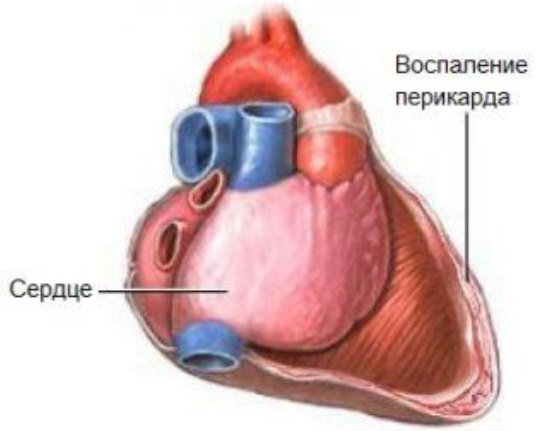
Локализации кардита

Эндокардит
(внутренняя оболочка сердца)

Миокардит
(сердечная мышца)



Перикардит
(внешняя оболочка сердца)



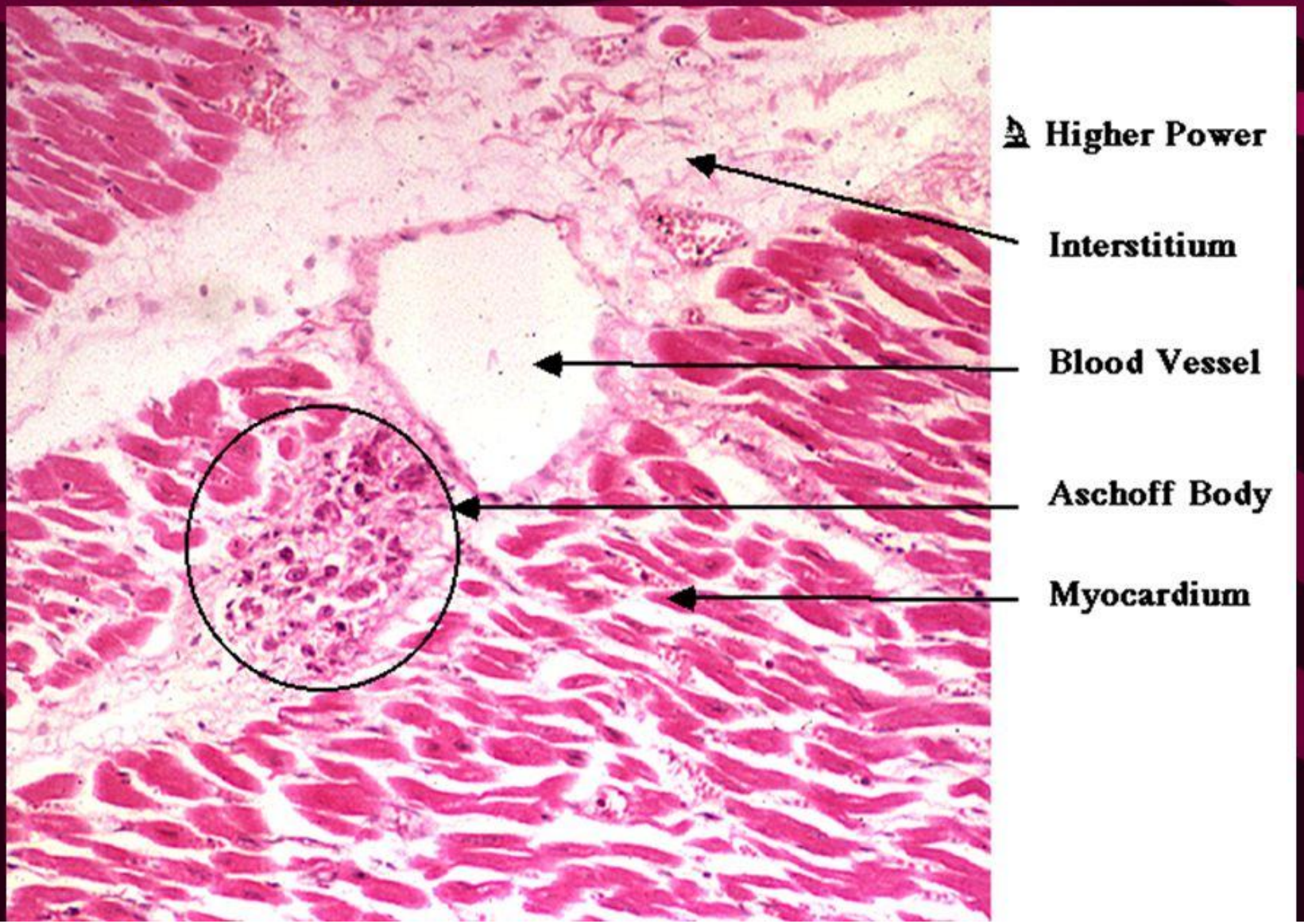
Rheumatic myocarditis

- Rheumatic myocarditis is characterized by dyspnea, the feeling of heaviness and pain in the heart, palpitation, and intermissions in the heart work. Percussion detects enlargement of the heart. Auscultation reveals decreased heart sounds (especially the first sound); gallop rhythms develop in severe affection of the myocardium. A soft systolic murmur can be heard at the heart apex. It is associated with relative incompetence of the valve or affection of the papillary muscles

Rheumatic myocarditis

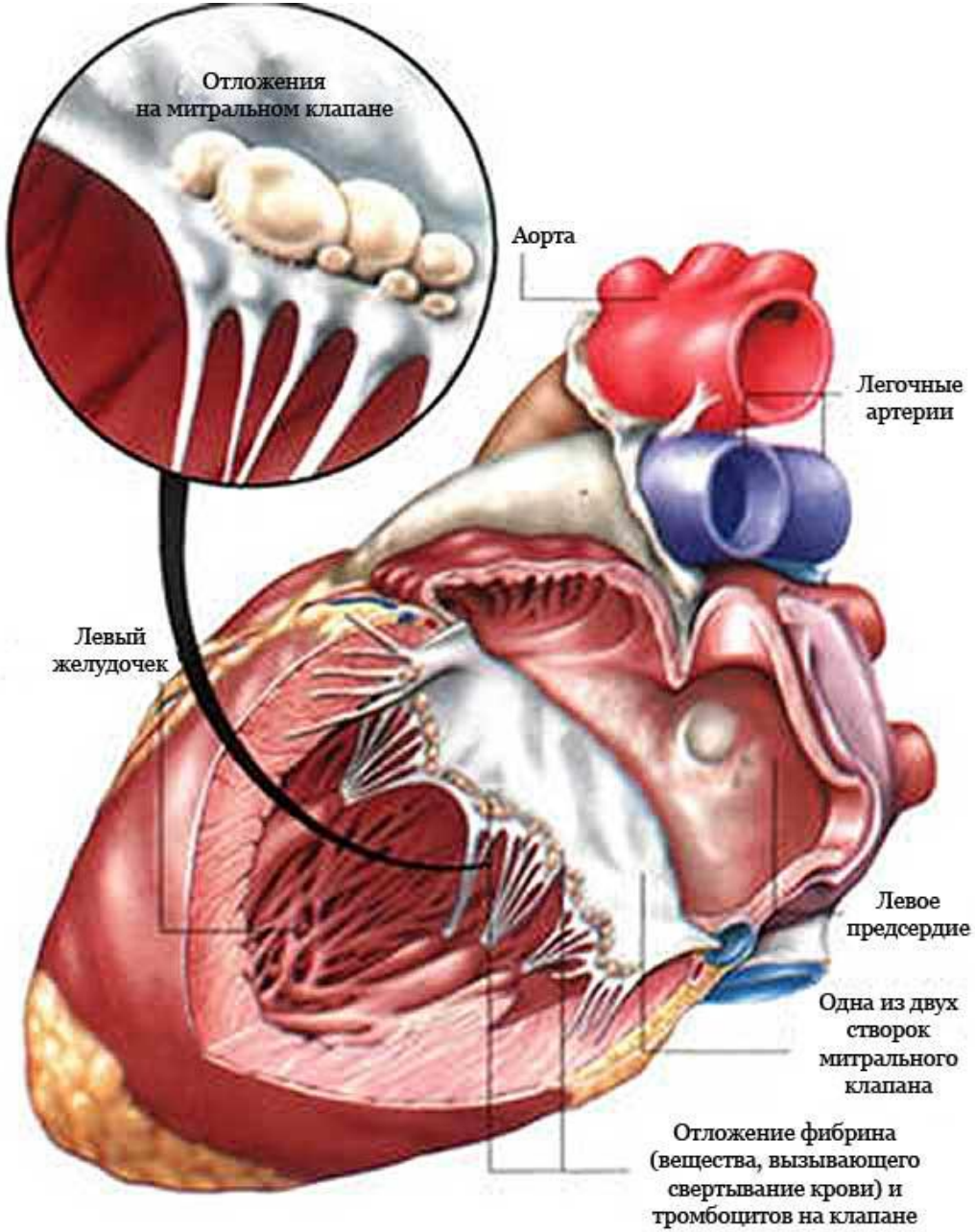
- The pulse is small and soft; tachycardia and arrhythmia are frequent. Blood arterial pressure is usually decreased. A prolonged PR interval may be present on ECG as well. Circulatory insufficiency rapidly develops in grave diffuse myocarditis. Myocardial cardiosclerosis develops in benign outcome of the disease.

Aschoff Body in Acute Rheumatic Carditis



Rheumatic endocarditis

- Rheumatic myocarditis usually concurs with rheumatic endocarditis (rheumocarditis). The mitral valve is mostly affected in endocarditis. Next in incidence follows the aortic valve; the tricuspid valve is affected still less frequently.


Отложения
на митральном клапане
Аорта
Легочные
артерии
Левый
желудочек
Левое
предсердие
Одна из двух
створок
митрального
клапана
Отложение фибрина
(вещества, вызывающего
свертывание крови) и
тромбоцитов на клапане

Rheumatic endocarditis

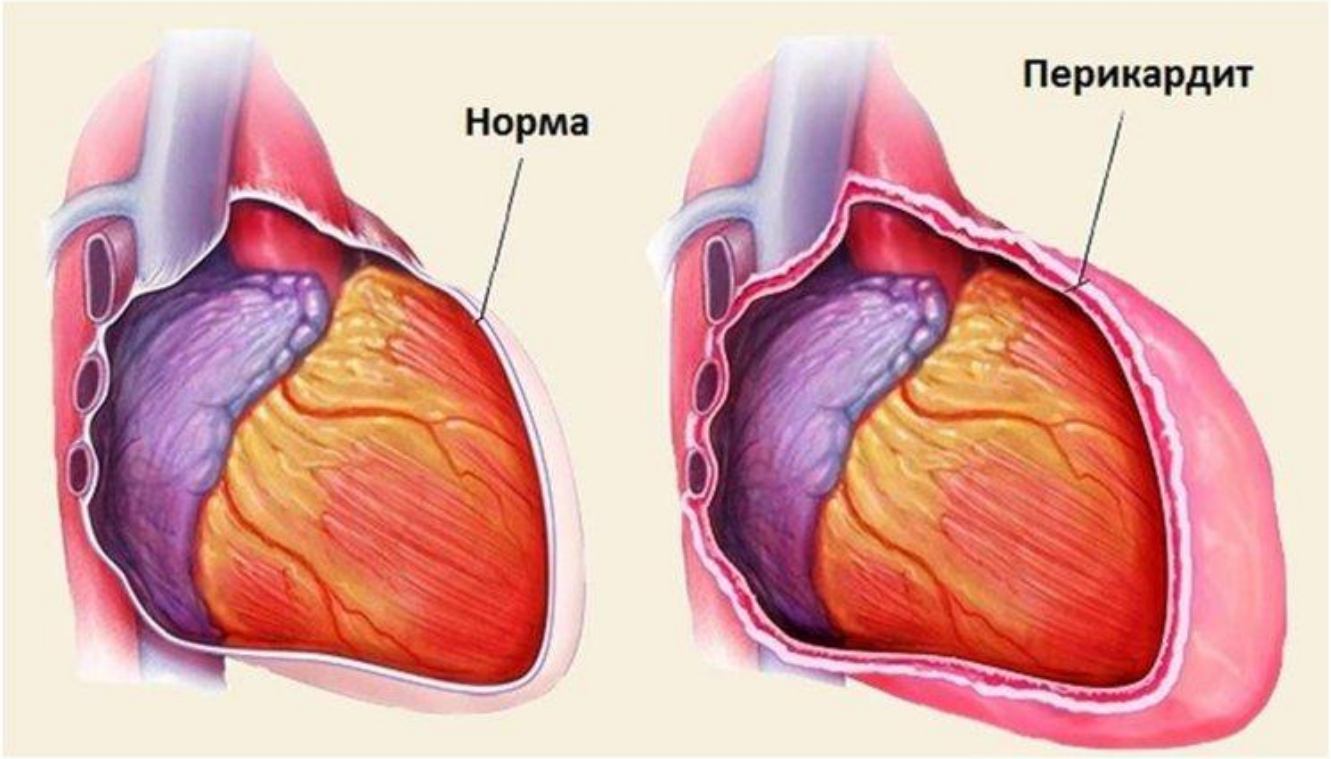
- Early endocarditis are not pronounced (symptoms of myocarditis prevail). At earlier stages of endocarditis systolic murmurs become coarser than in myocarditis; the murmur becomes louder after exercise; in some cases it becomes "musical". Diastolic murmur may be heard as well. It is probably explained by deposition of thrombotic mass on the valve cusps which produces turbulence in the blood flow as it passes from the atrium to the 71 ventricle. These thrombotic deposits on the valves can leave their seat and become the cause of embolism or infarctions in various organs (e.g. the kidneys or the spleen). If early attacks of rheumatic endocarditis are treated timely, development of the valvular heart disease may be prevented.

- The introduction of echocardiography has assisted in the identification of subtle abnormalities of the mitral valve, and these may be present in an additional 20% of patients who do not have an audible heart murmur. The mitral valve is involved most frequently, followed by the aortic valve. However, isolated aortic valve disease as a consequence of acute rheumatic fever is quite rare. In patients with aortic valve disease due to rheumatic fever, the mitral valve is almost always simultaneously affected.

Rheumatic pericarditis

- Rheumatic pericarditis is may be dry (fibrinous) or exudative. Rheumatic pericarditis may present with chest pain, dyspnea, fever, pericardial rub friction murmur, ECG changes, or radiologic changes or may be discovered incidentally in the course of a systemic illness. Dull or sharp precordial or substernal pain may radiate to the neck, trapezius ridge (especially the left), or shoulders. Pain varies from mild to severe and is usually aggravated by thoracic motion, cough, and respiration; it may be relieved by sitting up and leaning forward.

Rheumatic pericarditis

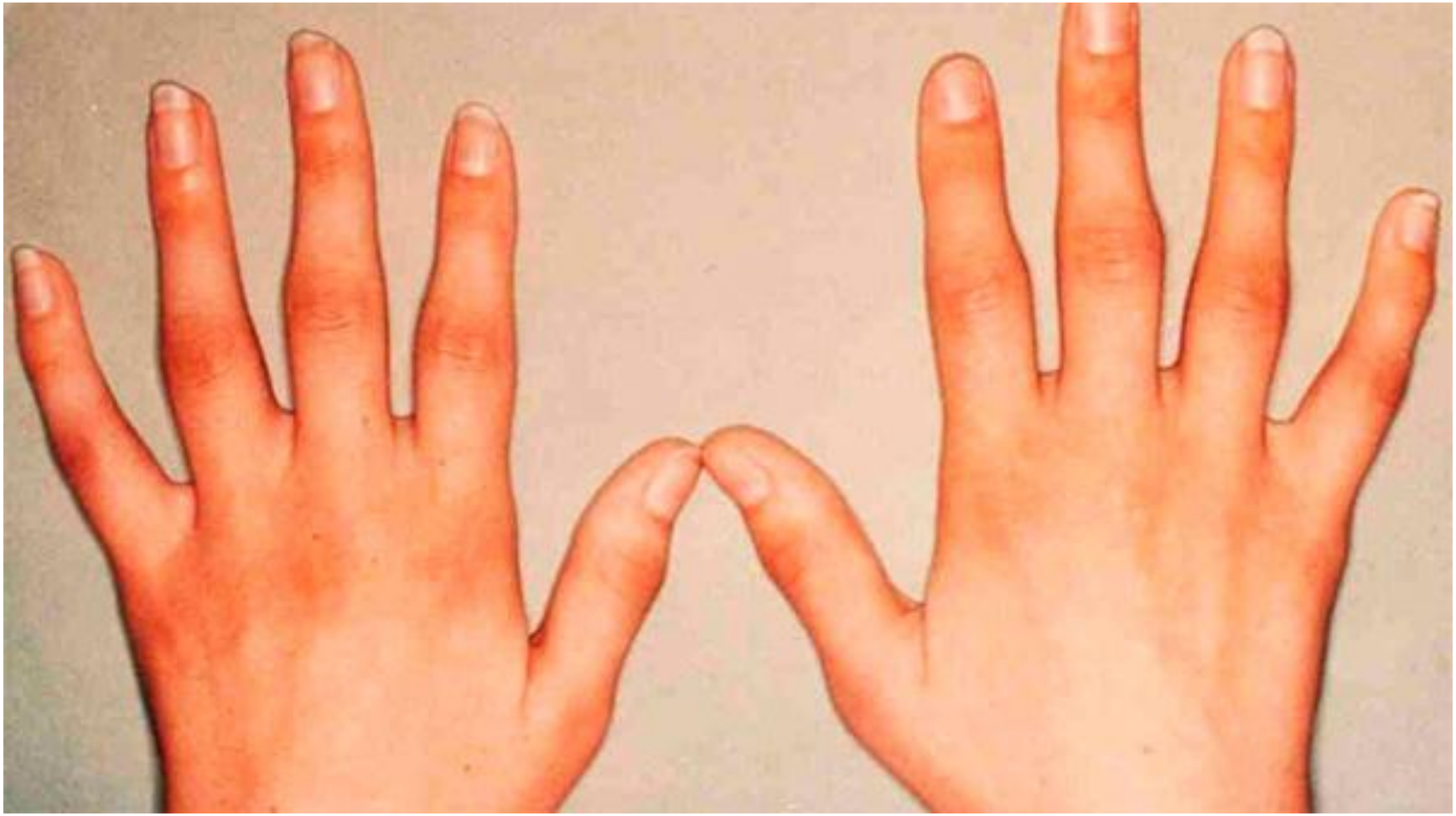


- Pericardial pain can usually be distinguished from ischemic coronary pain, which is not aggravated by thoracic motion or the recumbent position. Tachypnea and nonproductive cough may be present; fever, chills, and weakness are common. The most important physical finding is a triphasic or a systolic and diastolic precordial friction rub. However, it is often intermittent and evanescent or may be present only in systole or, less frequently, only in diastole. Considerable pericardial fluid may muffle heart sounds, increase the area of absolute cardiac dullness, and change the size and shape of the cardiac silhouette (trapezium or triangle configuration of the heart may be).

Cardiac tamponade

- is rare complication of the rheumatic pericarditis. Clinical findings of the cardiac tamponade are tachycardia, together with dyspnea and orthopnea and elevated systemic and pulmonary venous pressures. Severe cardiac tamponade is nearly always accompanied by an accentuated decline in systemic systolic BP on inspiration (pulsus paradoxus). A decline of > 10 mm Hg is usually significant. In advanced cases, the pulse may disappear on inspiration. Although rheumatic pericarditis can cause a serous effusion, fibrin deposits, and even pericardial calcification, it does not lead to constrictive pericarditis.

Rheumatic polyarthritis



Сустав поражён ревматизмом

Rheumatic migratory polyarthritis

- Migratory polyarthritis is present in as many as 75% cases of acute rheumatic fever. Migratory polyarthritis is characterized clinically by:72- symmetrical arthritis of large joints (ankles, wrists, knees, elbows). It usually does not affect the small joints of the hands or feet and seldom involves the hip joints;

Симптомы острой ревматической лиходарки у детей

Ревматический полиартрит: сильные боли в крупных и средних суставах

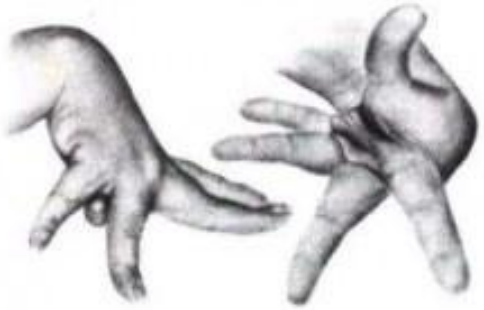
Боли и отечность суставов

Ревматический кардит и кольцевидная эритема

- - migrating character of pain; pain disappears in one joint and develops in others; - extremely painful, hyperemia, hyperthermia of joints; joints become swollen, sometimes with effusion; - polyarthritis is usually benign, all clinical finings are completely reversible. Acute inflammation subsides in a few days, although dull pain (arthralgia) in the joints may persist for a long time. The difference between arthralgia (subjective joint pain) and arthritis (joint pain and swelling) must be understood.

Rheumatic Chorea

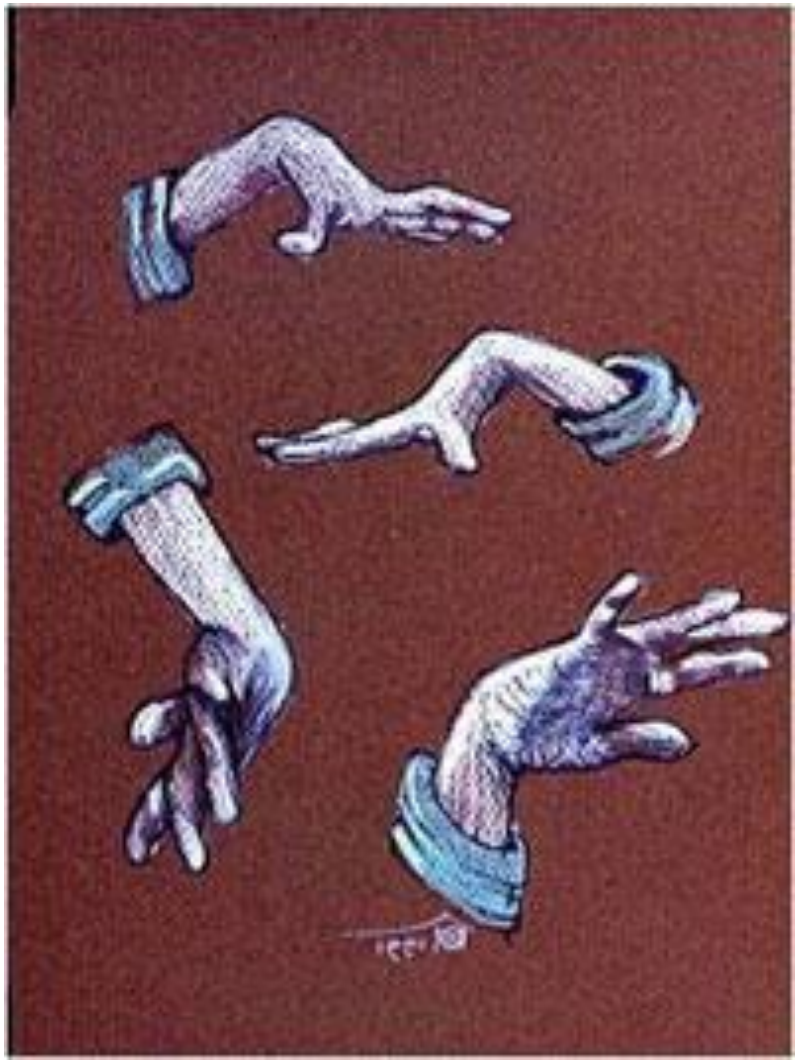
- Sydenham's chorea Sydenham's chorea (Rheumatic Chorea; St. Vitus' Dance) - a CNS disease, often of insidious onset but of finite duration, characterized by involuntary, purposeless, nonrepetitive movements and subsiding without neurologic residua



CHOREA MINOR

Поражение ЦНС – ревматическая хорея (малая хорея, пляска святого Витта)

- Проявление васкулита мелких мозговых сосудов.
- Встречается у детей и подростков, чаще у девочек и девушек.
- Частота поражения – 6 – 10%.
- Изолированное поражение в 5 – 7%.



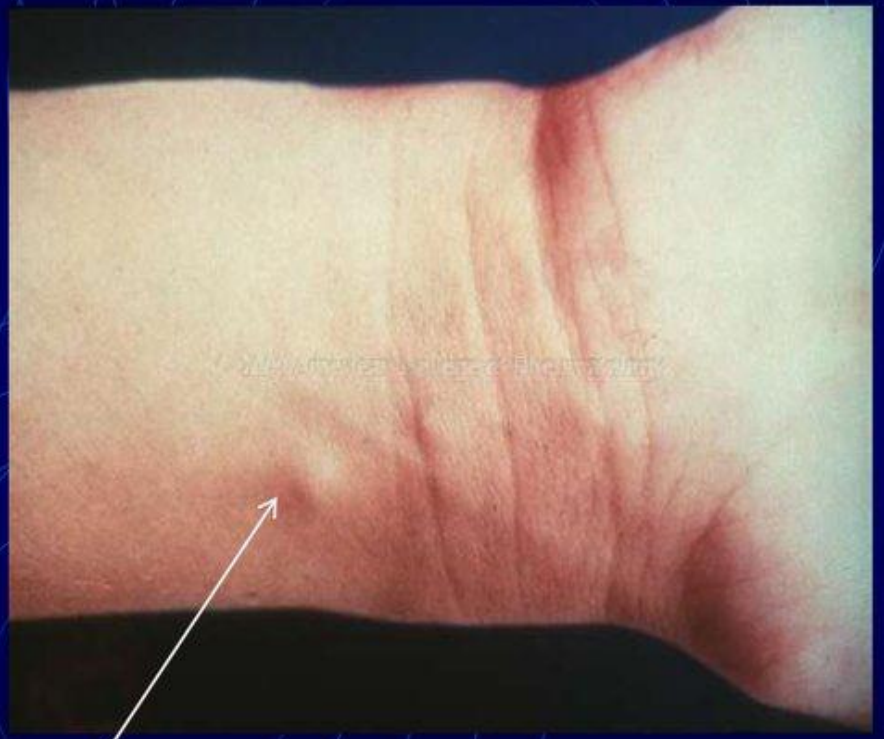
- Sydenham's chorea occurs in fewer than 10% of patients with rheumatic fever. This is due to either rheumatic vasculitis (attended by small hemorrhages or thrombosis of cerebral vessels) or inflammation of the brain and the spinal cord. Children (as a rule patients younger than 15 years) would develop encephalitis with predominant localization in the subcortical nodes (chorea minor). It is manifested by emotional lability and hyperkinesia (abnormal movements of the extremities, the trunk, and the facial muscles).

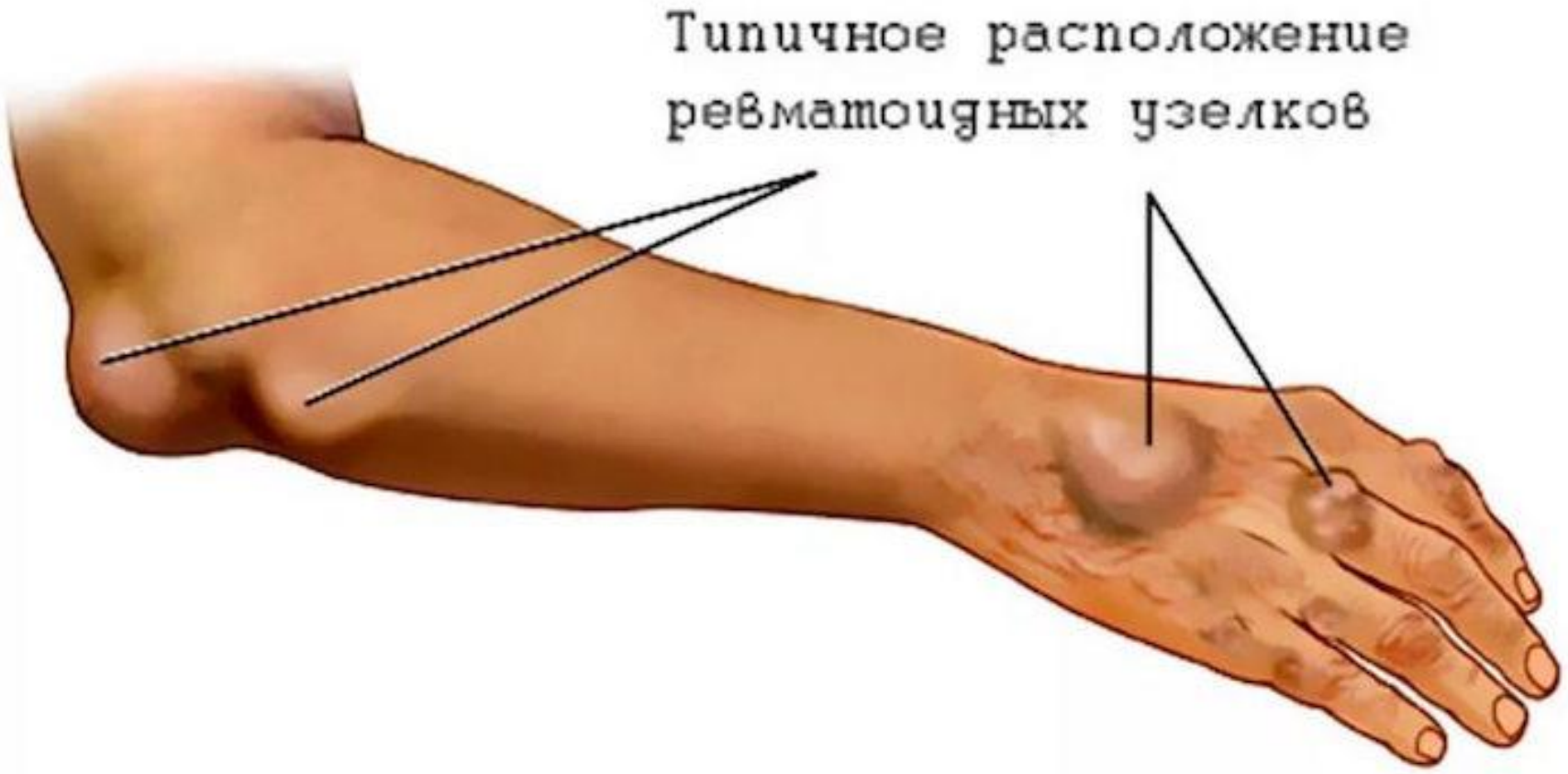
Skin signs and subcutaneous nodules

- Subcutaneous nodules and erythema marginatum are rare major manifestations of acute rheumatic fever, usually present in fewer than 10% of cases. Subcutaneous nodules are found over extensor surfaces of joints, are seen most often in patients with long-standing rheumatic heart disease, and are extremely rare in patients experiencing an initial attack. Subcutaneous 73 nodules are firm, painless formations varying in size from a millet grain to a bean, can be palpated, mostly on the extensor surfaces of the joints, along the course of tendons, and in the occipital region.

Subcutaneous nodule

Подкожные узелки




Типичное расположение
ревматоидных узелков

Erythema marginatum (annular erythema)

- Erythema marginatum (annular erythema) is an uncommon manifestation. It is an evanescent macular eruption with rounded borders (pale-pink painless rings not elevating over the surrounding skin). The skin of the chest, neck, abdomen, and the face is affected by annular erythema. In other cases nodular erythema develops: circumscribed indurated dark red foci on the skin varying in size from a pea to a plum; they are usually found on the lower limbs. If permeability of capillaries is increased, small hemorrhages into the skin sometimes occur.

annular erythema

Skin lesions

- Subcutaneous nodules or *Erythema marginatum* (present in 10 to 60% of cases, more often in children):

Subcutaneous nodules

- essentially giant Aschoff bodies, are most often
- located overlying the extensor tendons of the extremities at the
  - wrists,
  - elbows,
  - ankles,
  - and knees.

- Other clinical manifestations of acute rheumatic fever are very rare and usually completely reversible. Lungs are affected in very rare cases. This is specific rheumatic pneumonia. Dry pleurisy or pleurisy with effusion are more common. Heart affections may be the only clinical manifestation of rheumatism. The alimentary system is rarely affected. Acute pain in the abdomen (the abdominal syndrome) associated with rheumatic peritonitis (mostly in children) sometimes occurs. The liver is affected in certain cases (rheumatic hepatitis). Affections of the kidneys are also common. Protein or red blood cells can be found in the urine due to affections of the renal vessels and (less frequently) developing nephritis.

Laboratory and instrumental tests in acute rheumatic fever

- *Clinical blood analysis:* leucocytosis, shift in the leucocytic formula to the left, eosinophilia, mono- and lymphacytosis may further develop and accelerated ESR.
- *Biochemical analysis of blood. Special laboratory tests* help diagnose rheumatism.

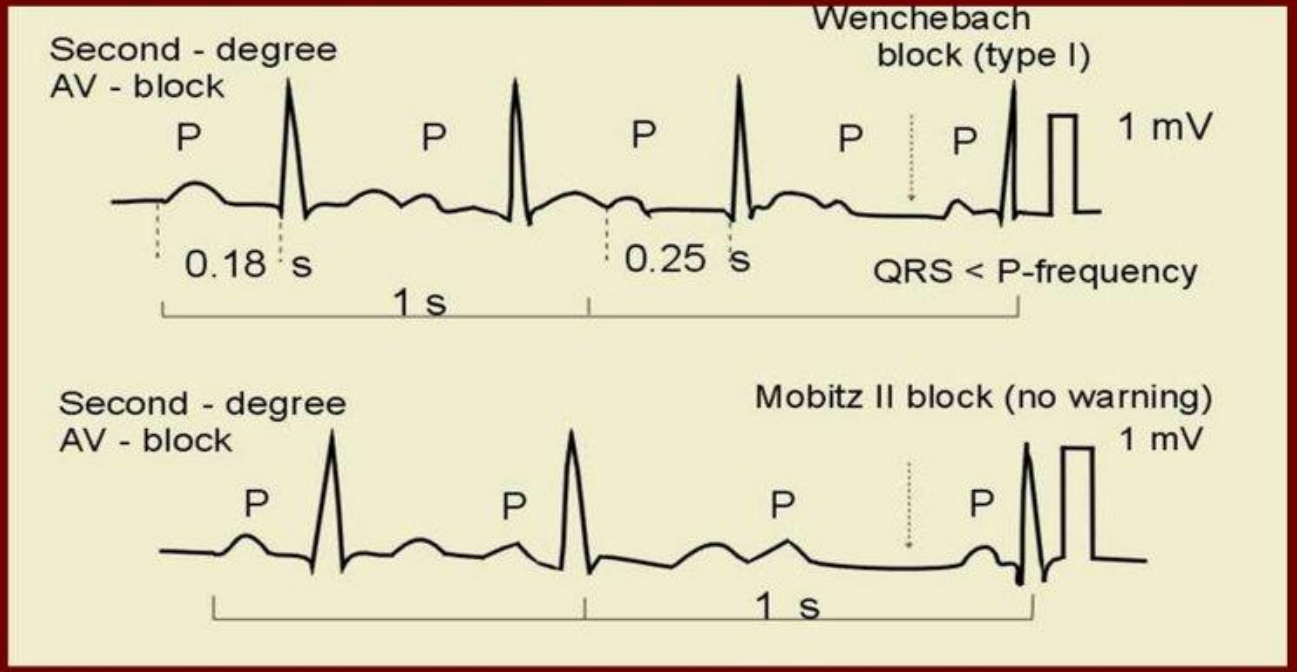
Microbiology study

- Microbiology study detects group A streptococci positive throat culture (throat swab or rapid antigen detection test). Immunological tests discovers increased titers of group A streptococcal antibodies (anti-streptolysin O, anti-deoxyribonuclease B, antihyaluronidase) in blood serum.

- *ECG:* premature contraction, atrial fibrillation or flatter, decreasing of ECG voltage, depression of ST-segment, appearance of negative, asymmetrical T-wave is possible; elevation of ST-segment due to pericarditis or subepicardial damages of myocardium; different degrees of AV-blockade.
- *X-ray of heart and Echo-CG* indicates enlargement of heart and its chambers.

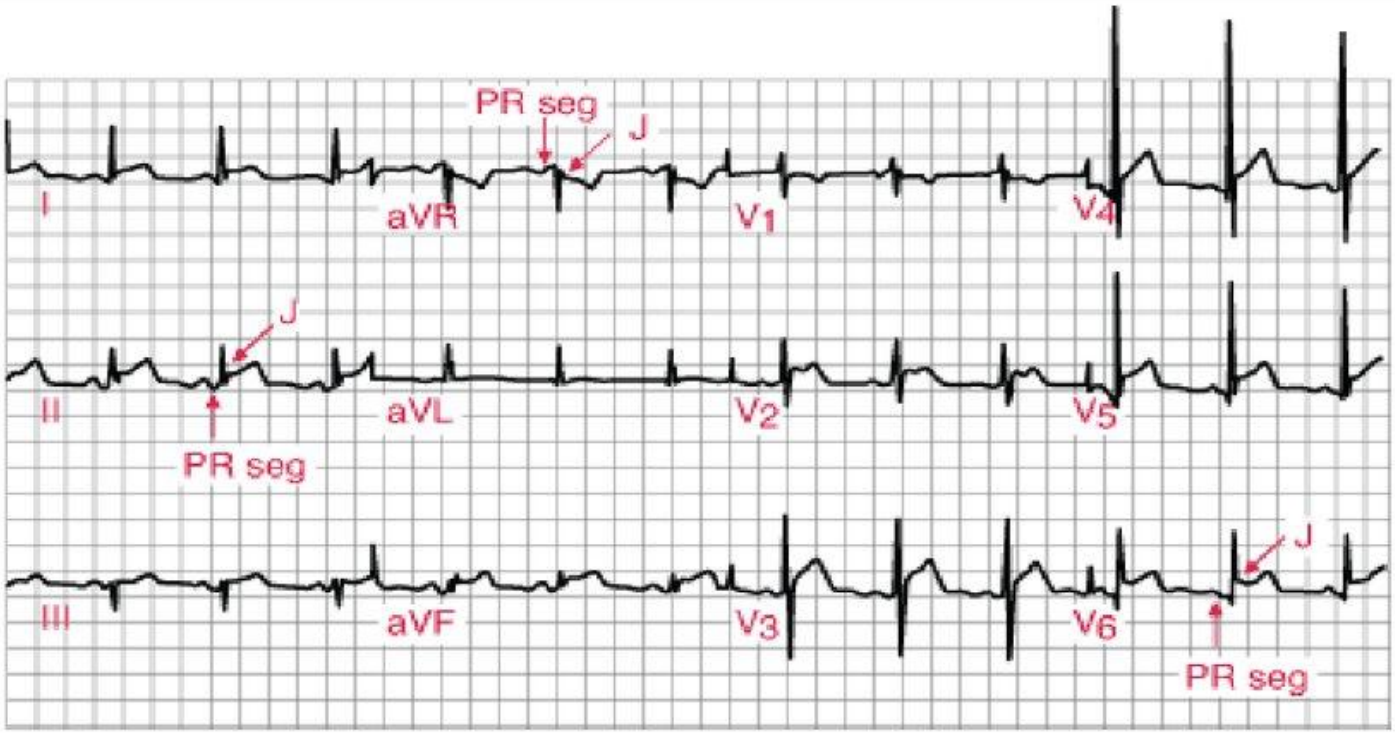
Atrioventricular block

- 2nd degree AV block- some of the P-waves are not followed by QRS-complexes
- Mobitz type I - PQ-interval is increased progressively until a P-wave is not followed by a QRS-complex. (Wenchebach block).
- Mobitz type II block - the ventricles drop some beats



ECG in acute pericarditis

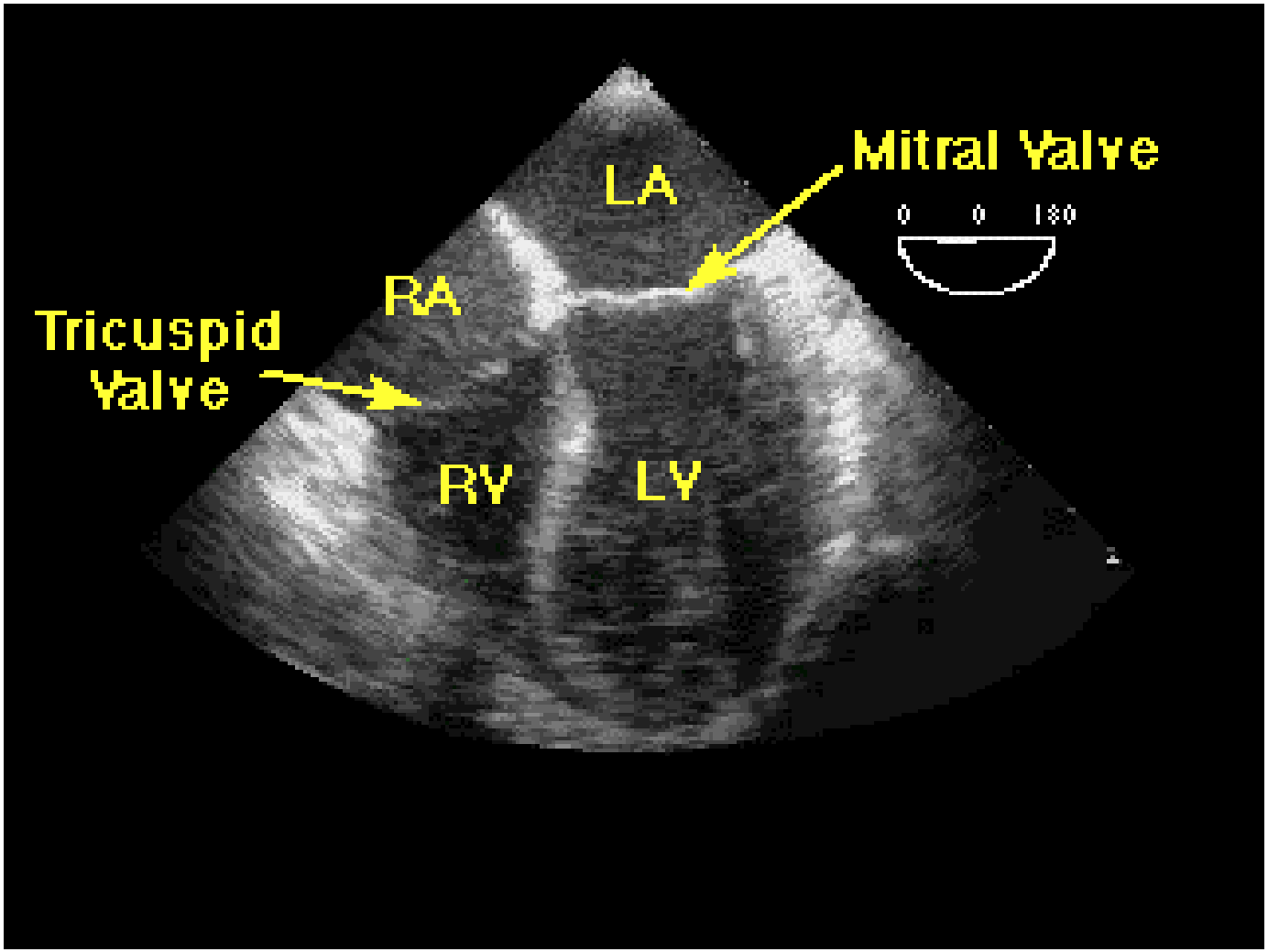
Acute pericarditis: Stage 1 ECG.



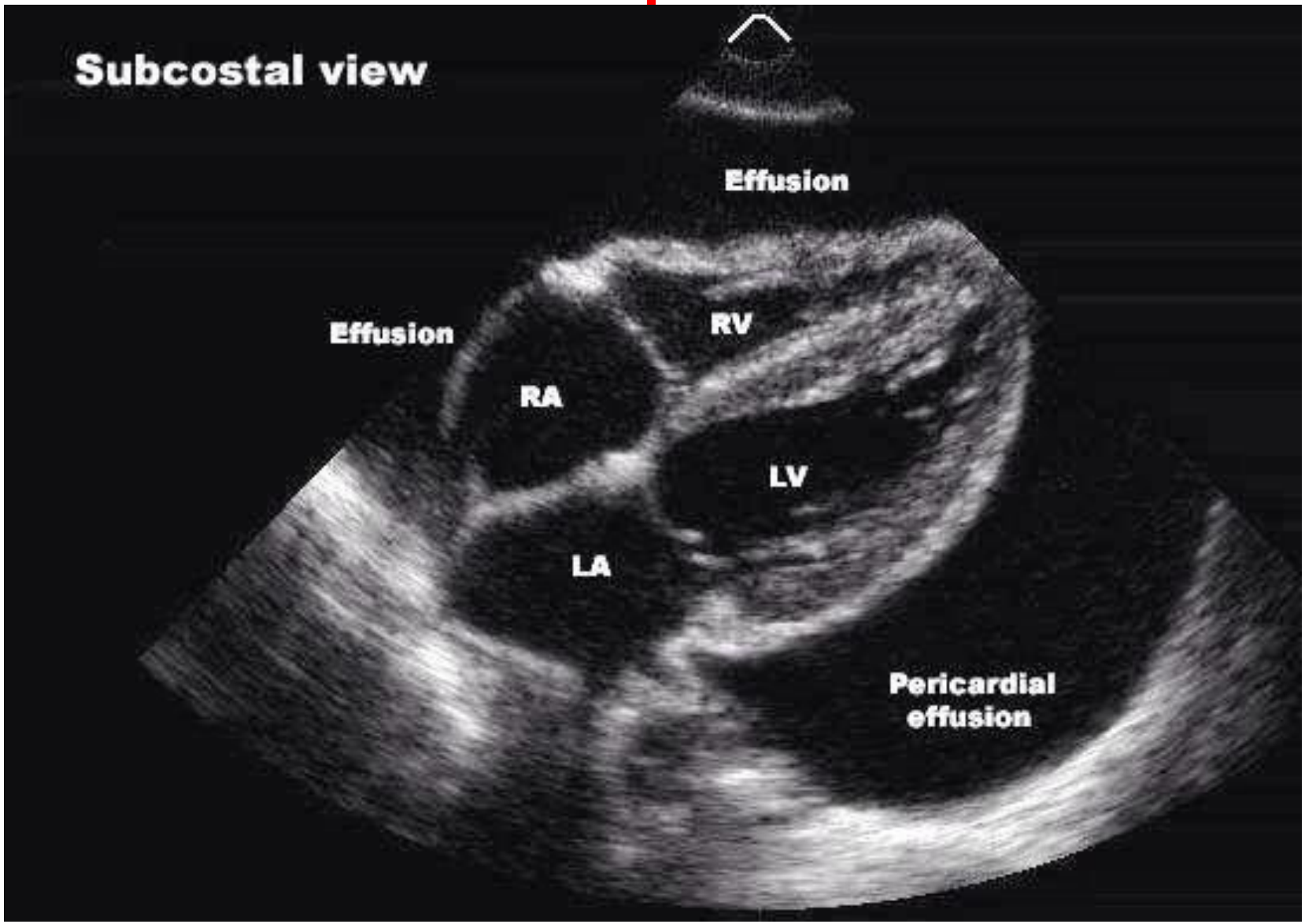
J points, except aVR and V_1, are elevated. T waves are essentially normal. PR segments, except aVR and V_1, are depressed. PR deviations are commonly absent in one limb lead (here, aVL).

- Phonocardiogram (PCG) show specific rheumocarditic changes in heart sounds, and the appearance of murmurs.
- Echocardiography detects abnormalities of the mitral (and other) valves, effusion in pericardium.

Echocardiography



Effusion in pericardium



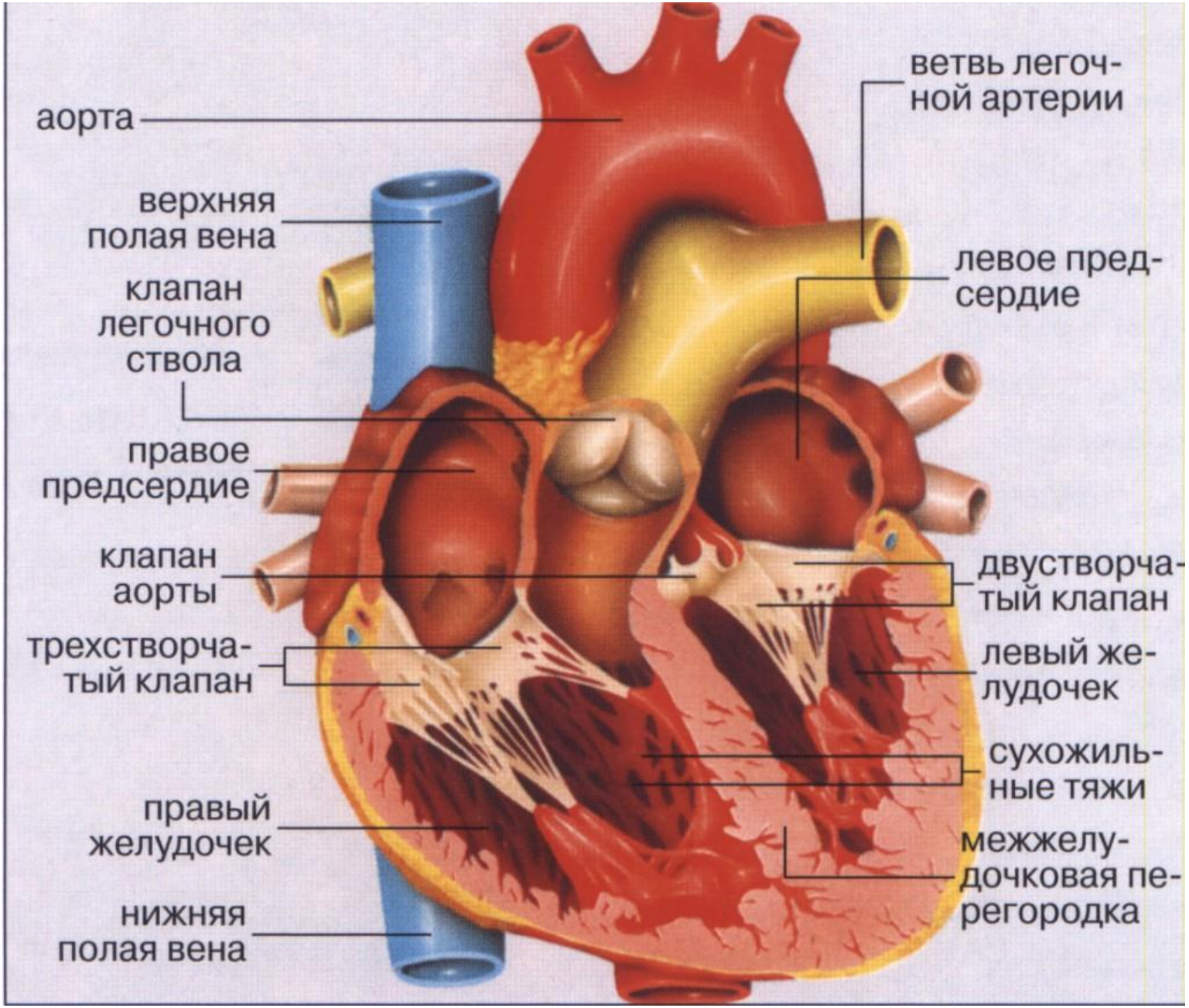
The Jones Criteria for Rheumatic Fever, Updated 1992

- *A. Major Criteria:*
- Carditis
- Polyarthritis
- Chorea minor - hyperkinesia (abnormal movements of the extremities, the trunk, and the facial muscles).
- Annular erhytema
- Rheumatic subcutaneous nodule

The Jones Criteria for Rheumatic Fever, Updated 1992

- *B. Additional (minor) Criteria:*
- Fever
- Pain in the joints
- C-reactive increased
- Increased the erythrocyte sedimentation rate

Acquired Heart Valves Diseases


аорта
верхняя полая вена
клапан легочного ствола
правое предсердие
клапан аорты
трехстворчатый клапан
правый желудочек
нижняя полая вена
ветвь легочной артерии
левое предсердие
двустворчатый клапан
левый желудочек
сухожильные тяжи
межжелудочковая перегородка

- **The acquired heart diseases**.
- Definition: Heart valves diseases are stable pathological changes in the structure of the heart that interfere with its normal function.

The acquired heart diseases

- organic changes in the valves or septal defects of the heart, resulting from diseases or injuries. Associated with heart defects violations of intracardiac hemodynamics form of pathological conditions characterized by supporting the pumping function of the heart and total hemodynamic compensatory mechanisms, the ineffectiveness of which develops circulatory failure, disabling the patient and often are the cause of death.

Etiology

- Etiology of acquired heart valves diseases: rheumatic endocarditis is the main cause of acquired heart defects; on second place - infective endocarditis and atherosclerosis; more rarely - syphilis, injuries, etc. Inflammatory processes occurring in the valve cusps often end in their sclerosis: deformation and shortening. An affected valve does not close completely to cause valves incompetence. The cusps of the valves may adhere to one another because of inflammation to narrow the orifice they close. This narrowing is called stenosis.

Classification of acquired heart valves diseases

- (1) According to etiology – rheumatic (I05-I08),
- non-rhumatic acquired valves diseases (I34-I37): infective endocarditis, atherosclerotic, syphilitic, etc.

- (2) According to pathogenesis – organic and functional heart valve disease. (3) According to morphology and homodynamic changes – incompetence (insufficiency, regurgitation), stenosis, combined heart valve disease (if incompetence and stenosis of the orifice simultaneously). (4) According to localization – mitral, aortic, tricuspid, pulmonary artery, concomitant (if two and sometimes three valves simultaneously) heart valve disease. (5) According to condition of blood circulation – compensated and decompensated (characterized by circulatory insufficiency) heart valve disease.

- Diagnosis of acquired heart valves diseases is based mainly on typical auscultative data confirmed by echocardiography, and in some cases heart catheterization.

Mitral incompetence (mitral insufficiency, or mitral regurgitation)

- **Definition: Incompetence of the mitral (bicuspid) valve (mitral insufficiency) is incomplete closure of the atrioventricular orifice during leftventricular systole. As a result, the blood is regurgitated from the ventricle back to the atrium. Mitral incompetence may be organic and functional.**

НашеСердце.ру

ПРИЧИНЫ МИТРАЛЬНОЙ НЕДОСТАТОЧНОСТИ

НОРМАЛЬНЫЙ МИТРАЛЬНЫЙ КЛАПАН

ПРОЛАПС МИТРАЛЬНОГО КЛАПАНА

ИНФЕКЦИОННЫЙ ЭНДОКАРДИТ

ФУНКЦИОНАЛЬНАЯ МИТРАЛЬНАЯ НЕДОСТАТОЧНОСТЬ

Etiology

- Organic mitral insufficiency arises as a result of rheumatic endocarditis. Connective tissue develops in the cusps of the mitral valve which then contracts to shorten the cusps and the tendons. The edges of the affected valve do not meet during systole and part of the blood is regurgitated through the slit into the left atrium from the ventricle during its contraction

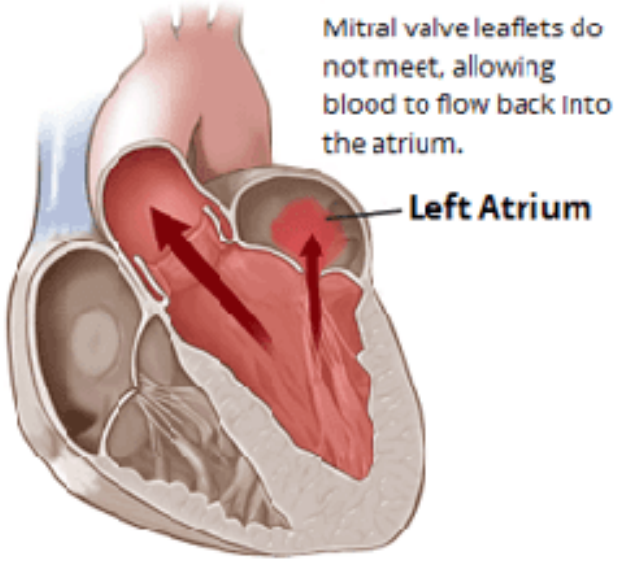
- In functional (relative) incompetence the mitral valve is not altered but the orifice, which it has to close, is enlarged and the cusps fail to close it completely. Functional incompetence of the mitral valve may develop because of dilatation of the left ventricle (in myocarditis, myocardial dystrophy, or cardiosclerosis) and weakening of the circular muscle fibres that form the ring round the atrioventricular orifice. Affection of papillary muscles may also cause functional mitral incompetence. Functional insufficiency thus depends on dysfunction of the muscles responsible for the closure of the valve.

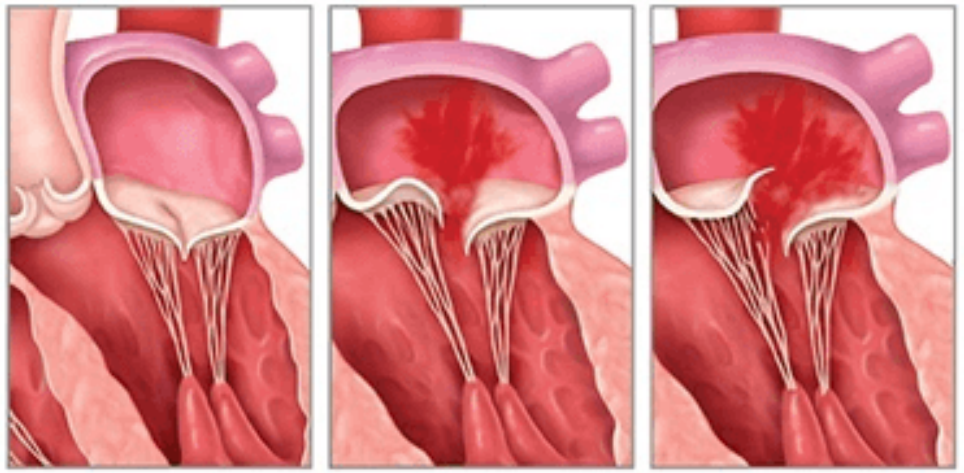
Hemodynamics

- If the mitral valve fails to close completely during systole of the left ventricle, part of the blood is regurgitated into the left atrium. Blood filling of the atrium thus increases (because of the blood from the pulmonary veins which is added to the normal blood volume). Pressure in the left atrium increases, the atrium is dilated and becomes hypertrophied.

Hemodynamics

Mitral Regurgitation

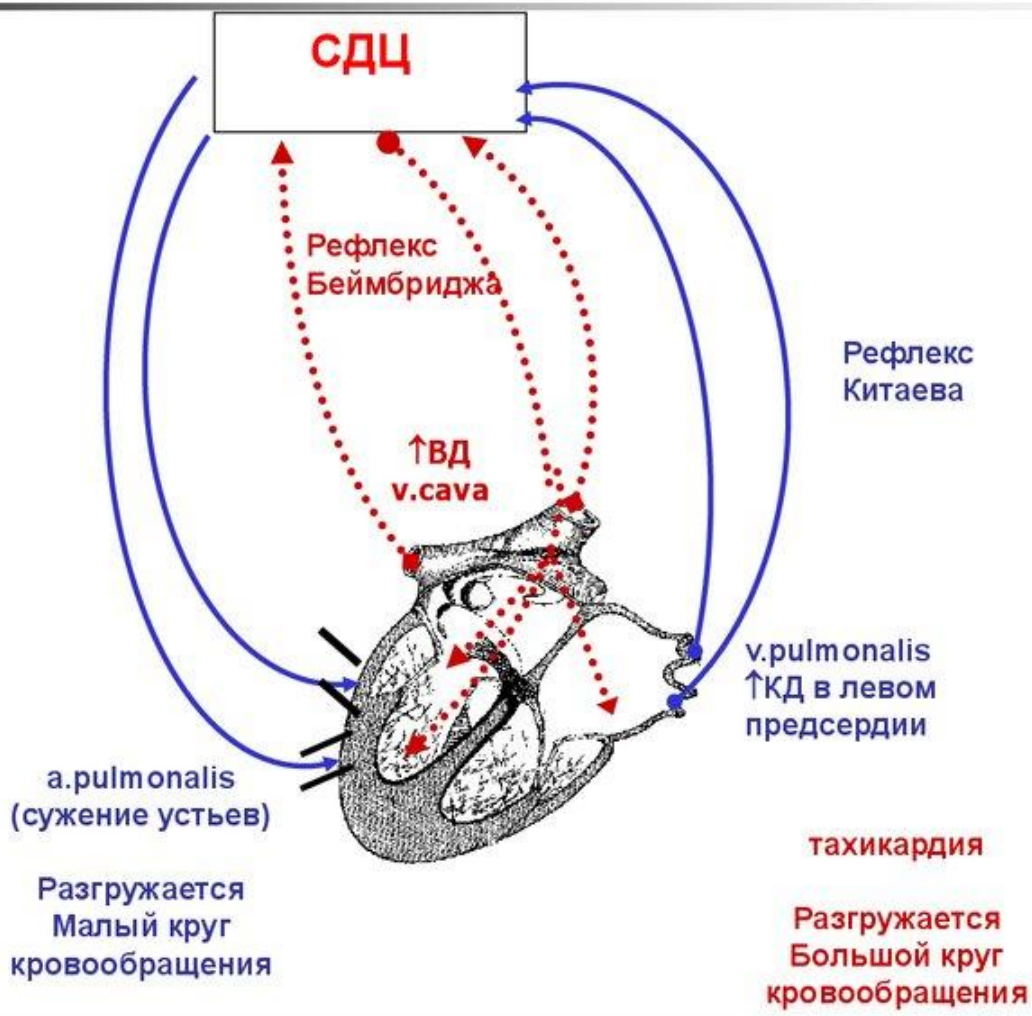




- The amount of blood that is delivered into the left ventricle from the overfilled left atrium during diastole exceeds normal and the atrium is thus overfilled and distended. The left ventricle has to perform excess work and becomes hypertrophied. Intensified work of the left ventricle compensates for 84 the mitral incompetence during a long time. When the contractile power of the left ventricular myocardium weakens, diastolic pressure in it increases and this in turn increases pressure in the left atrium

- Increased pressure in the left atrium increases pressure in the pulmonary veins and this in turn causes reflex contraction of the arterioles in the lesser circulation (Kitaev's reflex) due to stimulation of baroreceptors. Spasm in the arterioles increases significantly pressure in the pulmonary artery to intensify the load on the right ventricle which has to contract with a greater force in order to eject blood into the pulmonary trunk. The right ventricle can therefore also be hypertrophied during longstanding pronounced mitral incompetence.

РЕФЛЕКСЫ СЕРДЕЧНО-СОСУДИСТОЙ СИСТЕМЫ



Reflex Kitaeva

- Reflex Kitaeva (hypertension pulmonary arterioles in response to increased pressure in the pulmonary veins) is observed in defects, accompanied by increased pressure in the left atrium, especially in mitral stenosis. This reflex substantially prevents hypertension pulmonary capillaries, thus preventing the development of pulmonary edema.
- The phenomenon of centralization of blood flow is noted with a significant reduction of the effective stroke volume of the heart and the conditioned reflex (interoreceptors carotid sinus) activation of sympathoadrenal system with generalized tonic responses of peripheral resistance vessels - leading to redistribution of blood mainly the brain and heart by reducing blood flow in other organs, skeletal muscles and skin.

- Decompensation defect characterized by the occurrence of heart failure and circulatory problems, with certain heart defects the features. When gradually developing decompensation blemish it is based on the growing shortage of energy supply hyperfunctioning structures of the myocardium that is strongly associated with progressive myocardial dystrophy from hyperactivity. The role of compensatory reactions of the vascular system, in particular reflex Kitaeva or phenomenon of centralization of blood circulation, the progression of heart failure is increasing, and at some point, these compensatory mechanisms become essential, but at the same time, themselves become primary elements in the pathogenesis of secondary violations. So, in severe decompensation mitral defect reflex Kitaeva remains almost edinstvennym (at least leading) a mechanism to prevent life-threatening pulmonary edema, but the associated pulmonary arterial hypertension causes hypertrophy, stress and, ultimately, the appearance failure of the right ventricle of the heart

Clinical picture

- Most patients with mild or moderate mitral incompetence have no complaints for a long time and look very much like healthy subjects. As congestion in the lesser circulation develops, dyspnea, palpitation of the heart, cyanosis, and other symptoms appear.

Осмотр лица (митральный стеноз)

- As congestion in pulmonary circulation occurs, dyspnea, palpitation of the geart, cyanosis and other symptoms develop. When left ventricular function deteriorates, dyspnea, orthopnea, and paroxysmal nocturnal dyspnea become more prominent as well as a couph (dry or with small expectoration of sputum, sometimes with admixions of blood (haemoptoe)/ Quite often patients can feel pain in the heart area by various character: dull ache, strabbing or pressing pain of different intensity, which is not correspond th degree of overload. Palpitation, cyanosis may be revealed in such patients at rest or after physical examination. Peripheral edema may be apparent in patients with end-stage mitral incompetence/

- **Palpation.** Palpation of the heart area reveals displacement of the apex beat to the left and sometimes inferiorly. The beat becomes diffuse, intensified, and resistant, which indicates hypertrophy of the left ventricle.
- Blood pressure an arterial pulse. Blood pressure is usually normal. The peripheral arterial pulse is often normal but may be of small amplitude.

Percussion

- – displacement of heart borders upwards and leftwards may be revealed (so-called “mitral heart configuration” with indistinct heart waist) becouse of the enlarged left atrium and ventricle. At hypertrophy of the right ventricle the right heart border is displaced rightwards due to hypertrophy of the right ventricle.

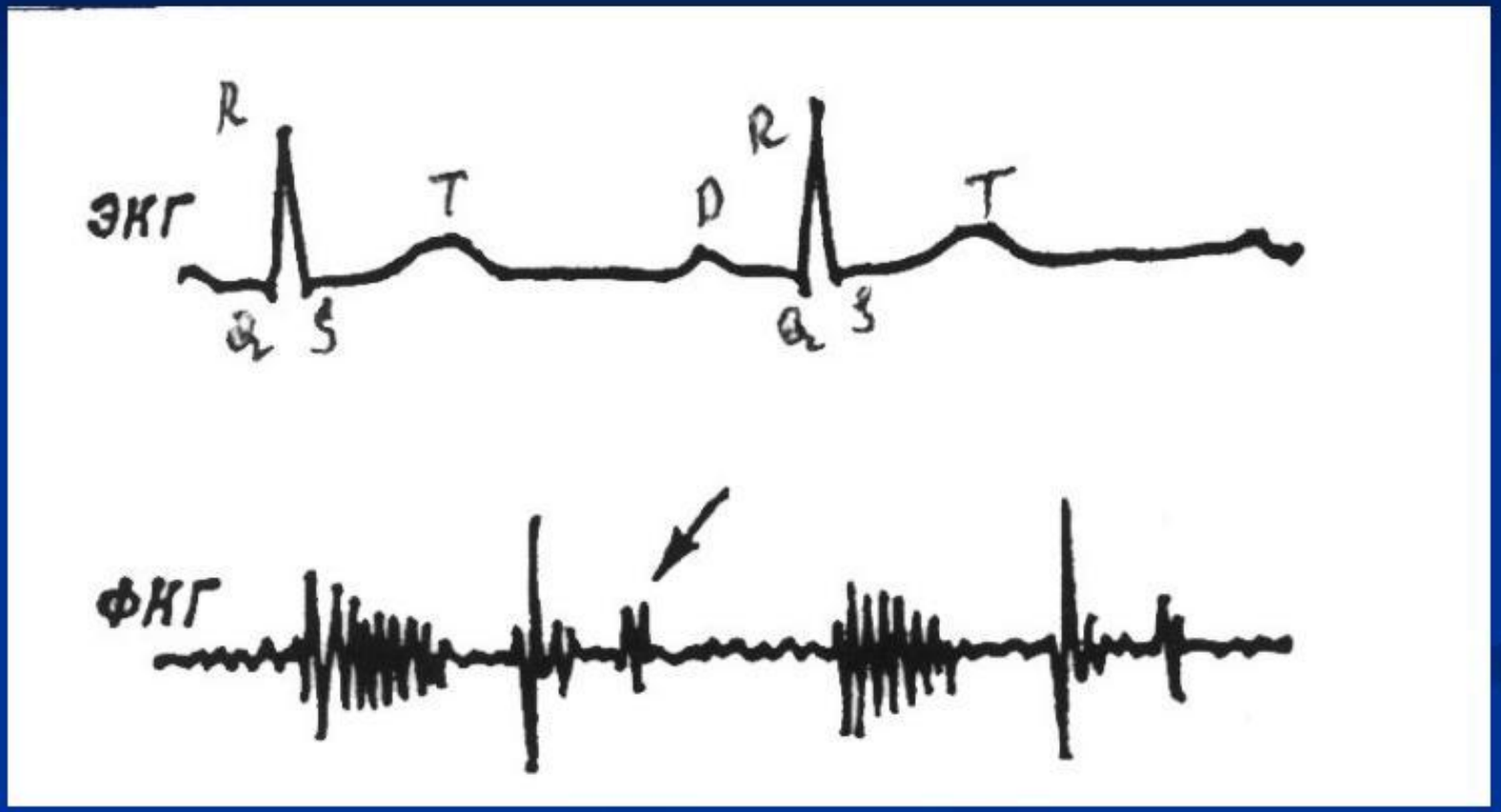
Auscultation

- Auscultation of the heart reveals decreased first sound at the heart apex because the valves never close completely in this disease. Systolic murmur can be heard at the same point, which is the main sign of mitral incompetence. It arises during systole when the stream of blood passes a narrow slit leading from the left ventricle to the left atrium. The systolic murmur is synchronous with the first sound. When the blood pressure rises in the lesser circulation, an accent of the second sound can be heard over the pulmonary trunk.

- **Auscultation.** In chronic mitral incompetence, the first heart sound usually is decreased in intensity. The weakening of the first heart sound occures because of absence of period of closured valves. . In uncomplicated mitral insufficiency the first sound is either faint or inaudible. The second heart sound usually is normal Accent of the second heart sound above the pulmonary artery is caused by increased blood pressure in pulmonary veins.

- The third heart sound heard at the apex with the bell of the stethoscope is common (protodiastolic gallop). It tends to occur especially in moderate or severe mitral incompetence, because of the high diastolic flow into the ventricle, and therefore does not necessarily imply significant left ventricular dysfunction or left ventricular filling pressures. It generally occurs 120 to 240 msec after the second heart sound.

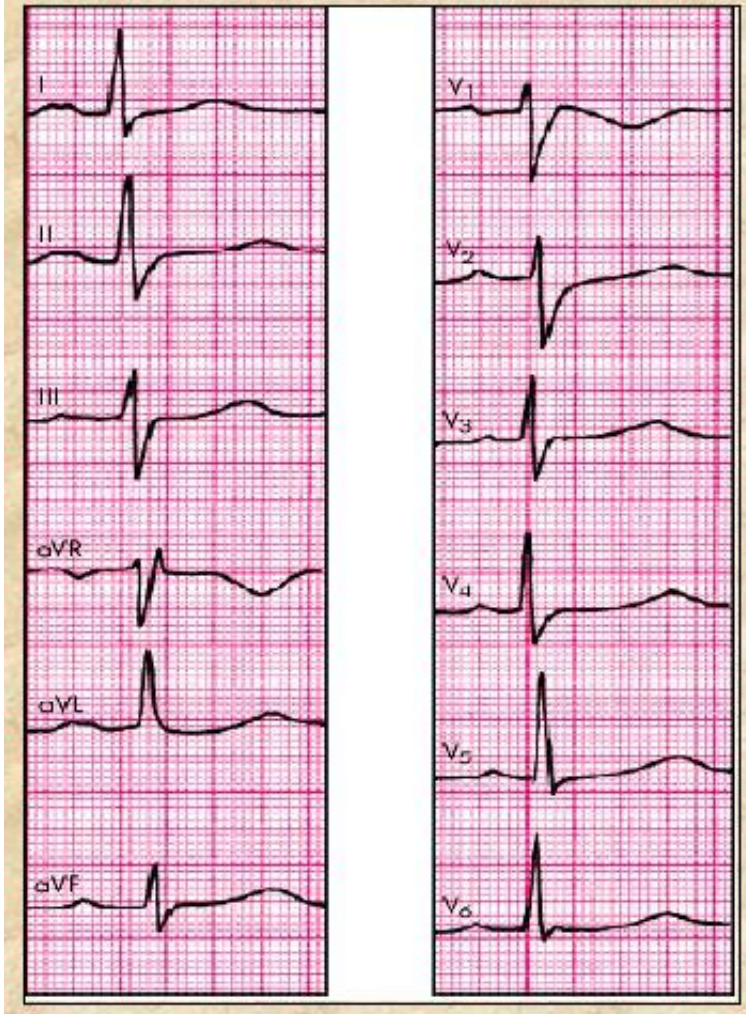
ФКГ при митральной недостаточности

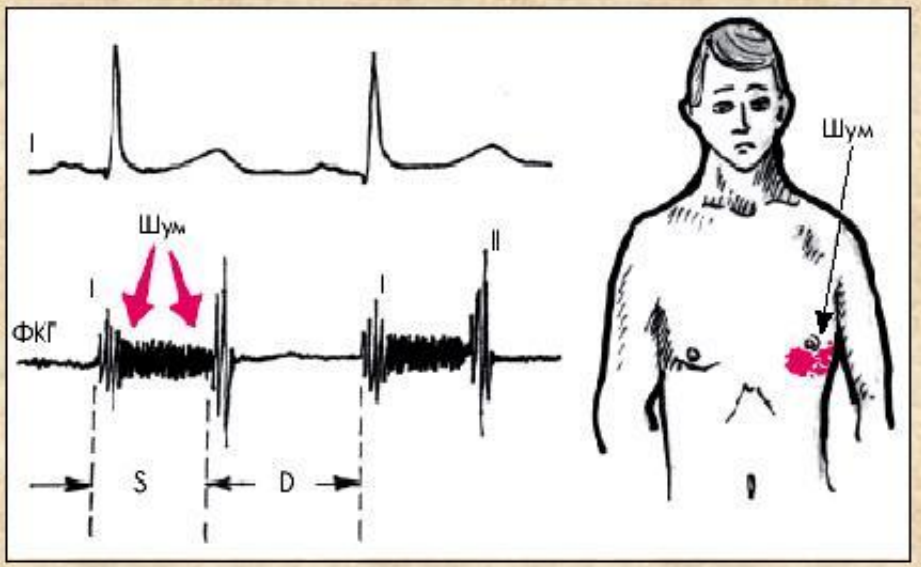


- The auscultatory hallmark of the mitral incompetence is a **sytolic murmur** that typically is **holosystolic,** starts with the first heart sound and remains at constant intensity throughout systole, ending at or after the aortic component of the second heart sound. Usually the murmur is **medium to high pitched, soft, blowing in quality.** The intensity of the murmur does not correlate well with the severity of regurgitant flow. The murmur is best heard with the diaphragm of the stethoscope placed firmly over the apex. Although in some patients the murmur may be located to one point, most frequently the apical murmur transmisses to the axilla. This murmur is usually transmitted to the left and toward the posterior axillary line.The murmur may be notably short, soft, or absent in acute severe mitral incompetence.
- The systolic murmur is better heared if the patient lies down on his left side and stops breathing.

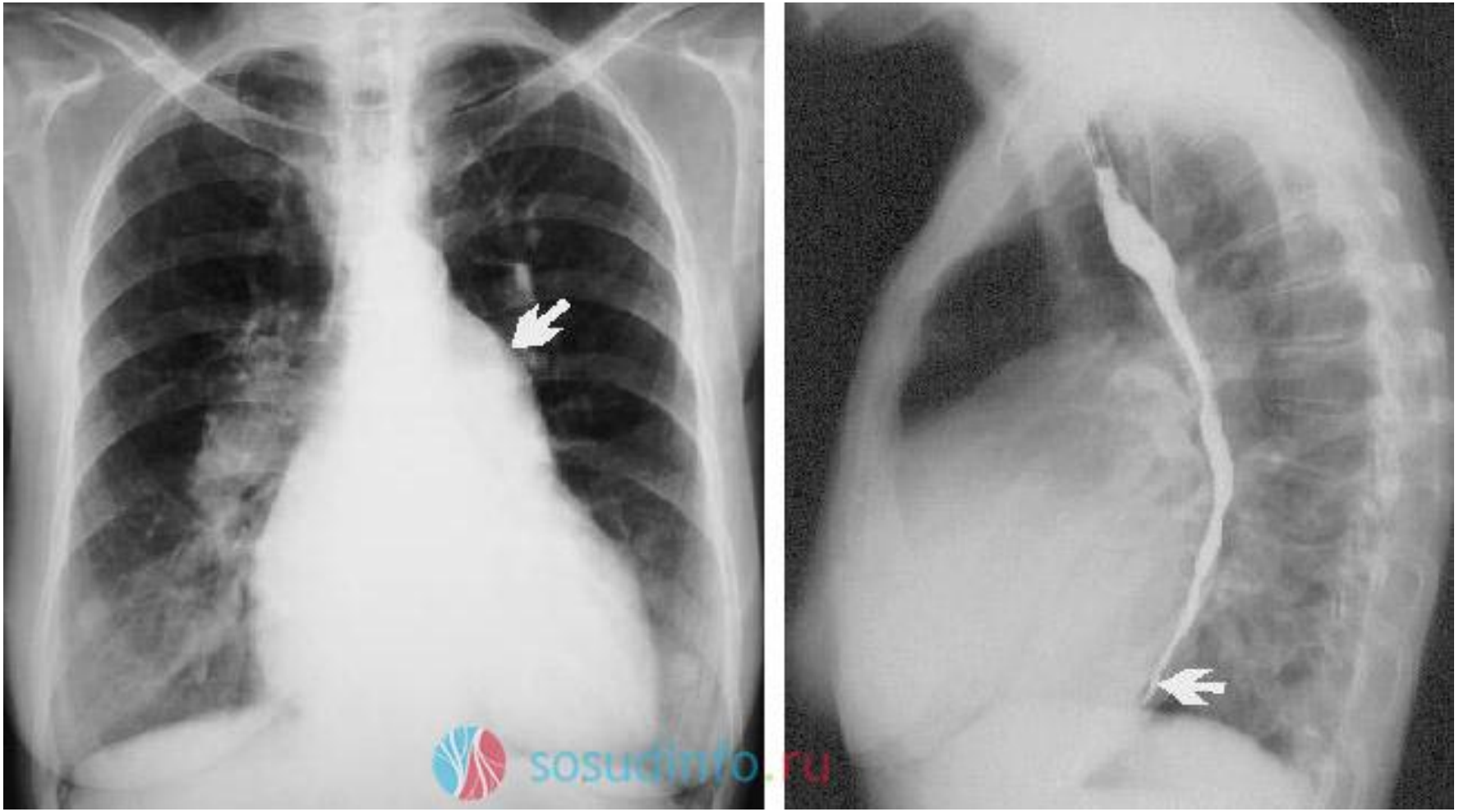
- The datas revealed during auscultation have to be improved and confirmed by **fonocardiogram (FCG).**
- **On X-ray** film the enlargement of heart sillhouette leftwards, upwards and downwards is observed. True cardiomegaly with left ventricular and left atrial enlargement is a common finding in significant chronic mitral incompetence. Left atrial enlargement may be recognized by straightening of the left heart border, by a characteristic right-sided double density (right and left atrial borders are superimposed), and, occasionally, by elevation of the left main-stem bronchus.

ЭКГ И ФКГ ПРИ МИТРАЛЬНОЙ НЕДОСТАТОЧНОСТИ





ЭКГ – гипертрофия ЛП и ЛЖ
ФКГ – уменьшение амплитуды I тона, систолический шум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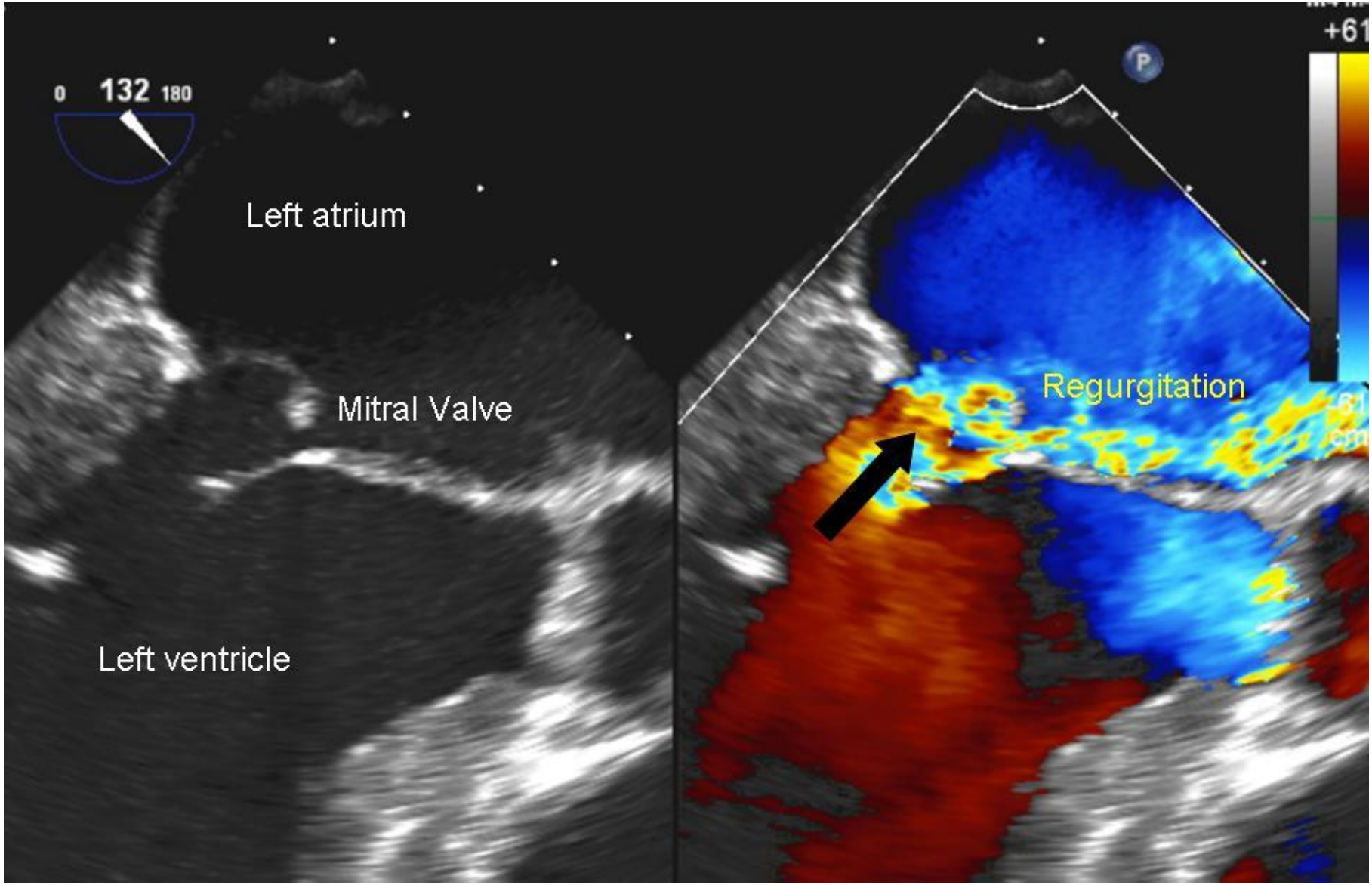
- The radiographic size the left atrium provides a rough guide to the severity and duration of the mitral incompetence. The atrium generally is more enlarged in mitral incompetence than in mitral stenosis; it may be gigantic if both lesions are present.
- In patients with mitral incompetence, the pulmonary vasculature is usually normal until late in the disease when significant venous pulmonary hypertension develops. When increased blood pressure in pulmonary circulation persists – dilatation of pulmonary arteria arch, hypertrophy, dilatation of the right ventricle are detected. The resulting redistribution of blood flow to the upper lobes and interstitial edema causes Kerley B lines.

- On ECG - hypertrophy of the left atrium may be revealed. About 75% of patients with moderate to severe mitral incompetence have atrial fibrillation, consistent with the presence of significantly enlarged left atrium. Patients with normal sinus rhythm nearly always have evidence of left atrial enlargement i.e., P mitrale configuration (broad notched P waves in lead II and biphasic P waves in lead V1). Left ventricular hypertrophy with leftward or normal axis has been said to be characteristic of mitral incompetence, but the QRS complex and axis usually are normal and associated with nonspecific ST-segment and T-wave alterations. Right ventricular hypertrophy is notably uncommon, and its presence indicates significant pulmonary hypertension.


0
132
180
Left atrium
Mitral Valve
Left ventricle
Regurgitation
+61

- Echocardiography reveales the enlargement of the left heart chambers, movement of the mitral valve cusps in the opposite direction, their thikening and the absence of full closure during systole. By Doppler mode turbulent blood stream into the cavity of the left atrium in the expressed cases of incompletence is recorded, that is the indirect sign of mitral incompletence.

Course

- Mitral incompetence may remain compensated for a long time. But a long-standing pronounced mitral incompetence and decreased myocardial contractility of the left atrium and the left ventricle cause venous congestion in the lesser circulation. Contractility of the right ventricle can later be affected with subsequent development of congestion in the greater circulation.

MITRAL STENOSIS

Mitral stenosis (stenosis of the left atrioventricular orifice)

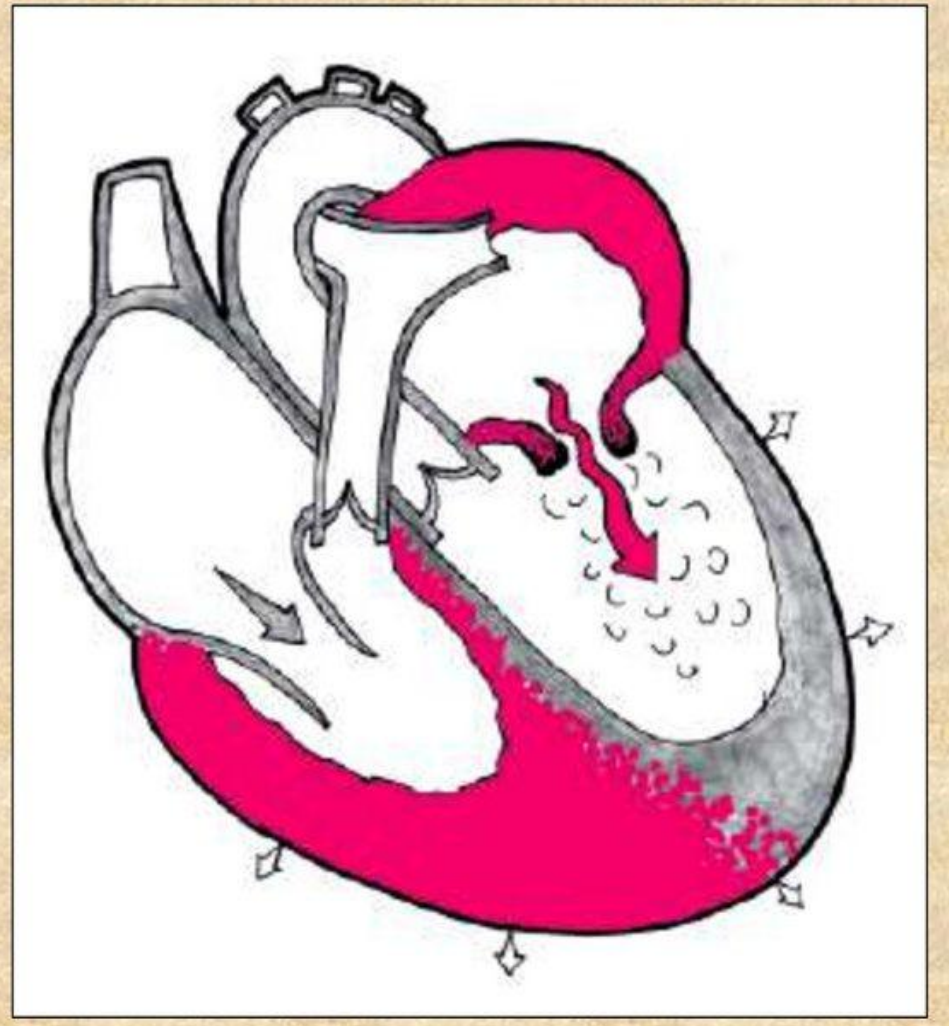
- Definition: Mitral stenosis is an obstruction of flow from left atrium to left ventricle because of a narrowed mitral orifice.

- **Three degrees of the mitral stenosis are distinguished:**
- 1) severe mitral stenosis (the square of mitral orifice is 0,5 cm2 or less);
- 2) the moderate one / the square of mitral orifice is about 1-0,6 cm2),
- 3) mild stenosis (the square of a mitral orifice is 1,5-1,1 cm2).

Etiology

- The left atrioventricular orifice usually narrows in a longstanding rheumatic endocarditis (stenosis ostii venosi sinistri). In very rare cases mitral stenosis may be congenital or secondary to infective endocarditis. The atrioventricular orifice narrows due to adhesion of the mitral cusps, their consolidation and thickening, and also shortening and thickening of the tendons. The valve thus becomes a diaphragm or a funnel with a slit in the middle. Cicatricial and inflammatory narrowing of the valvular ring is less important in genesis of mitral stenosis. The valve may be calcified in longstanding stenosis.

ИЛЛЮСТРАЦИЯ ИЗМЕНЕНИЙ ГЕМОДИНАМИКИ ПРИ МИТРАЛЬНОМ СТЕНОЗЕ



Disorders of hemodynamics

- Disorders of hemodynamics: due to pathological process, the adhesion of the mitral cusps, it consolidation, thickening and shortening narrow the left atrioventricular orifice. In patient with mitral stenosis orifice becomes 1.5 sm2 and less instead of normal 4-6 sm2. Narrowing of an orifice is a mechanical obstacle for a flow of blood from the left atrium to the left ventricle during diastole. The part of blood remains in the left atrium. Besides blood from pulmonary veins comes into the left atrium. In the left atrium the volume of blood is increased (in norm 50-60 ml, at narrowing 100-2000 ml), pressure raises (in norm - 5-7 mm Hg, at narrowing - 20-25 mm Hg). So the left atrium hypertrophies.

- However the muscle of a hypertrophied left atrium weak, therefore its contractile function reduces soon. It leads to dilation of the left atrium and increasing of venous pressure in pulmonary veins and capillaries. Increased pressure elevates in the pulmonary veins leads to irritation of baroreceptors, and causes reflex contraction of the arterioles in the lesser circulation (Kitaev's reflex), so pressure in the pulmonary trunk considerably rises, so called pulmonary hypertention. Pulmonary hypertension leads to a hypertrophy of the right ventricle, and subsequently and to its dilation. The left ventricle receives less blood in diastole, its size a little decreases and diastolic dysfunction develops.

Clinical picture

- When congestive changes occur in the lesser circulation, the patient complains on dyspnea and palpitation on physical exertion; he complains of pain in the heart, cough, and hemoptysis.
- When lung congestion drveelops, patients complain on evolving types of dyspnea, paroxysmal nocturnal dyspnea, orthopnea, as well as recumbent cough and hemoptysis that may be associated with rupture of bronchial veins during bronchitis or later be the frothy, bloody sputum or pulmonary edema.

facies mitrale

- **Inspection** reveiles acrocyanosis or cyanosis of cheeks, tip of the nose and the area above the bridge, the malar erythrocyanosis may be noted (lat. - “facies mitrale”). Visual examination of the heart region often reveales a cardiac beat consequent upon dilatation and hypertrophy of the right ventricle (lat. - gibbus cardiacus).
- The arterial pulse is usually normal, except that in patients with a low cardiac output and atrial fibrillation it will be reduced.

- If the defeat develops in childchood – patient’s growth slows down, infantilism develops /lat. - "mitral nanism”/.
- In the case of severe left atrial enlargement the last one presses on the nervus larynageus (its ramus recurens), therefore paresis of phonatory bands develops and the patient loses voice /aphonia/.

НАНИЗМ (карликовость)

от греч. nanos — карлик

низкий рост человека: для мужчин ниже 125 см, для женщин ниже 120 см

Причины:

- обусловлен поражением желез внутренней секреции: гипофиза, щитовидной и половых желез

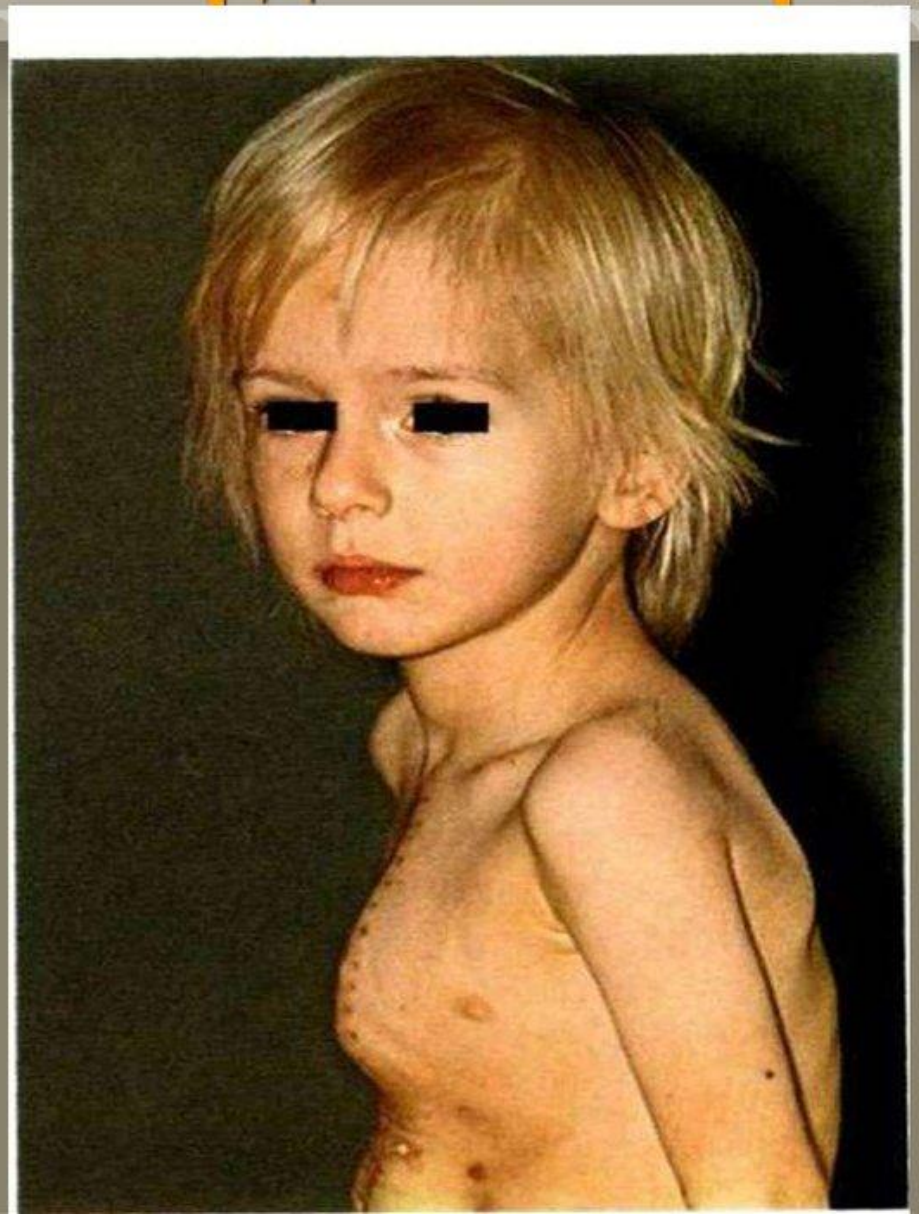
Карликовость митральная

Значение слова карликовость митральная по словарю медицинских терминов: карликовость митральная (n. mitralis) - К., наблюдаемая у лиц, с детства страдающих пороками левого предсердно-желудочкового (митрального) клапана сердца.

- Effort fatigue predominates early, followed by symptoms related to the hepatic and visceral congestion.
- The symptoms tend to progress gradually, but there will be brief periods of increased severity of symptoms due to respiratory infections, unusual physical or emotional stress, paroxysmal atrial fibrillation, pregnancy, or pulmonary embolism.

- Palpation: The left ventricular impulse is normal and somewhat difficult to detect except in the left lateral decubitus position. In presence of significant pulmonary hypertension, a hypertrophied right ventricle will produce an outward impulse that can be seen and palpated along the left sternal edge.
- The diastolic (presystolic) trill /lat. - fremissement cataire/ best of all is determined in the position of the patient on the left side at maximal expiration. It is caused by transit of blood through reduced apertura from the left atrium to the left ventricle.

«Сердечный горб»



- The pulse in mitral stenosis can be unequal on right and left arms. At considerable hypertrophy of the left atrium the left subclavial artery is pressed by the left ventricle and pulse filling on the left diminishes, (lat. - pulsus differens). If the left ventricle is not filled completely and the stroke volume is decreased, the pulse become small lat. - (pulsus parvus). At the case of arrhythmia pulse is atthythmic.
- Blood pressure is normal, sometimes systolic pressure slightly decreases and diastolic one - increases.

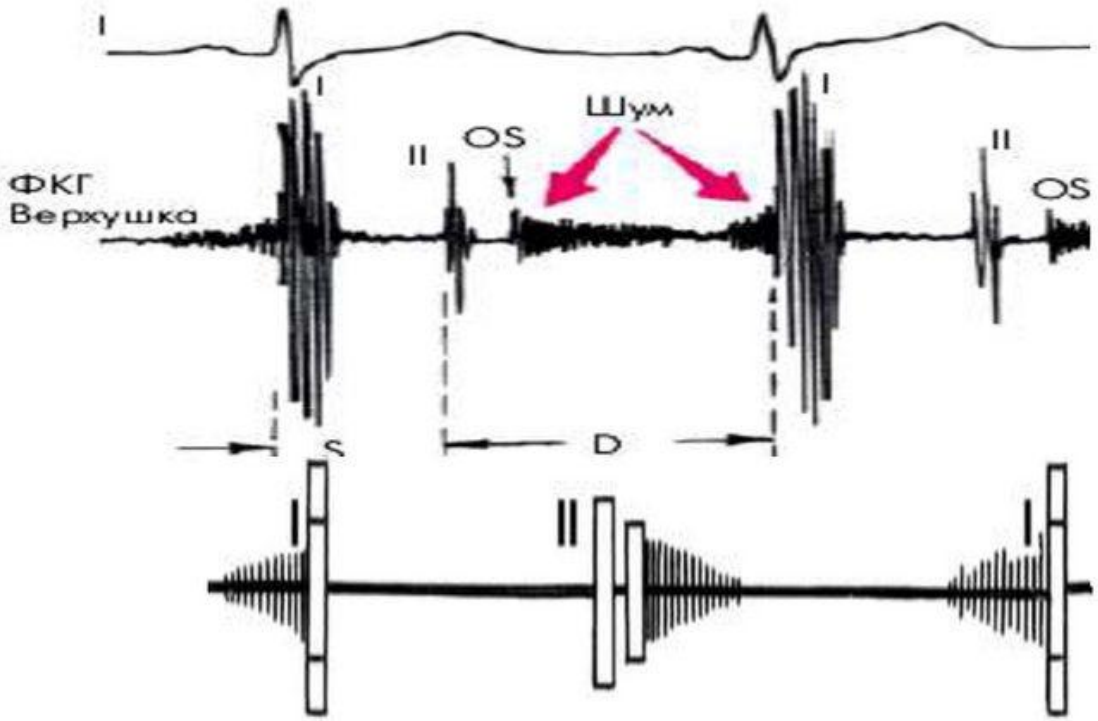
Сердечный толчок

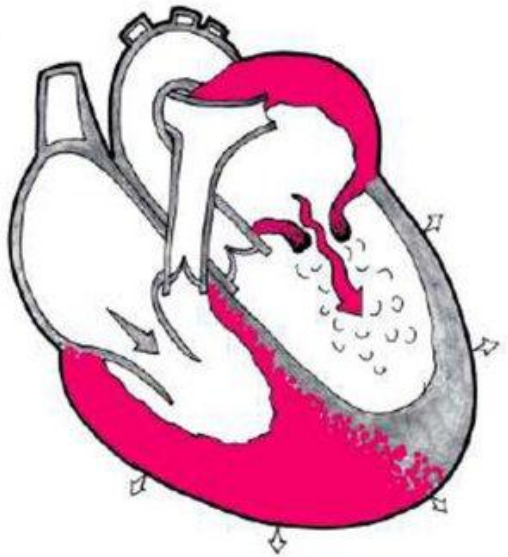
- Пальпируется только при увеличении правого желудочка
- Определяется по левому краю грудины у мечевидного отростка

- **Percussion:** displasement of heart's borders upwards and leftwards resulting in hypertrophy of the left atrium and right ventricle. Thus the heart is of "mitral" configuration.
- **Auscultation:** The cardinal auscultatory features of initial stenosis consist of accentuation of the first heart sound, early diastolic opening snap, and early, middiastolic crescendo-descendo murmur with presystolic accentuation. Mitral stenosis in its early or milder form is often unrecognized unless the physician listens carefully at the apex, with the patient in the left lateral decubitus position, and uses the bell of the stethoscope to detect the low-frequency diastolic murmurs. The patient should be examined both at rest and with exercise in order to estimate the severity of the stenosis property.

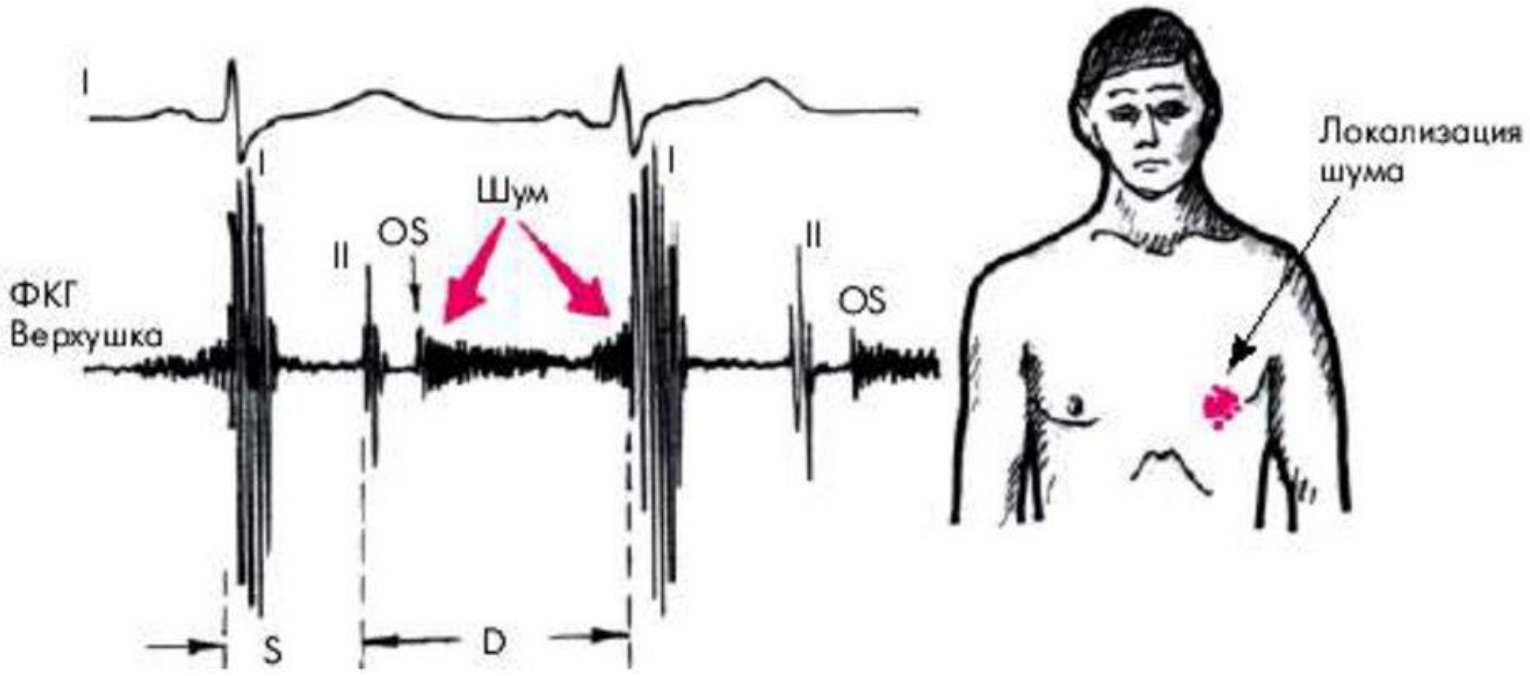
- The first heart sound characteristically is very loud, it is the result of two factors: (1) thickened chordae which are less elastic than normal and therefore cause a more abrupt termination of valve motion, and (2) the mitral valve is close to being maximally open at the onset of systole. Due to delay in mitral valve closure the left ventricle is not completly filled with blood and heart contraction descends promptly. The 1 sound sometimesn resembles the hue of the "sound in the empty pot".
- An adventitious sound due to opening of the mitral valve can be heard at the apex beat – so-called an opening snap sound which is an important auscultatory sign of mitral stenosis. This sound is heard between 30 and 120 msec after the aortic valve component of the second sound and occurs when left ventricular pressure drops below left atrial pressure in early diastole.

Мелодия митрального стеноза - «Ритм перепела»





Место выслушивания **диастолического шума** при стенозе левого атриовентикулярного отверстия



S - систола; **D** - диастола; **OS** - тон открытия митрального клапана

- It is a brief, high-pitched sound with wide transmission. It is caused by the "tambourine effect" of the valve with fused commissures .snapping into the ventricle rather than opening to allow free flow of blood. It is best heard at the point of maximal apical impulse; it also is heard easily along the left sternal border with the patient in the recumbent position. In addition to the implication of disease of the mitral valve, almost always rheumatic, the relationship of this sound to the aortic valve component of the second sound is helpful in assessing the severity of the mitral valve stenosis.

- The II sound normally occurs within 50 msec of the onset of the QRS. Because of the delay in mitral valve closure, the mitral component of the II sound is delayed. In mitral stenosis, it is usually delayed 70 msec or more. The delay of the II sound depends on the duration of the preceding cycle.
- The intensity of the second heart sound usually is normal in patients with mitral stenosis. However, one have to separate it from the opening snap before one can reliably define the qualities of its components.

- With severe mitral stenosis and the appearance of pulmonary hypertension, the pulmonary component of the second sound will increase in intensity (accentuation of the second heart sound above pulmonic artery). The loud first sound, second sound, and the sound of mitral valve opening give a specific murmur which is characteristic of mitral stenosis and resembles the song of a quail.
- A formula for estimating of the severity of the mitral stenosis is devised based on the delay in the first sound and the interval between the second sound and the opening snap of the mitral valve: the more severe the is stenosis, the greater the Q-to-first sound interval and the shoner the second sound-Q-to-opening snap interval. In calcific mitral stenosis, the first sound may be softer than normal, and then the opening snap is usually absent.

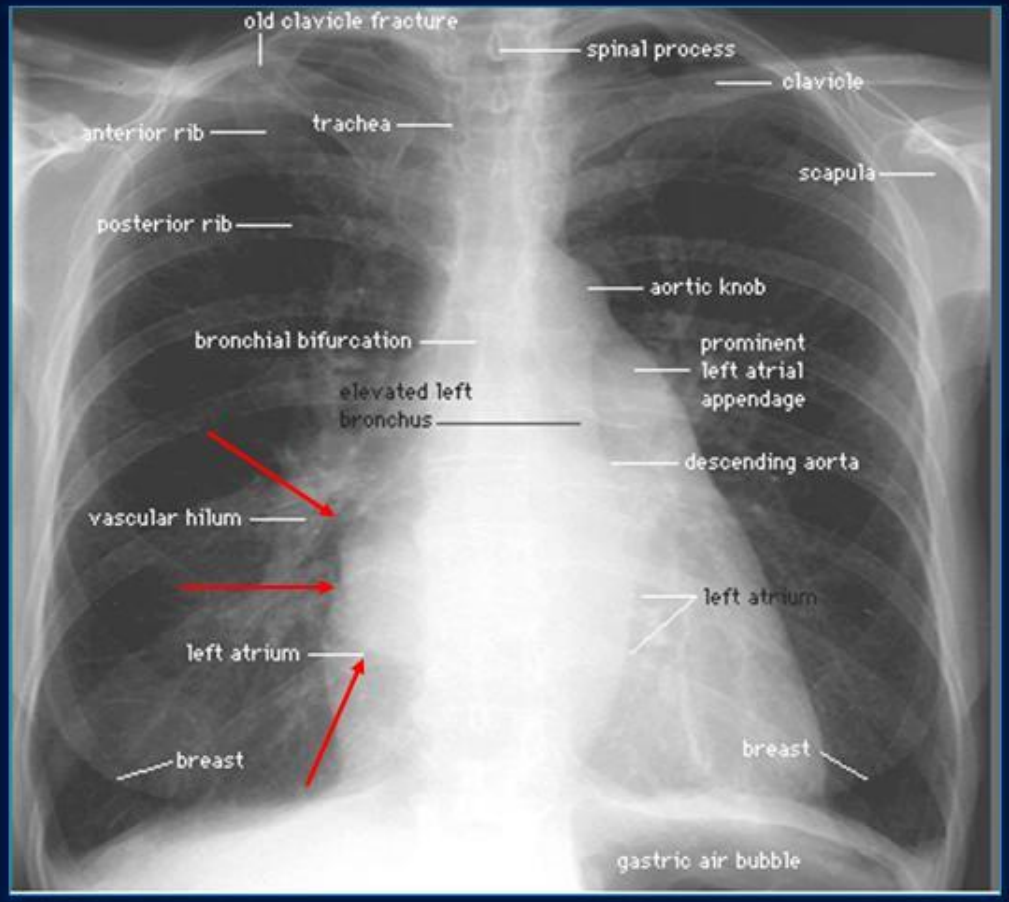
- **Murmurs.** The characteristic murmur of the mitral stenosis is a low-pitched diastolic creschendo- deschendo murmur heard best with the bell applied lightly to the skin over the heart apex. The murmur varies in length and may be only presystolic, middiastolic, or holodiastolic, can be heared during all diastole, strengthening before a systole (presystolic accentuation) and immediately merging with clapping first sound.: A holodiastolic murmur during long diastolic periods indicates a persisting gradient and hence severe stenosis. In the milder form of mitral stenosis it may be difficult to hear, and often the patient must exercise before it is clearly audible. The most characteristic murmur of mitral stenosis is one that begins immediately after the opening snap of the mitral valve, extends throughout diastole with presystolic accentuation, and ends with a loud first sound.

- Finally, in the presence of pulmonary hypertension complicating mitral stenosis, pulmonary valve incompetence will develop. Trills is associated with a high-pitched decrescendo diastolic murmur heared best along the left sternal border (Graham Steele murmur). When this murmur is present, it is associated with a. loud pulmonary component of the second sound that usually is palpable and implies severe mitral stenosis.
- In the case of calcification of the mitral valve rasping systolic murmur gains "irony" shade and sometimes a high “murmur-squeak" may be revealed. In the case of appearance of ciliary arrhythmia in patients with mitral stenosis heart sounds become absolutely inordinate. S.P.Botkin compared them with "a forge hum".

Chest roentgenogram

- On the chest roentgenogram –shows a specific enlargement of the left atrium, which leads to disappearance of the heart waist /the mitral heart configuration/ are detected. On the lateral film (the first oblique position) enlargement of the left atgrium is determined by the degree of displacement of the esophagus which is good visible after the patient have drunk baric suspension. The chest roentgenogram is helpful in both confirming the diagnosis and assessing the severity of the stenosis. A large left atrial sillhouette, dilated upper lobe veins, and evidence of alveolar or interstitial edema with 1- to 3-cm horizontal lines in the costophrenic angles (Kerley B lines) all are consistent with severe mitral stenosis.

Митральный стеноз



- Типичная картина стеноза митрального отверстия
- Обратите внимание! Признаки гипертрофии левого предсердия хорошо видны и без бокового снимка

Митральный стеноз

- In presence of pulmonary hypertension, the swelling of pulmonary artery arch and hypertrophy of the right ventricle are watched.The central pulmonary arteries are prominent, and there will be a sudden decrease in the vascular markings in the peripheral lung fields associated with right ventricular enlarge¬ment that will be seen best in the lateral film. Valvular calcification is visible on roentgenogram. At pulmonary prolonged hypertension pneumosclerosis develops.

Electrocardiogram

- The **electrocardiogram** in mitral stenosis maps signs of hypertrophy of the left atrium and left ventricle: deviation of heart electrical axis rightwards, high and prolonged waves P in the right thoracic leads and expressed waves f in the left thoracic leads may be detected. The ECG may be often normal except for a prolonged, bifid P wave in lead 2 and a prominent late negative deflection in V1 related to left atrial enlargement. Atrial fibrillation is the most common abnormal rhythm. Despite severe degrees of right ventricular hypertrophy, the ECG commonly will disclose only right axis deviation with S waves in leads 1 and V6 and an rSr in lead V1.

Митральный стеноз

Электрокардиограмма в I стандартном отведении (вверху) и **фонокардиограмма** (внизу)

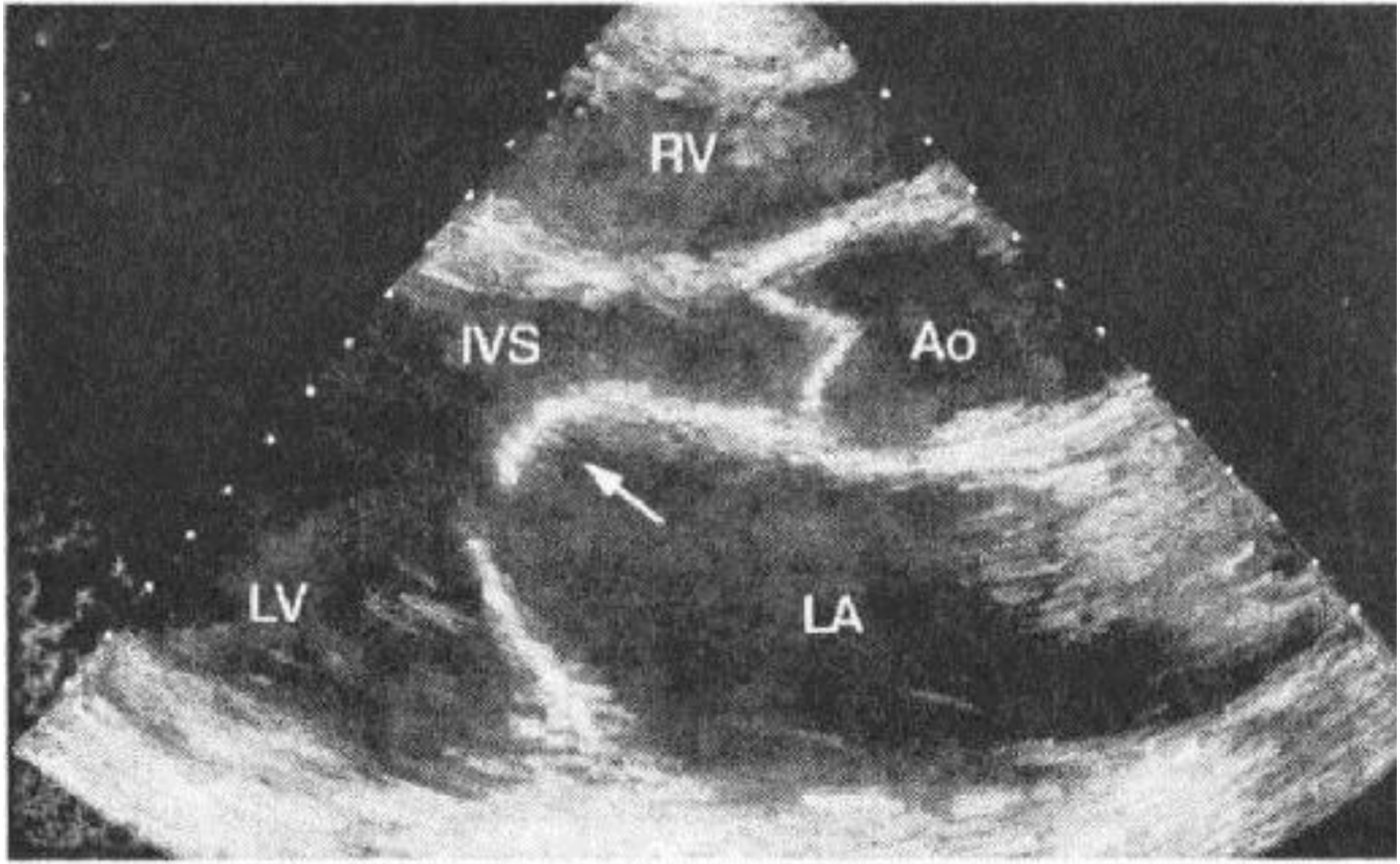
- на ЭКГ - широкий с двумя вершинами зубец Р (***P-mitrale***),
- Часто мерцательная артимия,
- на ФКГ — высокоамплитудный I тон, тон открытия митрального клапана (указан стрелкой),
- диастолический шум с пресистолическим усилением.

- On the **phonocardiogram** taken from the apex high voltage of the I heart sound is often detected; the second sound is often followed with the opening snup and diastolic murmur, the last one is better heard above the pulmonary artery; voltage of the П sound is enlarged in comparing with those above the aorta. The interval Q-to-I sound is enlarged.

Echocardiography

- Pertinent information obtainable from echocardiographic examination of a patient with mitral stenosis includes extent of calcification and fibrosis of the mitral valve, presence of atrial thrombi, enlargement of chambers in size and disorders of contractility, presence of other valvular abnormalities.The hypertrophy and augmentation of the left atrium, change of the mitral valve leaflets /induration, adhesion, calcification) may be revealed, the locomotion of leaflets becomes undirectional, gains the Π-similar form.

Echocardiography



- As the **surgical treatment** of the acquired heart valvular disease is successful now, it is necessity to precise diagnostics of degree of narrowing or degree of the valvular failure and detection of prevalence of one of them.

AORTIC INCOMPETENCE

Aortic incompetence (aortic insufficiency, aortic regurtation)

- Definition: Aortic incompetence is the failure of the aortic valve to close completely during ventricular diastole; blood thus leaks back into the left ventricle.

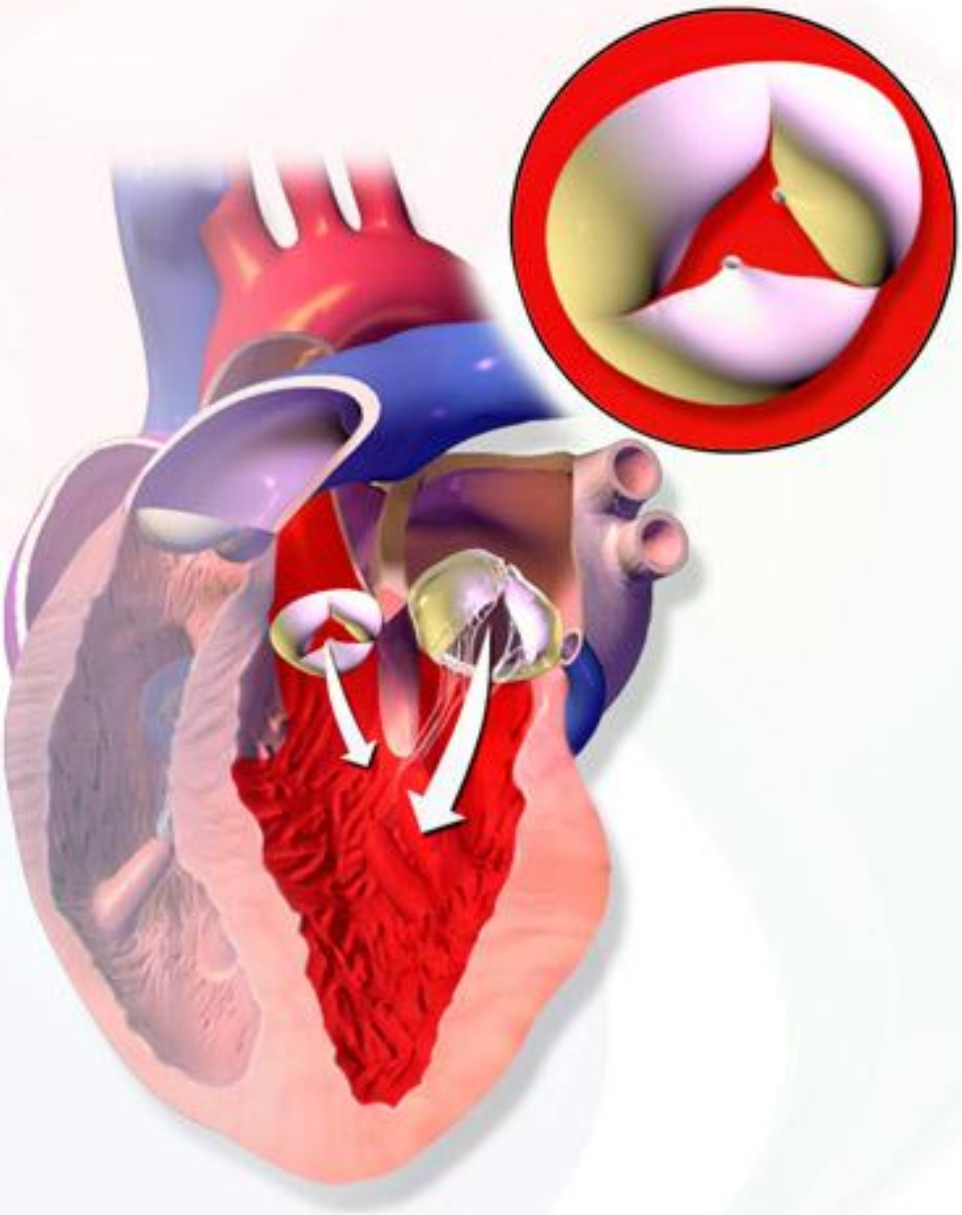
Etiology

- Aortic incompetence is usually secondary to rheumatic endocarditis, and less frequently infective (bacterial, septic) endocarditis, syphilitic affection of the aorta, or atherosclerosis. Inflammatory and sclerotic changes occurring in the base of the cusps during rheumatic endocarditis make them shrink and shorten. Atherosclerosis and syphilis can affect only the aorta (to distend it), while the valve cusps are only shortened. The cicatricial changes may extend onto the cusps to disfigure them. Parts of the valve disintegrate in ulcerous endocarditis associated with sepsis and the cusps are affected with their subsequent cicatrization and shortening

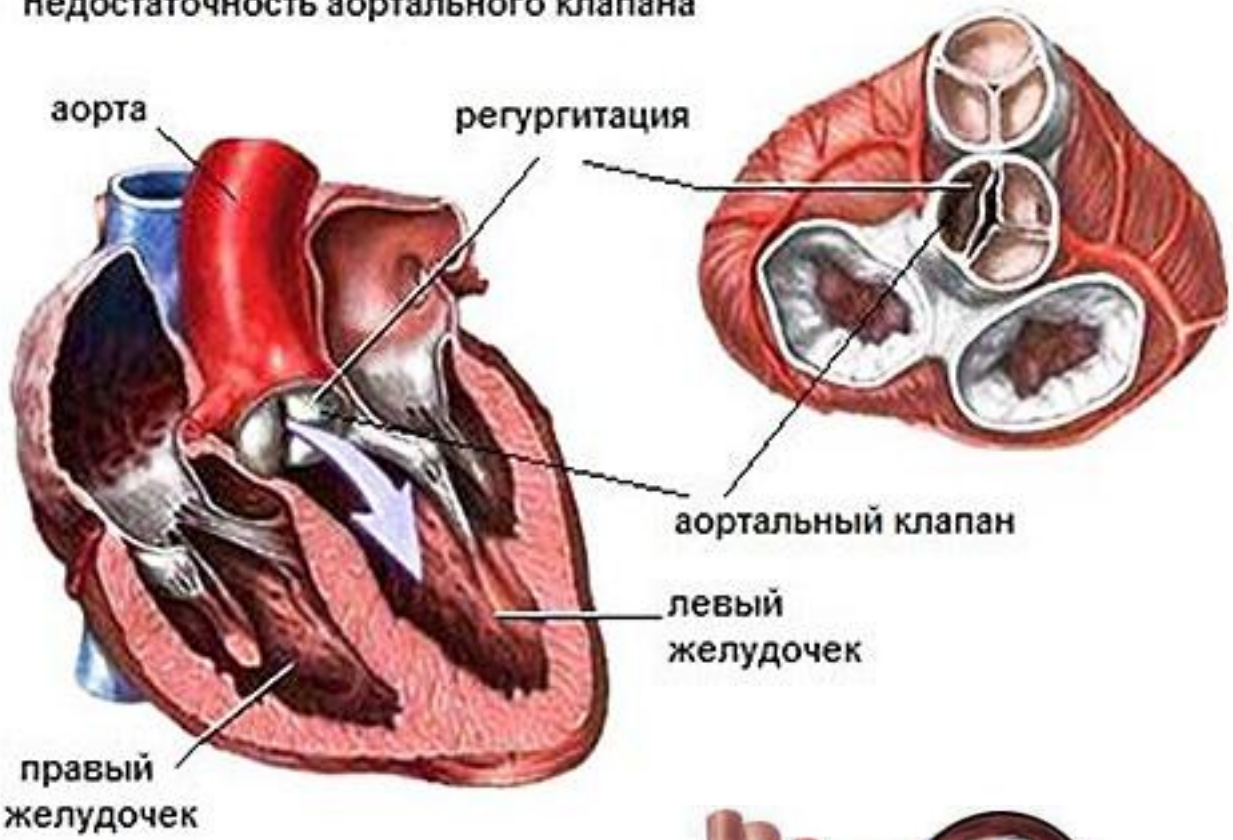
Disorders of hemodynamics

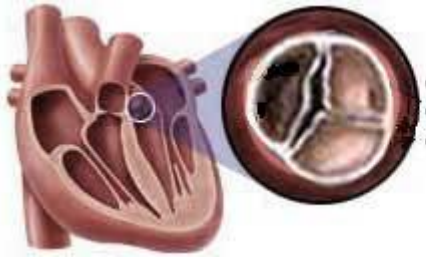
- During diastole the blood is delivered into the left ventricle not only from the left atrium but also from the aorta due to regurgitation, thus the left ventricle during diastole overfills. The amount of returning blood can reach from 5 up to 50 % of volume of the left ventricle. There is a significant overload of the left ventricle with volume (systolic volume may reach 200 ml and more).

АОРТАЛЬНАЯ НЕДОСТАТОЧНОСТЬ



Недостаточность аортального клапана

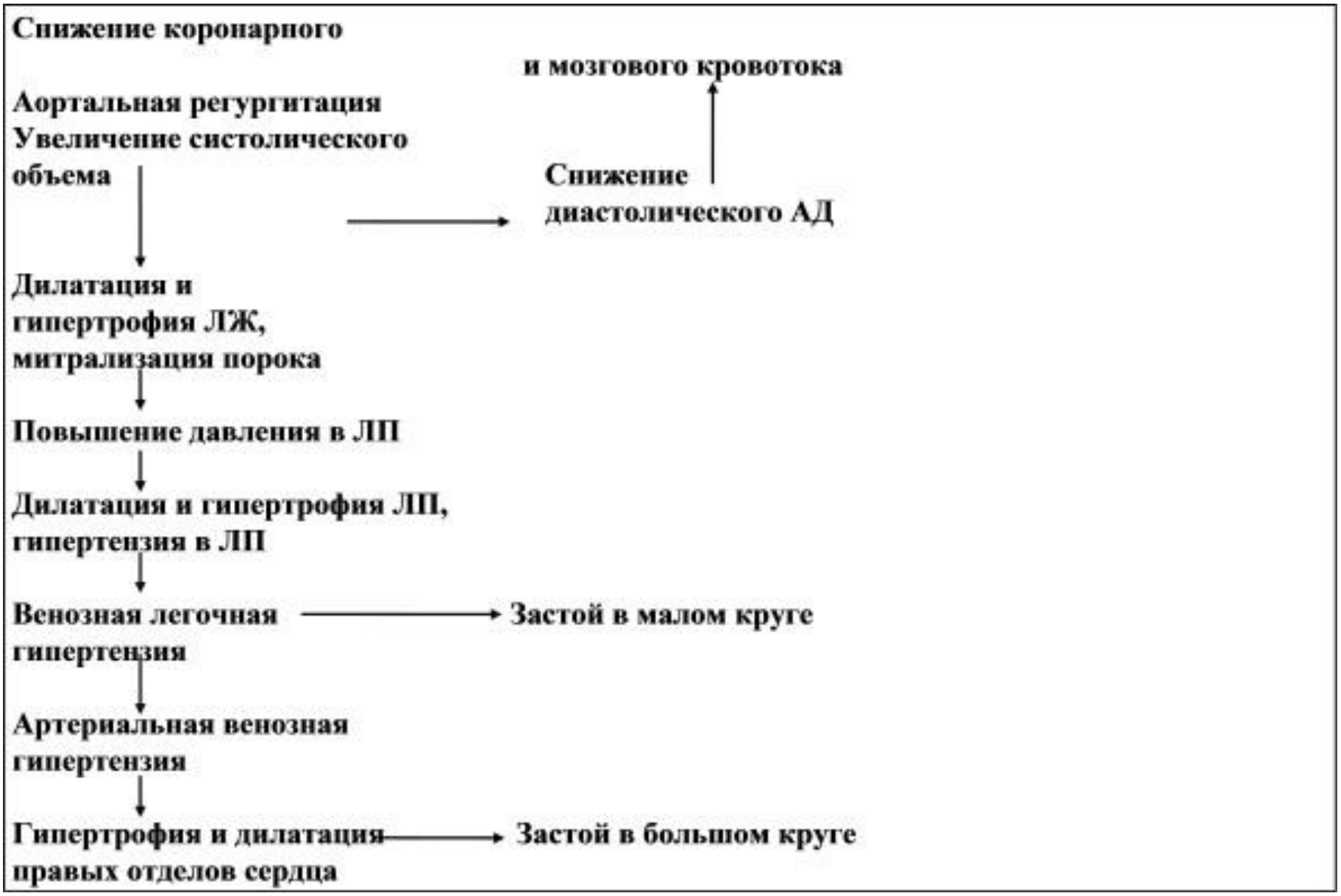




створки аортального клапана смыкаются не полностью, в связи с чем возникает регургитация

Disorders of hemodynamics

- During systole the left ventricle has to contract with a greater force in order to expel the large volume of blood into the aorta. Insufficiency of the aortic valve for a long time may be compensated by the strengthened work of hypertrophied powerful left ventricle. In case of intense regurgitation of the blood that moves aside the mitral valve is formed functional mitral stenosis. During progression of disease increased systolic volume at left ventricle causes its dilation. At longstanding course the disease may be accompanied by functional incompetence of mitral valve with regurgitation of blood in left atrium, which is hypertrophied as response to overloading. This is mitralization of aortic regurgitation.


Снижение коронарного
и мозгового кровотока
Аортальная регургитация
Увеличение систолического
объема
Снижение
диастолического АД
Дилатация и
гипертрофия ЛЖ,
митрализация порока
Повышение давления в ЛП
Дилатация и гипертрофия ЛП,
гипертензия в ЛП
Венозная легочная
гипертензия
Застой в малом круге
Артериальная венозная
гипертензия
Гипертрофия и дилатация
правых отделов сердца
Застой в большом круге

Clinical picture

- **Clinical picture.** Subjective condition of patients with aortic sncompetence may remain good for a long time because the defect is compensated for by harder work of the powerful left ventricle. Pain in the heart (anginal in character) may sometimes be felt; it is due to relative coronary insufficiency because of pronounced hypertrophy of the myocardium and inadequate filling of the coronary arteries under low diastolic pressure in the aorta. The patient may sometimes complain of giddiness which is the result of deranged blood supply to the brain (which is also due diastolic pressure).

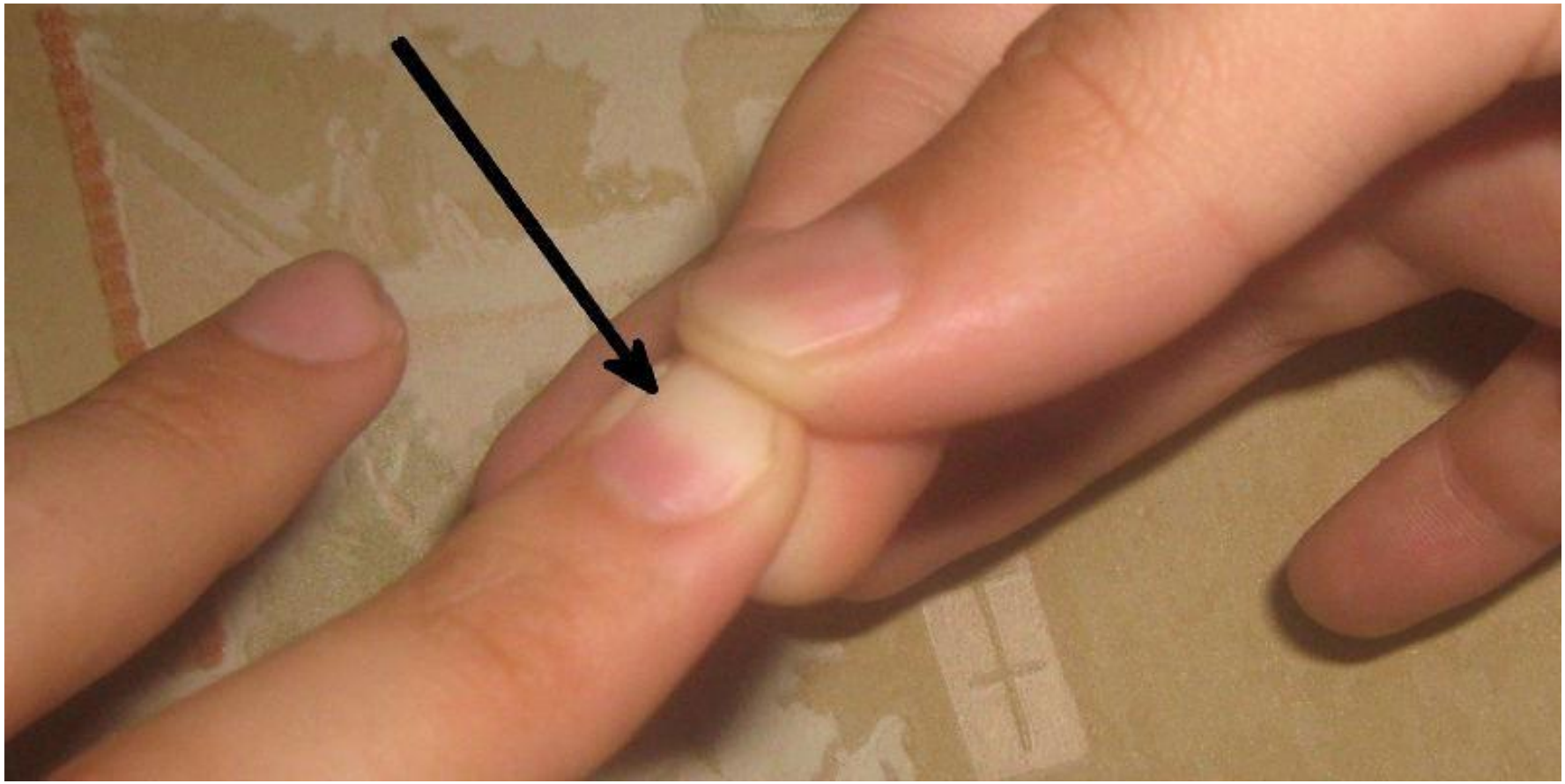
Complaints

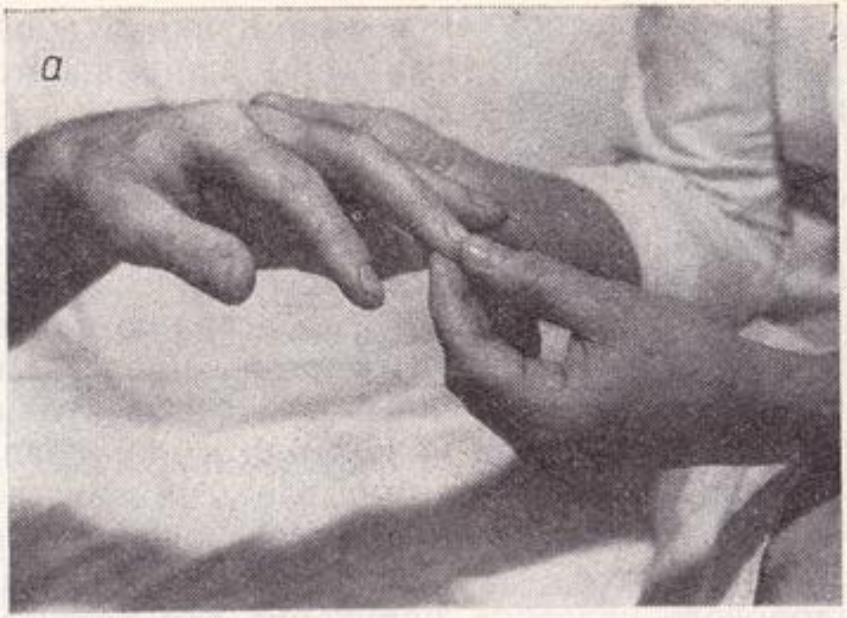
- Pain in the heart (anginal in character) may sometimes be felt; it is due to relative coronary insufficiency because of pronounced hypertrophy of the myocardium and inadequate filling of the coronary arteries under low diastolic pressure in the aorta. The patient may sometimes complain of giddiness which is the result of deranged blood supply to the brain (which is also due to low diastolic pressure). If contractility of the leftventricular myocardium is impaired, congestion in the lesser circulation develops and the patient complains of dyspnea, tachycardia, weakness, etc.

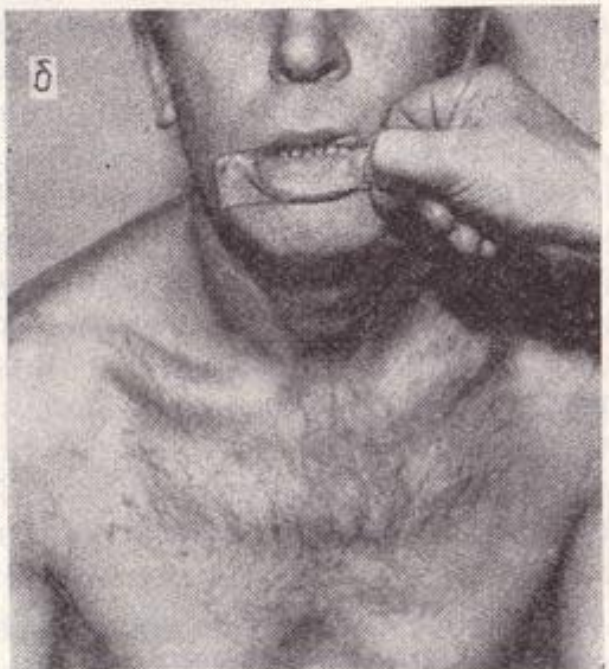
Inspection

- The skin of the patient is pallid due to insufficient filling of the arterial system during diastole. Marked variations in the pressure in the arterial system during systole and diastole account for the appearance of some signs, such as pulsation of the peripheral arteries, the carotids (carotid shudder), subclavian, brachial, temporal, and other arteries; rhythmical movements of the head synchronous with the pulse (Mussel's sign), rhythmical change in the colour of the nail bed under a slight pressure on the nail end, the so-called capillary pulse (Quincke's pulse), rhythmical reddening of the skin after rubbing, etc.

So-called capillary pulse (Quincke's pulse)




а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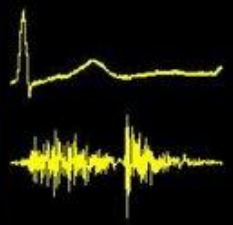
Симптомы усиления пульсации:



- “пляска каротид» ;
- Мюссе
  - ритмичное покачивание головы вперед и назад в соответствии с фазами сердечного цикла (в систолу и диастолу);
- Квинке
  - (“капиллярный пульс” - усиление пульсации ногтевого ложа.
- Ландольфи
  - пульсация зрачков в виде их сужения и расширения;
- Бекера > пульсации артерий сетчатки;
- Мюллера -> пульсация мягкого неба.
- Розенбаха -> пульсации печения;
- Герхарда -> пульсации селезенки;
- Траубе – двойной над бедренной артерией при ее небольшом надавливании;
- Дюрозье : систоло-диастолический шум над бедренной артерией;

Figure 75 Palpating common carotid artery.

Musset

Аортальная недостаточность

Клиника:

- Компенсированная АН, как правило, не ощущается больным
- Одышка при физической нагрузке, затем приступы сердечной астмы;
- сердцебиение, пульсация сосудов, головокружения, обмороки, в ряде случаев боли ангинозного характера;
- при внешнем осмотре аортальная бледность. Пульсация сосудов всех калибров (симптомы Мюссе, Мюллера, Ландольфи, Квинке);
- усиленный, разлитой верхушечный толчок, смещеннй влево и вниз;
- границы сердечной тупости смещены влево и вниз;
- при аускультации – диастоличесий шум над аортой, хорошо проводится влево и вниз до верхушки сердца, характеризуется высокой звучностью, мягкостью, возникат непосрдственно за 2 тоном, место наилучшго выслушивания – 2 межреберье справа и точка Боткина –Эрба. При декомпенсации продолжительность и интенсивность снижается., приглушенность 1 тона, ослабление или исчезновение 2 тона над аортой, дистолический шум Флинта.
- Пульс – большой, быстрый, высокий, короткий
- При аускультации над бедренной артерией двойной тон Траубе и двойной шум Дюруазье.
- АД – повышение САД и снижение ДАД, высокое пульсовое АД. АД на ногах превышающее АД на руках больше, чем на 60 мм, это свидетельствует о тяжелой АН – симптом Хилла.

- The apex beat is almost always enlarged and shifted to the left and inferiorly. Sometimes, along with the elevation of the apex beat, a slight depression in the neighbouring intercostal spaces can be observed The apex beat is palpable in the sixth and sometimes seventh intercostal space laterally of the midclavicular line. The apex beat is diffuse, intense, rising like a dome. This indicates significant enlargement of the left ventracle. The border of cardiac dullness can be found (by percussion) to shift to the left; the heart becomes "aortic" (with pronounced waist of the herat.

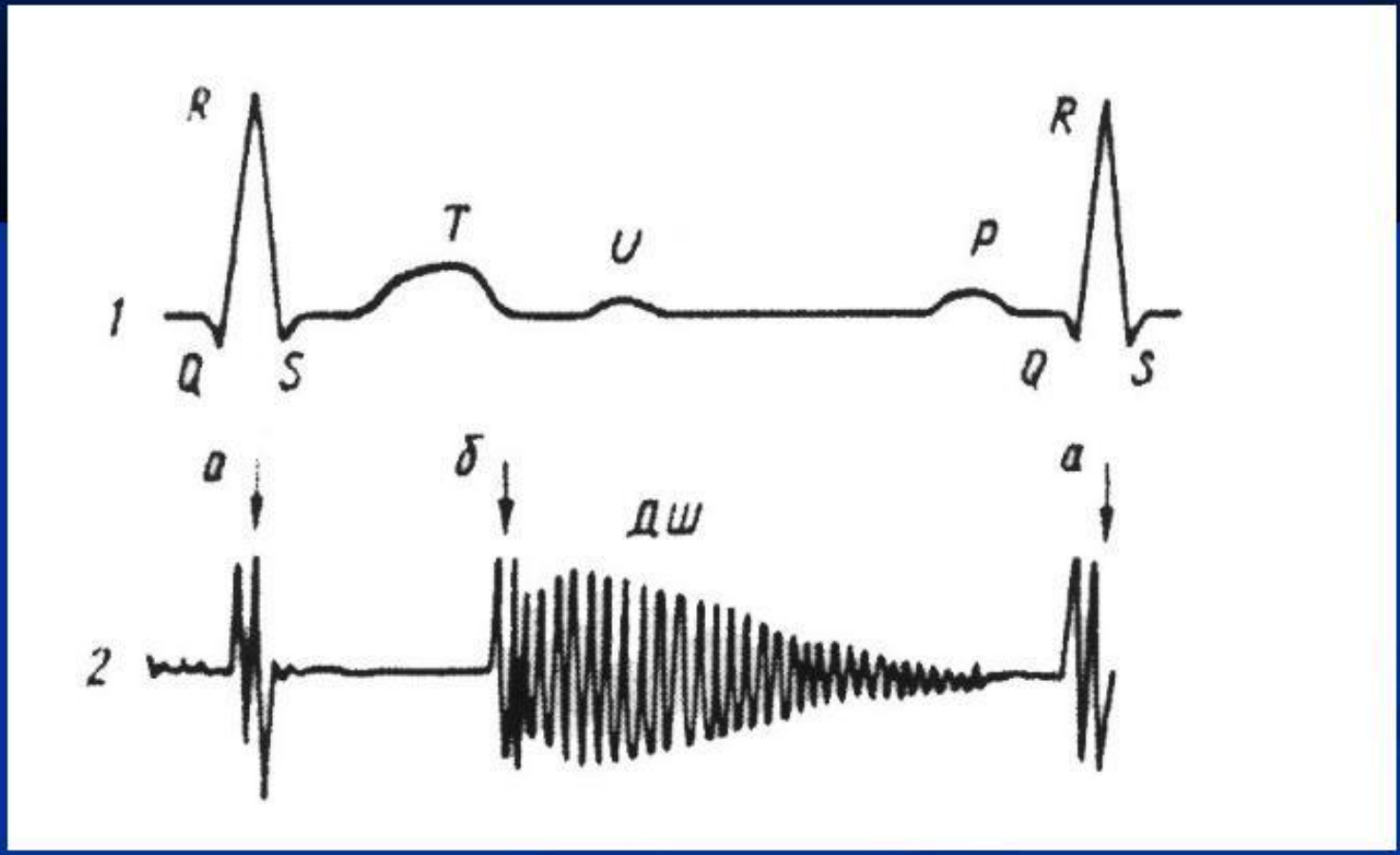
Auscultation

- Auscultation reveals decreased first sound at the apex, since during left-ventricular systole the period when the valves are closed is absent. The second sound on the aorta is also weak, and if the valve is damaged significantly, it can be inaudible. The second sound can be quite loud in atherosclerotic affection of the aorta. Diastolic murmur heard oer the aorta and at the Botkin-Erb listening point is characteristic. This is a low blowing protodiastolic murmur which weakens by the end of diastole sa the blood pressure in the aorta drops and the blood-flow rate decreases. The described changes in the sounds and murmurs are clearly visible on phonocardiogram.

Murmurs

- Murmurs of functional aetiology can also be heard in aortic incompetence at the heart apex. If the left ventricle is markedly dilated, relative mitral incompetence develops and systolic murmur can be heard at the apex. Diastolic murmur (presystolic or Austin-Flint murmur) can sometimes be heard. It arises due to an intence regurgitation of the blood that moves aside the mitral valve cusp to account for functional mitral stenosis. Doubled sound (Traube souble sound) nd doubled Vinogradov-Durozierz murmur can sometimes be heard over the femoral artery in this disease.

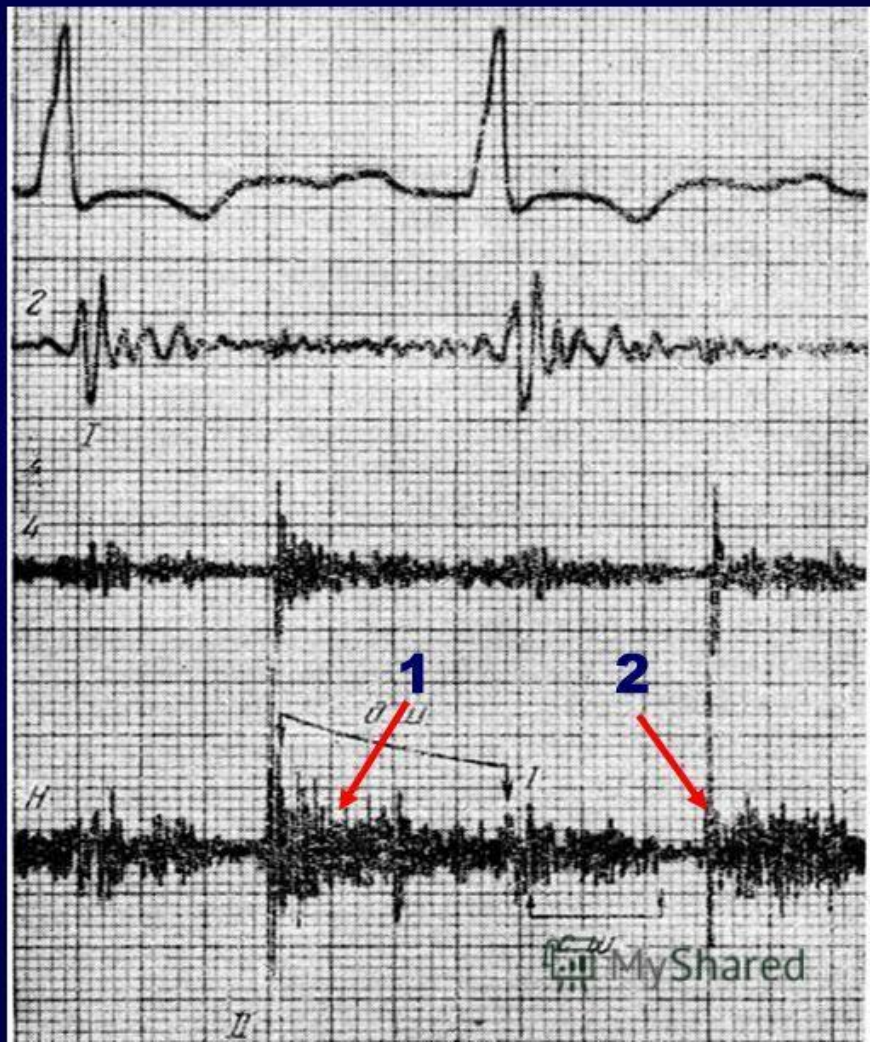
ФКГ при аортальной недостаточности



«КЛАПАННЫЕ» ПРИЗНАКИ НЕДОСТАТОЧНОСТИ АОРТАЛЬНОГО КЛАПАНА

1. Протодиастолический шум над аортой (во втором межреберье справа от грудины и в точке Боткина).

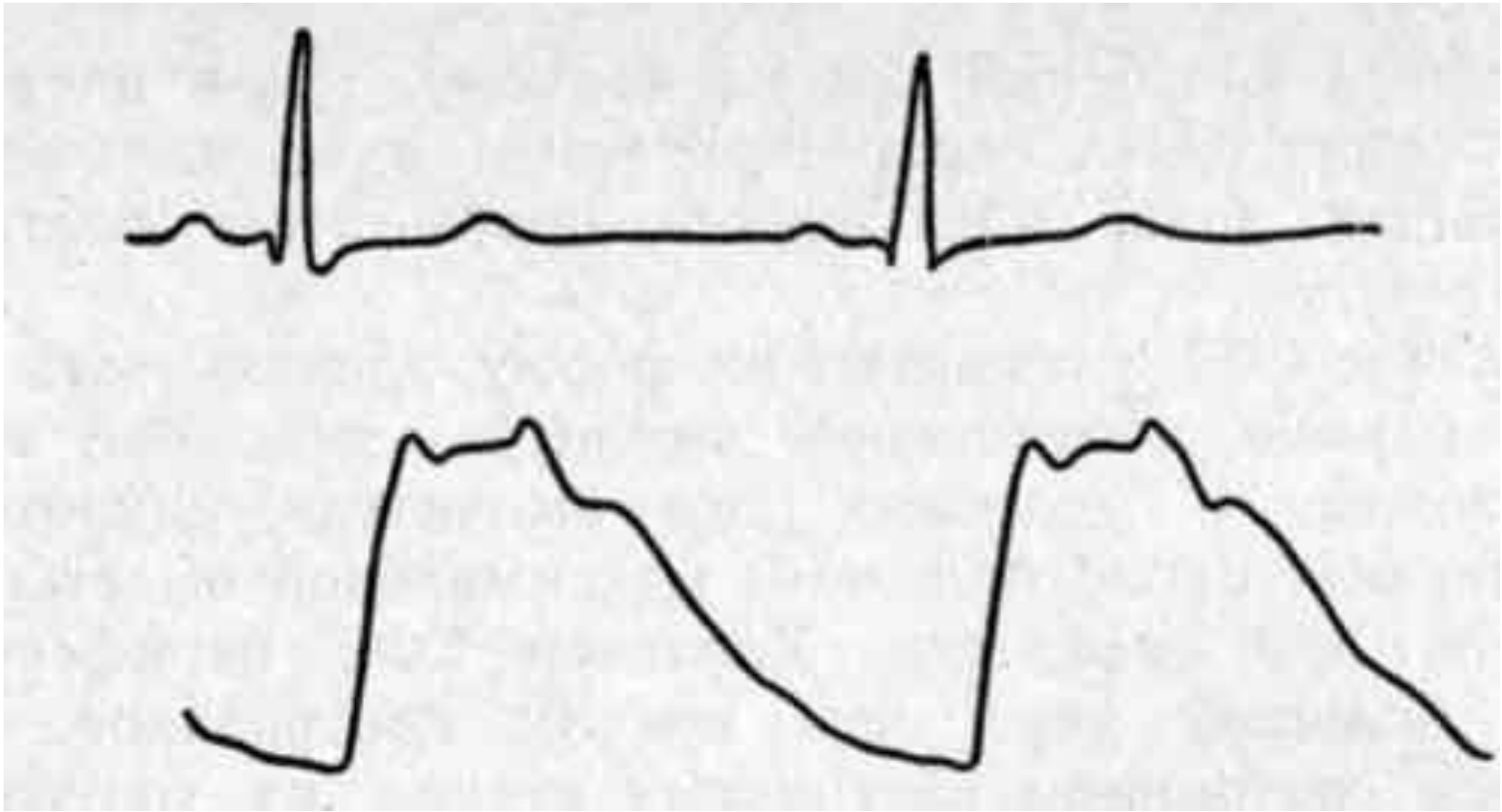
2. Ослабление (исчезновение) II тона за счет его аортального компонента.



Pulse is fast, full, and high

- The pulse is fast, full, and high, which is due to high pulse pressure and increased volume of blood deliverred into the aorta during systole. Arterial pressure constantly varies the systolic pressure rises and diastolic falls, and the pulse pressure is therefore high.

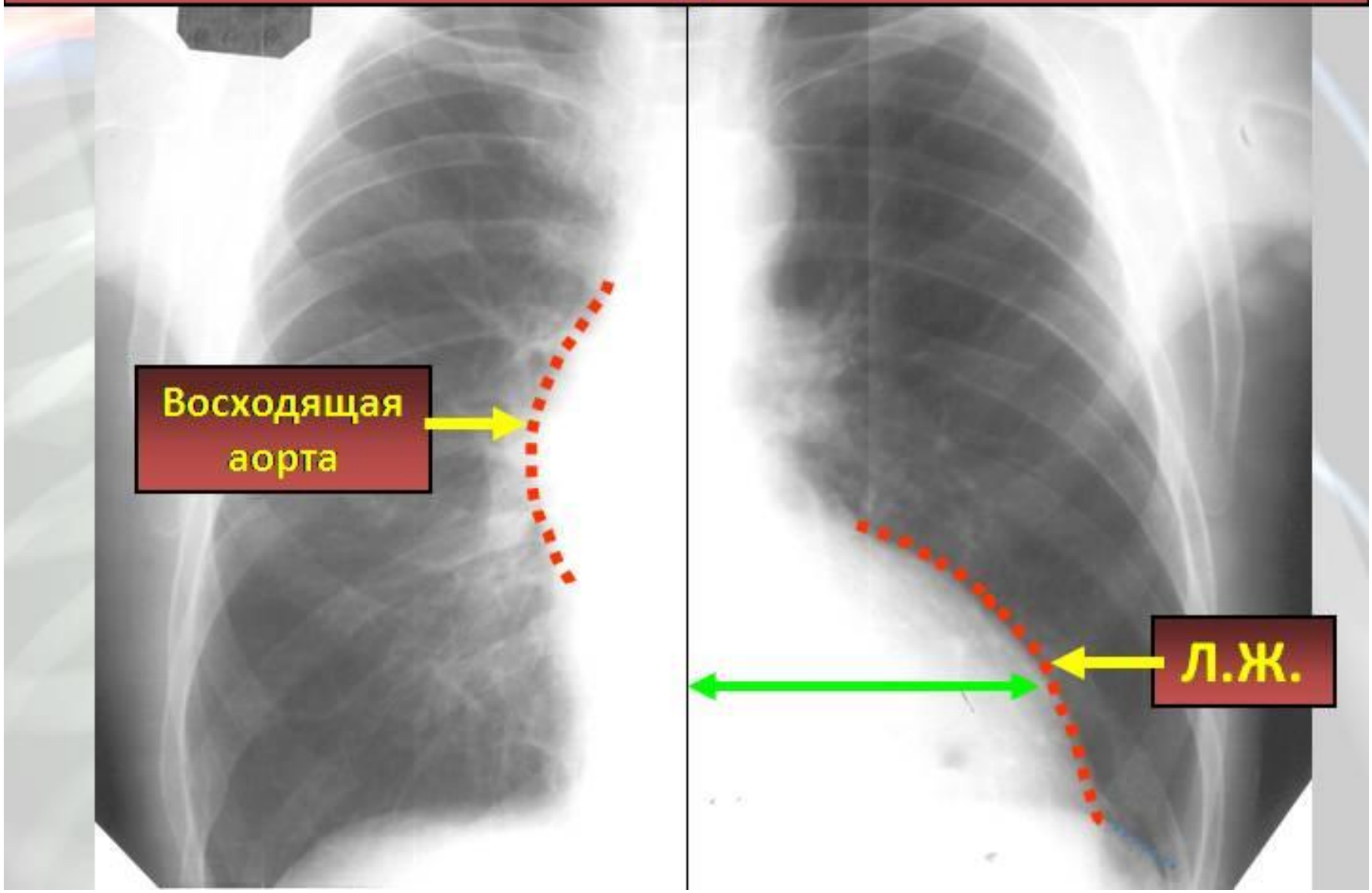
pulse is fast, full, and high



X-ray

- **X-ray** studies show an enlarged left ventricle with a distinct waist of the heart and dilatation of the aorta; pulsation of the aorta is intense.

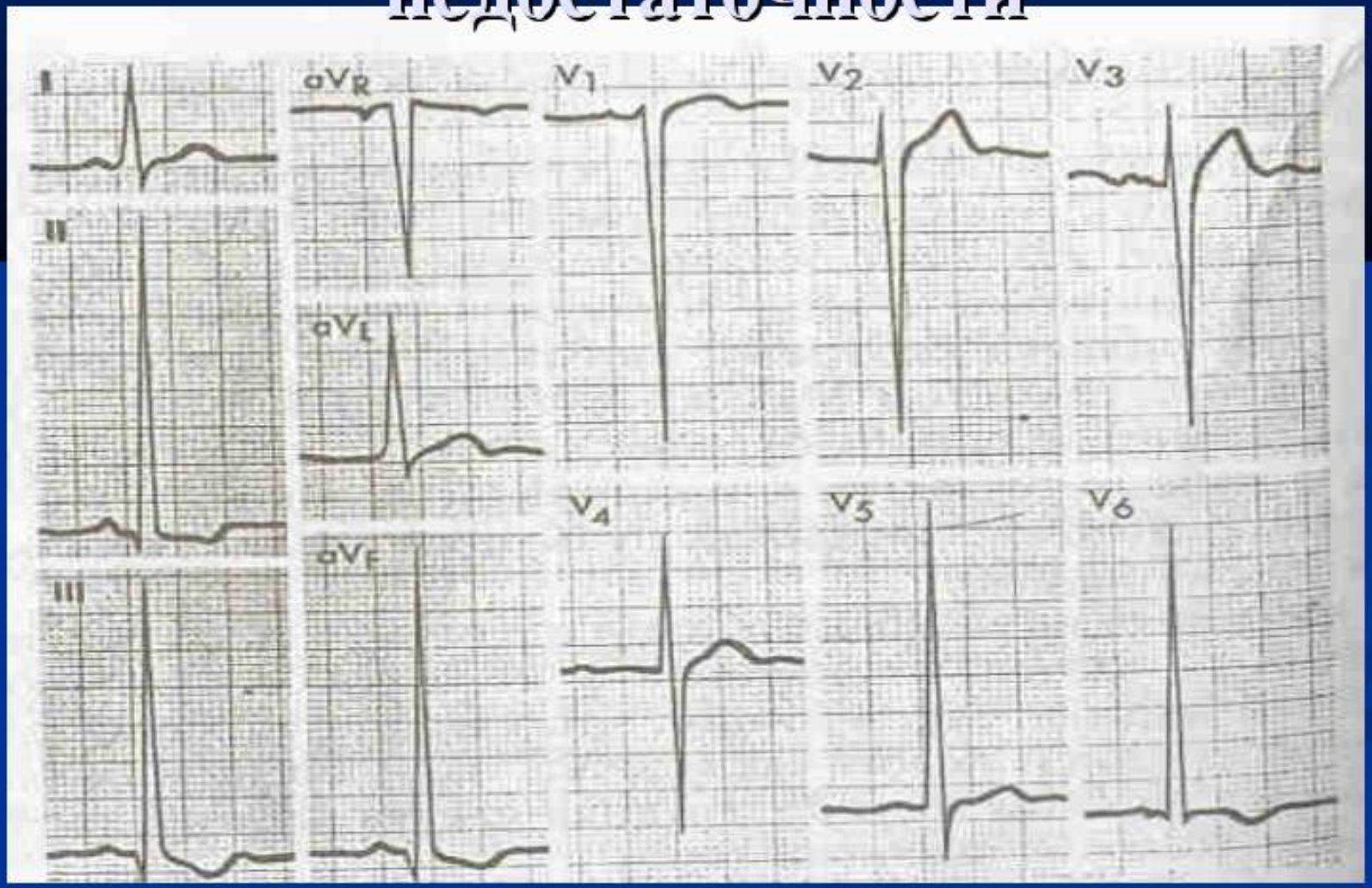
АОРТАЛЬНАЯ КОНФИГУРАЦИЯ
АОРТАЛЬНАЯ НЕДОСТАТОЧНОСТЬ



The ECG

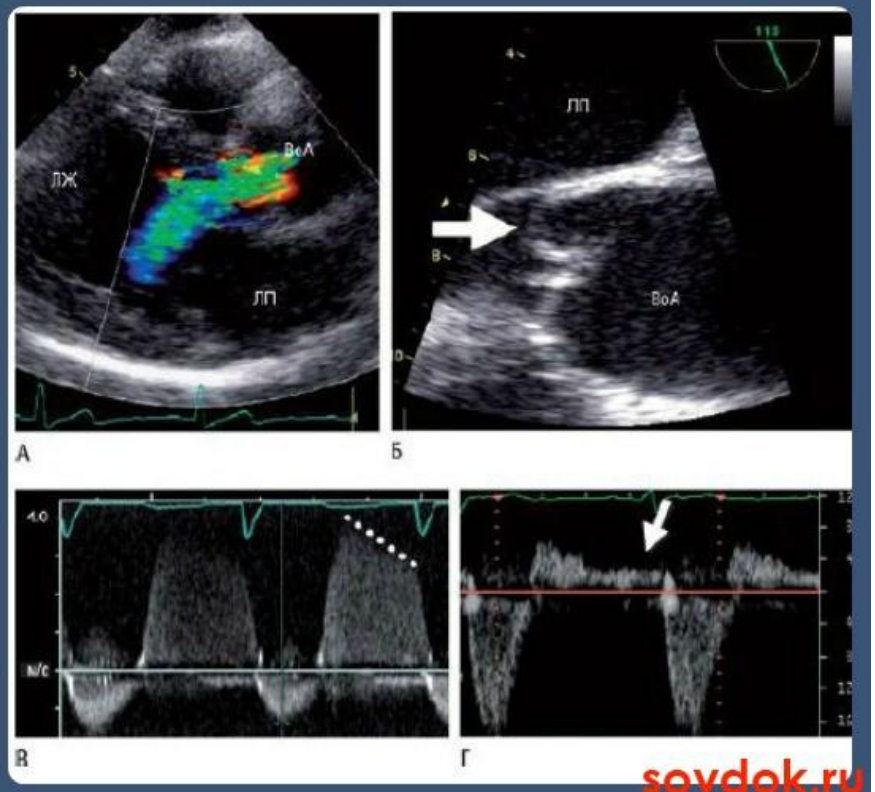
- **The ECG** also reveals various signs of hypertrophy of the left ventricle: the electrical axis is deviated to the left, the S waves in the right chest leads are deep and the amplitude of the R wave is higher in the left chest leads; these signs often combine with signs of overstrain in the left ventricle and relative coronary insufficiency (changes in the terminal part of the ventricular complex, displacement of the S-T interval, and the negative T wave).

ЭКГ при аортальной недостаточности



- **Echocardiograms** taken from patients with aortic failure show flutter of the anterior mitral cusp during diastole caused by the thrust of the blood regurgitated from the aorta into the ventricle.

Узи аортальной регургитации: проверка работоспособности аортального клапана



- Aortic incompetence can for a long time be compensated for by intensified work of the hypertrophied left ventricle. When its contractile force decreases, congestion in the lesser circulation develops. Acute weakness of the left ventricle sometimes develops and is manifested by an attack of cardiac asthma. Dilatation of the weakened left ventricle can cause relative mitral incompetence. This increases venous congestion in the lesser circulation associated with decompensated aortic incompetence and adds to the load on the right ventricle. This is mitralization of aortic incompetence, which may become the cause of venous congestion in the greater circulation.

AORTIC STENOSIS

- The narroving of the aortic orifice (aortic stenosis) interferes with expulsion of blood into the aorta during contraction of the left ventricle. Aortic stenosis is usually caused by rheumatic endocarditis; less frequently it develops due to bacterial endocarditis, atherosclerosis, or it may be congenital. Stenosis results from adhered aortic valve cusps or develops due to cicatricial narrowing of the aortic orifice.

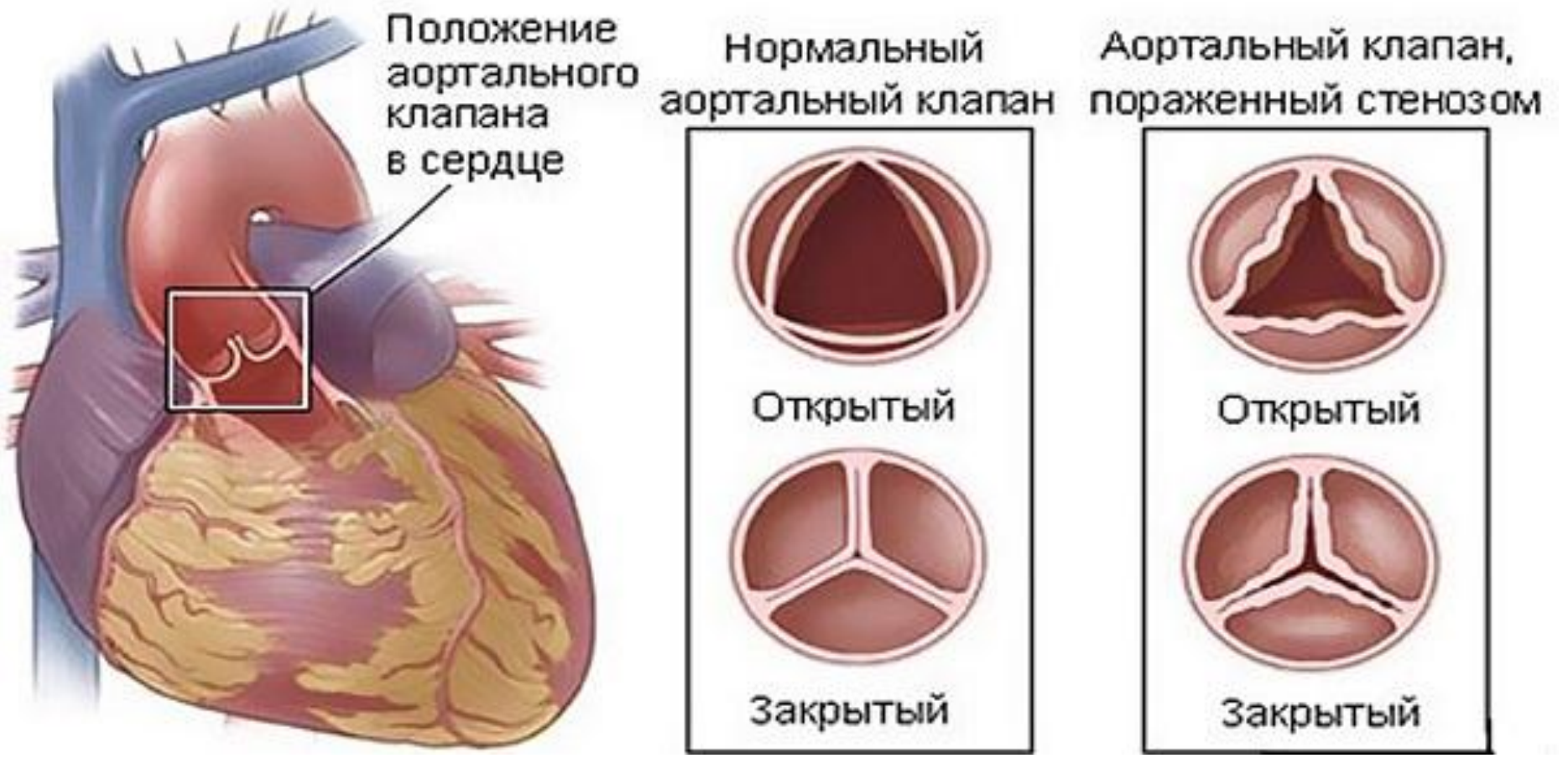
Disorders of hemodynamics

- At the expressed narrowing of the aorta orifice, when its area decreases up to 1.0-0.75 cm2 (in norm 3 cm2) during systole left ventricle does not empty completely. The gradient of systolic pressure between the left ventricle chamber and an aorta is increased. It exceeds 20 mm Hg. sometimes 100 and more.

Disorders of hemodynamics

- At narrowing of aortic orifice the minute volume of blood is reduced. In diastole to this remained blood in the ventricle the normal amount of blood from the left atrium is added that lead to overfilling of left ventricle with blood and to increase of pressure in it. Systolic pressure in the left ventricle raises proportionally degrees of narrowing of the aorta orifice (in norm 120 mm Hg, at narrowing raises in 1.5-2 times in comparison with a normal amount and may reach 250-300 mm Hg). This disorders of heart hemodynamics is compensated by the strengthened work of the left ventricle and causes its hypertrophy. Coronary blood flow may become inadequate.

- Due to good compensatory abilities of the left ventricle asymptomatic course of this disease may last 10-15 years. At reduction contractile abilities of myocardium develops dilatation of the left ventricle, later relative insufficiency of the mitral valve adds, with regurgitation of blood in the left atrium. First there is an intimate insufficiency in the lesser, and then in the lager circulation.


Положение аортального клапана в сердце
Нормальный аортальный клапан
Открытый
Закрытый
Аортальный клапан, пораженный стенозом
Открытый
Закрытый

Clinical picture

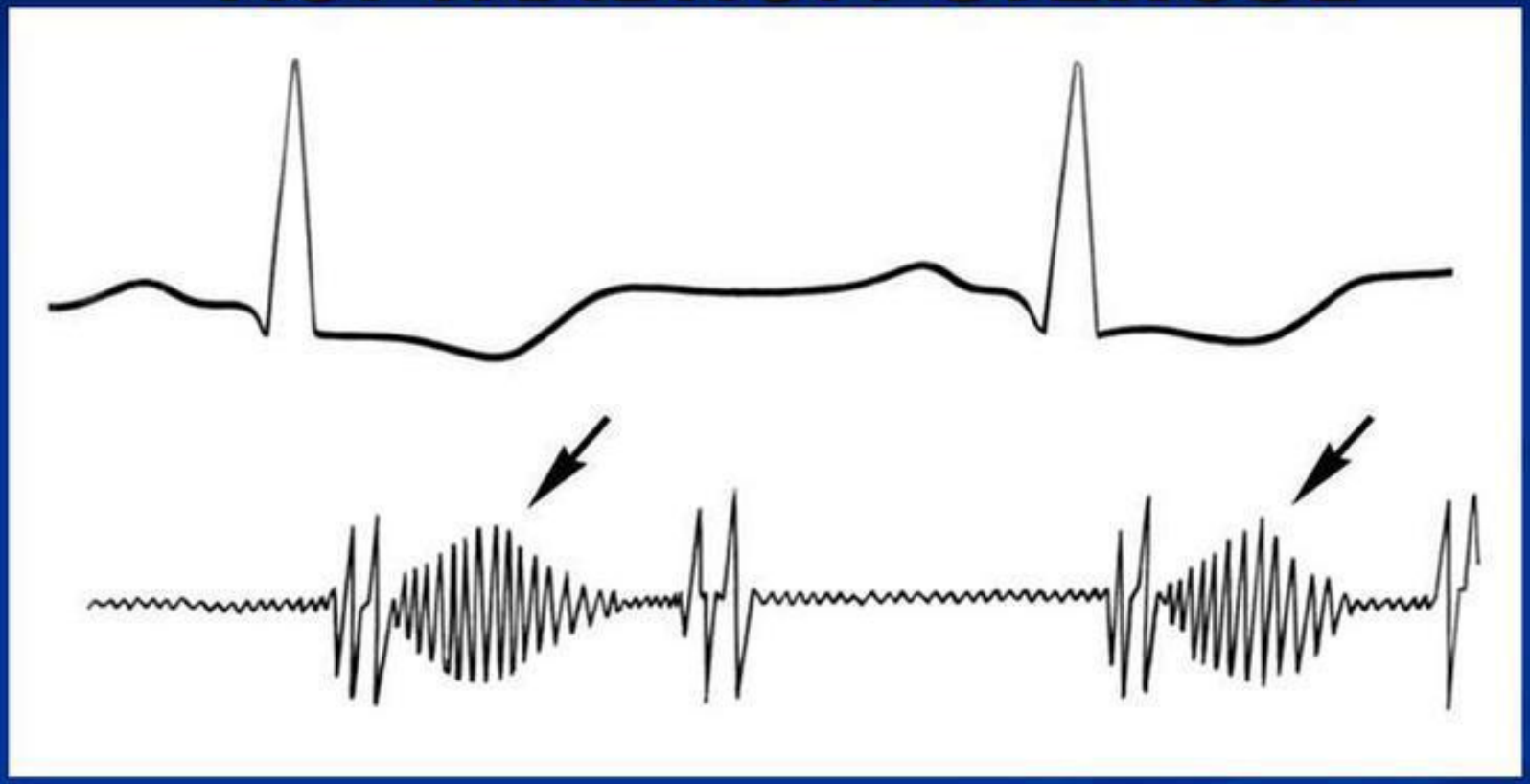
- Aortic stenosis can remain compensated for years and would not cause any unpleasant sublective sensations (even during intense physical exertion). If obstruction of the aortic orifice is considerable, infufficient blood ejection into the arterial system upsets normal blood supply to the hypertrophied myocardium and the patient feels pain in the heart (angina pectoris-type pain). Disordered blood supply to the brain is manifested by giddiness, headache, and tendency to fainting. These symptoms like pain in the heart would more likely occur during physical and emotional stress.

- The skin of the patient is pallid due to insufficient blood supply to the arterial system. The apex beat is displaced to the left, less frequently ineriorly; it is diffuse, high, and resistant. Systolic thrill (cat's purr) can be palpated in the region of the heart**. Percussion** reveals displacement of the left heart border; the heart is "aortic" due to hypertrophy of the left ventricle.

Auscultation

- **Auscultation** of the heart at its apex reveals diminished first sound due to overfilling of the left ventricle and prolongation of systole. The second sound is diminished over the aorta. If the aortic cusps adhere and are immobile, the second sound can be inaudible. Rough systolic murmur over the aorta is characteristic. This murmur is generated by the blood flow through the narrowed orifice. It is conducted by the blood onto the carotids and can sometimes be heard in the interscapular space. The pulse is small, slow, and rare, since the blood slowly passes into the aorta and its volume is decreased. Systolic arterial pressure is usually diminished, while diastolic remains normal or increases. The pulse pressure is therefore decreased.

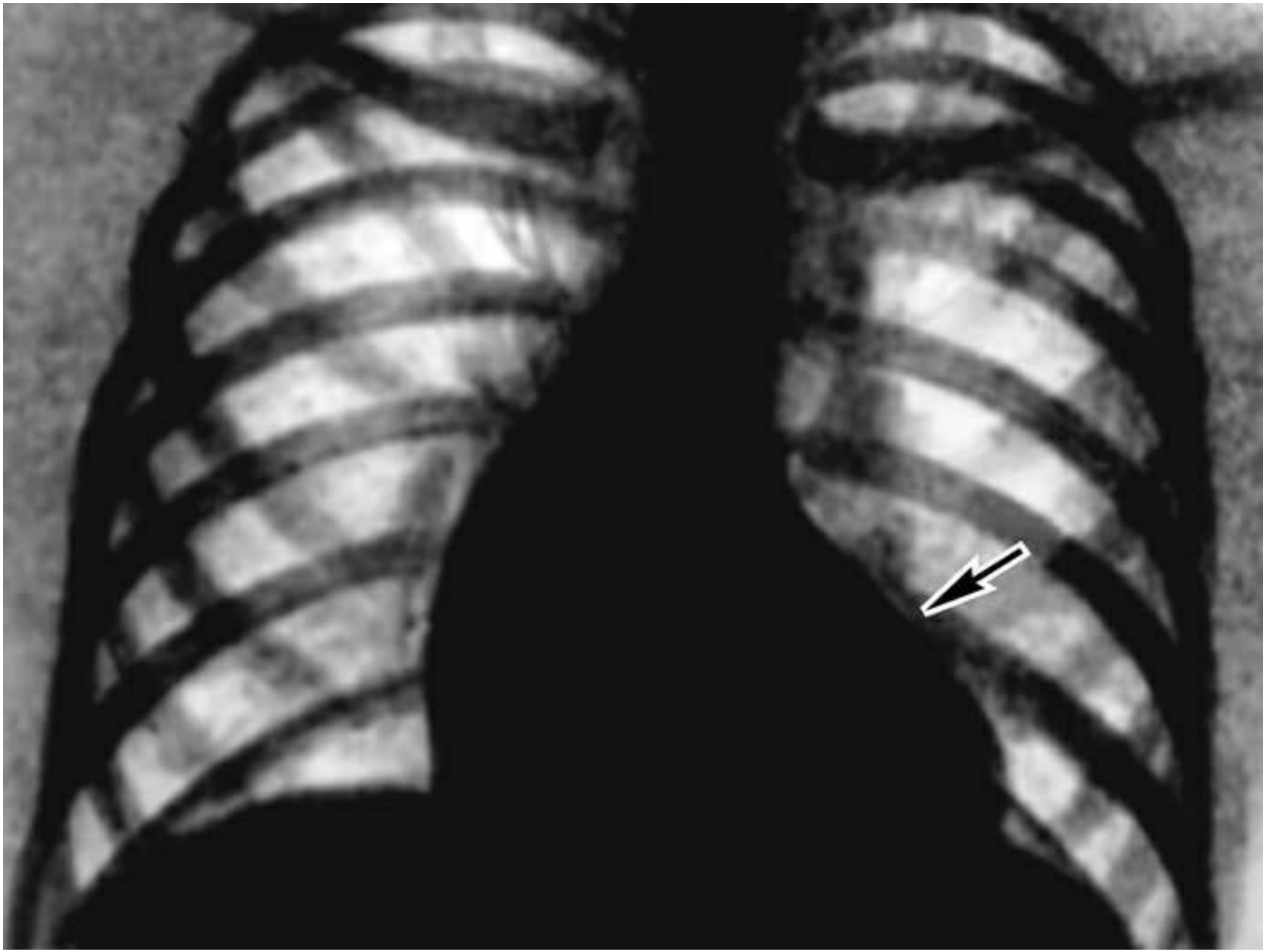
ФОНОКАРДИОГРАММА ПРИ АОРТАЛЬНОМ СТЕНОЗЕ



Фонокардиограмма при аортальном стенозе: систолический шум ромбовидной формы (указан стрелками).

примыкающий к II тону, 2 — мезодиастолический шум (регистрируется примерно в середине интервала между II и I тонами).

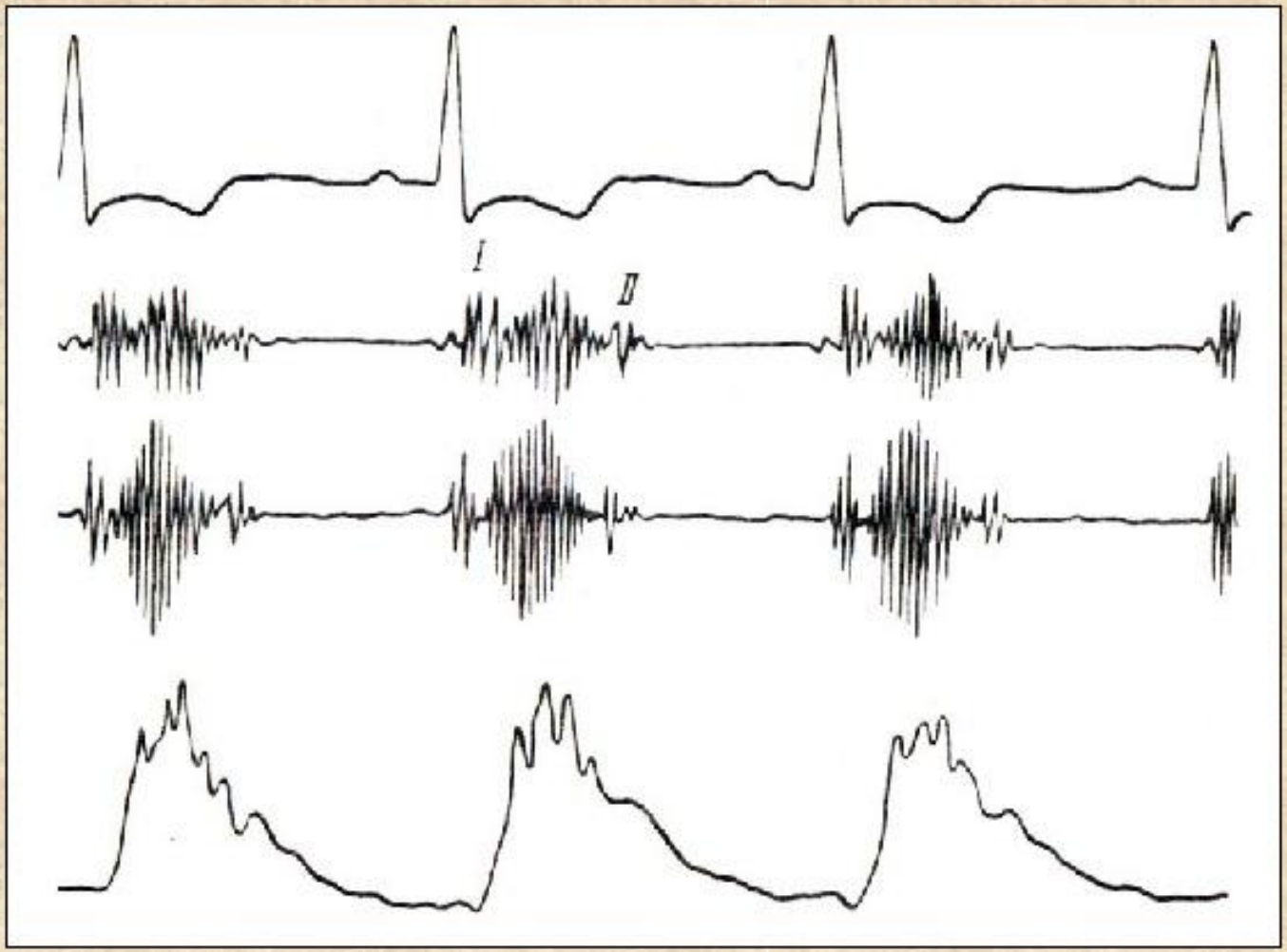
- **X-ray** examination shows hypertrophied left ventricle, "aortic" configuration of the heart, and dilatation of the ascending aorta (poststenotic); the cusps of the aortic valve are often calcified.



- **The ECG** usually shows signs of hypertrophy of the left ventricle and sometimes of coronary insufficiency.
- The phonocardiogram shows the specific changes in the heart sounds: diminished amplitudes of the first sound at the heart apex and of the second sound over the aorta. Systolic murmur over the aorta is typical; its oscillations are recorded in the form of - specific diamond-shaped figures.

- **Sphygmograms** of the carotids reveal slowed ascent and descent of the pulse wave (slow pulse), small amplitude of the pulse waves, and specific serrated pattern of their peaks (sphygmograms in the form of a cock's comb) showing oscillations associated with conduction of systolic murmur onto the neck vessels.

ИЛЛЮСТРАЦИЯ: ФКГ И СФИГМОГРАММА при АОРТАЛЬНОМ СТЕНОЗЕ



Echocardiograms

- **Echocardiograms** show decreased opening of the aortic valve during systole. Echoes from the cusps become more intense and signs of hypertrophy of the left ventricle appear.
- Aortic stenosis remains compensated for a long time. Circulatory insufficiency develops in diminished contractility of the left ventricle and it is manifested as in aortic incompetence.

Treatment

- Conservative treatment means management of heart failure. Operative treatment – implantation of artificial prostesis in incompetence or comissuritomy in stenosis.


THANK YOU !