SAINT PETERSBURG STATE PEDIATRIC MEDICAL UNIVERSITY
DEPARTMENT OF PROPAEDEUTICS OF INTERNAL DISEASES

SEMIOTICS OF ACQUIRED HEART VALVULAR DEFECTS

(tutorial for the 2-nd and 3-rd year students
of pediatric and general medical faculties)

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The tutorial contains the necessary information about acquired defects of mitral, aortic and tricuspid valves, including the features of hemodynamics and clinical presentation, as well as the data of physical examination (inspection, palpation, percussion and auscultation), and also current understanding of the results of insrumental investigations (electrocardiography, echocardiography, radiography). The tutorial is to be used by the 2-nd and 3-rd year students of pediatric and general medical faculties studying propaedeutics of internal diseases. The tutorial can also be used for training for state accreditation of GeneralMedicine Faculty graduates.

The tutorial was approved by Educational and Methodical Council of Saint Petersburg State Pediatric Medical University.

A GENERAL NOTION OF THE ACQUIRED HEART DEFECTS

The acquired heart defect is usually considered to be an irreversible valve apparatus structure disorder, newly occurring and resulting in a disturbed function, changed intracardiac hemodynamics, which causes overloading of the corresponding heart division, hypertrophied and dilatated chambers leading to blood circulation disturbances in the whole body.

Causes of the heart valve defects formation are:

- sclerodegenerative (atherosclerotic) processes in the valve apparatus (aortal stenosis in the elderly and old people)
- inherited disorders of the connective tissue (mitral valve prolapse resulting in a severe mitral insufficiency)
- inflammatory diseases in the valves endocardium: an infectious or rheumatic endocarditis
- rarer: chest injuries, syphilis etc.

Valve failure (insufficiency) is formed when the cusps\leaflets are markedly deformed, their area is decreased or the valve ring is dilated: in this case venous cusps close incompletely causing a regurgitation spurt (a reverse blood flow).

Valve stenosis is formed when the cusps fuse, or open incompletely due to above-mentioned causes.

There are two kinds of heart defects: 1) the one that combines stenosis with regurgitation in one valve, and 2) multivalvular disease when several valves are involved.

At present the aortic valve stenosis is the commonest one of the acquired heart defects. This is not unusually combined with aorta failure (a combined heart defect).

Nowadays mitral valve stenosis has becomes significantly rarer due to decreasing incidence of rheumatic endocarditis. In contrast, tricuspid valve defects incidence has lately increased. Infectious tricuspid valve endocarditis is a typical cause of death of heroin drug addicts, of patients with prolonged main veins catheterization, including those on permanent hemodialysis.

Pulmonary artery valve defects practically never occur on their own, more often they are associated with a tricuspid valve defect.

The **plan of describing acquired heart defects** is:

1. Short anatomical description of the defect

2. Etiology (causes of its development)
3. Hemodynamic changes (intra-cardiac ones, and also those in the systemic and pulmonary blood circulations)
4. Patient's complaints
5. Objective findings (those of patient's examination by inspection, auscultation, percussion, palpation)
6. Instrumental investigation methods: Echocardiography (EchoCG), electrocardiography (ECG), phonocardiography (PhCG) and X-ray examination.

All **the symptoms** of heart defects can be divided into two groups:

1. Directly valvular signs – those are data of auscultation (hearing changed heart sounds, extra sounds and characteristic murmurs appearing. EchoCG shows changed structure and function of the valve. PhCG demonstrates heart murmurs graphically.
2. Indirect signs are evidence of some changes of the heart chambers in response to overloading (myocardium hypertrophy and dilatated heart chambers, increased blood pressure in the main vessels and heart cavities. To evaluate these changes physical methods are used, such as palpation, percussion, as well as the findings of ECG, EchoCG, X-ray examination.

SYNDROME OF THE LEFT ATRIO-VENTRICULAR OPENING STENOSIS / MITRAL STENOSIS

Etiology. Rheumatic endocarditis (which results in the leaflets fusion and makes it impossible for them to open completely), degenerative changes of mitral valve leaflets and the ring leading to the leaflets being able for only little movement.

Hemodynamic changes. In mitral stenosis (narrowing of the left atrio-ventricular opening) during a diastole there is a difficulty for the blood to flow from the left atrium to the left ventricle. Normally the area of the opening is 4-6 cm^2 , however, in mitral stenosis it may decrease to 1-2 cm^2 , or even less. In this case the burden of resistance on the left atrium increases, intraatrium pressure increases, hypertrophy of it develops and Kitayev reflex works. When extra blood constantly stays in the left atrium it dilates. The pressure in the venous part of the pulmonary circulation grows (hypertension of the pulmonary circulation), the blood volume grows (hypervolemia of the pulmonary circulation). Then the pressure in the arterial part of the pulmonary circulation rises leading to the right ventricle hypertrophy and next, to its dilatation. Later congestion in the systemic circulation occurs. And occasionally, all signs of a

relative tricuspid valve failure develop. Finally, hypertrophy and dilatation of the three chambers (all except the left ventricle) may develop.

Kitayev reflex is one of the mechanisms compensating for a defect intended to preserve the normal pressure in the pulmonary circulation, which is necessary to maintain gas exchange in the lungs. When the left atrium and pulmonary veins walls are extended baroreceptors in them get irritated. This causes spasm of the pulmonary circulation arterioles by reflex, through the central nervous system. That contributes to lesser amount of blood coming to the pulmonary circulation capillaries and interferes with edema of the lungs development, but it also leads to even higher blood pressure in the pulmonary artery.

Complaints. The main complaint is breathlessness, first it starts with a moderate physical exercise, then it can begin at rest. There are also palpitations and rhythm gaps in the heart. In case of marked congestion in the pulmonary circulation bloody spitting (blood expectoration) is noted. Due to the significantly enlarged left auricle compressing the recurrent nerve, dysphonia (hoarse voice) or aphonia (low voice or whisper) may be observed in the patient. There may be seen dysphagia (disturbed swallowing process), if the esophagus is compressed.

Inspection. Cyanosis of cheeks, lips, the tip of the nose, as well as the cyanosis of fingers and toes (acrocyanosis) is noted. Facies mitralis (mitral face), which demonstrates cyanotic redness (blush) in the cheeks or ash-grey cyanosis due to a disturbed gas exchange in the lungs because of a marked pulmonary hypertension.

Palpation. Pulse is rapid, of small fulness. When the atria fibrillate the pulse gets arrhythmic (pulsus irregularis), its deficit (i.e. the difference between the heart beat frequency and that of the pulse) is noted.

When the left atrium is significantly enlarged and compresses the left subclavicular artery pulsus differens results, that is the pulse fullness on the left arm is smaller than it is on the right one. On palpation of the heart area the heart jerk appears due to the hypertrophy of the right ventricle. Sometimes diastolic thrill can be determined (the symptom of «cat's purr»), which is a palpatory equivalent to the diastolic murmur. In the epigastrium one can feel pulsation from the top downwards owing to the hypertrophy of the right ventricle.

Percussion. Extended borders of the relative dullness are observed. The upper one is extended owing to the dilated left atrium, the right one – due to the enlarged right ventricle.

Auscultation. The 1st sound is enhanced, it got the name «clapping sound». This is associated with the mitral valve leaflers hardening and rapidly increasing tension of the left ventricle lacking blood (volume). The 2nd sound is sharply accentuated on the

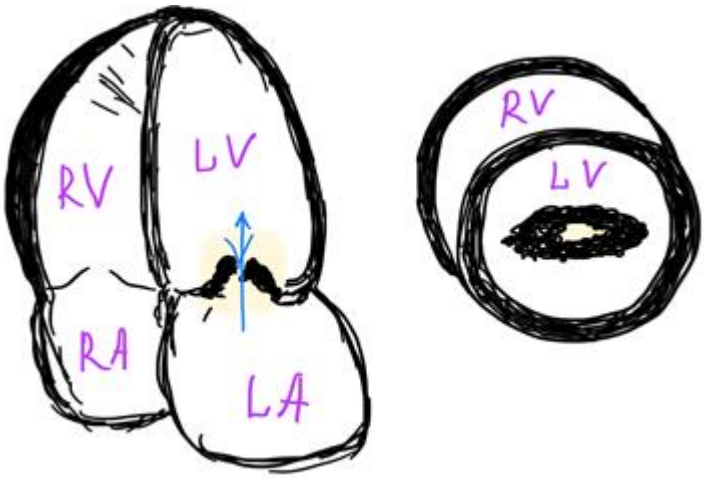
pulmonary artery. It is often split in two, or bisected (dualized) because of increased pressure in the pulmonary circulation and of non-simultaneous end of both left and right ventricles systole. The pulmonary artery valve closes later, but more intensely than the one of aorta.

A high-frequency sound phenomenon called «mitral snap», «opening snap» or the sound of mitral valve opening is heard 0.07-0.12 sec after the 2nd sound on the heart apex and in Botkin point. It appears with a sudden sharp opening of fused in the form of a funnel mitral valve leaflets at the start of a diastole. Combination of the enhanced 1st sound, the 2nd sound close to it in time and the mitral snap shows a melody characteristic of mitral stenosis, «quail's rhythm».

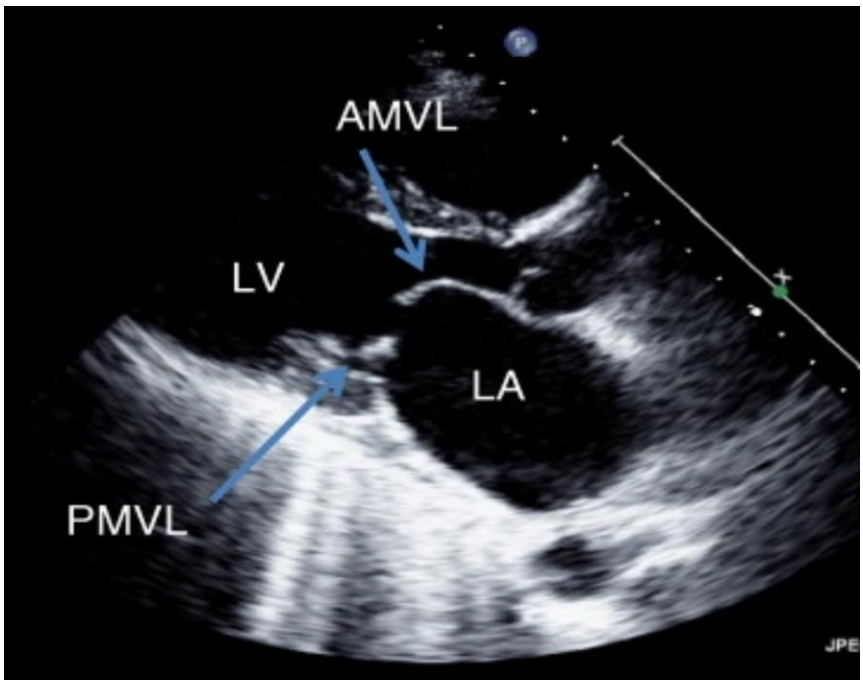
During a diastole diastolic murmur is heard at the heart apex and in Botkin point. The strength of the murmur depends on the severity of stenosis and on the degree of acceleration of the blood stream flowing through the narrowed opening of the mitral valve. The murmur is not irradiating. There can also be a presystolic murmur at the end of a diastole (which occurs due to decreased left atrium resulting in increased speed of blood flow through the narrowed mitral opening. This is absent during atria fibrillation.

Instrumental Investigation Methods

The main non-invasive method of certifying acquired heart defects is EchoCG. In mitral stenosis one can see limited mobility of the mitral valve leaflets which are often thickened, decreased area of the left atrio-ventricular opening during a diastole, enlarged size and volume of the left atrium. The Doppler test (measuring the blood flow velocity) reveals accelerated blood flow from the left auricle to the left ventricle through the mitral valve during a diastole. Later one can find an increased pressure in the pulmonary artery, the wall thickening and an increased size of the right ventricle.

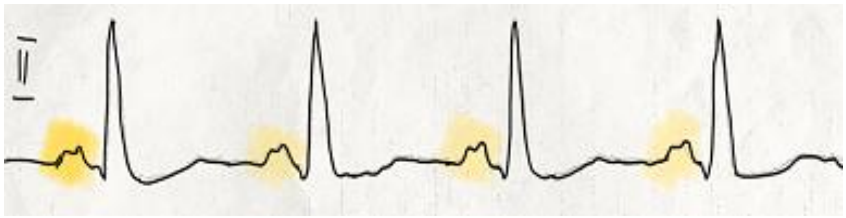


EchoCG scheme: LV- left ventricle, RV- right ventricle, LA- left atrium

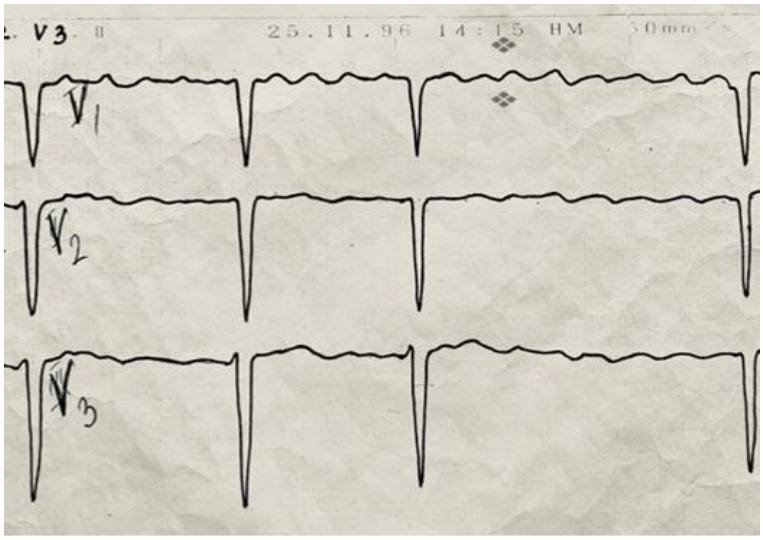


EchoCG in mitral stenosis (LV – left ventricle LA – left atrium, AMVL - anterior mitral valve leaflet, PMLV - posterior mitral valve leaflet)

ECG may record a sinus rhythm with P-mitrale (a split, or sometimes bisected P-wave in standard diversions. In the right thoracic diversions (V_1—V_2) two-phase positive-negative P-wave is observed. These are signs of hypertrophy and dilatation of the left atrium or atria fibrillation.



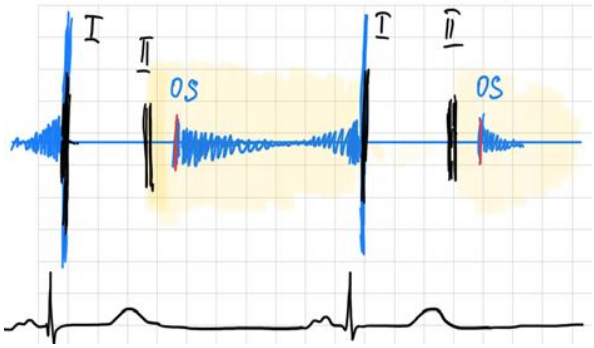
p-mitrale



Atria fibrillations

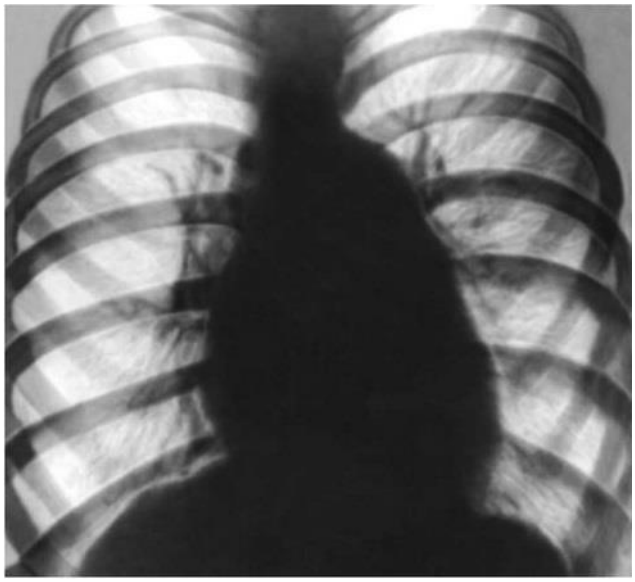
Besides that, signs of the right ventricle hypertrophy may appear (high R-wave in the diversion V1-2) with the electric cardiac axis shifting to the right ($R_{III}>R_{II}>r_I$, deep S_I).

PhonoCG records a high-amplitude 1st sound, a split in two or bisected 2nd sound, mitral snap (the sound of mitral valve opening) and diastolic murmur, diminishing and growing again (in saddle-shaped way, or as decrescendo–crescendo).



PhonoCG in mitral stenosis

X-ray shows a mitral heart shape (configuration), that is the heart shadow extended to the right and left in the intercostal spaces 3 - 4 (waist of heart flattened) due to dilated right ventricle, left atrium and dilated pulmonary artery; enhanced lung pattern due to the vascular component.



Radiograph (X-ray photograph) of the chest in mitral stenosis

SYNDROME OF MITRAL VALVE FAILURE / MITRAL REGURGITATION

Etiology of mitral failure is infectious or rheumatic endocarditis, mitral valve prolapse, ischemic dysfunction, chest traumas etc.

Hemodynamic changes are associated with an incomplete closure of mitral leaflets, those becoming deformed and shortened. Incomplete closure of mitral valve leaflers has the following results: during a left ventricle systole a part of blood returns to the left atrium (it is regurgitation). The left atrium experiences a load of additional volume because during a diastole it receives blood from the pulmonary circulation plus the regurgitated blood from the left ventricle. This creates a larger blood volume than normal in it. This results in a dilatation of the left atrium. The work of the left atrium increases too, with a larger blood volume, which, according to the law of Frank-Starling, leads to the hypertrophy of the left atrium myocardium. The left atrium also gets overloaded, a larger blood volume than usual gets into it. First it becomes dilated, then driving out more blood (the main one into the aorta and a part into the left atrium), its hypertrophy develops.

In case of hypertrophy and dilatation of the left heart chambers, if their function is sufficient, finally systolic output into the aorta does not suffer and there does not develop any essential congestion in the pulmonary circulation. Further hemodynamic changes are similar to mitral stenosis. However, the degree to which they are marked is incomparably lower.

In so called “pure” mitral valve failure the cardiac defect can be compensated for a long time. Only in an advanced case ability of the hypertrophied right ventricle to

contract starts gradually decreasing, it dilatates and congestion of the systemic circulation gradually develops. Thus, finally every heart chamber may be hypertrophied and dilatated.

In case of this defect, as well as in mitral stenosis, Kitayev reflex appearing can be noted.

Complaints of fatigue, decreased physical activity, shortness of breath, palpitation, feeling intermittent heart beat are present. In advanced cases peripheral edema may appear. Any complaints may be absent.

Inspection. There is sometimes a mild acrocyanosis, diffuse apex beat shifted to the left and right (to the breastbone) because of the hypertrophied left ventricle. One can see visible epigastral pulsation. If the right ventricular failure progresses edema appears on the legs and on the small of the back, ascites, hydrothorax.

Palpation. The pulse is not changed as the systolic outflow from the left ventricle into the aorta remains normal. In atria fibrillation the pulse is arrhythmic. In the heart area a diffuse apex beat shifted to the left due to a dilatated and hypertrophied left ventricle can be palpated. There is heart beat, epigastric pulsation from the top downwards because of the hypertrophied right ventricle.

Percussion. In most cases one can note the heart borders expended upwards and to the left. As the hemodynamic changes progress, **the right border of the heart dullness can also expand.**

Auscultation. The 1st sound is weakened at the heart apex due to:

- absent period of the mitral valve leaflets closure in the left ventricle during the systole
- a big end-diastolic volume of the left ventricle
- left ventricle hypertrophy

The 2nd sound is accentuated on the pulmonary artery, there is split in two, bisected 2nd sound on the pulmonary artery, which is associated with increased pressure in the pulmonary circulation.

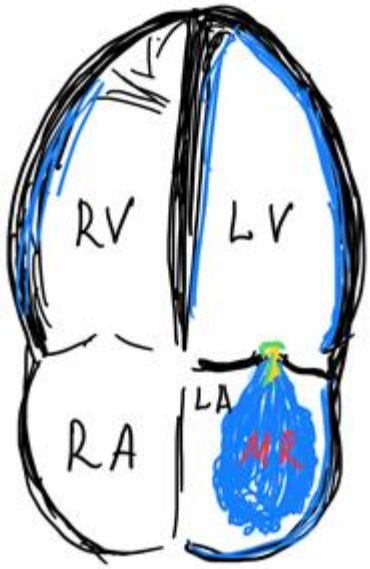
A systolic murmur can be heard in the 1st and 5th auscultation points. The murmur tends to enclose the whole of the systole. It is characterized by decrescendo, i.e. its intensity gradually decreases. The murmur is well conducted to the left axillary region. Appearance of the systolic murmur is due to the fact that during the left ventricle systole, in the phase of its isometric tension, a part of blood returns back to the left atrium through an opening formed owing to an incomplete closure of the deformed mitral valve leaflets.

X-ray examination shows mitral heart configuration, i.e. the heart waist is flattened because of the left atrium dilatation, as well as due to the dilated and hypertrophied left ventricle and sometimes also owing to forking of the pulmonary artery arch.

During the stage of the defect compensation the heart shadow gets larger upwards and to the left. As decompensation develops, the heart shadow gets larger to the right, too, at the expense of the hypertrophy and sometimes also dilatation of the right ventricle.

ECG demonstrates shift of the electric cardiac axis to the left (RiS_{III}) and signs of the left ventricle hypertrophy in this defect.

In the mitral valve failure EchoCG records an incomplete closure of the mitral valve leaflets, in prolapse there is sagging of one or both leaflets into the left atrium cavity. Application of the Doppler effect allows to reveal the regurgitating blood jet stream flowing from the left ventricle to the left atrium. Besides that, it can show enlarged left atrium size and hypertrophied left ventricle myocardium with a growing strike output of the heart.



Echocardiogram scheme Mitral Regurgitation, color Doppler

Check Up Questions to the Unit "Mitral Defects of Heart"

1. Name main etiologic factors resulting in acquired heart defects developing.
2. Describe the character of hemodynamic changes in the mitral valve failure and left atrioventricular opening stenosis.

3. Explain the essence of Kitayev reflex, positive and negative aspects of this reflex in mitral defects.

4. Explain the symptoms developing and revealed by inspection, palpation, percussion, auscultation in patients with mitral valve failure during the stages of compensation and decompensation.

5. Explain the mechanism of symptoms developed and revealed by inspection, palpation, percussion, auscultation jn the patients with left atrioventricular opening stenosis during the stages of compensation and decompensation.

6. Describe the mechanism of diastolic murmur appearance, its varieties prognosis in mitral stenosis (protodiastolic and presystolic ones).

7.Explain the mechanism of the emergence of the mitral snap.

TRICUSPID VALVE FAILURE SYNDROME / TRICUSPID REGURGITATION

Its etiology consists in dilatated right atrioventricular opening caused by a significantly enlarged size of the right ventricle (myocardium impairment with combined mitral defects at the background in case of the pulmonary heart), or in infectious bacterial, or rheumatic endocarditis. The group at risk of infectious endocarditis development includes heroin drug addicts, patients on prolonged catheterization of the major veins.

Hemodynamic changes. During the right ventricular systole blood does not only get into the pulmonary artery, but also returns back into the right atrium. This leads to its dilatation and hypertrophy. During the next diastole a larger amount of blood than normal gets into the right ventricle out of the right atrium. This results in its dilatation and hypertrophy. Thus, in this kind of defect both right heart chambers get enlarged. Congestion and hypertension develop in the venous part of the systemic circulation. Out of the right atrium blood regurgitates (due to the weak vein valve apparatus) into the upper and lower vena cava interfering with their emptying. Further on, it gets into the jugular and hepatic veins, which can be well seen on examination.

Complaints of patients include shortness of breath, pains in the right subcostal area (congestion of blood in the liver), intermissions, edema of lower extremities and in the small in the back, enlargement of abdomen (ascitis).

During inspec**tion** one can see cyanosis and puffiness of the face, acrocyanosis, jugular venous distention and positive venous pulse, i.e. filling of veins on the neck coincides with carotid pulse. Sometimes the liver pulsation is visible.

Palpation. During palpation of the pulse tachycardia can be noted. Other pulse indicators do not change in case of tricuspid failure.

Palpation of the heart area reveals the appearance of cardiac beat which can be identified along the right breastbone border due to significant enlargement of the right ventricle. Immediate appearance of two variants of epigastric pulsation - from top to bottom (due to hypertrophy of the right ventricle) and from the right to the left (due to positive hepatic pulse) is also characteristic. To identify the true hepatic pulsation the protruding liver edge should be clasped by fingers of one hand (they will diverge in case of systole). The symptom of "swings" when the doctor's palms identify asynchronous pulsation and even minor swinging movements if the doctor simultaneously places them above the cardiac beat and pulsating liver. One more sign of this defect is the symptom of Plesch – increased swelling of jugular veins when pressing the liver from below upwards.

Percussion. Only the right border (relative and absolute) of the heart is extended to the right due to hypertrophy and dilalatation of the right ventricle. The left border of the heart can be rarely extended due to some displacement of the left ventricle to the back side by the enlarged right ventricle.

Auscultation. Weakened I^{st} sound is identified at the points of auscultation of the tricuspid valve (4^{th} point - the base of xiphoid process or, in case of significant enlargement of the right ventricle in the 4^{th} intercostal space along the right edge of the breastbone) due to insufficiency of the tricuspid valve, hypertrophy of the right ventricle and because it is much filled during a diastole. There is also heard a systolic murmur as a result of retrograde (back) blood flow from the right ventricle to the right auricle during a systole, and swinging of the valve leaflets and the tissues surrounding heart. Systolic murmur has a decreasing character (decrescendo) and intensifies at breath-holding during an inhalation.

The liver enlargement and tenderness are identified during palpation and percussion.

Deviations of electric axis of the heart to the right and the signs of enlargement (hypertrophy) of the right ventricle ($r_I S_I R_{III}$) are identified by ECG.

X-ray examination identifies the enlargement of the right parts of the heart.

EchoCG allows evaluating the level of tricuspid failure and intensity of the right ventricle changes. An important sign of echocardiography investigation is absence of collapse of vena cava inferior during an inhalation. Doppler method allows to demonstrate blood regurgitation from the right ventricle to the right atrium.

Check Up Questions to the Unit "Tricuspid Valve Failure"

1. What hemodynamic changes occur in case of tricuspid valve failure?
2. What are the basic symptoms characteristic of tricuspid valve failure that can be found out by physical examination methods?
3. What are the data of instrumental investigation methods?

SYNDROME OF AORTIC STENOSIS

At present aortic stenosis is the most widespread defect among the acquired ones. By the frequency of surgical interventions it ranks the third after intra-coronary stents and bypass surgery. The leading mechanism of aortic stenosis is atherosclerotic degenerative process on cusps and rings of aortic valve. Among the causes of such calcification process is congenital heart disease - bicuspid aortic valve. Arterial hypertension increases the risk of the development of this defect. Besides, the causes of aortic stenosis can be rheumatic or infectious endocarditis (rarer) with calcification of valve vegetations.

Hemodynamic changes. In the presence of some barrier to the emptying of the left ventricle compensatory mechanisms are engaged: duration of the cardiac cycle increases (systole and diastole of the left ventricle are prolonged), pressure in its cavity is increased, the level of which is proportional to the level of narrowing of aortic opening. Hypertrophy of the left ventricle is developing, but without any significant enlargement of its cavities. For a long time this heart disease takes its course without any blood circulation disorders (years, decades). Weakened contractile ability of the left ventricle leads to its dilatation. Later the left atrium can get enlarged. The main hemodynamic feature of this defect is reduction of systolic blood output to aorta. Due to this, systolic pressure decreases and diastolic arterial pressure compensatory increases (pulse pressure decreases).

Complaints. Patients do not complain during the stage of compensation. The appearance of complaints is connected with deterioration of blood supply of the organs of systemic circulation, first of all, of the heart and brain. Patients complain of dizziness, headache, syncope, and of anginous pains even in case of low-intensity physical activity. Besides, patients can feel palpitation - strong beats of hypertrophied left ventricle against the front chest wall. Shortness of breath appears later. Genesis of anginous pains in case of aortic stenosis includes:

1. Reduction of blood output to the aorta, reduction of blood filling of coronary arteries
2. Compression of coronary arteries by hypertrophied myocardium of the left ventricle
3. Expansion of sclerous process from the cusps of aortic valve to ascending aorta including the opening of coronary arteries that leads to their narrowing and development of coronary failure.

During **inspection** one can notice a significant skin paleness caused by skin angiospasm associated with the reduction of cardiac output. During **pulse palpation** the pulse is low, slow and rare (pulsus parvus, tardus et rarus), systolic and pulse pressure are decreased. Bradycardia is one of the compensation factors. Compensation character of bradycardia means that diastole prolongation induces improvement of blood filling of coronary arteries.

Palpation of heart area: the apex beat is intensified and can be displaced to the left, which is caused by the development of significant hypertrophy of the left ventricle. Systolic thrill is palpated in the 2nd intercostal space to the right of the breastbone and it is connected with blood turbulence during its passage through the narrowed aorta opening. This gives evidence to the presence of low-frequency systolic murmur. Besides, patients with aortic stenosis often develop enlargement of the arch of aorta, so-called poststenotic enlargement of aorta associated with distention of the arch of aorta by intense blood stream released from the left ventricle. It can lead to retrosternal pulsation.

During **percussion** the left border of cardiac dullness can be displaced to the left. Sometimes the width of vascular bundle is increased due to poststenotic dilatation of ascending aorta.

During **auscultation** there is identified weakened the Ist sound at the heart apex due to left ventricle hypertrophy and weakened 2nd sound on aorta due to decrease of arterial pressure in this vessel.

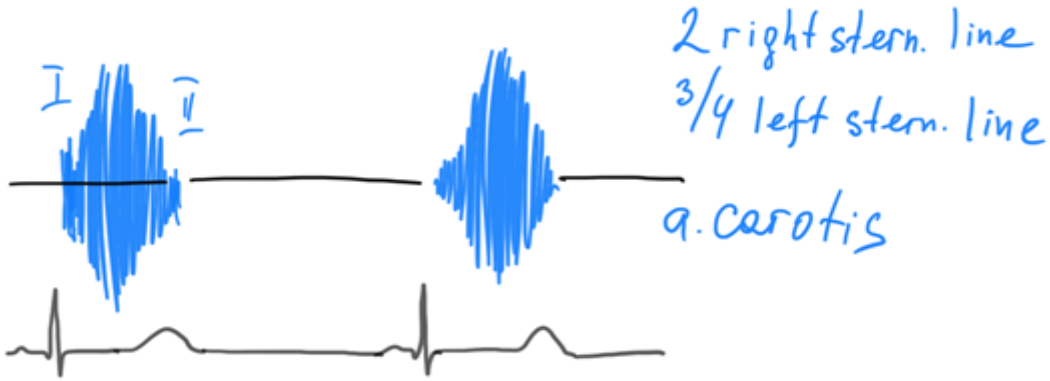
A characteristic feature of aorta stenosis is the presence of ejection harsh systolic murmur with its maximal intensity in the 2nd point of auscultation. Quite often the murmur is heard over the whole surface of the heart, however, its maximum is identified in the 2nd intercostal space along the right border of the breastbone. This murmur is well conducted along the vessels and bones: it can be heard in the right supraclavicular and subclavicular fossa, the carotid arteries, interscapular space and even in the field of the right bend of elbow. It is important to notice that the murmur intensity is associated not with the size of narrowing of aorta opening, but with the

energy of the left ventricle. Thus, the murmur can become weaker during decompensation of aortic stenosis and development of dilatation of the left ventricle.

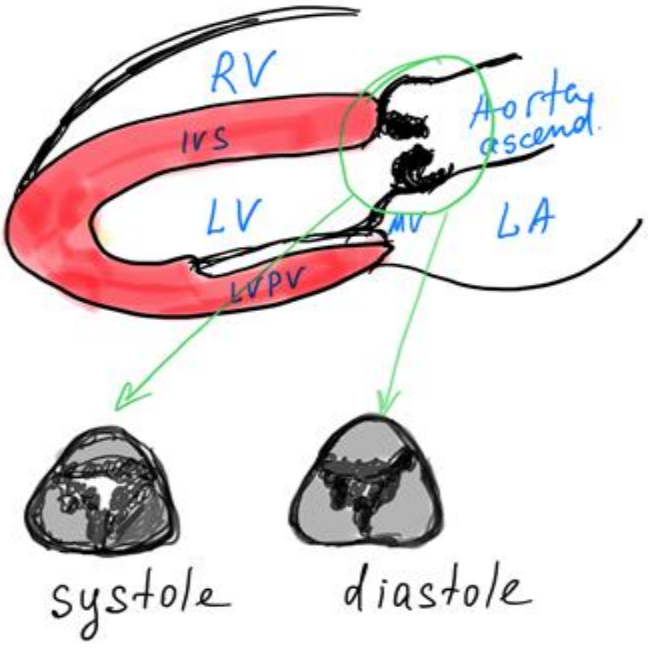
The chest X-ray usually reveals a relatively normal heart contour for many years; the heart is increased in its diameter when heart failure develops. There is also ascending aorta dilation and aortic valve calcification.

ECG shows: deviation of electric heart axis to the left, left ventricle hypertrophy, left atrium enlargement and later - atrial fibrillation or flatter.

Phonocardiogram identifies low sound 1, diamond-shaped systolic murmur in the second intercostals space to the right from the breastbone. Aortic component of the 2nd sound is considerably weakened.



Aortic regurgitation phonocardiogram scheme



Echocardiogram scheme. Aortic stenosis.

Echocardiogram identifies thickened, calcified and immobile aortic cusps, significant left ventricle hypertrophy. An important echocardiographic marker of the severe aortic stenosis is the gradient (difference) of pressure between the left ventricle and aorta.

SYNDROME OF AORTIC VALVE FAILURE (AORTIC REGURGITATION)

Among the causes of isolated aortic failure one should name bicuspid aortic valve, sclero-degenerative impairment, infectious endocarditis, syphilitic involvement of aorta with its extension, Marfan's syndrome, and Ehlers-Danlos syndrome.

Hemodynamic changes. The basic hemodynamic changes are caused by the reverse blood flow (regurgitation) from the aorta to the left ventricle during the diastole. From 5 to 50 % of systolic blood volume can be returned to the left ventricle. The reverse blood flow to the left ventricle contributes to the pressure increase in it by the time of systole beginning, it causes compensatory dilatation of the left ventricle, the degree of which is in proportion to the volume regurgitated. Left ventricle end-diastolic volume rises. Systolic release from the left ventricle is increased by the volume of blood which returns to the left ventricle during the diastole. The left ventricle is hypertrophied. Thus, the basic hemodynamic point of insufficiency of aortic valves is the pressure difference: arterial pressure increases during a systole and decreases during a diastole. Tachycardia appears in a compensatory way that contributes to the shortening of the diastole and decrease of regurgitation of blood volume. In progress there can be observed a considerable enlargement of the left ventricle, sometimes associated with the development of relative insufficiency of mitral valve that leads to hemodynamic overload and dilatation of the left atrium.

Symptoms. Patients do not complain for a long time during the stage of the defect compensation. Some patients complain of palpitation aggravating with physical activity. In case of significant defect of valves when arterial pressure decreases considerably during the diastole angina pectoris is a frequent complaint; dizziness, tendency to faint that is caused by impairment of coronary and brain blood supply.

With decrease of left ventricle contractile function patients begin to complain of breathlessness at first during physical exhertion, and later at rest.

During **inspection** one can reveal aortic paleness of the skin caused by quick blood outflow from arterioles and low filling of arterial vessels of the skin during the period of diastole. Examining the neck one can observe carotid shudder - an intensive

pulsation of carotid artery, pulsation of pupils and head shaking simultaneous with the heart systole (de Musset's sign). If you slightly press the patient's finger nail bed to make a white spot, or if you make a hyperemic spot on the patient's forehead by rubbing it with your stethoscope, it is possible to observe their pulsation. This is a pseudo-capillary Quincke's pulse when not capillaries, but arterioles are pulsating. All these symptoms can be explained by a big difference in systolic and diastolic pressure and, therefore, by high pulse pressure. An intensive apex beat caused by the left ventricle hypertrophy is seen when inspecting the heart region.

Palpation can demonstrate pulsus celer, altus et frequens (fast, high, and frequent pulse) caused by difference of systolic and diastolic arterial pressure, the palpation of heart region identifies an intensive raising apex beat displaced downwards (the 6-7th intercostal space) and to the left, that is, the main clinical sign of dilatation and hypertrophy of the left ventricle. In case of very severe regurgitation diastolic thrill can be identified simultaneously giving evidence of the presence of low-frequency diastolic murmur.

P**ercussion** reveals enlargement of the left heart border due to dilatation and hypertrophy of the left ventricle. Sometimes there is shifting of relative and absolute dullness of the heart upwards in case of the development of relative failure of the mitral valve that leads to dilatation and hypertrophy of the left atrium.

Auscultation finds out the I st sound is weakened at the heart apex as a result of big diastolic filling and hypertrophy of the left ventricle. The 2nd sound in the 2nd intercostal space to the right from the breastbone is also weakened due to incomplete connection of aortic cusps. An organic murmur is heard during the whole diastole period with the maximum of its force on aorta. It is well conducted to the heart apex and to Botkin's point. The character of murmur is decrescendo (decreasing). In case of expressed deformation and consolidation of the semilunar aorta valves due to aorta valves insufficiency a functional systolic murmur can be heard together with diastolic murmur. It appears due to turbulent blood movement during its passage in the period of systole in the field of changed valves. With the weakened first and second sounds in the background, and presence of systolic and diastolic murmur, there is a peculiar melody that was called "sawyer's murmur".

Sometimes a functional presystolic murmur (Flint's murmur) can be heard on the heart apex, it appears because a return blood flow from aorta to the left ventricle during a diastole pushes aside the anterior leaflet of the mitral valve. This contributes to relative narrowing of mitral opening. In case of expressed dilatation of the left ventricle

there can be heard a systolic murmur at the apex caused by relative failure of the mitral valve.

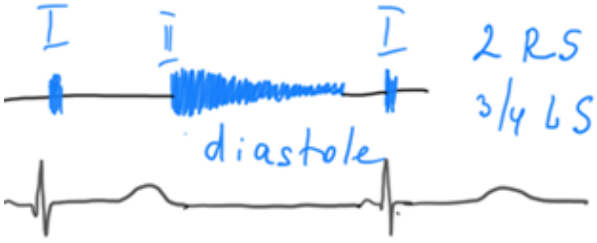
During auscultation of femoral artery below Poupart's ligament there can be heard a vessel sound - one clapping sound or even two sounds (Traube's double sound) which is not very frequent. It is usually observed as a result of big difference in the arterial pressure, especially when diastolic pressure decreases.

If you forcefully press the femoral artery with a stethoscope you can hear 2 murmurs (during a systole and a diastole) - Vinogradov-Durozier's double murmur. In a healthy person in case of artery compression there can be heard only one murmur during a systole. Vinogradov-Durozier's double murmur can be explained by turbulent blood flow in the area of compressed femoral artery that swings its wall and causes the sound, besides, blood flows from the heart to periphery during a systole, and during a diastole a part of blood moves to the area of narrowing, from periphery to the heart, due to retrograde blood flow in case of aorta valves failure.

Thus, along with other physical findings, in case of aorta valve failure it is possible to identify a number of so-called *vascular symptoms* caused by high pulse pressure:

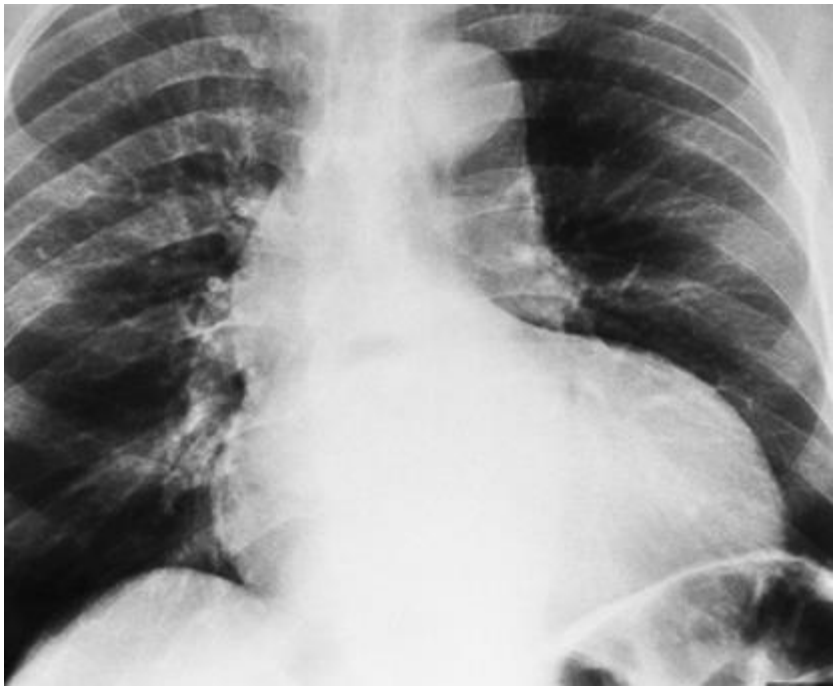
1. Carotid shudder
2. Musset's sign
3. Pulsation of pupils, uvula, tonsils and soft palate
4. Pulsus celer, altus et frequens
5. Sounds of arteries - Traube's double sound
6. Vinogradov-Durozier's double murmur
7. Quincke's sign

The ECG identifies a deviation of electric heart axis to the left and signs of hypertrophy of the left ventricle. Phonocardiogram illustrates low sound 1, low sound 2 in aorta point and diastolic descending murmur in auscultation points 2 and 5.



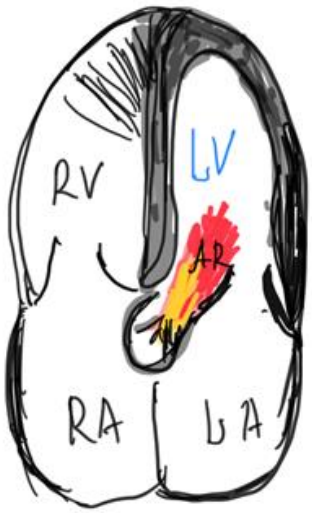
Phonocardiogram scheme. Aortic stenosis

Chest X-ray can identify the *aortic heart configuration* when the heart waist is seen well due to enlargement of the left ventricle.

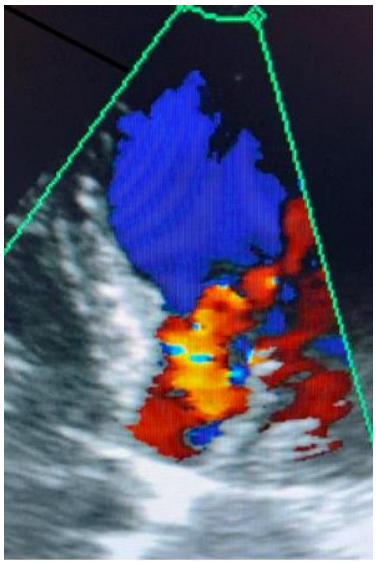


Chest X-ray, aortic heart configuration (LV enlargement) and ascending aorta dilatation / from Valvular Heart Disease, Radiology Key

Echocardiogram demonstrates left ventricle enlargement and its vigorous contraction. An important symptom of severe aortic regurgitation is diastolic fluttering of anterior mitral valve leaflet. But the main sign is detection by Doppler of aortic regurgitation.



Aortic stenosis. Echocardiogram scheme.



EchoCG, color Doppler

Aorta valves failre remains to be a compensated defect for a long time; insufficiency of blood circulation does not appear because the load is directed to the strongest part of the heart – the left ventricle. The signs of cardiac failure (the left ventricle one) can develop in acute form and later quickly progress. A significant aortic failure (with acute decrease of diastolic pressure) has a less favorable course because a considerable hemodynamic overload of the left ventricle leads to the decrease of its contraction function.

Check Up Questions to the Unit "Aortic Heart Defects"

1. Hemodynamics disorders in case of insufficiency of aorta valves.
2. Vascular symptoms in case of aorta valves failure.
3. Auscultation symptoms in case of aorta valves failure.
4. Findings identified by instrumental investigations in case of aorta valves failure.
5. Compensation factors in case of aortic stenosis.
6. Symptoms of aortic stenosis.
7. Findings identified by instrumental investigations in case of aortic stenosis.
8. Systolic and diastolic murmurs can be heard on the aorta in case of aorta valves failure and combined aorta defect. Characterize the symptoms that make it possible to differentiate these defects.

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