Acquired heart defects: clinical picture, diagnosis

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Abstract. In acquired heart defects, they are defined as the occurrence of an irreversible violation in its structure, that leads to a complete loss of its function, changes in intracardiac hemodynamics, as well as change in intracardiac hemodynamics, as a result of which there is an overload of the corresponding parts of the heart, hypertrophy and dilatation of the chambers and circulatory disorders throughout the body. The main causes of valvular heart disease formation: the hereditary connective tissue disorder (mitral valve prolapse, leading to severe mitral insufficience) and other connection tissue disorders (mitral valve prolapse leads to severe mitral insufficiency) • infectious or rheumatic endocarditis.

1 Introduction

Medical manifestations of acquired heart defects (APD) are consistent with the presence and development of permanent organic lesions, accompanied by disfunctioning valve systems in intracardiac hemodynamics. Clinical manifestations of acquired heart defects (APD) consist in the presence and development of persistent organic lesions, together with disfunctioning valves and intracardiac hemodynamics. During the development of Acquired Heart Defects (APD) a disease manifested by permanent organic lesions in the valvular apparatus, together with organogenic lossions from itsvalvular system. As well as intracardiac dysfunction is caused by an unstable function of the affected valves and intracardiac pressure. More than 2–4 times more likely to be affected by PPS is the general population. The general population is approximately 2–4 times more likely to be affected by PPS. The general population is approximately 2–4 times more likely to be affected by PPS. According to various authors, PPS are found on average from 1–2 to 4–5% in the general population. E.E V.E.V e v. E.V, according to the information of E-V, said: "They said that." This is the Shlyakhto et al. The Shlyakhto, - The Shl - they are Shl. The prevalence of PPS in the population is 5–10 per 1000 population, the prevalence of PPS in the population is 5–10 cases per 1000 population, the prevalence of PPS in the population is 5–10 cases per 1000 population. Frequency of these events is generally greater with the development of life. The frequency of these events is generally greater with the development of age. It significantly increases with the age of birth. At the same time, as a rule, it significantly increases with age [1]. On an average, the number of patients with

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aortic heart disease in the elderly and senile population is about 10.7%, with an increase of 25–48% for lesional valves. Although the prevalence of this disease is more than 10% [2], it also occurs hypersclerosis of the aortic valve (AV) - up to 25.5%. For hypersclerosis of the aortic valve (AV) - up to 25.5%. Lesions of aortic and mitral valves are most frequently detected in clinical practice. Aortic stenosis or Mitral Insufficiency is one of the most common defects in clinical practice. It is considered to be one of the most common defects in clinical practice. According to the Euro Heart Survey on VHD, AV lesion was detected in 44.3% of patients with valvular heart disease (33.9% - aortic stenosis, 10.4% - AK insufficiency), mitral valve loss (in 34.3% of people (1.3% -mitral stenosis, 9.5%) - in 34.3% of patients (2.2% - mitral valve insufficiency), mitral valve disruption - in 34.3% of patients (2.2% - mitral valve insufficiency), mitral valve injury - Aortic stenosis in 81.9% and AK insufficiency in 50.3% of patients were degenerative. For the majority of cases, mitral stenosis was rheumatic, and mitral valve insufficiency was degenerative in 61.2% of cases. The main method of examination is still used as echocardiography (EchoCG) and the development of qualitative criteria for assessing severity invalvular disease, cardiac auscultation. As well as EchoCG and the development of qualitative criteria for assessing severity invalvular disease, cardiac auscultation as the main method of examination is still used.

In acquired heart defects, they are defined as the occurrence of an irreversible violation in its structure, that leads to a complete loss of its function, changes in intracardiac hemodynamics, as well as change in intracardiac hemodynamics, as a result of which there is an overload of the corresponding parts of the heart, hypertrophy and dilatation of the chambers and circulatory disorders throughout the body. Causes of the formation and development of valvular heart disease:

• The sclerodegenerative (atherosclerotic) processes in the valvular apparatus (aortic stenosis in the elderly and senile age) are caused by: • sclerodegenerative (atherosclerotic) processes in the valvular apparatus (aortic stenosis in the elderly and sen)
  - I have hereditary connective tissue disorders (mitral valve prolapse, that leads to mitral insufficiency) and the symptom of hereditary connective organ disorders (mitrale valve prolapse in response to severe mitral insu)
  - The valve endocardium inflammation: infectious or rheumatic endocarditis, the disease of the valve endocardium.
  - Less frequent: chest trauma, surgical injury (or syphilis) and other conditions. Valve inefficiency occurs due to the development of deformation of the valves, a decrease in their area or expanding the valve-ring: incomplete opening of the valves is required. The formation of an incomplete shutdown happens, causing the formation of a jet of regurgitation (reverse blood flow). In the case of Valve stenosis, cusps are fused and incompletely opened due to the above reasons, valve stenosis is formed. There are also combined and mixed heart defects: Combine- with the simultaneous presence of stenosis and insufficiency of one valve; combined- with the simultaneous presence of stenosis and insufficiency of several valves; combined- with simultaneous damage to several valves.

Today, the most common acquired defect is aortic valve stenosis, which can be combined with aortic heart disease (combined aortic heart disease). [4]. Mitral valve stenosis is now much less common due to a decrease in the incidence of rheumatic endocarditis, as well as an increase in the incidence of rheumatic endocarditis. For the last time, it has increased frequency of tricuspid valve defects: an infectious endocarditis that occurs at the tricuspid valve is one of the most common reason for death of drug addicted people, patients with prolonged catheterization of the main veins, and even who received hemodialysis. During the treatment of pulmonary valve defects, there is no independent significance. Mostly we are discussing a combined lesion of tricuspid and respiratoryvalve systems: more often we talk about a combined lesion of the tricuspid and pulmonary valves.
2 Research Methodology

The main non-invasive method for verifying acquired heart defects is echocardiography. With mitral stenosis, the limitation of mobility of the often thickened mitral valve cusps, a decrease in the area of the left atrioventricular orifice in diastole, and an increase in the size and volume of the left atrium are visualized. Doppler studies (measurement of blood flow velocities) reveal an acceleration of blood flow through the mitral valve in diastole from the left atrium to the left ventricle. Later, it is possible to determine increased pressure in the pulmonary artery, identify wall thickening and increase in the size of the right ventricle [5].

On the ECG, a sinus rhythm with P-mitrale can be recorded (splitting, and sometimes even bifurcation of the P wave in standard leads, in the right chest leads (V1-V2) there is a biphasic positive-negative P wave - signs of hypertrophy and dilatation of the left atrium (Fig. 1) or atrial fibrillation (Fig. 2).

![Fig. 1 P-mitrale](image-url)
3 Results and Discussions

When excess blood remains permanently in the left atrium, it dilates. Increased pressure in the venous part of the pulmonary circulation (hypertension of the pulmonary circulation) and increased blood volume (hypervolemia of the pulmonary circulation). Then the pressure in the arterial part of the pulmonary circulation rises, which leads to hypertrophy of the right ventricle, and then its dilatation [6]. In the future, stagnation develops in the systemic circulation, and sometimes all signs of relative insufficiency of the tricuspid valve. As a result, hypertrophy and dilatation of the three chambers of the heart can occur - all except the left ventricle. The Kitaev reflex is one of the defect compensation mechanisms aimed at maintaining normal pressure in the pulmonary circulation, which is necessary to maintain gas exchange in the lungs. When the walls of the left atrium and pulmonary veins are stretched, the baroreceptors embedded in them are irritated [7].

Complaints. The leading complaint is shortness of breath, first with moderate physical exertion, then at rest, palpitations and interruptions in the region of the heart. With severe stagnation in the pulmonary circulation, hemoptysis is noted. Due to a significant increase in the left atrium and compression of the recurrent nerve by it, dysphonia (hoarseness of voice) or aphonia (quiet whispering of the patient), as well as dysphagia (impaired swallowing) can be observed during compression of the esophagus. Inspection. Cyanosis of the cheeks, lips, tip of the nose, cyanosis of the fingers, toes (acrocyanosis). Facies mitralis (mitral face) - a cyanotic flush of the cheeks, or ashen (gray) cyanosis due to a violation of gas exchange in the lungs due to severe pulmonary hypertension. Palpation. The pulse is frequent, small filling. When joining atrial fibrillation, an arrhythmic pulse (pulsus irregularis), its deficiency (difference in heart rate and pulse rate) is determined. With a significant increase in the left atrium and compression of the left subclavian artery, pulsus differens occurs, i.e., the filling of the pulse on the left hand is less than on the right. On palpation of the heart region due to hypertrophy of the right ventricle, a cardiac impulse appears. Sometimes diastolic trembling is determined (a symptom of “cat’s purr”) - the palpatory equivalent of diastolic murmur. In the epigastric region - pulsation from top to bottom due to hypertrophy of the right ventricle.
Percussion. There is an increase in the boundaries of relative cardiac dullness of the upper (due to the expansion of the left atrium) and right (enlargement of the right ventricle). Auscultation. I tone is strengthened and received the name of a clapping tone. This is due to the compaction of the mitral valve leaflets and the rapid tension of the left ventricle, which is insufficiently filled with blood [8]. II tone on the pulmonary artery is sharply accentuated, often split or bifurcated due to increased pressure in the pulmonary circulation and the non-simultaneous end of the systole of the left and right ventricles; the pulmonary valve closes later, but more intensively than in the aorta. After the second tone, after 0.07-0.12 s, a high-frequency sound phenomenon is recorded at the apex of the heart and at the Botkin point, which is called the mitral click or mitral valve opening tone. It occurs with a sudden sharp opening of the cusps of the mitral valve fused in the form of a funnel at the beginning of diastole. The combination of an amplified first tone with a second tone close in time and a mitral click gives a characteristic melody of mitral stenosis - the rhythm of a quail. During diastole, a diastolic murmur is heard at the apex of the heart and at the Botkin point. The strength of the noise depends on the severity of the stenosis and the degree of acceleration of blood flow through the narrowed opening of the mitral valve. Noise does not radiate. There is also a presystolic murmur at the end of diastole (due to contraction of the left atrium, leading to an increase in blood flow through the narrowed mitral orifice) which will, accordingly, be absent in atrial fibrillation.

Etiology - expansion of the right atrioventricular orifice due to a significant increase in the size of the right ventricle (myocardial damage, against the background of combined mitral defects, with cor pulmonale) or due to bacterial or rheumatic endocarditis. Heroin addicts and patients with long-term catheterization of the main veins are at risk for developing infective endocarditis of the tricuspid valve [9]. hemodynamic changes Thus, with this defect, both right chambers of the heart increase and stagnation and hypertension develop in the venous part of the systemic circulation. From the right atrium, blood regurgitation (due to the weakness of the valvular apparatus of the veins) into the superior and inferior vena cava, preventing their emptying, and then into the jugular and hepatic veins, which is clearly visible on examination. Complaints of patients on shortness of breath, pain in the right hypochondrium (stagnation of blood in the liver), interruptions, swelling of the lower extremities and in the lumbar region, an increase in the abdomen (ascites) (Fig. 3).

Fig. 3 Tricuspid valve insufficiency syndrome
On examination - cyanosis and puffiness of the face, acrocyanosis, swelling of the neck veins and the presence of a positive venous pulse, i.e. the filling of the veins in the neck coincides with the pulse of the carotid artery. Sometimes the pulsation of the liver is visible. Palpation. Palpation of the pulse may cause tachycardia. The rest of the quality of the pulse in case of insufficiency of the tricuspid valve does not change [10]. Palpation of the heart region determines the appearance of a cardiac impulse, which can also be determined along the right edge of the sternum due to a pronounced increase in the right ventricle. The appearance of two variants of epigastric pulsation at once is also characteristic - from top to bottom (due to right ventricular hypertrophy) and from right to left (due to a positive hepatic pulse). To determine the true pulsation of the liver, the protruding edge of it must be clasped with the fingers of one hand, which will diverge during systole. The symptom of “swing” is determined when the doctor’s palms, simultaneously located above the heart beat and the pulsating liver, determine asynchronous pulsation and even make small rocking movements. Another sign of this defect is Plesh’s symptom - increased swelling of the jugular veins with pressure on the liver from the bottom up [8]. Percussion. Only the right border (relative and absolute) of the borders of relative and absolute dullness of the heart expands to the right due to hypertrophy and dilatation of the right ventricle. The left border of the heart may also be enlarged due to the displacement of the left ventricle posteriorly by the enlarged right ventricle.

Auscultation. At the points of auscultation of the tricuspid valve (the base of the xiphoid process or, with a significant increase in the right ventricle in the 4th intercostal space along the right edge of the sternum), a weakened 1 tone is determined due to insufficiency of the tricuspid valve, hypertrophy of the right ventricle and its large diastolic filling. A systolic murmur is also heard here, due to retrograde blood flow from the right ventricle to the right atrium during systole, and the fluctuation of the valve leaflets and surrounding tissues of the heart. Systolic murmur is decreasing in nature (decrescendo), increases with holding the breath during inspiration [9]. Palpation and percussion of the liver is determined by its increase in size and pain on palpation.

Aortic stenosis is currently the most common of the acquired defects. In terms of the frequency of surgical interventions on the heart, it ranks third after coronary artery stenting and coronary artery bypass grafting. The leading mechanism of aortic stenosis is a sclerodegenerative lesion of the leaflets and annulus of the aortic valve. Among the causes of such a calcifying process is a congenital heart disease - a bicuspid aortic valve (bicuspid aorta). Increases the risk of developing this defect arterial hypertension. In addition, causes of aortic stenosis may be rheumatic or less commonly infective endocarditis with calcification of valvular vegetations. Hemodynamic changes [10]. Hypertrophy of the left ventricle develops, but without a pronounced increase in its cavities. This heart disease proceeds for a long time without circulatory disorders (years, decades). With the weakening of the contractile force of the left ventricle, its dilatation develops. In the future, in advanced cases, dilatational insufficiency of the bicuspid valve, an increase in the left atrium, an increase in pressure in the pulmonary circulation may develop - mitralization of aortic stenosis occurs. The main hemodynamic manifestation of this defect is a decrease in systolic ejection of blood into the aorta. As a result, systolic blood pressure decreases and diastolic blood pressure increases compensatory (pulse decreases). Complaints. In the stage of defect compensation, patients do not complain. The appearance of complaints is associated with a deterioration in the blood supply to the organs of the systemic circulation, primarily the heart and brain itself. Patients complain of dizziness, headaches, fainting, as well as anginal pain in the region of the heart and behind the sternum, even with little physical exertion. In addition, patients may feel palpitations - strong blows of the hypertrophied left ventricle against the anterior chest wall [7].
The compensatory nature of bradycardia lies in the fact that the lengthening of the diastole improves the blood filling of the coronary arteries. In addition, patients with aortic stenosis often develop an expansion of the aortic arch, the so-called, post-stenotic expansion of the aorta associated with stretching of the aortic arch by a strong jet of blood ejected from the left ventricle. This can lead to the appearance of retrosternal pulsation. During percussion, the left border of cardiac dullness is shifted to the left.

![Echocardiographic signs of aortic stenosis](image)

**Fig. 4.** Echocardiographic signs of aortic stenosis - compaction and calcification of immobile aortic crescents - marked with an arrow (LV - left ventricle, LA - left atrium, MV - mitral valve, IVS - interventricular septum)

Sometimes the width of the vascular bundle increases due to post-stenotic expansion of the ascending aorta. Auscultation determines the weakening of the 1st tone at the apex of the heart due to left ventricular hypertrophy and the weakening of the 2nd tone on the aorta due to a decrease in blood pressure in the systemic circulation. A characteristic feature of aortic stenosis is the presence of a coarse systolic murmur with a maximum force in the 2nd intercostal space on the right side of the sternum [8]. Thus, with decompensation of aortic stenosis and the development of left ventricular dilatation, the murmur may weaken. An x-ray examination determines an increase in the heart in diameter due to the expansion of the shadow of the left ventricle, the waist of the heart is pronounced, i.e. the heart acquires an aortic configuration, there is also a post-stenotic expansion of the aorta. The ECG reveals a deviation of the electrical axis of the heart to the left, signs of left ventricular hypertrophy, and with mitralization of the defect, signs of an increase in the left atrium (P-mitrale), as well as atrial fibrillation and flutter [10]. On FCG, a diamond-shaped systolic murmur is recorded with an epicenter in the second intercostal space to the right of the sternum. The aortic component of the II tone is significantly weakened. EchoCG reveals thickening of the aortic valve cusps, pronounced hypertrophy of the posterior wall of the left ventricle without significant expansion of the cavity (Fig. 4). An important echocardiographic marker of the severity of aortic stenosis is the pressure gradient (difference) between the left ventricle and the aorta.

### 4 Conclusions

After analyzing these forecasts, we can say that the main prospects in the field of business model transformation as part of business digitalization are increased attention to data
cybersecurity, business models and processes based on artificial intelligence technologies, the Internet of things and new software. The prospects and trends for the development of new business models indicate that companies that keep pace with technological development, invest in their digital growth and create their own technologies will be able to build new sustainable competitive advantages and outperform existing competitors. Digital giants that form their own business ecosystems are already influencing industries around the world, setting the technological pace and vector of economic progress and forcing lagging competitors out of the market. Ignoring the introduction of new technologies and changes in business processes by a business can lead to the fact that in the future the company will lose the interest of its consumers in their products or services, as there will be a company on the market that is more responsive to their changing needs and global technological trends. Timely response to trends in the digitalization of business models, analysis and application of emerging progressive technologies that, according to researchers, will be relevant in the market as part of optimizing business processes and improving the value chain, can help businesses remain competitive and have sustainable advantages in the long term.

References

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