Valvular heart diseases

The obtained valvular defects include:

1. **stenosis** (narrowing)
2. **insufficiency** (regurgitation) – its result is a return flow of blood

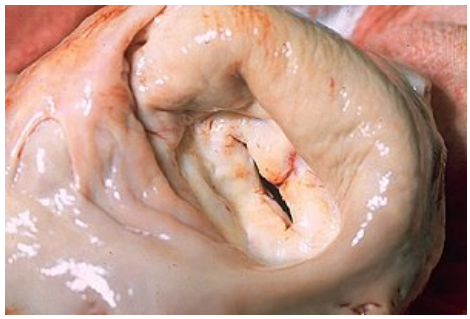
They most often affect the left valves (the mitral and aortic valves). Both defects are often combined on one flap (combined defect - the flap is both stenotic and insufficient), other times it is a pure defect (more often pure insufficiency). The term isolated defect means that only one flap is affected, while the other is normal.

Relative insufficiency occurs without organic damage to the valve during dilatation of the heart or aorta , when the edges of otherwise unchanged valves do not abut each other (eg during dilatation of the right ventricle, this results in relative insufficiency of the tricuspid valve or dilatation of the aorta due to syphilitic mesoartotitis.

Valve defects can be **compensated by** myocardial muscle hypertrophy, which maintains normal cardiac output. With more severe or acute valve defects **decompensation** and heart failure occur (morphological manifestation of decompensation is dilatation of the relevant parts of the heart).

Mitral stenosis

Mitral stenosis most often arises as a result of **rheumatic endocarditis** (adhesions of the commissures and tendons), or in large vegetations infectious endocarditis on the valves, tumors of the left atrium (mainly myxoma), massive annular sclerosis of the mitral ring. Blood stagnates in the left atrium, which expands and forms wall with thrombi (especially in its auricula), which can embolize into the great circulation, the dilated left atrium then exerts pressure on the esophagus and the left laryngeal nerve recurrens. Blood congestion is transmitted to the small circulation , congestion occurs and later rusty induration of the lungs, pulmonary hypertensioninduces subsequent right ventricle hypertrophy. It later dilates and causes a relative insufficiency of the tricuspid valve with subsequent dilatation of the right atrium and transfer of congestion into the large circulation - in the late stages there is generalized venostasis (increased filling of the jugular veins, hepatomegaly …). Consequently hypertension in the pulmonary circulation is pulmonary arterial sclerosis.


Mitral stenosis

Macroscopically the heart has mitral stenosis with dilated left atrium, hypertrophic right ventricle (this is not a cor pulmonale! - this term is reserved for hypertrophy of the right ventricle with pulmonary hypertension with cause in the lungs) and dilated right atrium, while the size of the left ventricle is not changed, clinical consequence is a **diastolic murmur**.

Mitral insufficiency

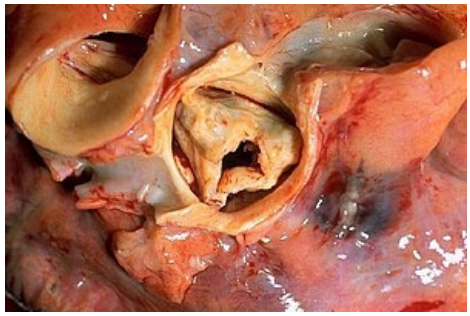
It is caused by a **disorder of the valve tips** (rheumatic endocarditis, mitral prolapse - myxoid degeneration of the valve, infectious endocarditis) or the suspension apparatus (scarring or rupture of the papillary muscle or tendon). During systole, blood penetrates back into the left atrium, which spreads and hypertrophies , from where stagnation is transmitted as in mitral stenosis to the small circulation, where it has the same consequences with right ventricle hypertrophy, but left ventricle behaves differently than in mitral stenosis - hypertrophies and dilates.

Macroscopically the heart has hypertrophied and dilated all sections, clinically **systolic murmur** in mitral insufficiency.

Aortic stenosis

The causes of aortic stenosis can be threefold:

- **rheumatic endocarditis** (10 %) – adhesions of commissures (younger adults)
- **bicuspid aortic valve** (40 %) – without commissure adhesions, dystrophic calcification of the valve (5th-6th decade)
- **senile (sclerotic) type** (50 %) – without commissures, dystrophic calcification of the valve (8th-9th decade)


Rheumatic aortic stenosis

Narrowing of the left ventricular outflow tract leads to an increase in afterload, to which the heart responds with left ventricular hypertrophy. With the worsening of the defect, there is anginal problems and the risk of sudden death - aortic stenosis **is the deadliest valve defect!**

Aortic insufficiency

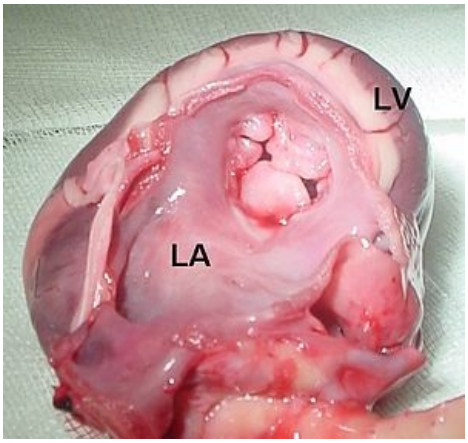
The cause is both the **involvement of the own valve** (rheumatic or infectious endocarditis), and the **involvement of the aorta** (luetic aortitis, Marfan's syndrome , aortic dissection , ankylosing spondylitis). During diastole, non-locking valve blood flows back to the left ventricle (**diastolic murmur**), which fills up more and therefore hypertrophies and then dilates. The disorder is also transmitted to other cardiac compartments, so in the final stage we find hypertrophy and dilatation of all cardiac compartments (the most pronounced hypertrophy of the whole heart, its weight can exceed 1000 g - cor bovinum). In the wall endocardium of the left ventricle septum below the aortic mouth, there are regurgitation algae - **Zahn's valves**.

Tricuspid stenosis

A rare defect, it occurs **in rheumatic endocarditis** or **carcinoid syndrome**.

Tricuspid insufficiency

It is usually **relative in right ventricle dilatation**, or occurs **in post-rheumatic shortening of the tips, carcinoid syndrome, infectious endocarditis and Ebstein's malformation**.



Mitral insufficiency caused by myxoid degeneration of the valve

Mitral prolapse (myxoid degeneration, Barlow's syndrome)

Enlargement of mitral valve tips(which, during systole, arch - weaken - into the left atrium). Anulus fibrosus sinister is dilated, the tendons thin and elongated.

Microscopically the flap content of mucous materials (acidic mucopolysaccharides) is increased.

Links

Related articles in czech

- https://www.wikiskripta.eu/w/Chlopenn%C3%AD_vady/Repetitorium

Source

-