
# PATHOGENETIC MODELS OF CHRONIC HEART FAILURE

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**ABSTRACT**

*Chronic heart failure as a complication causes disability and death of many people. Despite the improvement of treatment methods and the abundance of modern medicinal methods, chronic heart failure remains one of the most urgent problems of human society. Even in the most developed countries of the world, the incidence of chronic heart failure remains high. The pathogenesis of chronic heart failure develops on the basis of several complex mechanisms and models, and requires a pathogenetic approach to treatment.*

Chronic heart failure as a complication causes disability and death of many people. Today, the pandemic of chronic heart failure is growing at high rates throughout the world. According to the data of the European Society of Cardiology in 2016, the risk of developing chronic heart failure at the age of 55 was 33% in men and 28% in women. 52% of the cause of the development of chronic heart failure was found to be due to high risk factors of hypertension, diabetes, obesity and smoking among the population.

Chronic heart failure is a syndrome that develops as a result of various diseases of the cardiovascular system, which leads to the inability of the heart to provide blood circulation corresponding to the metabolic needs of the body, which is accompanied by intracardiac and peripheral hemodynamic disturbances, systemic changes in the heart, blood circulation violation of neurohumoral control, dimming of large and small blood circulation. We can divide the causes of chronic heart failure into the following 3 groups. The first group of causes is caused by myocardial damage. In the case of myocardial infarction, cardiomyopathies, which lead to myocardial necrosis and later develop in the form of cardiosclerosis, the second reason is the volume and pressure strain of the heart ventricles. As a result of hypertension, congenital and acquired heart defects, the volume and resistance of the heart increases, i.e. it is strained by pressure. Rheumatic heart lesions can be examples of mixed causes.

In the cardiac model of the pathogenesis of chronic heart failure, heart failure occurs as a result of hemodynamic disturbances caused by a decrease in myocardial contractility, i.e., systolic dysfunction. Myocardial hypertrophy, due to the growth of connective tissue in the last stage, the myocardium cannot perform the contractile function, in chronic heart failure, hemodynamic disturbances are invisible in the form of a decrease in systolic volume and

minute volume, despite an increase in the number of heart contractions, and low arterial blood pressure.

Based on this model, cardiac glycosides are used in the pathogenetic approach. Cardiac glycosides increase myocardial contractility (positive inotropic effect). Cardiac glycosides are especially important in chronic heart failure with atrial fibrillation.

In the cardiorenal model of the pathogenesis of chronic heart failure, water and electrolytes are retained in the body and swelling is observed, along with a decrease in myocardial contractile function. As a result, swellings appear in the body in the form of hidden - up to 5 liters, obvious, in the tissues (legs, heels, genitals, anterior abdominal wall, waist, lung edema) and in the cavity (ascites, hydropericardium, hydrothorax). The mechanism of swelling is a decrease in the hydrostatic heart pump function, which is an increase in the hydrostatic pressure in the venous capillaries.

Based on this model, diuretics are used in combination in pathogenetic approach. The use of diuretics should be started with the smallest dose and of course it is necessary to constantly control the amount of potassium in the blood. In severe cases, loop diuretics can be used in combination with carbonic anhydrase inhibitors and aldosterone inhibitors.

In the cardiocirculatory model of the pathogenesis of chronic heart failure, compensatory hemodynamic changes caused by myocardial damage lead to pre- and post-systolic stress of the heart. Due to the activation of the sympathoadrenal system, the positive chronotropic effect of catecholamines increases. As a result of the increase in the concentration of noradrenaline, angiotensin-II, aldosterone in the blood plasma of a patient with heart failure, the force of heart contraction increases, the number of heart contractions increases, venous blood return increases, and O2 utilization in tissues increases.

Compensatory mechanisms in heart failure include:

- Frank-Starling's law - the heart is aimed at increasing the force of contractility, and it is activated when it is strained by volume.
- Bowdich phenomenon-increasing the force and speed of myocardial contraction with an increase in the number of heart contractions.
- Anrep effect-isometric hyperfunction occurs when the resistance increases, the myocardium increases the force of contraction.
- Activation of the sympathoadrenal system - vasoconstriction under the influence of catecholamines is aimed at centralizing blood circulation.
- Myocardial hypertrophy-in 3 stages: accident, complete hypertrophy and cardiosclerosis (according to F.Z. Meerson)
- Activation of the renin angiotensin aldosterone system occurs as a result of deterioration of blood circulation in the kidney.
- Endothelin production-constricts peripheral blood vessels.
- Production of sodium uretic peptide (NUP) - an endogenous diuretic reduces the pre-systolic tension of the heart. By increasing the production of nitric oxide, it expands the resistance vessels and reduces the afterload.

Cardiac and extracardiac compensatory mechanisms are activated in heart failure and long-term cardiac contractile function helps to maintain the minute volume in accordance with the body's needs. But over time, the compensatory mechanisms themselves create side effects. For example:

Side effects of the Frank-Starling mechanism - As a result of excess volume and stretching of cardiomyocytes, dilatation of the heart cavities occurs;

Renin angiotensin aldosterone system side effects - An increase in total peripheral vascular resistance due to reflex narrowing of peripheral arterioles. Increased resistance to the heart's blood flow. Deterioration of organ and tissue perfusion. Microcirculation disorders and hypoxia occur as a result of reduced organ and tissue perfusion.

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