**THE ROLE OF STRESS IN THE PATHOGENESIS OF CORONARY HEART DISEASE.**

**Introduction.** An analysis of the living conditions of persons suffering from coronary heart disease showed that the etiological factor is stress that provokes the development of myocardial infarction (one form of CHD). When it comes to the etiology of ischemic heart disease, it is common to talk about so-called risk factors for this disease. Among these factors are the most important - hereditary factors, in all likelihood related to the features of cholesterol exchange combined with food consumption, cholesterol-rich, adynamics, smoking, sometimes alcohol and, finally, stressful situations.

At the moment, various studies have shown that stress, ischemia and a combination of these factors play a leading role in the occurrence of major heart diseases. The whole complex of cardiovascular system lesions (atherosclerosis, blood vessel thrombosis, coronospasm, myocardiodystrophy, myocardial infarction, arrhythmias and sudden cardiac death, arterial hypertension) is based on stress damage to myocardial and blood vessels [1]. Myocardial ischemia itself, which occurs in coronary artery occlusion, is also a powerful stress factor that not only enhances ischemic damage, but can cause primary non-coronarogenic damage to non-ischemic heart regions. The stress response that occurs when new or extreme environmental factors act on the organism largely determines the development of biochemical and functional myocardial changes in ischemic or post-ischemic reperfusion heart damage [3]. F. Z. Meerson [2-3] analyzed in detail the mechanisms by which stress response can be important in the pathogenesis of ischemic heart disease and myocardial ischemia.

The first attack of the disease causes a person to feel painful and fear of death, which leads to stress. The stress response has the following effects:

• Liver damage, atherogenic dyslipidemia;

• Arrhythmia of heart rate of healthy heart areas;

• Increased blood creability and thrombus formation;

• Decreased resistance of the muscle layer of the heart to hypoxia and reoxygenation;

• Increased vascular resistance of a large circulation and as a consequence heart overload.

All these factors cause repeated attacks of myocardial ischemia, each of which again triggers a stress response, and as a result such a cyclic process leads to myocardial infarction. One of the factors contributing to the development of atherosclerosis is considered stress damage of the liver. In favour of this provision, evidence suggests that so-called familial hypercholesterolemia, leading to the coronary atherosclerosis, is associated with a genetic liver defect manifested in the disruption of the capture mechanism and catabolism of low-density atherogenic lipoproteins. It has been found that stress damage to the liver leads to disruption of cholesterol oxidation processes in it, its conversion to bile acids and their removal from the body, which in turn contributes to hypercholesterolemia. As a result, even in the absence of excess cholesterol in food, stress can lead to the development of stenotic coronary sclerosis and plays an important role in the development of coronary heart disease. At the same time, the stress reaction causes excess calcium, multiple zones of conduction disturbance and depolarization. The combination of these disorders leads to a breakdown of electrical stability of the heart, severe arrhythmias, fibrillation and cardiac arrest. The resulting ischemic area is accompanied by pain, which increases the stress response and can complicate the course and outcome of the primary ischemic injury. Myocardial ischemia is accompanied by shutdown of the respiratory chain of mitochondria in the ischemia zone, inhibition of the tricarboxylic acid cycle, deficiency of macroergic compounds and activation of glycolysis. All this naturally causes pain syndrome, which increases or again causes stress response, increasing the activity of the sympathetic-adrenal system and the release of catecholamine. At present, it has been found that in the development of stress or ischemic heart damage, the disorder of neurohormonal regulation of the function of this organ is of great importance. In neurodystrophic myocardial damage caused by extreme irritation on various reflexogenic zones or hypothalamic centers, the nature of nerve and hormonal influences on the heart changes dramatically. Thus, one of the key links of stress damage to the myocardium is the lipotropic effect of cateholamines and glucocorticoids, which directly or indirectly enhance the activity of lipases, phospholipases, lipid peroxidation intensity. [4]

Several mechanisms can be identified that contribute to the development of CHD:

1. Prolonged, frequent stimulation of the sympathetic nervous system leads to myocardial damage. In this injury, an important role is given to increasing intracellular calcium content. Calcium mechanisms of damage are usually considered in accordance with the following phenomena: activation of phospholipases, increase of LPO activity, weakening of sarcoplasmic reticulum (SPR) power and myofibrill contractions, deterioration of mitogodria due to their calcium overload. All of these disorders of myocardial compressive function mechanisms inevitably lead to disorders of the cardiac cycle-systole and diastole phases.

2. Under the influence of catecholamines, the demand of the myocardium for oxygen grows, which can lead to ischemic damage to the myocardium.

3. Catecholamines, having the ability to activate blood coagulation processes, can thereby contribute to the development of coronary artery thrombosis by potentiating ischemic myocardial damage.

4. The reduction of coronary blood flow may be facilitated by hyperventilation of the lungs during stress, which leads to an increase in oxygen tension in the blood, followed by the development of hypocapnical alkalosis, which in turn increases the tone of the coronary arteries.

5. The deterioration of coronary blood flow is facilitated by stress-characteristic hyperlipidemia caused by excessive enhancement of the adaptive lipotropic effect of stress. In the blood, the content of UEFA (unesterified fatty acids) is increased, followed by triglycerides resynthesized in the liver from UEFA. Production and content of β-lipoproteins containing cholesterol is increased in blood. Coronary artery atherosclerosis develops, whose role in CHD pathogenesis cannot be overestimated [5].

According to the given data, it becomes obvious that stress has a great impact on the development of pathology of the heart muscle and cardiovascular system. And if not a stressful reaction caused primary damage, it can strongly affect further development of the disease and cause its relapses. In addition to affecting the heart muscle, stress also affects the blood itself, increasing its coagulability, which can cause thrombus formation within the blood pathways and cause serious problems.

**List of references**

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