

# Heart failure (pediatrics)/acute

Acute heart failure is accompanied by a decrease in the ratio of *cardiac output / cardiac index* (  $CO/CI$  ), an increase in systemic venous pressure and a reflex sympathoadrenal reaction. In acute failure, myocardial dysfunction occurs suddenly, we detect clinical signs of failure with the application of sympathetic activation and the Frank-Starling law .

In case of right- or left-sided failure, dilation of the atria and venous bed leads to the activation of the so-called stretch receptors. This increases sympathetic tone and subsequently stimulates  $\alpha + \beta$  adrenergic and cholinergic receptors. **Stimulation of  $\alpha$ -adrenergic receptors** leads to vasoconstriction in the GIT, kidneys, muscles, and skin. Clinical signs are peripheral hypoperfusion and oliguria . **Stimulation of  $\beta$ -adrenergic receptors** has a positive chronotropic (tachycardia) and inotropic effect. **Stimulation of cholinergic receptors** is clinically manifested by sweating. Overall, it can be said that sympathetic activation leads to an increase in contractility and heart rate, i.e. it applies **positive inotropic and chronotropic effect**.

A direct increase in myocardial contractility is mediated by increased release of noradrenaline from sympathetic nerve endings in the myocardium and increased secretion of catecholamines from the adrenal medulla . If the compensatory mechanisms of the myocardium are insufficient and the cardiac output decreases, peripheral compensatory mechanisms are involved. It is  $\alpha$ -adrenergic stimulation with peripheral vasoconstriction and redistribution of blood with preferential supply to the myocardium and CNS. The degree of peripheral compensation also depends on the degree of decrease in cardiac output. In a mild form, it manifests itself only in a reduction of physical load tolerance (decrease in blood flow through the skeletal muscles), in severe forms it leads to a decrease in perfusion of the kidneys , liver ,pancreas , etc., with the development of oliguria , edema , hepatopathy . With further progression , cardiogenic shock develops with dilation of the heart muscle.

## Links

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### Source

- HAVRÁNEK, J.: *Akutní srdeční selhání*.