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Role of a2-adrenoceptors in rat heart with the model of myocardial infarction

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Abstract

Chronic adrenergic and angiotensin-energic stimulation of the heart muscle is one of the main causes of the onset and development of heart failure. α2-AR is widespread in the cardiovascular system and can be further applied and introduced into the heart's regenerative cell therapy. This study's objective was to study the effect of α2-AR stimulation on the performance of an isolated heart in rats with a model of myocardial infarction. To study the mechanisms of MI, experiments were carried out according to the classical technique of H. Selye-ligation of the anterior branch of the left coronary artery and perfusion of a Langendorff heart with a model of myocardial infarction. We studied the parameters of myocardial activity by changes in heart rate, as well as the left ventricle pressure (LVP), and the CF (dp/dtmax)/(dp/dtmin) of the left ventricle. Coronary dilator activity was assessed by measuring the outflow of the perfusion solution through the coronary arteries. The amplitude of the left ventricle's pressure wave after application of the α2-AR agonist clonidine hydrochloride at a concentration of 10-6 M to the perfused solution increased by 44% ($p \leq 0.05$) from the initial values. The rate of contraction and the rate of relaxation of the left ventricular myocardium also increased by 20% ($p \leq 0.05$) and 37% ($p \leq 0.05$), respectively. The activation of α2-AR in adult rats' isolated hearts with a model of myocardial infarction caused a decrease in heart rate by 18% ($p \leq 0.05$) and CF by 2% ($p \leq 0.05$).

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Keywords

Isolated heart, Model, Myocardial infarction, Rat, α -adrenoreceptors 2

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