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Carbon monoxide affects electrical and contractile activity of rat myocardium

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Abstract

Background: Carbon monoxide (CO) is a toxic gas, which also acts in the organism as a neurotransmitter. It is generated as a by-product of heme breakdown catalyzed by heme oxygenase. We have investigated changes in electrical and contractile activity of isolated rat atrial and ventricular myocardium preparations under the influence of CO. Methods. Standard microelectrode technique was used for intracellular registration of electrical activity in isolated preparations of atrial and ventricular myocardium. Contractions of atrial myocardial stripes were registered via force transducer. Results: CO (10-4 - 10-3 M) caused prominent decrease of action potential duration (APD) in working atrial myocardium as well as significant acceleration of sinus rhythm. In addition CO reduced force of contractions and other parameters of contractile activity. Inhibitor of heme oxygenase zinc protoporphyrin IX exerts opposite effects: prolongation of action potential, reduction of sinus rhythm rate and enhancement of contractile function. Therefore, endogenous CO, which may be generated in the heart due to the presence of active heme oxygenase, is likely to exert the same effects as exogenous CO applied to the perfusing medium. In ventricular myocardium preparations exogenous CO also induced shortening of action potential, while zinc protoporphyrin IX produced the opposite effect. Conclusions: Thus, endogenous or exogenous carbon monoxide may act as an important regulator of electrical and contractile cardiac activity.

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