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EFFECT OF ADAPTATION TO SHORT-TERM STRESS ON RESISTANCE OF PARAMETERS OF MYOCARDIAL ENERGY METABOLISM AND CONTRACTILE FUNCTION TO ACUTE HYPOXIC HYPOXIA AND REOXYGENATION

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Adaptation to short-term stress regularly increases the resistance of the heart to ischemic and reperfusion arrhythmias [3] and limits depression of the contractile function and disturbances of the electrical stability of the heart in experimental myocardial infarction [8]. However, it has not yet been settled whether this protective effect is due purely to limitation of the stress reaction, which is always observed during adaptation to short-term stress [2], or whether this adaptation involves a direct increase in the resistance of the heart to acute hypoxia and subsequent reoxygenation.

The aim of this investigation was to assess the effect of preliminary adaptation to stress on the resistance of the parameters of the energy metabolism and contractile function of the heart to acute hypoxia and subsequent reoxygenation.

EXPERIMENTAL METHOD

The investigation was conducted on male Wistar rats weighing 200-250 g. Adaptation to stress was carried out by immobilizing the animals in the supine position for between 15 min and 1 h, 8 times at intervals of 1 day. Acute experiments were then carried out on the adapted and control animals, under pentobarbital anesthesia (50 mg/kg) and artificial respiration. The rats' hearts were frozen actually in the chest with Wollenberger's forceps: in the animals of group 1 in a state of relative physiological rest, in those of group 2 in a state of hypoxia (4 min after stopping artificial respiration), and in group 3 during reoxygenation (5 min after the resumption of respiration). The frozen hearts were used to determine the parameters of myocardial energy metabolism. ATP, ADP, AMP, and lactate were determined with the aid of kits from "Boehringer," and creatine

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