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EFFECT OF NEUROGENIC INJURY TO THE HEART ON ITS GLUCOSE-6-PHOSPHATE DEHYDROGENASE CONTENT

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Neurogenic damage to the rat heart muscle by electrical stimulation of the arch of the aorta leads to the development of a deficiency of tissue noradrenalin (NA), the mediator of the sympathetic nervous system. This is accompanied by a marked increase in glucose-6-phosphate dehydrogenase (G6PD) activity. Preliminary administration of actinomycin D, an inhibitor of RNA synthesis, completely prevents the increase in G6PD activity in the heart muscle tissue following neurogenic injury. The results indicate the more rapid induction of this enzyme in the tissue of the myocardium after neurogenic injury, which correlate with changes in the tissue NA balance.

KEY WORDS: neurogenic injuries; heart muscle; noradrenalin; glucose-6-phosphate dehydrogenase; actinomycin D.

Previous investigations have shown that destruction of the tissue of the myocardium through its neurogenic injury is preceded by metabolic disturbances [1, 4, 5], based on changes in the velocity of enzyme reactions [2, 3, 7]. Simultaneously with disturbance of the tissue noradrenalin balance, on account of exhaustion of its reserves, a marked increase was found in the activity of hexokinase and lactate dehydrogenase, with a consequent increase in the rate of glycolysis, and also in glucose-6-phosphate dehydrogenase (G6PD) activity, limiting the pentose phosphate metabolic pathway [37]. However, the mechanisms of the changes in enzyme activity in the heart muscle associated with neurogenic lesions have not yet been explained. It was accordingly decided to study changes in G6PD activity in the soluble fraction of heart muscle after neurogenic injury with the aid of actinomycin D, an inhibitor of protein synthesis.

EXPERIMENTAL METHOD

Experiments were carried out on 36 male albino rats weighing 180-200 g. Neurogenic damage to the myocardium was caused by electrical stimulation of the arch of the aorta [1]. The series of investigations was undertaken on four groups of animals: 1) 10 intact rats (control); 2) 5 rats undergoing a mock operation, the operative control; 3) 10 rats subjected to electrical stimulation of the arch of the aorta for 3 h on the second day after the operation; 4) 11 rats receiving an intraperitoneal injection of actinomycin D in a dose of 100 μg per rat before electrical stimulation.

G6PD activity in the soluble fraction of heart muscle tissue (20,000 g, 40 min) was determined spectrophotometrically at 340 nm, 24 h after stimulation of the arch of the aorta, from the rate of reduction of added

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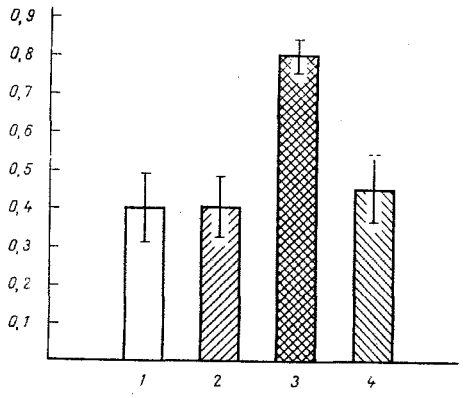

Fig. 1. G6PD activity in heart muscle. Abscissa: 1) control animals, 2) animals undergoing mock operation, 3) 24 h after stimulation of arch of aorta for 3 h, 4) 24 h after stimulation preceded by injection of actinomycin D; ordinate, G6PD activity (in μmoles NADPH/mg protein [8]).

NADP [10]. The protein concentration was determined by Lowry's method [11] and the noradrenalin (NA) level by a fluorometric method [6].

EXPERIMENTAL RESULTS AND DISCUSSION

The results showed that after neurogenic injury to myocardium, simultaneously with the sharp decrease in the tissue NA concentration in the heart muscle there was a considerable rise in G6PD activity. For instance, in animals undergoing electrical stimulation of the arch of the aorta, the myocardial NA level fell to 0.1 ± 0.015 μg/g compared with the normal value of 0.5 ± 0.04 μg/g wet weight of tissue, whereas G6PD activity was doubled (Fig. 1).

The results of these experiments agree with those of other workers [8, 9] who found changes of a similar type in G6PD activity on account of particular molecular forms in denervated liver tissue of animals with a sharply reduced catecholamine concentration in the organ.

Preliminary administration of actinomycin D, the inhibitor of protein synthesis, completely prevented the rise in G6PD activity in the soluble fraction of the heart muscle after neurogenic injury. The G6PD activity in these animals did not differ significantly from that in the controls or in animals undergoing the mock operation without subsequent stimulation of the arch of the aorta (Fig. 1).

Prevention of the increase in G6PD activity in the myocardium by actinomycin D is evidence of the more rapid induction of the synthesis of this enzyme which correlate with changes in the tissue NA balance following neurogenic injury to the myocardium.

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