CHOLINERGIC INFLUENCES ON METABOLISM IN THE DAMAGED MYOCARDIUM*

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UDC 616.127-001-008.9-092: 612.819.911

Two days after injury to the myocardium produced by injection of adrenalin into rabbits, strengthening of cholinergic influences increased the rate of protein resynthesis in the myocardium, while weakening of these influences was accompanied by slowing of protein resynthesis.

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Metabolic processes in the heart are under the control of trophic regulation effected largely through cholinergic mechanisms [4-6].

The object of this investigation was to study the effect of these mechanisms on the course of metabolic processes in the damaged myocardium.

EXPERIMENTAL METHOD

Experiments were carried out on 64 rabbits. Damage to the myocardium was produced by repeated intravenous injections of caffeine and adrenalin [2]. Cholinergic influences on the heart were potentiated by intramuscular injections of 0.1% eserine solution (0.8 ml), followed after 5-10 min by acetylcholine (AC) solution in a dilution of $1:10^5$ (1 ml). Cholinergic influences were weakened by intramuscular injection of 0.1% atropine solution (1 ml) into rabbits after preliminary division of the left vagus nerve in the neck. In all the experiments, injections were given three times a day for 6 days until the myocardium was damaged, and then until the animals died.

The state of metabolism in the myocardium was assessed from incorporation of methionine-S^{35} and sodium phosphate-P^{32} into its composition. Indicator solutions of each isotope were injected into the marginal vein of the ear (in doses of 20 μCi P^{32} and 40 μCi S^{35}/kg body weight). The animals were sacrificed by air embolism 10 min later, the heart was quickly removed and freed from blood, and four weighed samples were taken from the walls of the atria and ventricles.

Radioactivity for P^{32} was determined on a type MST-17 end-window counter, and for S^{35} on a type BFL-25 counter. Both counters were connected to a B-2 apparatus.

The results were expressed in pulses/min/100 mg fresh tissue. The results were analyzed separately for the atria and ventricles, the differences between them lying within the limit of experimental error. Accordingly, combined results for the heart as whole are given in this paper.

EXPERIMENTAL RESULTS

Incorporation of P^{32} into the myocardium 2 days after adrenalin damage to the heart was increased from 270 ± 63.4 to 558 ± 82.2 pulses/min/100 g tissue ($M \pm mt$; $P < 0.05$), but the retention of S^{35} was unchanged.

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Kazakh Research Institute of Maternal and Child Welfare, Alma-Ata. (Presented by Academician of the Academy of Medical Sciences of the USSR S. E. Severin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 68, No. 12, pp. 23-24, December, 1969. Original article submitted July 24, 1968.

Strengthening of cholinergic influences at this stage led to a more rapid incorporation of P^{32} (764 ± 234.8 pulses/min) and, in particular, of S^{35} (103 ± 22 pulses/min) into the myocardium two days after adrenalin damage (60 ± 20.6 pulses/min).

Suppression of cholinergic regulation had no effect on the level of phosphorus incoporation into the damaged myocardium, but definitely reduced the incoporation of sulfur (34 ± 19.6 pulses/min/100 g tissue; $P < 0.01$).

The results confirm the role of cholinergic mechanisms in the regulation of tissue repair processes and, in particular, in protein synthesis. In the early stage of adrenalin damage to the myocardium, when processes of destruction and death of muscle fibers are predominant in the tissues [3], protein resynthesis does not exceed the normal level [1].* Under these conditions, cholinergic mechanisms, by stimulating protein resynthesis, promote compensatory repair processes.

The level of P^{32} incorporation into the myocardium was increased 7 days after adrenalin injury while that of S^{35} was normal. However, at this stage of development of disturbances in the myocardium, interference with cholinergic regulation could no longer influence the metabolic rate.

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*In Z. I. Vedeneeva's experiments [1] on rats, using a different modification of myocardial damage, resynthesis of protein-S^{35} was doubled after only 24 h; the level of significance of the difference is not given.