PROTEIN FRACTIONS OF THE MYOCARDIUM IN DOGS WITH EXPERIMENTAL MYOCARDIAL INFARCTION

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UDC 616.127-005.8-092.9-07:616.127-008.935.6-074

Despite the fact that the metabolic processes in myocardial infarction have been investigated by many authors, little attention has been paid to the study of the protein metabolism of the myocardium. Experimental results have been obtained indicating a decrease in the myosin content and in the ATPase activity in the zone of ischemia in the early period after ligation of the coronary artery [7].

The object of the present investigation was to study the fractional composition of the myocardial proteins and the ATPase activity of the chief contractile protein (actomyosin) at various periods of development of experimental myocardial infarction in dogs (from 24 h to 6 months).

EXPERIMENTAL METHOD

Myocardial infarction was produced in dogs by ligation of the anterior descending branch of the left coronary artery*.

In 15 control animals, the tissues of the left and right ventricles were investigated, and in 25 dogs undergoing the operation tissue was taken for examination from the undamaged portions of the right and left ventricles and also from the region of the infarct.

The heart was extracted from the animals under artificial respiration and ether anesthesia. It was perfused with ice-cold physiological saline and placed in ice. The tissue was manipulated in a cold room (2-4°). The fractional composition of the proteins was studied by I. I. Ivanov's method [4].

The ATPase activity of the actomyosin was investigated by P. M. Zubenko's method [3] and the phosphate subsequently determined by Lowry's method [10].

The protein was precipitated with 10% trichloroacetic acid solution and the total nitrogen and the protein nitrogen were determined by Kjeldahl's method. The numerical results were analyzed statistically by Student's method [6].

EXPERIMENTAL RESULTS

The protein content, and the enzyme activity in the zone of ischemia and the portions of the heart not affected by ischemia, were compared with the corresponding indices for the myocardium of the healthy dogs.

It is clear from Table 1 that in the zone of the infarct, the content of sarcoplasmic and myofibrillary proteins fell gradually with an increase in the length of time after formation of the infarct.

The decrease in the content of sarcoplasmic proteins was evidently due partly to loss or total disappearance of enzymes from the zone of ischemia [1,2,9].

The changes in the content of the myofibrillary proteins took place on account of the chief contractile protein actomyosin, the content of which fell gradually, so that 6 months after ligation of the coronary artery it was 52% below normal.

The decrease in the content of actomyosin during the first day after the development of the infarct was evidently attributable to its breakdown caused by necrosis of the tissue. However, for long subsequent periods after the creation of the infarct, the process of necrotic breakdown of protein (its restoration was impossible as a result of the

*The operations were performed by B. F. Fedorov, Senior Scientific Assistant at the Institute of Physiology, and E. F. Lushnikov, Assistant at the 1st Moscow Medical Institute.

Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Active Member of the Academy of Medical Sciences of the USSR V. V. Parin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 62, No. 8, pp. 40-44, August, 1966. Original article submitted December 14, 1964.

TABLE 1. Fractional Composition of Myocardial Proteins in Dogs with Experimental Myocardial Infarction

Nitrogen (in mg/g moist tissue)	Statistical index	Normal		Time after ligation of coronary artery					
				1 day			4 days		
		LV	RV	ZI	LV	RV	ZI	LV	RV
Sarcoplasmic proteins	$M \pm m$	6.3±0.5	6.2±0.4	5.5±0.4	6.0±0.5	5.6±0.5	4.9±0.4	6.9±0.6	6.88±0.5
	P			0.05			<0.002	0.5	0.5
Myofibrillary proteins	$M \pm m$	5.5±0.6	5.4±0.5	4.89±0.45	4.88±0.6	4.8±0.5	4.12±0.4	5.7±0.5	4.9±0.3
	P			0.05	0.05	0.05	<0.05		
Actomyosin	$M \pm m$	3.99±0.4	3.97±0.55	2.92±0.3	3.46±0.5	3.50±0.6	3.1±0.3	3.85±0.5	3.77±0.4
	P			<0.05			<0.05		
Stroma	$M \pm m$	5.8±0.6	5.5±0.5	5.5±0.4	5.8±0.6	6.2±0.5	7.4±0.4	6.0±0.5	5.8±0.4
	P						<0.01		
Solid residue (in %)	$M \pm m$	22.6±0.6	22.5±0.4	15.9±0.7	20.0±0.5	20.0±0.4	16.2±0.6	20.8±0.3	21.0±0.4
	P			<0.02	<0.05	0.05	0.02	0.05	<0.5

Nitrogen (in mg/g moist tissue)	7 days			15 days			6 months		
	ZI	LV	RV	ZI	LV	RV	ZI	LV	RV
Sarcoplasmic proteins	4.9±0.3	6.5±0.5	6.8±0.5	5.0±0.3	6.1±0.4	6.1±0.4	4.3±0.5	6.5±0.3	6.2±0.4
	<0.02			<0.02			0.01		
Myofibrillary proteins	3.9±0.25	6.83±0.7	7.0±0.68	3.6±0.3	6.68±0.5	6.1±0.4	3.0±0.1	5.8±0.5	5.5±0.5
	0.01	<0.02	<0.02	<0.01	<0.02	<0.5	<0.01		
Actomyosin	2.4±0.2	4.7±0.5	4.8±0.4	2.8±0.8	4.5±0.5	4.3±0.4	1.9±0.35	4.4±0.4	4.2±0.5
	<0.01	0.02	<0.02	<0.01			0.001	<0.05	0.5
Stroma	7.5±0.7	5.9±0.4	6.2±0.5	8.0±0.5	5.2±0.4	5.7±0.5	11.0±0.6	6.6±0.3	6.0±0.4
	<0.01			0.001			0.001		
Solid residue (in %)	20.2±0.2	22.4±0.5	22.2±0.4	22.4±0.5	22.3±0.4	22.5±0.5	24.5±0.7	22.0±0.4	22.7±0.4
							<0.02		

Legend: Here and in Table 2: ZI) zone of infarct; LV) left ventricle; RV) right ventricle.

TABLE 2. ATPase Activity of Actomyosin in Dogs with Experimental Myocardial Infarction

	Statistical index	Normal		Time after ligation of coronary artery					
				1 day			4 days		
		LV	RV	ZI	LV	RV	ZI	LV	RV
Phosphorus (in mg/g tissue/ 10 min)	$M \pm m$ P	4.46±0.35	4.88±0.41	3.35±0.3 <0.05	4.6±0.4	5.8±0.37 <0.05	3.6±0.25 <0.01	6.7±0.5 0.01	6.6±0.55 0.001
Phosphorus (in mg/mg actomyosin)	$M \pm m$ P	1.1±0.09	1.2±0.1	1.1±0.07	1.35±0.08 0.05	1.6±0.1 0.02	1.1±0.08	1.7±0.2 <0.02	1.7±0.15 <0.02

	7 days			15 days			6 months		
	ZI	LV	RV	ZI	LV	RV	ZI	LV	RV
Phosphorus (in mg/g tissue/ 10 min)	2.3±0.21 <0.01	11.2±0.87 0.001	10.7±0.9 0.001	2.8±0.2	4.5±0.4	4.6±0.31	1.7±0.2 <0.001	3.7±0.3 0.01	4.0±0.55 0.01
Phosphorus (in mg/mg actomyosin)	0.9±0.08 <0.05	2.3±0.3 0.001	2.2±0.25 <0.001	0.95±0.09	1.0±0.05	0.98±0.06	0.94±0.05 0.5	0.84±0.07 <0.05	0.99±0.05

disturbance of ATP resynthesis [5,8,11,12]) was joined by a process of replacement of the remaining active contractile protein by scar connective tissue. This is confirmed by the appearance of collagen fibers in the zone of ischemia [2] and by the increase in the nitrogen content of the stroma and of the solid residue of the tissue (see Table 1).

The ATPase activity of the actomyosin (Table 2) and the content of actomyosin gradually fell with an increase in the length of the period after ligation of the coronary artery, i.e., the content of actomyosin per gram of tissue of the ischemic area containing active enzyme fell. The activity of the actomyosin remaining in the zone of ischemia was almost the same as the actomyosin activity of the healthy heart, for its ATPase activity, expressed per milligram of protein nitrogen, remained essentially unchanged throughout the period of the experiment.

In the tissue of the myocardium outside the zone of ischemia, in both the left and the right ventricle, the content of sarcoplasmic proteins remained unchanged throughout the period of investigation.

The content of myofibrillary proteins and of actomyosin likewise was unchanged, except on the 7th-15th day after ligation of the coronary artery, when the content of contractile proteins rose (see Table 1). This may evidently be regarded as a compensatory, adaptive reaction to the loss of the contractile function by a certain part of the myocardium. Processes of compensation and adaptation may probably also account for the increase in the ATPase activity of the actomyosin during the first 7 days after creation of the infarct (see Table 2).

The results described above are in full agreement with those obtained by other investigators who found an intensification of the processes of glycolysis and respiration [7] and an increase in the absorption of oxygen [8] and metabolism of organic phosphorus compounds [5-8] in the tissues of the heart outside the zone of ischemia in the early periods of development of an infarct.

Six months after ligation of the coronary artery, a slight increase in the actomyosin content with a fall in ATPase activity were observed in the left ventricle outside the zone of ischemia (see Tables 1 and 2), and these findings may be regarded as a decrease in the ability of the actomyosin to utilize the energy of high-energy bonds.

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All abbreviations of periodicals in the above bibliography are letter-by-letter transliterations of the abbreviations as given in the original Russian journal. *Some or all of this periodical literature may well be available in English translation.* A complete list of the cover-to-cover English translations appears at the back of the first issue of this year.
