GLYCOGEN, LACTIC ACID, AND PYRUVIC ACID CONTENTS OF THE MYOCARDIUM UNDER CONDITIONS OF COMPENSATORY HYPERFUNCTION

A. P. Bozhko

Department of Normal Physiology, Vitebsk Medical Institute
(Scientific Director: Docent G. M. Pruss)
(Presented by Member AMN SSSR V. V. Parin)
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Further research on myocardial biochemistry is needed for the understanding of changes in the activity of the heart under conditions of compensatory hyperfunction, and this applies in particular to its carbohydrate metabolism, which bears a direct relation to the energetics of muscle contraction.

F. Z. Meerson and his coworkers have demonstrated the existence of considerable changes in glycogen and lactic acid contents of the myocardium at various stages of compensatory hyperfunction, developing under the conditions of experimental aortic stenosis in rabbits [2,3].

We have been unable to trace any references in the literature to biochemical changes taking place in the myocardium under conditions of compensatory hyperfunction caused by exclusion of part of the working musculature of the left ventricle from the contractile act. Such studies may be of practical importance since this model of hyperfunction develops following resection of part of the left ventricle, which is of interest to surgeons as one of the possible variants of surgical treatment of certain heart diseases.

We examined the dynamics of changes in the contents of glycogen, lactic acid, and pyruvic acid in muscle tissue of the left ventricle under conditions of hyperfunction caused by substantial reduction in the amount of working heart muscle.

EXPERIMENTAL METHOD

For our experiments we used 109 rabbits. Parts of the 5th and 6th ribs were resected to the left of the midline, under general urethane anesthesia and with local procaine anesthesia, and the heart was exposed, without opening the pleural cavity. A ligature was applied round the lower third of the left ventricle, which, when tightened, excluded the corresponding part of the ventricle from the act of contraction. A dummy operation, involving exposure of the heart, but without ligation, was performed on the animals of the control group.

The mediastinum was opened at different times after the operation (1, 3-4, 15, and 30 days), the heart was quickly removed, washed free of blood with cold physiological saline, the part below the ligature was cut off, and the remainder of the left ventricle was fixed, for determination of its glycogen, lactic acid, and pyruvate contents. The time elapsing between opening of the mediastinum and fixation of the heart muscle did not exceed 60 seconds.

For glycogen determination portions of myocardium were hydrolyzed in 30% potassium hydroxide solution, and glycogen was precipitated from the hydrolyzate by adding ethanol [7]. Its amount was estimated from the intensity of its color reaction with phenol and sulfuric acid [8].

For lactic and pyruvic acid estimation portions of myocardium were frozen in a stream of carbon dioxide delivered from a cylinder, using the device described in [1], for 5-6 seconds. The frozen tissue was weighed, and ground up with 5% trichloroaectic acid at 0°. The suspension was centrifuged, lactic acid was determined colorimetrically in the supernatant by the reaction with p-hydroxydiphenyl [5], and pyruvic acid by the method of Friedemann and Haugen [6].

The results were treated by the method of statistics of variance.

EXPERIMENTAL RESULTS

The glycogen content fell abruptly, from 609 to 339 mg-%, over the first few days after the operation. Its content remained at the same low level over the whole period of observation (see the table).

Glycogen, Lactic Acid, and Pyruvic Acid Contents at Different Stages of Compensatory Cardiac Hyperfunction

Substance determined	Statistical Coefficient	Control	Days after operation			
			1	3–4	15	30
Glycogen	M	609	339	438	460	416
	m	50	31	30	38	42
	P		0,001	<0,01	<0,03	0,001
Lactic acid	M	34	45	36	43	34
	m	3,6	5,3	1,7	3,4	6,0
	P		>0,5	>0,5	0,1	1,0
Pyruvic acid	M	0,84	0,97	0,93	0,86	0,95
	m	0,05	0,1	0,07	0,09	0,06
	P		>0,5	>0,2	>0,5	>0,20

There were no substantial changes in lactic and pyruvic acid contents (see the table), but the ratio of these two acids, which characterizes the relative proportions of the processes of anaerobic and aerobic utilization of glycogen rose, with a high degree of significance (P< 0.005), for two weeks after the operation (see the table). This ratio had reverted to the normal value a month after the operation.

Compensatory myocardial hyperfunction following exclusion of a substantial part of the working muscle from the act of contraction presents initially a picture analogous to that of the so-called traumatic phase found in experimental aortic stenosis of rabbits [4].

In our experiments, the traumatic state persisted for two weeks, during which time about 30% of the animals died, with symptoms of cardiac insufficiency (ascites, hydrothorax, pulmonary, and renal congestion).

The glycogen content of the myocardium fell steeply during the first few days after operation, and the rise in the ratio of lactic to pyruvic acid observed after two weeks is clear evidence of the preponderance of anaerobic carbohydrate metabolism. A similar activation of anaerobic resynthesis of adenosine triphosphate, due to intensified expenditure of high-energy phosphates for functional and plastic purposes, was observed in experimental aortic stenosis [2].

Following ligation of the ventricle, the part of the myocardium situated above the ligature underwent hypertrophy, which was evident, both micro- and macro-scopically, a month after the operation.

Selye et al. [9] also found pronounced hypertrophy of that part of the myocardium which was situated above the ligature.

The traumatic state was followed by one of more or less stable compensation (relief of ascites, hydrothorax, and of pulmonary and renal congestion).

However, the glycogen content of the myocardium remained at its low level. The normalization of the lactate: pyruvate ratio pointed t o the absence of hypoxic phenomena, in the given case. The lowered glycogen content may be ascribed to the increased requirement by the myocardium of carbohydrate metabolites. It also indicates lowering of glycogen reserves, and hence of the potential energy resources of the heart.

This conclusion is supported by the results of experiments involving partial resection of the left ventricle of dogs.

SUMMARY

Compensatory hyperfunction of the left ventricle after ligating off part of its muscle results in cardiac insufficiency, followed by a relatively stable compensation. The glycogen content of the working part of the myocardium of the left ventricle fell to a low level soon after ligation, and was still at this level a month after operation. This is evidence of diminution in the potential energy resources of the heart. The lactic and pyruvic acid contents did not vary substantially.

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