CONTRACTILE FUNCTION OF THE HEART MUSCLE IN EXPERIMENTAL MYOCARDIAL INFARCTION

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Seven days and one month after ligation of the descending branch of the left coronary artery the maximal pressure in the left ventricle (P_{max}) during compression of the aortic orifice and the maximal intensity of function of its structures (IFS_{max}) were below normal; the index of contractility (IC) also was lowered. Six months after ligation, P_{max} and IC had returned to the normal level while IFS_{max} remained low.

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The fate of the patient surviving immediately after an attack of myocardial infarction is largely determined by the level of compensatory hyperfunction of regions of the hyperfunction of regions of the heart outside the necrotic area. However, the contractile function of undamaged parts of the myocardium after development of a myocardial infarct has received little study. In recent years an attempt has been made by cardiologists to replace determination of hemodynamic indices characterizing the contractile activity of the heart by parameters directly reflecting the contractile function of the myocardium [2-6]. The object of the present investigation was to assess quantitatively the state of the contractile function of undamaged areas of the heart in rabbits at various times after the development of experimental myocardial infarction.

EXPERIMENTAL METHOD

Experiments were carried out on 52 rabbits, 20 of which formed the control group. Ischemic necrosis of the myocardium was produced in the remaining animals by ligation of the descending branch of the left coronary artery, and the animals were investigated 7 days and 1 and 6 months after the operation. To assess the contractile function of the heart, acute experiments were carried out on all the rabbits. Under urethane-chloralose anesthesia the chest was opened, controlled respiration instituted, and the pressure inside the left ventricle and carotid artery was recorded by means of electromanometers. In the course of the experiment the aortic orifice of each animal was completely occluded for 30 sec ten times at intervals of 5 min. During occlusion, the left ventricle contracted with the strongest possible force, and these contractions took place under almost isometric conditions.

Three indices characterizing the force and velocity of myocardial contraction at the 25th second were determined from the curve of left intraventricular pressure recorded during aortic occlusion. The first of these indices was the maximal systolic pressure which the left ventricle could develop under these conditions, reflecting the maximal force of isometric contraction which its muscle as a whole could develop. The second index was the ratio between the maximal pressure developed by the ventricle during occlusion of the aorta and the weight of the ventricle, excluding the weight of the necrotic zone in animals with experimental infarction. This index we describe as the maximal intensity of function of the myocardial structures [1], and it describes the capacity for work of one unit of myocardial tissue. The third index, the index of contractility, describes the velocity of the contractile process under isometric conditions [2]. We calculated the index of contractility by dividing the mean rate of development of isometric contraction by the integrative isometric tension. The mean rate of development of isometric contraction was calculated as the ratio between the pressure developed* and the time measured from the beginning of increase in pressure to its maximum. The integrative isometric tension was calculated as the area bounded by the rising part of the pressure curve, the

*The pressure developed was equal to the difference between the maximal systolic pressure and the pressure at the end of diastole.

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TABLE 1. Dynamics of Indices of Force and Velocity of Myocardial Contraction during Experimental Myocardial Infarction, M ± m

Index	Group of animals	Serial No. of aortic occlusion			
		I	II	V	X
Maximal systolic pressure (in mm Hg)	1 (control)	213 ± 10.0	214 ± 10.9	201 ± 10.8	177 ± 11.7
	2 (infarct; 7 days)	189 ± 11.9	183 ± 10.7	163 ± 10.3	156 ± 15.8
	3 (infarct; 1 month)	199 ± 9.6	173 ± 4.3	159 ± 5.6	145 ± 3.6
	4 (infarct; 6 months)	205 ± 8.9	199 ± 8.9	191 ± 12.2	191 ± 16.5
	Significance of differences	$P_{1-2} = 0.05$	$P_{1-2} < 0.05$	$P_{1-2} < 0.05$	$P_{1-2} > 0.2$
		$P_{1-3} > 0.5$	$P_{1-2} < 0.01$	$P_{1-3} < 0.01$	$P_{1-3} < 0.02$
		P_{1-4}	Not significant		
Maximal intensity of function of structures (in mm Hg)	1 (control	50.8 ± 2.4	53.4 ± 2.4	48.4 ± 2.6	43.4 ± 2.9
	2 (infarct; 7 days)	49.2 ± 3.3	47.5 ± 2.4	41.8 ± 2.6	38.7 ± 2.0
	3 (infarct; 1 month)	48.4 ± 2.3	44.3 ± 1.7	40.4 ± 2.0	37.7 ± 1.6
	4 (infarct; 6 months)	40.4 ± 1.9	39.1 ± 1.6	37.5 ± 2.2	36.6 ± 2.8
	Significance of differences	$P_{1-2} > 0.2$	$P_{1-2} = 0.05$	$P_{1-2} = 0.05$	$P_{1-2} > 0.05$
		$P_{1-3} > 0.2$	$P_{1-3} < 0.01$	$P_{1-3} < 0.02$	$P_{1-3} > 0.2$
		$P_{1-4} < 0.01$	$P_{1-4} < 0.001$	$P_{1-4} < 0.01$	$P_{1-4} > 0.1$
Index of contractility (in sec^{-2})	1 (control)	804 ± 73	626 ± 58	424 ± 47	352 ± 76
	2 (infarct; 7 days)	393 ± 25	369 ± 27	307 ± 35	238 ± 30
	3 (infarct; 1 month)	547 ± 62	460 ± 60	280 ± 46	276 ± 43
	4 (infarct; 6 months)	785 ± 82	638 ± 84	660 ± 76	541 ± 121
	Significance of differences	$P_{1-2} < 0.001$	$P_{1-2} < 0.01$	$P_{1-2} = 0.05$	$P_{1-2} > 0.1$
		$P_{1-3} < 0.02$	$P_{1-3} = 0.05$	$P_{1-3} < 0.05$	$P_{1-3} > 0.2$
		P_{1-4}		Not significant	

perpendicular dropped from the pressure maximum, and the horizontal straight line corresponding to the level of the pressure at the end of diastole. The value thus obtained (in mm^2) was multiplied by a coefficient dependent on sensitivity of the instrument recording the pressure and speed of movement of the paper.

EXPERIMENTAL RESULTS

The dynamics of the indices for the force and velocity of myocardial contraction at different times after development of a focus of ischemic necrosis in the myocardium is shown in Table 1.

Table 1 shows that 7 days and one month after ligation of the coronary artery the maximal pressure in the left ventricle during complete occlusion of the aorta fell every time by 10-20%. Six months after the development of experimental infarction, despite the presence of an aneurysm or an extensive scar in the left ventricle, the maximal pressure developed by the left ventricle during aortic occlusion was considerably greater and not significantly different from normal.

The maximal intensity of function of the structures 7 days and 1 month after creation of the experimental infarct likewise was reduced by 15-20%.

Six months after the creation of the experimental infarct this index had fallen still further and remained 20-25% below normal during all occlusions of the aorta. The relative weight of the left ventricle of these animals was appreciably higher than the relative weight of the heart of control animals of the same body weight kept in the animal house for six months, and varied between 0.0015 and 0.0020, compared with a range of 0.0012 to 0.0013 in control "old" animals. Consequently, moderate hypertrophy of the heart was present in the rabbits with an ischemic focus of necrosis in the myocardium six months after its onset.

The decrease in force of contraction of the undamaged areas of the myocardium was evidently directly connected with changes in their contractility, because the pressure at the end of diastole, which reflects to some extent the degree of initial stretching of the myocardial fibers, was not lower in the rabbits with an ischemic focus of necrosis in the myocardium, but sometimes higher than in intact animals. Under normal conditions the pressure at the end of diastole during aortic occlusion varied from 10 to 45 mm, and in the experimental animals from 20 to 48 mm. Consequently, the decrease in force was not dependent on compensatory reduction in the working load on the heart by the reduced inflow of blood, but was due entirely to changes in the functional state of areas of the myocardium unaffected by necrosis.

The index of contractility 7 days after the onset of experimental infarction during the first five occlusions was 40-50% below normal. During development of fatigue of the heart, the difference between these indices in the control and experimental animals diminished and ceased to be statistically significant. One month after the onset of experimental infarction the index of contractility rose slightly but still remained 25-35% below normal. Six months after the onset of experimental infarction this index no longer differed by a significant margin from normal.

Changes in the index of contractility 7 days and one month after the onset of experimental myocardial infarction were not connected with changes in the cardiac rhythm, which remained within normal limits at all times of the investigation.

Hence, the state of contractile function of the undamaged areas of myocardium 7 days and one month after creation of an ischemic focus of necrosis in the myocardium of the left ventricle was characterized by a decrease in the indices of force and velocity of contraction, the velocity being affected rather more than the force. Six months after the creation of the experimental infarct, when moderate compensatory hypertrophy of the myocardium had developed and the maximal developable pressure and index of contractility had returned to their normal level, the maximal attainable intensity of function by structures of the undamaged areas of the myocardium remained low.

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