DYNAMICS OF STRENGTH OF THE LEFT VENTRICULAR WALL OF RATS IN UNCOMPLICATED EXPERIMENTAL MYOCARDIAL INFARCTION

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KEY WORDS: myocardial infarct; strength of left ventricular wall.

Aneurysms and ruptures of the heart are of the most common causes of death from myocardial infarction (MI) and they are associated not only with disturbances of the hemodynamics, but also with a reduction in the strength of the heart muscle in the zone of infarction [1-3]. However, no systematic study of the dynamics of strength of the heart wall in MI has been undertaken.

The aim of this investigation was to study changes in strength of the left ventricular wall of rats with uncomplicated MI.

METHODS

Four series of experiments were carried out on 131 noninbred middle-aged albino rats, receiving the same care and attention as laboratory animals. In each series from 2 to 10 aniamsl took part for the duration of the experiment. In series I (10 rats) the strength of the heart wall was studied under normal conditions, and the heart was tested immediately after death of the animals. In series II (19 rats) the strength of the heart wall was tested in rats with uncomplicated MI 6, 12, and 24 h and 2, 3, 5, 7, and 15 days after production of the infarct. MI was produced by ligation of the anterior coronary artery. In series III (78 rats) the dynamics of the strength properties of the left ventricular wall was studied during total ischemia, as a model of the ischemic phase of MI. In the first half of the observations in series III the heart was incubated before testing under aseptic condtions in physiological saline at 37°C for 1, 3, 5, and 12 h and 1, 2, 4, and 6 days. In the second half of the observations in series III, heart preparations surviving under sterile conditions in physiological saline, were subjected to further (for the period of survival) to periodic changes of pressure and volume, at the rate of 140 cycles/min, with fluctuations of pressure from 30 to 140 mm Hg. The duration of the experiments was 1-7, 9, 12, and 24 h. In the series IV (24 rays) the strength properties of the left ventricular wall were studied in epxerimental MI. In this series, however, the heart preparations were incubated before testing for 12 h under conditions corresponding to those in the first half of the obsservations in the experiments of series III. The duration of MI in the experiments of series IV was 12, 24, and 38 h and 2, 3, 5, and 7 days. The strength of the left ventricular wall was judged by the normal force (pressure) at which it ruptures. To measure the destructive forces a rubber balloon connected by a system of tubes to a source of positive pressure, a manometer, and a reservoir for physiological saline, was introduced into the chamber of the ventricle. The solution was injected into the ventricle at the rate of 0.1 ml/sec until its wall ruptured. The value of the destructive force was recorded at the moment of rupture. After the end of the experiments the place and size of the zone of rupture were investigated, and the internal volume and thicknes s of the walls of the ventricle were measured. The accuracy of measurement of the destructive force was $2.5 \cdot 10^3$ Pa, the internal volume of the ventricle was 0.5 and 10^{-2} ml, and the thickness of the ventricular wall was 0.01 mm. The data were subjected to statistical analysis with observance of the requirements for physical experiments.

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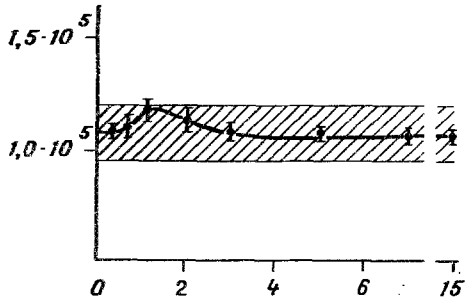

Fig. 1

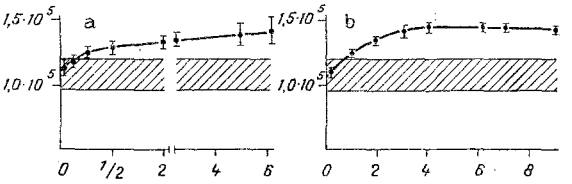

Fig. 2

Fig. 1. Dependence of destructive force (at which rupture of the ventricular wall takes place) on duration of uncomplicated experimental MI. Abscissa, duration of experiment (in days); ordinate, destructive force (in Pa).

Fig. 2. Strength of left ventricular wall in total ischemia, in the absence (a) and presence (b) of periodic changes of pressure and volume. Abscissa, duration of experiment (a, in days, b, in h). Remainder of legend as to Fig. 1.

RESULTS

The left ventricular wall of the normal rat heart has a high reserve of strength. Rupture of the wall required a force of $(1.1 \pm 0.06) \cdot 10^5$ Pa, more than twice the maximal possible force exerted during the animal's life. The results of the tests do not correlate with the internal volume of the ventricle and the thickness of its wall. Ruptures were observed more often at its boundary with the ventricular septum or along the edges of the papillary mucles, as a result of which these areas of the heart wall must be regarded as "natural" concentrators of tension.

The strength of the left ventricular wall in rats with uncomplicated MI corresponded to its normal values, and showed no tendency to decrease at any stage of the experiments (Fig. 1). These results are in agreement with data in the literature [6]. The authors cited, who used a similar technique studied the breaking strength of the left ventricular wall at various stages during the first 8 days of expermental MI, likewise found no decrease. The present writers showed previously [4] that the strength of the left ventricular wall in patients dying in the acute period of MI often varies within normal limits.

The fact that the strength of the left ventricular wall is preserved in rats with uncomplicated MI in the necrotic phase can be explained in two ways: by an increase in its strength in the ischemic phase and by the prior (relative to necrosis) onset of repair processes.

Evidence of an increase in strength of the heart wall in the zone of infarction in the ischemic phase is given by the results of experiments on totally ischemic heart preparations. In the experiments of series III an increase in strength of left ventricular wall was recorded; in the case of periodic mechanical work, moreover, simulating the biomechanics of the heart, the rate of this increase was significantly greater (Fig. 2).

According to data in the literature [3], in experimental animals with uncomplicated MI repair processes in the zone of infarction for a large proportion of the necrotic phase are accompanied by necrotic changes, which begin as early as on the first day of the experiments.

The importance of synchronization of necrotic and reparative processes of maintaining the strength of the heart wall in the zone of infarction is demonstrated by the results of the experiments of series IV. Delays (by 12 h) tests on heart preparations revealed a decrease of strength in the first 8-36 h of the experiment (Fig. 3), whereas in the earlier and later stages strength was within normal limits. In these experiments the conditions provided allowed necrosis to take place, whereas repair processes were completely blocked. Destruction of necrotic tissues in the zone of infarction took place on a considerable scale through secretion of hydrolytic, proteolytic, and lipolytic enzymes of polymorphonuclear leukocytes, a pool of which was formed in the zone during the first 2 days of MI. In the latter stages reparative processes predominated over necrotic, and the strength of the ventricular wall was not reduced.

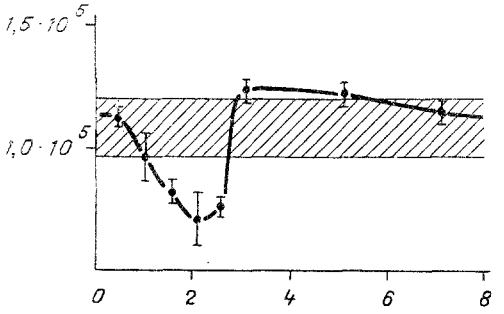

Fig. 3. Strength of left ventricular wall at different times of experimental MI during successive tests. Legend as to Fig. 1.

Under normal conditions the rat heart is thus characterized by high reserves of strength, and in uncomplicated MI, even in the stage of marked necrotic changes in the myocardium in the zone of infarction, strength is not reduced. The mechanisms whereby this strength is maintained include an increase in the ischemic phase and an early onset of repair, following necrotic changes.

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PREVENTION OF ISCHEMIC AND REOXYGENATION ARRHYTHMIAS AND VENTRICULAR FIBRILLATION WITH THE ANTIOXIDANT IONOL

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KEY WORDS: ventricular fibrillation; transient myocardial ischemia; reperfusion of the myocardium; antioxidants.

Reoxygenation after hypoxia or ischemia of the myocardium leads to activation of lipid peroxidation (LPO) [2, 6], which plays an important role in reoxygenation injury to membranes of the myocardium [2, 5] and disturbance of its contractile function [2, 3, 6]. These phenomena can be prevented by antioxidants [1, 2, 5, 13]. Recent investigations have shown that induction of LPO in the myocardium regularly leads to bradyarrhythmia and cardiac arrest, which can also be prevented by antioxidants [4]. These facts are important because relatively transient ischemia followed by reperfusion (RP) and reoxygenation constitute an unavoidable stage of the process in any coronary attack, in the course of which reduction or cessation of the coronary blood flow is followed by reactive hyperemia, adenosine-induced in its origin [7]. Under these circumstances cardiac arrhythmias and fibrillation may develop [9, 12]. The role of activation of LPO in the genesis of these arrhythmias is highly prob-

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