CHANGES IN CONTRACTILE ACTIVITY OF THE RABBIT MYOCARDIUM IN BURN SHOCK

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In vitro experiments on the papillary muscles of the rabbit heart showed that 1 h after burn trauma myocardial contractility is disturbed in 50% of cases. It is concluded that this may be one cause of the severe disturbance of cardiac output observed in the same experiments.

KEY WORDS: hemodynamics; contraction of myocardium; burn shock.

Clinical and experimental burn shock is accompanied by a sharp decrease in cardiac output [2-9, 13, 19]. One cause of this decrease is considered to be the development of insufficiency of the heart muscle [7, 8, 11, 17, 21]. It is very difficult to obtain direct proof of changes in the contractility of the heart in response to burn trauma in the intact organism when the hemodynamics is disturbed, and conclusions in the literature on this problem are based on indirect evidence [1, 8, 11, 17]. Some workers [10, 12] have found myocardial depressants in the blood serum of animals and persons afflicted with burns.

To obtain direct data on the effect of thermal trauma on the myocardium the contractile activity of isolated papillary muscles obtained from the heart of animals in a state of burn shock was studied.

EXPERIMENTAL METHOD

Experiments were carried out on 19 rabbits anesthetized intravenously with urethane (1 g/kg body weight). The control group consisted of nine animals. Burn trauma affecting 30-35% of the body surface was inflicted on 10 rabbits [4]. In all the animals the minute volume of the heart (MV), arterial blood pressure (BP), central venous pressure (CVP), and total oxygen consumption (TOC) were measured at intervals. The total peripheral vascular resistance (TPVR) was calculated. After measurement of the initial parameters in the control animals and after burn injury to the experimental animals dextran (2% of body weight) was injected. MV was measured by the thermodilution method; the cold Ringer's solution was injected into the right atrium and changes in the blood temperature were recorded by means of a thermistor introduced into the arch of the aorta. To measure TOC continuously a closed system was used, with absorption of CO_2 and automatic supply of O_2. The CVP was measured in the right atrium with a water manometer. The thorax of the animals was quickly opened 1 h after burning and the heart was removed and placed in oxygenated Tyrode solution. After not more than 15 min a papillary muscle was isolated from the right ventricle and placed in a perfusion chamber through which flowed Tyrode solution saturated with carbogen (95% O_2 + 5% CO_2) and heated to 35-36°C. In the course of 1 h before the investigation began the papillary muscle was stimulated at a frequency of 1 Hz. Stimulation was carried out with two silver electrodes placed at the edges of the preparation. The muscle was stimulated with a continuous series of pulses with frequency varying within the range from 0.1 to 5 Hz. Above-threshold stimuli 5-10 msec in duration were used. Contractions were recorded by means of a 6MKhIS mechanotron on a Mingograph-81 recorder

EXPERIMENTAL RESULTS

Before removal of the heart, MV for the rabbits of the control group was 243 ± 25.7 ml·min/kg, BP was 76 ± 4.4 mm Hg, TPVR was 10.0×10^3 dynes·sec·cm^{-5}, CVP varied from −1 to 48 mm Hg, and TOC was 19.7 ± 0.77 ml/min.

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TABLE 1. Changes in Parameters of Hemodynamics and Total Oxygen Consumption in Rabbits in Response to Burn Trauma

	Time after burning, min	MV	BP	TPVR	TOC
Initial data		237±18,9 ml/min/kg	80±3,8 mm Hg	11.4·10³±1· 10^3 dyn·sec·cm^{-5}	20,9±1,4 ml/min
After burns	1—3	62±3,4%	88±6,7%	121±16,0%	85±3,1%
	30	28±2,5%	50±4,0%	180±27,6%	70±3,8%
After injection of dextran	1—3	139±16,5%	93±3,5%	73±6,6%	96±2,4%
	30	76±13,4%	94±2,9%	132±17,6%	86±4,6%

Legend. Initial values taken as 100%.

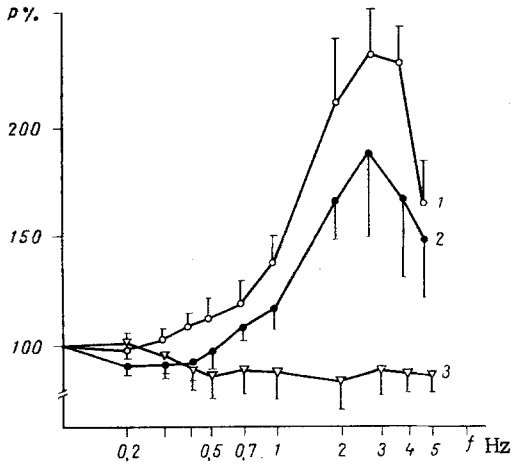

Fig. 1. Effect of burn trauma on character of frequency—power curves recorded in papillary muscles of rabbit heart. 1) Control (mean of nine experiments), 2 and 3) 1 h after burns (mean of five experiments). Abscissa, frequency of stimulation of preparations (in Hz); ordinate, power of contraction (in %); power of contractions during stimulation of preparations at frequency of 0.1 Hz taken as 100%).

Changes in parameters of the hemodynamics and TOC observed in the rabbits in response to burn trauma are shown in Table 1. Burns caused a rapid and very sudden fall of cardiac output. The mean value of MV 30 min after trauma was 28 ± 2.5% of the initial level. The TPVR at this time was doubled, TOC was reduced by 30% of the initial level, and CVP changes in either direction. These results point to an extremely severe disturbance of the circulation. Injection of dextran in a dose of 2% of the body weight (about 30% of the blood volume) restored the normal hemodynamic parameters within a short time. After the end of dextran administration a progressive decrease in MV was resumed and corresponding changes were observed in the other parameters.

The contractile activity of the papillary muscles of the right ventricle during rhythmic stimulation in animals of the control group was characteristic of the normal myocardium of warm-blooded animals [14, 15, 20]. The graph in Fig. 1 shows how the amplitude of isometric contractions depended on the frequency of stimulation of the preparations (frequency—power or f—p curves). In the animals of the control group (curves 1) a sharp increase in amplitude of the contractions was observed within the frequency range from 0.5 to 3 Hz (the amplitude in response to stimulation at 0.1 Hz was taken as 100%). During stimulation with a frequency of 4-5 Hz the amplitude of contractions fell.

In the group of burned animals (10 rabbits) two types of preparations were distinguished. In five preparations the f—p relationships (curve 2) were similar to those observed in the

animals of the control group. In five other preparations the f–p curve (curve 3) was monophasic in character [14, 16, 18]. In these preparations no increase in amplitude of the contractions took place with an increase in the frequency of stimulation but, on the contrary, the amplitude fell.

Frequency–power curves recorded under steady-state conditions, as has been shown [14, 16], may be an indicator of myocardial function. The normal myocardium of warm-blooded animals has frequency–power curves of a triphasic character [14, 15, 20]. In response to repetitive stimulation of preparations of injured or incompetent myocardium, monophasic f–p curves are formed [14]. In the present experiments, in 50% of papillary muscles removed from the rabbit heart 1 h after infliction of burn trauma on the animals monophasic f–p curves were recorded. In accordance with data in the literature, this type of curve points to the development of myocardial insufficiency. Burn trauma caused sharp changes in the hemodynamics and, in particular, a decrease in cardiac output. One cause of this could be myocardial insufficiency. The fact established in this investigation, namely that myocardial insufficiency may arise even in isolated fragments of heart muscle, is evidence of changes in its contractile function in burn shock.

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