CHANGES IN PARAMETERS OF THE PUMPING FUNCTION OF THE HEART AND MYOCARDIAL CONTRACTILITY IN EXPERIMENTAL CARDIAC TAMPONADE

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KEY WORDS: cardiac tamponade; myocardial contractility; pumping function of the heart.

In cardiac tamponade the presystolic length of the heart muscle is reduced, and this narrows the limits of function of the fundamental mechanism of regulation of cardiac activity — the Frank—Starling mechanism [6-8]. Under these conditions it is interesting to study changes in the parameters of myocardial contractility, for potentiation of the action of the sympathico-adrenal system on the myocardium may be one of the basic mechanisms of compensation of hemodynamic disturbances in tamponade. Data in the literature on the character of changes in the indices of myocardial contractility in cardiac tamponade are few in number and highly contradictory in nature [4, 5, 12].

The object of this investigation was to compare changes in the parameters of the pumping function of the hear with changes in the indices of myocardial contractility, determined in the phase of isometric contraction of the ventricles, during the development of cardiac tamponade.

EXPERIMENTAL METHODS

Experiments were carried out on mongrel dogs weighing 14-25 kg anesthetized with chloralose and urethane. A polyethylene catheter was introduced into the pericardial cavity 6-7 days before the experiment. During the experiment the pressure in the aorta and left ventricle was recorded electromanometrically by means of catheters introduced through the right brachial and carotid arteries. The natural frequency of the catheter-manometer system was not less than 40 Hz. The first derivative of pressure in the left ventricle was obtained by means of a differentiator with linear output up to 75 Hz. To estimate myocardial contractility, besides dp/dt_{max} the indices of contractility calculated during the isovolemic phase of systole also were used: the index $[dP/dt_{max}]/Pd$ [11], the modified index $[dP/dt_{max}]/IIP$ [9], and V_{max} [10]. The indices of contractility were calculated on the M-4030 computer and by means of the specialized Indeks computer system, yielding continuous information about changes in the contractility indices immediately, in the course of the experiment. Technical details of the method of evaluating myocardial contractility and determining the parameters of the pumping function of the heart (cardiac output, end-diastolic volume of the left ventricle) were described previously [1-3]. Cardiac tamponade was created by slow injection (50 ml in the course of 20-30 sec) of isotonic sodium chloride solution, heated to 38°C, into the pericardial cavity. Three stages of cardiac tamponade were modeled, with a mean pressure inside the pericardial cavity of 3, 6, and 9 mm, by stabilization of the intrapericardial pressure at an assigned level (for 10 min). The duration of each stage did not exceed 30 min. To record the parameters of the cardiohemodynamics of myocardial contractility, the Mingograph-34 and the ÉK 6T-01 electrocardiograph were used.

EXPERIMENTAL RESULTS

The period of transitional changes of the hemodynamics arising as the pressure in the pericardial cavity increased was characterized by a transient fall in the mean aortic pressure and systolic pressure in the left ventricle, the appearance or enhancement of the respiratory wave of aortic pressure, by a gradual increase in the end-diastolic pressure in the left ven-

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TABLE 1. Changes in Parameters of Cardiodynamics and Myocardial Contractility in Experimental Cardiac Tamponade (M ± m)

Parameter studied	Initial state	Stage of cardiac tamponade		
		I	II	III
Cardiac output, ml/min	1719±154	1544±151	1342±98	1149±105
End-diastolic volume of left ventricle, ml	52±3	46±3	34±2	26±4
End-systolic volume of left ventricle, ml	40±3	37±2	28±2	21±3
Heart rate, beats/min	142±9	157±9	211±9	249±13
End-diastolic pressure in left ventricle, mm Hg	4,1±0,4	5,8±3,8	9,2±0,7	10,2±0,9
Mean aortic pressure, mm Hg	126±3	122±3	120±4*	100±5
$\frac{dP/dt_{max}}{Pd}$, sec^{-1}	45±5	50±5*	54±5	52±3
$\frac{dP/dt_{max}}{IIP}$, sec^{-2}	3414±678	4109±806*	4739±872	4048±507
Vmax, sec^{-1}	4,9±0,4	5,0±0,4	6,0±0,7	5,1±0,5
dP/dt_{max}, mm Hg/sec	2966±288	3422±386**	3560±54**	3169±343*

Legend. Criterion of significance of difference from initial level (P) determined by difference method; no asterisk $P < 0.05$; one asterisk $P > 0.1$; two asterisks $0.1 > P > 0.05$.

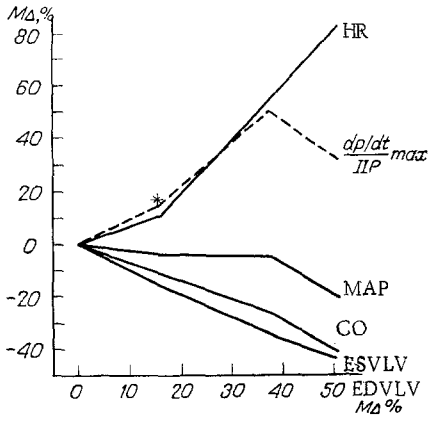

Fig. 1. Changes in end-diastolic volume of left ventricle and in parameters of cardiodynamics and myocardial contractility during cardiac tamponade (three stages of tamponade respectively). MΔ, %) Arithmetic mean of changes in parameter expressed in % of initial values; HR) heart rate; MAP) mean aortic pressure; CO) cardiac output; ESVLV) end-systolic volume of left ventrile; EDVLV) end-diastolic volume of left ventricle. Asterisk indicates differences from initial level not significant, by difference method (P > 0,05).

tricle, and by an increase in the heart rate. The indices of myocardial contractility usually increased appreciably toward the end of injection of fluid into the pericardial cavity (especially when stage II of cardiac tamponade was modeled), but during the next 5-10 min they fell a little, although they still remained above their initial level. During modeling stages I and III of tamponade the indices of myocardial contractility could fall, especially when a marked slowing of the rhythm developed. Rapid withdrawal of fluid from the pericardium caused the cardiac activity to recover. Changes in the parameters of the circulation and myocardial contractility as the pressure in the pericardial cavity fell slowly were the opposite of those observed when the intrapericardial pressure rose.

Characteristics of periods of stable measurements of the hemodynamics established while the pressure was maintained at an assigned level in the pericardial cavity for 5-10 min, are given in Table 1. With a fall in the end-diastolic volume of the left ventricle on average by 16, 30, and 50% of the initial level, to correspond to stages I, II, and III of cardiac tamponade, the mean values of the cardiac output fell proportionally. Only in four of the 13 experiments did the cardiac output remain close to its initial level in stages I and II of cardiac tamponade. The indices of contractility in these cases were appreciably increased.

The mean aortic pressure was practically unchanged in the first two stages of cardiac tamponade and was 20% below the initial level in stage III of tamponade (Fig. 1).

The decrease in the end-systolic volume of the left ventricle (by 16, 33, and 44% of the initial level) did not enable maintenance of the ejection fraction, which was reduced on average by 10, 25, and 35% of the initial level; in conjunction with limitation of diastolic filling of the left ventricle this led to a marked decrease in the stroke volume of blood (on average by 25, 50, and 65% of the initial value).

A significant and considerable increase in the indices $[dP/dt_{max}/]Pd$ and $[dP/dt_{max}]/IIP$ was observed in stage II (by 21 ± 7 and 50 ± 18% respectively) and stage III of cardiac tamponade. The increase in V_{max} and dp/dt_{max} was less substantial.

The considerable increase in the indices of myocardial contractility, combined with a decrease in the presystolic length of the heart muscle and some decrease in the mean aortic pressure are without doubt manifestations of increased myocardial contractility due to strengthening of the positive inotropic factors acting on the myocardium during cardiac tamponade.

The opposite changes in heart rate and in the indices of myocardial contractility, both in individual experiments and also on averaging of the data (during the transition from stage II to stage III of tamponade) are evidence that an increase in myocardial contractility aimed at compensating disturbances of the pumping function of the heart in tamponade takes place as a result, not of rhythm-dependent inotropic influences but on direct inotropic influences on the heart, evidently of adrenergic nature.

The results thus indicate that an essential role in the adaptive reactions of the body to limitation of realization of the length–tension relationship in cardiac muscle (the Frank–Starling mechanism) is played by an increase in myocardial contractility which, however, cannot ensure sufficiently effective maintenance of the cardiac output.

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