PHASE STRUCTURE OF THE CARDIAC CYCLE IN THE COURSE OF ANAPHYLACTIC SHOCK

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Experiments on dogs showed that anaphylactic shock (injection of a reacting dose of horse serum) is followed, both in the first few minutes and, in particular, in the stage of persistent hypotension, by shortening of the cardiac cycle, lengthening of the total period of contraction on account of the phase of isometric contraction, and shortening of the ejection period. In the experimental animals the index of myocardial tension also was reduced.

Numerous experimental [6-8] and clinical [4, 5, 10, 11] investigations have demonstrated considerable electrocardiographic changes and disturbances of the microcirculation in the heart [9] during the development of anaphylactic shock.

The object of this investigation was to study changes in the phase structure of the cardiac cycle and contractile power of the myocardium in the course of anaphylactic shock.

EXPERIMENTAL METHOD

Experiments were carried out on 10 mongrel dogs weighing 10-15 kg and previously sensitized with normal horse serum. The phase structure of the cardiac cycle was analyzed by a polycardiographic method with simultaneous recording of the ECG, phonocardiogram, sphygmogram of the carotid artery, and kineto-cardiogram in position 4 on the NEK-6 electrocardiograph. These parameters were recorded at the time of stopping the artificial respiration apparatus for 10-15 sec in the initial state, during the first minutes of shock, and 15-20 min after injection of the reacting dose of serum, i.e., in the phase of persistent hypotension.

A dynamic transducer, developed and tested in Professor L. B. Andreev's Laboratory [1-3], was used for kinetocardiography.

EXPERIMENTAL RESULTS AND DISCUSSION

Analysis of the results (Table 1) showed that in the initial phase of development of anaphylactic shock tachycardia was accompanied by lengthening of the total period of contraction on account of the phase of isometric contraction. The ejection period was reduced by about half. Mechanical and total systole were shortened. Diastole was reduced on account of a shortening of all phases of the filling period. Meanwhile the period of relaxation was virtually unchanged.

After 15-20 min, in the phase of established persistent hypotension, the cardiac cycle was considerably shortened. The period of total contraction was lengthened on account of the phase of isometric contraction, but the phase of asynchronous contraction was reduced. The ejection period continued shortened. Mechanical and total systole were reduced. Diastole was reduced through shortening of the filling period.

The results thus indicate considerable changes in certain phases of the cardiac cycle, more marked in the period of persistent hypotension.

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TABLE 1. Phase Analysis of Cardiac Cycle of Left Ventricle

	Init backg	ial round	First few minutes of shock			15-20 min of shock		
Parameters	М	± m	М	±m	P	М	±m	P
Pulse rate (beats/min) Cardiac cycle	110	28,7	183	41,1	0,001	199	36,4	0,001
(in sec)	0,45	0,14	0,353	0,097	0,1-0,05	0,313	0,057	0,02-0,01
Electrical systole (in sec)	0,209	0,047	0,191	0,019	0,25	0,191	0,018	0,5
Period of total con- traction (in sec)	0,077	0,02	0,104	0,02	0,01-0,002	0,093	0,007	0,05—0,02
Asynchronous con- traction (in sec)	0,038	0,009	0,044	0,017	0,25-0,1	0,031	0,006	0,002
Isometric contrac-								
tion (in sec) Ejection period (in sec)	0,038	0,014	0,06	0,014	0,001	0,063	0,01	0,001
Mechanical systole		,	.,	-,.	,		-,-	,,,,
(in sec)	0,144	0,026	0,109	0,016	0,01-0,002	0,108	0,023	0,002—0,001
Total systole (in sec)	0,183	0,032	0,154	0,023	0,05-0,02	0,139	0,024	0,010,002
Diastole (in sec)	0,241	0,01	0,158	0,045	0,05-0,02	0,162	0,039	0,05
Period of relaxation (in sec)	0,063	0,02	0.061	0.024	0,5-0,1	0,051	0,014	0,25—0.1
Period of filling (in sec)	0,187	0,087	0,097	0,028	0,01-0,002	0,109	0,039	0,05-0,02
Intrasystolic index (in percent)	73,6		49,1			42,6		
Index of myocar - dial tension	41,5		66,8			66,8		

The changes discovered in the cardiac cycle of the left ventricle correlated with disturbances of the microcirculation in the heart. Whereas during the first few minutes of shock the circulation was considerably limited in the endocardial and intramyocardial layers of the heart, in the stage of established persistent hypotension the blood flow was also limited in the epimyocardial layer.

Disturbances of the microcirculation in the heart and the resulting hypoxia adversely affect both electrical activity of the heart and the contractile power of the myocardium.

In anaphylactic shock the total period of contraction was in fact lengthened and the period of ejection shortened. Considerable changes occurred both in the intrasystolic index and the index of myocardial tension, indicating the development of a syndrome of cardiac hypodynamia. These disturbances evidently play an important role in the mechanism of formation of anaphylactic shock.

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