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PATHOGENESIS OF ACUTE CARDIAC FAILURE IN CLOSED CHEST INJURIES

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KEY WORDS: acute cardiac failure; closed chest injury; arterial hypoxemia.

The pathophysiology of disturbances of the cardiodynamics in closed chest injuries has been inadequately studied. The few data in the literature on the work of the heart under these conditions are contradictory in character: Both an increase [2, 7] and a decrease in cardiac output in the immediate post-traumatic period [3, 4, 9] have been reported. The undertaking of such investigations under clinical conditions, of course, may be very difficult because of the severity of the patient's state and the fact that he is subject to a whole group of unfavorable factors, capable of influencing cardiac function, all at the same time.

It was accordingly decided to undertake an experimental study in which, in order to examine the possibility of onset of acute cardiac failure (ACF) in closed chest injuries, and its pathogenesis, the cardiodynamics was investigated in animals exposed to single standard artificially produced components of thoracic trauma: partial blood loss, contusion of the lungs, disturbance of the integrity of the thoracic cage, and a combination of all three.

EXPERIMENTAL METHOD

Experiments were carried out on 54 dogs weighing 8-23 kg in which a "floating" microcatheter was introduced into the right ventricle through the jugular vein and a special thermistor probe into the arch of the aorta through the carotid artery. It was thereby possible to record the maximal systolic pressure (SP) and the end-diastolic pressure (EDP) in the right ventricle, the pulse rate (PR), the cardiac output and stroke volume (CO and SV, respectively) by the thermodilution method, and also to take samples of arterial blood for measurement of the partial pressure of oxygen (p_aO_2) and the acidity (pH), on an Astrup gas microanalyzer, at intervals over a period of 7 days.

In five series of experiments the injury factors (IF) were external blood loss in a volume of 20 ml/kg body weight (IF_1, eight experiments), displacement of a fragment of the thoracic cage prepared previously (IF_2, 14 experiments), contusion of the lung tissue (IF_3, 12 experiments), a combination of blood loss with displacement of the fragment of the thoracic cage (IF_4, 10 experiments), and a combination of blood loss with contusion of the lung (IF_5, 10 experiments). During exposure to IF the animals were anesthetized superficially with hexobarbital (0.02-0.03 g/kg). The various indices were recorded in the initial state and subsequently at intervals: every hour for the first 4 h after the beginning of IF and once daily thereafter. The results were subjected to statistical analysis by Student's method.

EXPERIMENTAL RESULTS

The results are given in Table 1. Indices obtained in animals withstanding the effects of IF favorably are classed in group A, those for animals dying in the course of the experi-

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TABLE 1. Indices of Cardiodynamics and Arterial Blood Gases in Experiments Reproducing Traumatic Factors of Closed Chest Injury

Group of animals	Number of animals	Phase	SP, mm Hg		EDP, mm Hg		SV, ml/min·kg	
			I	II	I	II	I	II
	54	Initial value	21,5±0,03		0,82±0,4		1,12±0,09	
A	7	IF_1	15,6±1,25*	21,5±2,86	0,46±0,32	0,3±0,27	0,67±0,06*	0,91±0,05
	6	IF_2	21,5±1,74	22,4±2,06	2,5±1,36	1,2±0,54	0,81±0,06*	0,93±0,05
	9	IF_3	22,2±1,16	23,4±0,66	5,4±0,86*	1,24±0,53	0,83±0,09*	0,92±0,07
	5	IF_4	19,6±0,51*	20,5±1,91	1,4±0,82	1,4±0,82	0,71±0,04*	1,12±0,15
	5	IF_5	19,4±2,09	19,6±4,09	2,73±0,47*	1,6±0,53	0,91±0,14	1,27±0,18
B	7	IF_2	24,6±1,87	16,2±1,25*	2,8±0,88	8,4±1,16*	0,82±0,08*	0,66±0,05*
	5	IF_3	16,3±4,67	17,6±2,47	4,2±0,8*	7,5±0,50*	0,97±0,15	0,88±0,15
	5	IF_4	15,7±2,31*	11,1±6,33*	1,7±1,04	2,45±0,67*	0,92±0,12	0,54±0,10*
	5	IF_5	15,5±3,43*	10,5±1,15*	0,3±0,28	1,4±0,98	0,91±0,10	0,42±0,06*

(continuation)

PR, beats/min		CO, ml/min·kg		pH		P_aO_2, mm Hg	
I	II	I	II	I	II	I	II
123±9,22		138±12,07		7,41±0,01		84±1,90	
141±7,27	161±14,05	95±7,14*	147±20,8	7,34±0,01*	7,36±0,01*	80±1,9	82±3,16
162±10,3	133±10,45	124±9,18	137±27	7,40±0,03	7,37±0,03	80±3,27	81±1,54
147±6,06	142±4,2	124±13,33	143±13,03	7,39±0,01	7,40±0,08	78±1,92	81±2,21
196±21,5*	159±11,09	132±8,34	184±14,36*	7,35±0,04	7,36±0,03	80±2,92	85±1,72
165±16,7	132±9,18	146±10,45	162±17,95	7,34±0,04	7,39±0,01	71±4,25*	78±2,6
162±15,32	163±13,95	129±7,03	108±11,23	7,38±0,02	7,38±0,09	80±5,56	54±1,97*
172±23,49	144±24,79	154±30	120±2,71	7,37±0,03	7,40±0,02	76±0,6*	56±9,79*
157±11,3	160±15,1	141±20,6	92±26,36	7,31±0,02*	7,24±0,03*	71±3,83*	86±7,53
147±6,52	145±31,4	136±15,31	63±12,81*	7,34±0,02*	7,12±0,01*	77±3,6	59±7,42*

*Values differ statistically significantly from initial values ($P < 0.05$).

ment in group B. Each index was studied in the initial phase, i.e., during the first few hours of observation (I), and in the final phase, near the time of the animal's death or of stabilization of its state (II). It will be clear from Table 1 that under IF_1 conditions there was a very small decrease in all indices (except PR) in phase I, followed by their recovery in phase II. No increase in EDP was observed. All indices returned completely to normal inthe course of 24 h. All the animals survived.

The action of IF caused a uniform cardiodynamic response in the animals of group B, expressed as a tendency for EDP to rise and for SP, SV, and CO to fall. In phase I of the process, because of an increase in PR, the value of CO remained at its initial level. In phase II a further decrease in SV was not accompanied by a corresponding increase in PR, and for that reason a decrease was observed in CO which was greatest under IF_4 and IF_5 conditions, when the values of SV were significantly below the initial levels.

The above changes in indices of the cardiodynamics were accompanied by arterial hypoxemia, which increased and reached maximal severity under IF_2 and IF_3 conditions. In the experiments with IF_4 and IF_5 acidosis increased, whereas p_aO_2 in phase II showed a tendency to return toward normal in some experiments.

For animals of group A under experimental conditions IF_2-IF_5, against the background of a definite tendency for SP in phase I to remain unchanged, some decrease in SV and an increase in PR were characteristically observed, but in phase II there was a tendency for these indices to return to normal. The course of EDP revealed a tendency to rise in phase I and to return to normal in phase II. These changes were accompanied by a decrease in p_aO_2 and pH in phase I, after which these indices also tended to return to normal.

Investigation of the circulating blood volume showed that even in experiments with a hemorrhagic component the deficit of CBV did not exceed 25% of its initial value.

Analysis of the results indicates that injuries to the thoracic cage and lung tissue, complicated or uncomplicated by partial blood loss, can cause disturbances of the cardiodynamics.

Both the severity and the outcome of the pathological processes under conditions of these standard experimental models were evidently determined largely by integrity of the protective-adaptive mechanisms of the body as a whole and of the heart in particular. So far as the heart is concerned, these protective mechanisms are realized through an increase in the force (SP) and frequency (PR) of the contractions in response to IF. Because of the integrity of these responses the animals in group A were able to maintain the necessary level of the volume blood flow.

In the animals of group B the functional reserves and intensity of the compensatory reactions of the heart could not ensure adequate protection against the effects of IF. Even in phase I in most experiments a marked decrease in the functions of contraction and relaxation of the myocardium was noted. The results suggest that under these conditions maintenance of the cardiac output even at close to the initial level threatened the collapse of its compensatory mechanisms. This was clearly demonstrated in phase II, when a further increase in EDP and also a decrease in SP and SV were observed. The PR level in this period evidently did not make good the deficiency of the stroke volume. This is particularly characteristic of IF_4 and IF_5 conditions. Depression of the functional capacity of the myocardium in these series of experiments could be deduced, in particular, from the tendency of EDP to rise, which was not found under IF_1 conditions, when the volume of the blood loss was similar.

On the whole, the disturbances of the cardiodynamics in the animals of group B can be interpreted as ACF. Its manifestations were largely universal for all series of experiments. Besides a decrease in SV, another characteristic feature of ACF was a definite order of increasing severity of the signs (an increase in EDP — a fall in SP — a fall in EDP), in agreement with existing views on the stages of formation of myocardial depression during exposure to a high load [6].

Analysis of the results showed that the increase in EDP in some experiments preceded a fall in cardiac output; this suggests that disturbance of diastole can be regarded as a trigger stage in the formation of ACF.

Comparison of the results reveals the pathogenetic dependence of changes in the cardiodynamics on the blood gas composition. For instance, the marked inverse relationship between EDP and p_aO_2 will be noted. From this point of view the results agree with data in the literature on the role of hypoxemia in the development of ACF [6, 10, 11]. Probably arterial hypoxemia may also be a trigger factor in the development of ACF, and as the intensity of the latter increases it may play an ever-increasing role in its development. On the other hand, when ACF has already developed, disturbances of the gas exchange in the lungs may arise through a decrease in the pulmonary blood flow, leading to an even greater fall in p_aO_2 [1, 5]. If the condition runs an unfavorable course conditions could thus be created under which pulmonary and cardiac insufficiencies would aggravate one another. Furthermore, acidosis, which was most marked in IF_4 and IF_5, evidently also played an important role in the pathogenesis of ACF. The possibility of this type of relationship has been demonstrated by a number of experimental and clinical studies [5, 8, 12].

It can thus be concluded that a deep or lasting decrease in pH and p_aO_2 depressed the adaptive capacity of the heart during exposure to the various components of severe thoracic trauma and, in some cases, determined the development of acute cardiac failure under these conditions.

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RESPONSE OF THE HEART TO FUNCTIONAL LOADS IN CHEMICALLY DESYMPATHIZED ANIMALS

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Information on the regulatory and adaptive-trophic functions of the sympathetic nervous system has been obtained by recent investigations involving the use of immunochemical and chemical desympathization methods [3, 4, 11]. The question of the state of myocardial contractile function and the powers of adaptation of the heart of chemically desympathized animals under conditions of increased loads has so far received little study, despite its importance if the role of the sympathicoadrenal system in cardiopathology is taken into account.

In the investigation described below the reaction of the heart of chemically desympathized animals to functional loads was studied.

EXPERIMENTAL METHOD

Experiments were carried out on 22 chemically desympathized and 20 control male Wistar rats weighing 135-175 g. The substance Isobarin, from Pliva (Yugoslavia) [2, 12] was used to produce desympathization. The animals were aged 2 months. Functional loads consisted of imposing on the heart increasing frequencies of contraction [8], and a maximal resistance load was created by compressing the aorta for 10 sec. Parameters of the contractile function of the heart were recorded after 2, 5, and 10 sec. The systolic pressure (SP) was measured in the left ventricle, the maximal rate of its development and fall were determined, and the end-diastolic pressure (EDP) recorded. The intensity of contractile function (ICF) [6], the contractility index [16], and the relative duration of the diastolic pause (as a percentage of the total duration of the cycle) were calculated. Acute experiments were carried out under urethane anesthesia (160 mg/100 g) under open chest and artificial ventilation conditions. The pressure in the left ventricle was recorded by means of a catheter inserted through the apex of the heart and connected to the probe of a Mingograph-34 electromanometer (Elema, Sweden). The ECG was recorded simultaneously with the first derivative of pressure by means of a DE-1 differentiator. The results were subjected to statistical analysis by Student's method.

EXPERIMENTAL RESULTS

The initial indices of contractile function of the heart — the force of contraction of the muscle of the left ventricle, the rate of development and fall of pressure, and ICF of the myocardium — were lower in chemically desympathized rats than in control animals with the same cardiac frequency (Table 1). Imposing an increasing frequency of contractions on the heart gave rise to pulsus alternans in a high proportion (41.7%) of desympathized animals at a frequency of stimulation (320-330 beats/min) at which this phenomenon was not observed in the control animals. The control animals developed pulsus alternans in some cases at frequencies exceeding 480 beats/min. An increase in the cardiac frequency to 420-450 beats/min caused an increase in ICF by 13-16% in the control rats. In desympathized animals, under these

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