ROLE OF HEMORHEOLOGIC DISORDERS IN DISTURBANCE OF THE CENTRAL HEMODYNAMICS FOLLOWING EXPERIMENTAL ACUTE OCCLUSION OF THE TERMINAL PORTION OF THE AORTA

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Despite considerable advances in the treatment of acute occlusions of the bifurcation of the aorta and the main arteries of the limbs [2, 3, 7], mortality from this disease still remains high. The main cause of this mortality is cardiovascular failure, and an important role in its genesis is ascribed to hemorheologic disorders [4, 5].

The object of the present investigation was accordingly to study the effect of changes in blood rheology on the state of the central hemodynamics following experimental acute occlusion of the terminal portion of the aorta.

EXPERIMENTAL METHOD

In total, 32 experiments were carried out on adult mongrel dogs of both sexes weighing 16-22 kg. Occlusion of the bifurcation of the aorta was produced by the method developed in clinical practice [1]. In the course of the experiment the blood pressure was recorded in the aorta, left ventricle, and the orifice of the venae cavae by means of the Mingograph-82 apparatus (Siemens). The cardiac output and circulating blood volume (CBV) were determined by a radiocardiographic method using ^{131}I-albumin.

To assess the rheologic properties of the blood, the viscosity of the blood, intensity of aggregative activity of the erythrocytes, hematocrit index, and the plasma protein spectrum were investigated. Hemodynamic indices and hemorheologic parameters were investigated immediately beforeocclusion and 3, 6, and 12 h after obstruction of the blood flow to the lower limbs.

EXPERIMENTAL RESULTS

Data on changes in the hemodynamic parameters and rheologic properties of the arterial and venous blood are given in Tables 1 and 2.

The blood plasma protein composition showed the following changes: The albumin content fell from 66.5 to 58.2% of the total protein (by 12.5%); slow globulins from 197 to 85 mg%; the γ-globulin level rose from 166 to 432 mg%; the content of fibrinogen and macroglobulins in the plasma remained within normal limits.

As a result of occlusion of the terminal portion of the aorta the cardiac output fell as early as 3 h after interruption of the blood flow. This was due, firstly, to a fall in the venous return, as shown by a fall in the venous pressure and CBV, and secondly, to an increase in the total peripheral vascular resistance as a result of activation of the sympathicoadrenal system.

Hemorheologic disorders are known to give rise primarily to circulatory changes in the system of the microcirculation, which are accompanied by elevation of the total peripheral resistance in the venous section of the vascular system. The possibility cannot therefore be ruled out that the changes observed in the rheologic properties of the blood 3 h after the experiment began were to a certain extent connected with marked disturbances of the central

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TABLE 1. Hemorheologic Indices of Arterial and Venous Blood (M ± m)

Index	Blood	Normal	Duration of occlusion, h		
			3	6	12
Maximal flowability, dynes/cm^2	Arterial	0,94±0,05	1,85±0,02*	1,61±0,14*	1,85±0,11*
	Venous	0,72±0,07	1,99±0,17*	1,85±0,11*	2,31±0,13*
Apparent viscosity, cP	Arterial	9,80±0,26	12,53±0,67*	10,24±0,45	9,55±0,40
	Venous	9,12±0,37	9,55±1,02	11,02±0,31*	14,29±0,43*
Dynamic viscosity, K^2, cP	Arterial	4,66±0,34	4,76±0,39	3,72±0,36	2,99±0,41
	Venous	4,71±0,31	2,37±0,64*	3,84±0,56	5,11±0,57
Coefficient of aggregation, A·10, dynes/cm	Arterial				
	Venous	0,31±0,06	0,69±0,12*	0,42±0,07	0,45±0,06
Degree of aggregation of erythrocytes	Arterial	25,4±13,9	41,8±13,8	28,6±4,2	15,7±4,1
	Venous	25,4±2,0	35,1±9,2	33,9±14,3	48,91±13,1
Hematocrit index	Venous	35,5±1,12	37,6±0,74	42,3±1,31*	44,1±1,38*

*Here and in Table 2, $P < 0.05$.

TABLE 2. Indices of Central Hemodynamics (M ± m)

Index	Initial	Duration of occlusion, h		
		3	6	12
PR	77,5±6,25	95,3±7,4	102,0±10,1*	146,0±14,1*
Mean arterial pressure, mm Hg	131,0±4,9	140,5±3,9	145,0±5,1*	157,0±6,8*
Central venous pressure, mm water	90,5±6,5	73,6±10,3	40,4±9,0*	36,2±4,93*
	1,7±0,13	1,02±0,12*	0,99±0,16*	0,95±0,09*
Total peripheral vascular resistance, dynes·cm·sec^{-1}	5907±465	9569±832*	12 150±1730*	15 200±1730
CBV	2,02±0,09	1,43±0,08*	1,36±0,06*	1,51±0,09

hemodynamics and that they were the result of the state of stress developing in response to total occlusion of the aorta.

The fact that a tendency toward restoration of the normal hemodynamics and blood rheology was observed after 6 h of occlusion points to the intensity of the compensatory mechanisms of the body at this stage of the experiment. The increase in pulse rate (PR), aimed at maintenance of the cardiac output, and also elevation of the arterial blood pressure lead to improvement of perfusion of the tissues at a time when the cardiac output is reduced.

However, after 12 h of interruption of the blood flow in the main arteries of the hind limbs the heart was unable to restore the reduced cardiac output through a considerable rise in PR. The cause of the reduced cardiac output was a decrease in the venous return as a result of the increased resistance in the venous section of the vascular system.

In the opinion of Dormandy [5] and Dintenfass [4], changes of this sort in the central hemodynamics are associated with rheologic disorders and, in particular, with increased viscosity of the blood. The reasons for the increased viscosity of the blood in the dogs after 12 h of occlusion were evidently, firstly, a reduction in CBV and an increase in the hematocrit index, secondly, a change in the plasma protein spectrum toward an increase in the content of high-molecular-weight proteins and, thirdly, increased aggregative activity of the erythrocytes in the blood. This last factor is known to lead to the development of hypoxic states in the tissues and organs [4, 6].

Analysis of the central hemodynamics and the rheologic parameters of the blood in the different stages of these experiments showed that definite correlation exists between the intensity of the hemodynamic disorders and the degree of disturbance of the blood rheology: The higher the blood viscosity and the intensity of erythrocyte aggregation, the more severe the hemodynamic disturbances. The possibility cannot be ruled out that in dogs with experimentally induced embolism, hemorheologic disorders arising during the first hours of the experiments are preceded by hemodynamic disturbances; by the 12th hour of occlusion, however, disturbances of the suspension stability of the blood is already beginning to exert its own influence on the indices of the central hemodynamics through irreversible changes in the microcirculatory system.

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PATHOGENESIS OF ACUTE CARDIAC FAILURE IN CLOSED CHEST INJURIES

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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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