loading takes place at the "input," so that adaptive mechanisms can be brought into play successively and coordinated intracardiac hemodynamics ensured when the load on the myocardium is increased. In coarctation of the aorta, this overloading is observed at the "output," which places the intracardiac adaptive mechanisms in an extremely unfavorable situation in which adaptation to function under the new conditions becomes necessary simultaneously and rapidly, and this leads to discoordination of relations between the right and left sides of th e heart.

A different situation arises in acute focal ischemia of the myocardium of the left and right ventricles. The myocardium is damaged in both cases. In acute focal ischemia of the right ventricle, however, the damage arises at the "input" (not overloading of the intact myocardium, but the actual injury), which upsets the whole system of adaptive mechanisms of the heart and leads to desynchronization of activity of its right and left sides. In ischemia of the left ventricle, this "upset" takes place at the "output." Adaptive mechanisms with earlier times of activation remain intact, and reorganization is possible whereby the heart adapts itself (including the left ventricle) to new conditions of functioning, and it maintains the correlations between its left and right sides that are characteristic of the normal situation.

Thus when the load on the intact myocardium of the left ventricle is sharply increased, or in cases of primary injury to the right ventricle, a disturbance of the normal trend of adaptive reactions in the heart takes place, and this leads to a change in the character of correlations between its left and right sides. Disturbance of these correlations may lead, in turn, to disturbance of the activity of the heart as a system.

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TREND OF THE CARDIAC OUTPUT AND BLOOD RHEOLOGY IN THE EARLY PERIOD OF RESUSCITATION AFTER PREAGONAL AND AGONAL STATES

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KEY WORDS: blood loss; postresuscitation period; volume of the heart.

The study of the mechanism of formation of postresuscitation circulatory insufficiency after various terminal states occupies an important place in the analysis of the pathogenesis of postresuscitation sickness and prevention and treatment of its complications [3, 6-9, 13]. The aim of this investigation was to study the trend of the cardiac output (CO) and of the flow properties of the blood in the early postresuscitation period after preagonal and agonal states induced by blood loss.

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TABLE 1. Changes in Some Parameters of Hemodynamics and Blood Rheology in Animals in Postresuscitation Period after Terminal States (M ± m)

Parameter studied	Original state			Terminal period			Postresuscitation period								
	1	2	3	1	2		5			1			3		
	n=7	n=8	n=4	4 ч	2 ч	Agony for 6,6 ± 1.2 min	1	2	3	1	2	3	1	2	3
CO, ml/min·kg	139±15	116±15	158±12	71±6*	61±6*	—	267±24*	176±19	161±13**	116±10	109±12	154±16	84±9*	67±9*	124±9,5**
BP, mm Hg	109±7	118±11	112±15	40±4*	40±2*	6,1±1,7	108±13	111±13	91±7	101±9	113±7	90±7	90±8	112±7	90±3,6
Heart rate, beats/min	98±13	71±5	68±15	206±8*	190±20*	--	161±11*	111±9*	150±10*	144±12*	117±9*	153±10*	169±16*	130±22*	142±7,3*
Viscosity of blood (in mPa/sec) with shear velocities of:															
1,34 sec^{-1}	17,3±1,7	16,0±1,9	14,4±0,4	33,9±5*	19,2±3	8,6±0,7*	43,0±6*	22,3±2,2***	10,6±5,0**	31,2±5	36,7±2,3	12,8±5,0**	42,5±9	35,7±4,6	14,9±4**
54,2 sec^{-1}	6,5±0.2	5,6±0,3	5,6±0,5	7,1±0,3	5,4±0,3	4,5±0,2*	7,3±0,3**	6,1±0,3	4,3±0,5*	7,5±0,2	6,6±2,3	5,0±1,0**	7,8±0,3	6,7±0,02	4,8±0,2**
Hematocrit, liter/liter	0,53±0,02	0,51±0,02	0,51±0,05	0,55±0,02	0,46±0,01*	0,39±0,01	0,55±0,02**	0,50±0,07	0,42±0,02*	0,59±0,01	0,58±0,02	0,42±0,02	0,60±0,01	0,57±0,02	0,46±0,06**

Legend. *P < 0.05 compared with original values, **) with values between groups 1 and 3, ***) with values during terminal state; n) number of observations.

EXPERIMENTAL METHOD

Experiments were carried out on 15 heparinized (500 IU/kg) dogs of both sexes weighing 10-15 kg. Under superficial pentobarbital anesthesia with trimeperidine premedication (8 mg/kg) the animals were quickly exsanguinated (for 3-7 min) from the femoral artery, lowering their blood pressure (BP) on average to 40 mm Hg, at which level it was maintained for 4 h (preagonal state, group 1) or for 2 h, followed by further blood loss and lowering of BP until a terminal pause ensued (transient respiratory arrest, group 2). The total volume of blood lost by the animals of group 1 was 45 ± 2 ml/kg, of group 2, 50 ± 5 ml/kg, and of group 3, 47 ± 5 ml/kg. The vital functions of the dogs of group 1 were restored after hypovolemic hypotension lasting 4 h by intra-arterial injection of blood into the femoral artery. Resuscitation of the animals of group 2 began in the period of agony. Artificial ventrilation of the lungs (AVL) was carried out with 100% oxygen, a tidal volume of 40 ml/kg, and respiration rate of 16-20 cycles/min. Blood was reinfused through the femoral artery. After the blood loss had been made good and with the appearance of spontaneous respiration, AVL was stopped. In the experiments of group 3, after arterial hypotension for 4 h, resuscitation continued with intra-arterial injection of a mixture of blood (60%) with dextran (40%). Infusion therapy was given with monitoring of the pulmonary arterial and central venous pressure, aortic pressure, and the ECG, which were recorded on a polygraph (San-Ei, Japan). The viscosity of the blood was determined with a VIR-75 rotary viscometer, designed and produced at the Gor'kii Physicotechnical Institute. The viscosity of the blood was determined in millipascals per second, with shear stress of between 2 and 200 dynes/cm^2, corresponding to a shear velocity of between 1.3 and 52.4 sec^{-1}. The surface configuration of the erythrocyte membrane was investigated in three-dimensional representation, by scanning electron microscopy. The morphological characteristics of the erythrocytes were in accordance with the classification in [5]. CO was determined by the thermodilution method [2].

EXPERIMENTAL RESULTS

The value of CO was considerably reduced after 2 and 4 h of hypovolemic hypotension and it rose sharply during the first minutes of the postresuscitation period in animals revived from the preagonal and agonal states (Table 1). The degree of increase of CO in animals of these two groups differed: The blood flow was doubled in the animals of group 1 and increased by 1.5 times in the animals of group 2. The difference between these two groups is significant.

Postischemic hyperperfusion was thus greater in animals subjected to a longer period in the terminal state.

After 1 h of the postresuscitation period the value of CO stabilized at the same level as initially. However, this stabilization was not permanent, and by the 3rd hour of the postresuscitation period CO in the animals of groups 1 and 2 had fallen to 60 and 58% (hypoperfusion) of its initial value respectively. Similar phasic changes in CO have been found in the resuscitation period after clinical death [3, 9].

We know that the relative contribution of disturbances of the rheologic characteristics of the blood to pathology of the cardiovascular system is very great [1, 8, 11, 12, 14]. In the present experiments, at the end of the terminal state the viscosity of the blood was significantly higher in animals exposed to hypovolemic hypotension for 4 h, compared with that in the animals after hypotension for 2 h, with an agonal period when spontaneous hemodilution took place (Table 1).

During the first few hours of the postresuscitation period in animals exposed to arterial hypotension for 4 h the viscosity of the blood did not differ significantly from that at the end of the terminal period. Conversely, in animals revived from an agonal state, it rose sharply, and by the 5th minute after resuscitation it was 2.5 times higher than in the agonal state. In the agonal period, despite the low viscosity of the blood and fall in the hematocrit index, considerable disturbance of the rheologic properties of the erythrocytes was found. This was expressed as a significant decrease in the number of discoid forms (from 91.11 ± 1.45 to 65 ± 6.2%) and an increase in the number of nondiscoid, nontransitional forms (from 3.66 ± 1.3 to 25.4 ± 6.8%) in the initial and agonal states respectively. Disturbance of the shape of the erythrocytes continued for the next 3 h of the postresuscitation period: discoid forms accounted for 61.7 ± 7.0% and nondiscoid, nontransitional forms for 25.3 ± 3.4%. Profound and stable disturbances of the shape of the erythrocytes and, consequently, deformability of these cells were thus an important mechanism of the rheologic changes of the

blood developing actually during the terminal stage. A further increase in viscosity of the blood in the postresuscitation period took place on account of a significant increase in the hematocrit index (Table 1). It can be tentatively suggested that the fall in CO in this period took place not only because of disturbances of the rheologic properties of the erythrocytes themselves, but also of a decrease in the effectively circulating fluid volume. It must be noted that preliminary injection of heparin (500 IU/kg body weight) did not prevent the profound changes in rheologic properties of the blood. When the degree of parallel changes in visocisity of the blood CO is assessed by the 5th minute of the postresuscitation period for animals of groups 1 and 2, it will be noted that postischemic hyperfusion was more marked ($P < 0.05$) in animals of group 1 with very high viscosity ($r = 0.6$; $P = 0.05$).

To improve the flowability of the blood for intra-arterial infusion a mixture of blood and dextran (hematocrit 0.3 liter/liter) was used in a volume equal to that of the blood loss (group 3). By the 5th minute of the postresuscitation period the viscosity of the blood in the animals of this group was significantly lower than that in the animals of group 1, and this was combined with absence of postischemic hyperperfusion. The BP level in animals resuscitated with blood alone (or blood with dextran) did not differ significantly (Table 1). There was likewise no significant difference in the values of pH in these groups (7.08 ± 0.04 and 7.11 ± 0.02 respectively). It can be tentatively suggested that the presence of high viscosity of the blood in the first minutes after resuscitation is a pathogenetic factor, forming postischemic hyperperfusion (reactive hyperemia). The results confirm the hypothesis [10, 15] on the role of high viscosity of the blood in the mechanism of vasodilatation in the period of postischemic hyperperfusion. It must also be pointed out that the inclusion of hemodilution with dextran in resuscitation of hypovolemic states also prevented development of a low cardiac ejection syndrome during the first hours after resuscitation. For instance, the cardiac ejection in animals of group 3 after 3 h of the postresuscitation period was significantly higher than that after infusion of whole blood. As we known [1, 3, 4, 12], this was due not only to hemodilution, but also to the ability of dextran to maintain the effectively circulating fluid volume and vascular tone for a long time. Further improvements in resuscitation measures for use after lethal blood loss demand optimization of methods of infusion therapy and introduction of the principles of gravitation surgery of the blood in order to correct its composition.

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