CHANGES IN SOME RHEOLOGICAL PROPERTIES OF THE BLOOD AFTER RESUSCITATION

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In experiments on cats anesthetized with pentobarbital, the dynamic viscosity of the blood and the aggregative power of the blood cells were studied during the first 2 h after resuscitation by means of a capillary loop viscosimeter. An increase in viscosity was found both in the zone of small (1.82 dyn/cm^2) and in the zone of large (10.94 dyn/cm^2) shear stresses. The increase in the hematocrit index and in the aggregative power of the blood cells plays a definite role in the increase in viscosity.

KEY WORDS: resuscitation; viscosity of the blood; rheological indices.

The importance of changes in the rheological properties of the blood in the genesis of the circulatory disorders in shock has been demonstrated [1, 7, 12]. In the postresuscitation period the circulating blood volume is reduced, mainly on account of plasma [4]. This fact, together with disturbance of the ratio between the protein fractions [6], suggests the possibility of changes in the rheological properties of the blood.

Considering that the dynamic viscosity is one of the most important indices of flowability of the blood, it was decided to study the character of its changes and also the aggregative power of the blood cells in the early period after resuscitation.

EXPERIMENTAL METHOD

Experiments were carried out on cats of both sexes anesthetized with pentobarbital (45 mg/kg). The dynamic viscosity of the blood (η) was determined on a capillary loop viscosimeter [9] with a diameter of the working part of 736 nm and a length of 9.9 cm at 37° C. The viscosity was studied at shear stresses (τ) of between 1.82 and 10.94 dyn/cm^2, so that characteristics could be obtained in non-Newtonian and Newtonian regions. The shear stress and shear velocity ($\bar{U}$) were determined by the appropriate equations [9]. By plotting graphs of $\bar{U}$ against τ in Casson's system of coordination [8] the minimal shear stress causing the blood to flow – the yield point (τ_0) – was determined. The hematocrit index was obtained and the index of aggregation calculated [1, 10]. Clinical death was produced by arterial bleeding; the animals were resuscitated after 4 min by intraarterial infusion of the removed blood and by artificial respiration (V. A. Negovskii's method). The rheological properties of the blood were studied in 10 animals in the initial period and again 15, 60, and 120 min after restoration of cardiac activity. One drop of heparin was added to each blood sample (1.3 ml). In eight animals, six to a frame, not subjected to bleeding (control group), the same parameters were studied at times corresponding to those in the experimental series. Statistical analysis of the results was carried out with the aid of Student's criterion and nonparametric criteria [3].

EXPERIMENTAL RESULTS AND DISCUSSION

At the 15th minute of resuscitation a marked increase in the viscosity of the blood took place in the zone of small and large shear stresses and velocities (Table 1; Fig. 1). In the control at this period

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TABLE 1. Dynamic Viscosity of Blood, Hematocrit Index, and Aggregation Index in Early Period after Resuscitation

Index studied	Group of animals	Initial data	Postresuscitation period (minutes after resumption of cardiac contractions)		
			15	60	120
Viscosity (in cP) with sheer stress of:					
1.82 dyn/cm²	Experimental	5,68±0,69	6,88±0,83 †	7,71±0,93 †	9,77±1,15*
	Control	6,75±1,02	7,18±0,74	8,37±1,16 †	9,41±1,39 †
10,94 dyn/cm²	Experimental	2,56±0,16	2,90±0,18 †	2,88±0,15†	3,52±0,22*
	Control	2,65±0,20	2,77±0,24	3,02±0,34†	3,33±0,36 †
Hematocrit index (in %)	Experimental	34 (27—45)	41 (32—53) †	41 (32—51) †	45 (34—53) †
	Control	36 (31—48)	37 (31—45)	41 (36—50) †	43 (37—53) †
Index of aggregation	Experimental	0,30 (0,13—0,59)	0,30 (0,19—0,59)	0,33 (0,22—0,41)	0,46 (0,26—0,77) †
	Control	0,33 (0,06—0,60)	0,40 (0,13—0,73)	0,43 (0,17—0,78)	0,46 (0,23—0,64) †

* P< 0.05 0.001 after Student.
† P < 0.05 0.01 after Wilcoxon.

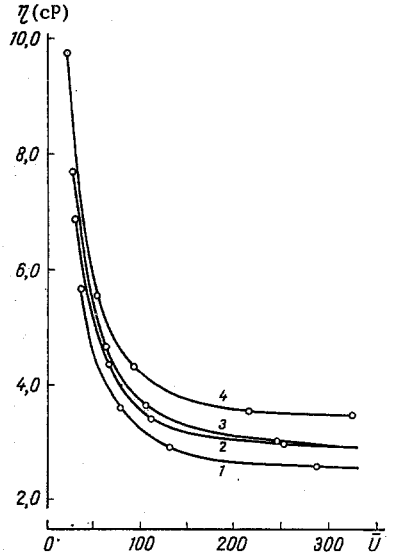

Fig. 1. Viscosity of blood (η) as a function of shear velocity ($\bar{U}$): 1) initial viscosity; 2–4) 15, 60, and 120 min after resuscitation.

no significant increase in viscosity was observed. By the end of the first hour the blood viscosity remained at about the same level, and some increase was found only in the region of low shear velocities. During the next hour the viscosity of the blood increased again. The increase in viscosity in the control experiments was less marked. For instance, at $\tau = 1.82$ dyn/cm², the viscosity of the blood in the experimental animals increased by the end of the investigation on the average by 1.72 times, which differed significantly from the increase in the control group (criterion $\bar{U}$) which was by 1.39 times. These differences were smaller in the region of higher shear velocities.

The value of the yield point (τ_0) was also substantially altered in the experimental animals. Whereas initially it was 0.17 dyn/cm², in the postresuscitation period it was increased gradually to 0.19, 0.24, and 0.26 dyn/cm². In the control group in the initial period it was 0.24 dyn/cm², and later it remained virtually unchanged.

Simultaneously with the increase in viscosity, the hematocrit index also increased. The coefficient of correlation between the viscosity of the blood, for $\tau = 1.82$ dyn/cm² and an initial hematocrit index of 0.81, fell in the postresuscitation period to 0.70, 0.74, and 0.51; i.e., 2 h after resuscitation the correlation between the hematocrit index and blood viscosity was no longer significant. This applied also to the zone of high shear stresses (10.94 dyn/cm²) for which the initial coefficient of correlation was 0.73, falling to 0.59 by the end of the investigation.

Hence, whereas the increase in viscosity during the first hour of the postresuscitation period was connected mainly with hemoconcentration, later other factors assumed importance. One of them could be an increase in the aggregative properties of the blood cells, as reflected in an

increase in the index of aggregation. The coefficient of correlation between the blood viscosity and index of aggregation rose from 0.18 in the initial period to 0.61 2 h after resuscitation. The increase in aggregation of the blood cells may be connected with changes in the ratio between the blood protein fractions, which moved in the direction of an increase in coarse-dispersed proteins [8], and as the result of a fall of the ζ-potential.

Comparison of the dynamic viscosity with the general peripheral resistance studied previously [5] shows that only in the earliest stages of the postresuscitation period (until 15-20 min) were their changes in different directions. At this stage of the posthypoxic period increased extravasation of fluid [4] evidently took place, accompanied by an increase in the hematocrit index and viscosity. At the same time the tone of the resistive vessels was lowered ("postischemic hyperemia") and, as a result, the peripheral resistance was lowered. Later, when the phenomena of postischemic hyperemia had subsided or disappeared completely, the viscosity and the general peripheral resistance increased parallel to each other.

An increase in viscosity has its greatest effect on movement of the blood in the microvessels and, in particular, in the postcapillary venules and veins, the shear velocity in which is characterized by low values [11]. It must be remembered that the lumen of these vessels, with a reservoir function, is reduced because of the developing deficiency of the circulating blood volume [4]. This may lead to a marked increase in resistance of the venous part of the vascular system. At the same time, an increase in resistance of the venous portion [2] is known to have an effect on the return of blood to the heart several times greater than an increase in the arterial resistance. Presumably, therefore, reduced flowability of the blood is an important factor determining the fall in the cardiac output and facilitating the development of circulatory hypoxia in the postresuscitation period.

The facts described above are evidence of the need for treatment correcting changes in the rheological properties of the blood in the resuscitation period.

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