CHANGES IN HEMORHEOLOGIC INDICES IN THE COURSE OF HEMORRHAGIC SHOCK

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Relations between rheologic, hemodynamic, and metabolic indices during hemorrhagic shock were studied in experiments on dogs. The mechanical properties of the blood were found to change immediately after blood loss parallel with the macrohemodynamic disorders, and they preceded the development of metabolic acidosis. It is concluded that determination of the blood fluidity threshold can be used to establish the degree of the circulatory disturbance in shock.
KEY WORDS: *shock; rheologic changes; fluidity threshold; Casson's model.*

An important role in microcirculatory disturbances in shock is played by changes in the rheologic properties of the blood: the deformability and adhesiveness of the red cells, the dynamic viscosity of the plasma, the fluidity threshold of the blood [1, 4, 7, 9, 12]. Most experimental and clinical studies have been concerned with the apparent blood viscosity during high shear speeds [1, 4, 12]. Investigations of other indices have usually been undertaken on physical models in vitro, and the possibilities of using the results to estimate the state of the circulation are thus limited.

TABLE 1. Changes in Some Blood Indices during Hemorrhagic Shock (M ± m)

Index studied	Original data	30 min of shock	90 min of shock
Fluidity threshold (in dynes/cm²):			
heparinized animals	0,027±0,005	0,039±0,009*	0,049±0,008‡
nonheparinized animals	0,043±0,005	0,060±0,008*	0,152±0,007‡
pH:			
artery	7,37±0,01	7,37±0,01	7,31±0,02
vein	7,34±0,01	7,23±0,02	7,12±0,03*
BE (in meq/liter):			
artery	+5,1±0,02	−2,5±2,2*	−10,5±1,5‡
vein	+3,3±3,1	−5,2±1,5†	−14,8±1,0‡
PO_2 (in mm Hg):			
artery	100,0±1,2	114,6±2,0*	133,3±1,7†
vein	45,7±1,2	34,9±1,1†	32,3±1,2†
PCO_2 (in mm Hg):			
artery	74,4±2,1	52,7±1,8*	44,8±1,9†
vein	90,3±1,9	69,5±2,5*	70,1±2,5*
Hematocrit (vein)	56,0±2,8	52,0±3,1	55,4±2,8

*P 0.05.
†P 0.005.
‡P 0.001.

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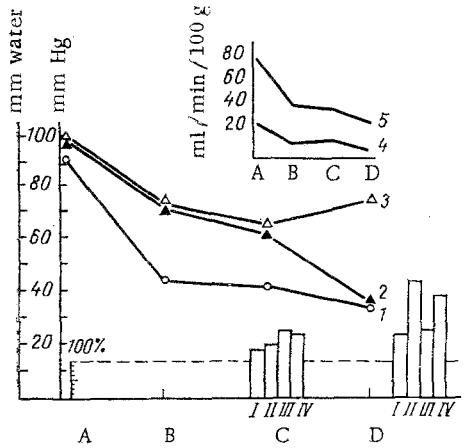

Fig. 1. Changes in hemodynamic indices during hemorrhagic shock: A) original data; B) 30 min; C) 60 min; and D) 90 min of shock. 1) arterial pressure; 2) portal venous pressure in heparinized animals; 3) portal venous pressure in nonheparinized animals; 4) blood flow along hepatic artery; 5) along portal vein. I and II) fluidity threshold of heparinized and nonheparinized dogs respectively; III and IV) portal resistance of heparinized and nonheparinized dogs respectively.

The rheologic properties of the blood were studied together with macrocirculatory changes and the dynamics of the acid–base balance during experimental hemorrhagic shock induced in preliminarily heparinized or nonheparinized animals.

EXPERIMENTAL METHOD

Experiments were carried out on 40 mongrel dogs, half of which were given preliminary injections of heparin in a dose of 300 units/kg. The abdomen was opened under morphine-hexobarbital anesthesia and the portal and hepatic veins, the caudal vena cava, and the aorta were catheterized. The pressure in these vessels, the blood flow along the portal vein and hepatic artery, measured by means of an electromagnetic flowmeter, and the ECG in three leads were recorded synchronously on a Mingograph-81 apparatus. The resistance of the organs to the blood flow and the debit in the vessels were calculated per 100 g weight of liver. Simultaneously with recording of the physiological parameters, blood samples were taken for investigation of the acid–base balance and the hematocrit index (Ht). The pH and pO_2 were investigated with the micro-Astrup apparatus and pCO_2 and the blood–buffer systems were determined from the Siggaard–Andersen nomogram. The blood fluidity curves were obtained with the aid of a rotation viscosimeter [2] at shear speeds of between 0.05 and 1.5 sec^{-1}, i.e., in the region of greatest nonlinearity, and with temperature stabilization at 25°C. The blood samples were heparinized at the rate of 5 units/ml. The fluidity curve was plotted between Casson's coordinates [7]:

$$\tau^{1/2} = f\left(\dot{\gamma}^{1/2}\right),$$

where τ is the shear stress (in dynes/cm^2), and $\dot{\gamma}$ the shear velocity (in sec^{-1}). The intercept on the $\tau^{1}/_{2}$ axis by the fluidity curve when $\dot{\gamma}$ tends to 0 was taken as the fluidity threshold (τ_0). The results were subjected to statistical analysis [3]. Hemorrhagic shock was induced by measured bleeding [13] and the hypotension was maintained at the level of 35-45 mm Hg for 90-120 min.

EXPERIMENTAL RESULTS AND DISCUSSION

The shock followed the typical course for this particular model and was characterized by stages of compensation ("early" shock) and decompensation ("late" shock) of the cardiovascular system [13]. During the period of early shock correlation was found between the pressure drop in the vessels and the blood flow along the portal vein and hepatic artery. In the period of decompensation, when the debit was low, the portal resistance to the blood flow increased sharply (Fig. 1) and metabolic acidosis developed: pO_2 in the arterial blood rose, pO_2 and pCO_2 in the venous blood fell, the buffer systems were exhausted, pH fell, and the arterio-venous pH difference increased (Table 1). A rapid increase of portal resistance and of pressure in the portal vein was observed in the nonheparinized animals and decompensation of their cardiovascular system developed earlier than in heparinized animals.

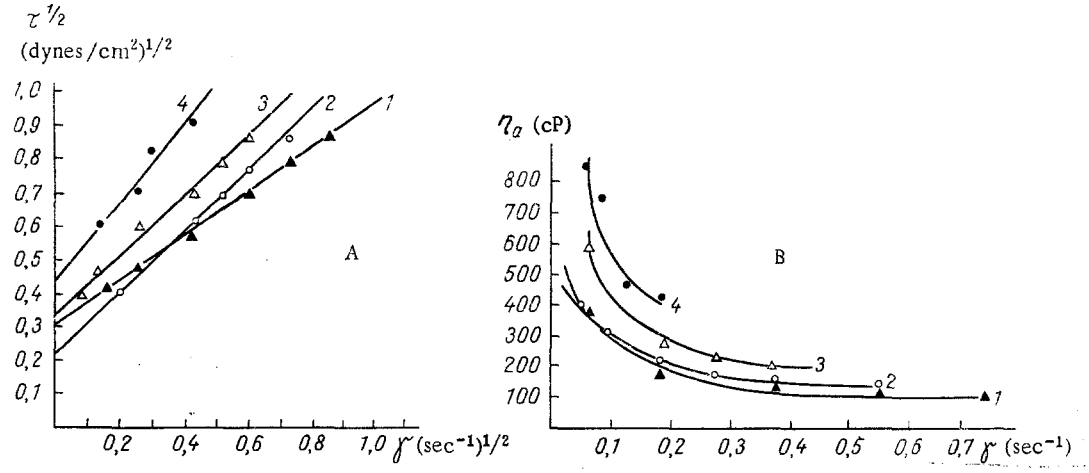

Fig. 2. Changes in blood flow curves of dogs during hemorrhagic shock: A) in Casson's coordinates: intercept made on ordinate by flow curve for velocity γ (in $\sec^{-1})^{1/2} = 0$ taken as fluidity threshold; B) apparent blood viscosity as a function of shear velocity in shock. 1) Original data, 2) 30 min, 3) 60 min, and 4) 90 min of shock.

The flow curves, except at a few points, were easily converted into straight lines in Casson's coordinates (Fig. 2). As Table 1 shows, substantial differences were present in the original values of the fluidity threshold of the heparinized and nonheparinized animals. During the period of "late" shock (90-120 min) the increase in τ_0 of the nonheparinized dogs was much more marked than in the heparinized dogs.

The dynamics of the Casson viscosity (K) corresponded to changes in the hematocrit index. During the period of "early" shock, for instance, K fell to 74 ± 5.5% of its initial value ($P < 0.01$), with a tendency for Ht to fall. Both K and Ht regained their original values after 90-120 min. This is perfectly understandable for, according to Casson's theory [6], the gradient of the flow curve is determined entirely by the index of volume concentration of the suspension and the viscosity of the fluid part. The viscosity of blood serum is less variable than Ht. The functional relationship between these values is described by the equation:

$$K = \frac{\eta_0}{(1 - Ht)^{2.5}},$$

where K is the Casson viscosity, Ht the hematocrit index, and η_0 the dynamic viscosity of the plasma. The index K in the assessment of the mechanical properties of the blood is analogous to the dynamic viscosity of a non-Newtonian fluid and it can characterize the blood flow along the great vessels. It is meaningless to speak of any viscosity of the blood during its flow along vessels whose diameter is commensurate with that of the red cells. In that case the fluidity threshold is important, for this index is defined by the strength of the spatial structure of the aggregates and by the degree of aggregation [9-11]. Accordingly, the dynamics of τ_0 objectively reflects the shock-specific disturbances of the microcirculation, i.e., the degree of pathological aggregation of the blood cells. Characteristically rheologic changes arise virtually simultaneously with a decrease in the circulating blood volume, the intravascular pressure gradient, and the velocity of the blood flow. Hemorheologic pathology precedes the appearance of metabolic acidosis, but the stage of decompensation develops when severe changes have occurred in the mechanical properties of the blood. Aggregation of the red cells is a result of the slowing of the blood flow [5, 7, 12] and is one of the main causes of the development of tissue hypoxia in shock [4, 12]. The increase in the portal resistance during the period of "early" shock may be mainly due to vasoconstriction [12]. However, in the period of "late" shock also, despite the reduction in vascular spasm and the appearance of vasoconstrictor metabolites [4], the resistance to the blood flow in the liver is increased. This may be the result of intensifi-

cation of pathological aggregation. Differences between the degree of increase of τ_0 in the heparinized and nonheparinized dogs confirm the role of fibrinogen in the formation of the fluidity threshold and they emphasize the role of disturbances of coagulation in the etiology of organic disturbances in the microcirculatory system in shock [4, 8, 10].

The results of these investigations thus indicate that a well-marked hemorheologic pathology develops in shock. The fluidity threshold index varies with an increase in the duration of hypotension, it precedes the appearance of metabolic acidosis, and it reflects the severity of the shock syndrome. The high sensitivity and simplicity of the corresponding tests allow them to be used as a means of establishing the degree of the hemodynamic disturbances in shock.

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