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EFFECT OF AUTOLOGOUS BLOOD REINFUSION ON THE SYSTEMIC AND PORTAL CIRCULATION AFTER ACUTE BLOOD LOSS IN RATS

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UDC 616-005.1-036.11-092.9-085.388-036.8-07:616.149-008.1

KEY WORDS: acute blood loss; portal macrocirculation and microcirculation; reinfusion of autologous blood.

Clinical indications for the use of autologous blood reinfusion, eliminating the risk of development of various complications connected with transplantation of homologous blood, have widened considerably at the present time [1, 5, 8]. However, the particular features of the action of autologous blood on the macro- and microcirculation in vital organs have not been adequately explained.

The aim of this investigation was to study the effect of reinfusion of autologous blood on the portal macro- and microcirculation, the systemic arterial pressure (BP), and the duration of survival of rats after acute blood loss (ABL).

EXPERIMENTAL METHOD

Experiments were carried out on 52 male Wistar albino rats weighing 200-250 g. The microcirculation in the liver and intestine was studied by contact luminescent biomicroscopy under general urethane anesthesia [7]. Simultaneously with visual inspection of the hepatointestinal microcirculation, the volume velocity of the blood flow in the portal vein of the liver and the linear velocity of the blood flow in the hepatic artery were measured by an ultrasonic method [3]. ABL was induced by one session of bleeding from the femoral artery in a volume equivalent to 2.5% of the animal's body weight in the course of 5 min. The blood was collected and stabilized with heparin, added at the rate of 2 U to 1 ml blood [5]. The heparinized autologous blood, warmed to 37°C, was reinfused after 10 min intravenously into the animal in the course of 5 min. As an integral criterion of the state of the cardiovascular system BP in the carotid artery, measured with a micromanometer, was used [6]. The duration of survival after reinfusion of autologous blood was used as an indicator of the general state of the animals.

EXPERIMENTAL RESULTS

The writers showed previously that the state of the portal macro- and microcirculation under experimental conditions correlates with the severity of the course of the posthemorrhagic period (PHP). A marked and persistent reduction of the portal fraction of the total hepatic blood flow after ABL in rats is evidence of a decompensated type of course of PHP, whereas during rapid quantitative restoration of the portal blood flow to a subnormal level PHP follows a benign course [4]. Intravenous injection of autologous blood into rats with a compensated type of course of PHP directly during reinfusion caused a rapid increase in the systemic BP and in the velocity of the hepatic portal and arterial blood flow to values 20-25% higher than initially, and an improvement of the microcirculation in the terminal vascular bed of the liver and intestine (Fig. 1). The degree of filling of the microvessels of

Research Institute of General Pathology and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR G. N. Kryzhanovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 100, No. 12, pp. 652-655, December, 1985. Original article submitted January 31, 1985.

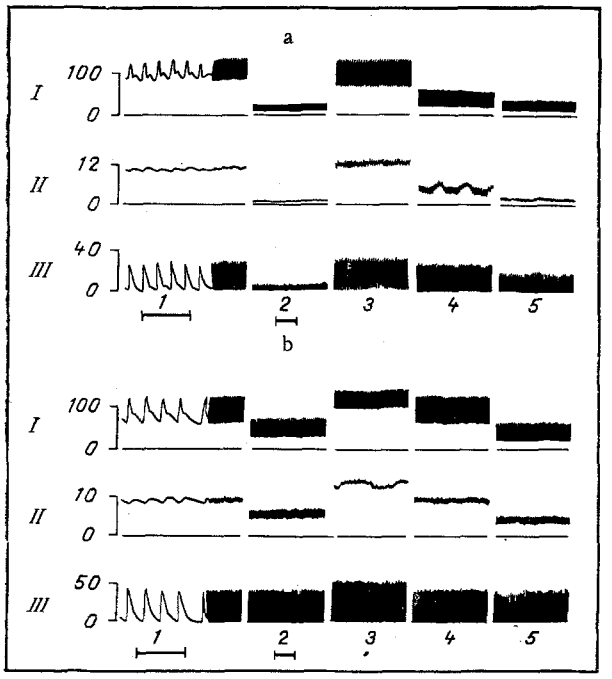

Fig. 1. Character of changes in systemic BP and velocity of blood flow in the portal vein of the liver and hepatic artery in rats after acute blood loss and reinfusion of the autologous blood. a) Decompensated type of course of acute blood loss, b) compensated type. I) BP in carotid artery (in mm Hg), II) volume of blood flow in portal vein (in ml/min), III) linear velocity of blood flow in hepatic artery (in cm/sec). 1) Before blood loss; 2) 10 min after blood loss; 3) at end of reinfusion of autologous blood; 4, 5) 1 and 2 h respectively after reinfusion of autologous blood. Time marker 0.5 and 10 sec.

the liver and intestine with blood was found to be restored on biomicroscopic examination, and the linear velocity of the blood flow in the microcirculation under these circumstances increased considerably and, on visual inspection, exceeded the initial velocity. Parameters of the systemic BP and the portal macro- and microcirculation 10-15 min after reinfusion fell to the control values and were respectively: BP 100 mm Hg, volume velocity of the portal blood flow 12 ml/min, linear velocity of the arterial blood flow 40 cm/sec. These parameters remained comparatively stable at this level for 1 h, after which they began to fall gradually, and 2 h after reinfusion they were not more than 50% of the control values. Intravenous injection of autologous blood into the animals led to the development of focal changes in the microcirculation, discovered by the writers previously [4] in PHP, in the form of the appearance of fragmented microthromboses, microstasis, and increased vascular and tissue permeability, in the terminal microvessels of the liver and intenstine. It is a noteworthy fact that the focal microcirculatory disturbances in the liver were clearly localized topographically in a manner which corresponded to the zones of the functional element of the organ described by the writers previously [2]. They were found in the region of the afferent hepatic microvessels, and gradually the whole central zone of the functional element appeared disconnected from the microcirculation. Parallel with the fall of the systemic BP and of the velocity of the portal and arterial hepatic blood flow, the general character of the blood flow in the microvessels changed, with an increase in microhemoconcentration, so that the blood flow became granular in type, and signs of intravascular aggregation of erythrocytes were intensified. The animals died when there was a simultaneous fall in the level of the systemic BP and in the velocity of the portal and arterial blood flow to zero, and when, at the microcirculatory level, a progressive decrease in the velocity of the blood flow was observed, followed by its complete arrest without any additional microcirculatory structural changes. The minimal

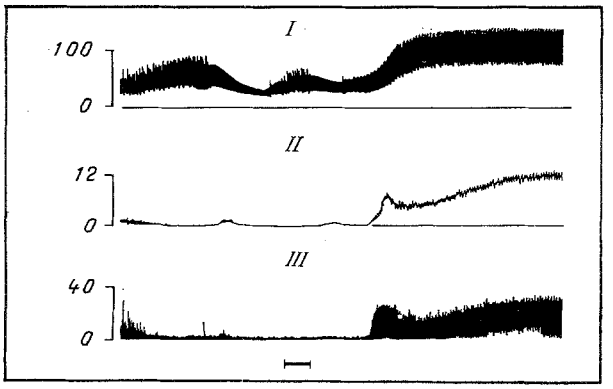

Fig. 2

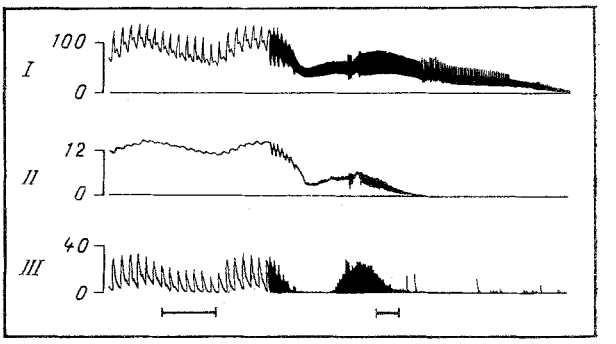

Fig. 3

Fig. 2. Dynamics of changes in systemic BP and hepatic circulation in rat with rapid, decompensated type of course of PHP during reinfusion of autologous blood. Legend as to Fig. 1. Time marker 10 sec. Trace of systemic BP shows disturbances of cardiac rhythm (extrasystoles).

Fig. 3. Sudden and irreversible worsening of parameters of systemic and hepatic circulation in rats with rapid decompensated type of course of PHP 15 min after reinfusion of blood. From top to bottom: BP in carotid artery; volume velocity of blood flow in portal vein; linear velocity in hepatic artery; time marker 1 sec and 10 sec.

duration of survival was 5 h.

The initial dynamics of recovery of the systemic BP and hepatic macrocirculation was similar in animals with a decompensated type of course of PHP, when the duration of survival of the untreated animals was 1 h [4]. Reinfusion of autologous blood into these animals led to a rapid rise of the systemic BP and velocity of the portal and arterial blood flows, values of which usually exceeded their initial levels. The heterogeneity of the microcirculatory changes in the liver, characteristic of this type of course of PHP and described by the writers previously [4], underwent the following transformation. In vascular zones with primary generalized constriction of the hepatic microvessels and with a reduction in the velocity of the sinusoidal blood flow in them, rapid recovery of the blood flow in them, on visual inspection, was higher than in the background. In macrosegments in which dilation of the sinusoids was observed in PHP, there was initially only an increase in the degree of filling of the microvessels with blood without any appreciable changes in the velocity of the blood flow, which remained greatly reduced for 5-7 min. This was followed by recovery of the original microcirculation of blood in these zones also. As a rule, in animals of this group, unlike in animals with compensated blood loss, stabilization of the parameters of the systemic BP and of the portal macro- and microcirculation was unstable, and by the end of the first hour after reinfusion the parameters recorded showed a decrease of 50-60% (Fig. 1). The subsequent trend of changes in the hepato-intestinal macro- and microcirculation corresponded to disturbances of the hemodynamics described previously in the splanchnic circulation following reinfusion of autologous blood into animals with a compensated type of course of PHP. The length of survival of the animals of this group did not exceed 2-3 h after reinfusion.

In rats with a rapid decompensated typed of course of PHP, when the untreated animals died 10-15 min after blood loss, signs of instability of cardiac activity were found during the first minutes after the beginning of reinfusion. These took the form of considerable fluctuations in the systemic BP, both upward and downward (Fig. 2). The BP trace also revealed disturbances of the cardiac rhythm in the form of extrasystoles. The volume velocity of the portal blood flow and the linear velocity of the blood flow in the hepatic artery decreased during this period to zero. This was followed by a progressive increase in these parameters of the systemic and hepatic circulation to the subnormal level. A sudden and irreversible fall of the systemic BP and in the velocity of the blood flow in the portal vein and hepatic artery took place 10-15 min after the beginning of reinfusion of autologous blood (Fig. 3). The terminal vascular bed of the liver and intestine became ischemic, the diameter of the microvessels decreased, and the animals died in the course of 30 min after reinfusion of autologous blood.

The state of portal macro- and microcirculartion thus reflects not only severity of the course of acute blood loss, but also the effectiveness of its treatment with autologous blood. Making good the circulating blood volume after acute blood loss by reinfusion of the autologous blood is not an effective method of converting a rapid decompensated type of course of PHP a compensated type. Reinfusion of autologous blood into animals after acute blood loss intensifies posthemorrhagic intravascular aggregation of erythrocytes. In the liver, at the level of the functional elements, infusion of autologous blood can also lead to the development of local posthemorrhagic disturbances of the microcirculation, in the form of the appearance of fragmented microstases and microthromboses, disturbances of vascular and tissue permeability, and exclusion of the central zone of the functional elements of the liver, responsible for protein synthesis, for oxidation-reduction process, and also for glycogenolysis [2], from the microcirculation. This may lead to a reduction in the energy potential of the organ and to partial functional hepatic failure; treatment with autologous blood should therefore best be combined with specific pathogenetic correction of the zonal disturbances of the hepatic microcirculation and blood rheology described above.

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