CHANGES IN VOLUME OF CIRCULATING BLOOD AND ITS COMPONENTS IN THE ACUTE PERIOD OF THE PROLONGED LIMB CRUSH SYNDROME

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Experiments on 13 adult dogs showed that immediately after trauma there is a short period of hypervolemia with hypotension, an increase in the velocity of the blood flow, while the hematocrit index remains normal. The hypervolemia is then followed by hypovolemia accompanied by a parallel decrease in the volume of circulating plasma and erythrocytes. The development of hypovolemia and the decrease in the blood flow velocity precede a fall of blood pressure. The hypervolemia is explained by mobilization of the blood reserves (spasm of the vessels) and the development of hemodilution. The hypovolemia probably develops as a result of pathological retention of the blood (vasodilatation) and the activation of arteriovenous shunts.

Very little information can be found in the literature on changes in the circulating blood volume (CBV) in the prolonged crush syndrome of the soft tissues (PCS) [8, 10, 11].

This paper describes the results of experiments to study the CBV and its components, together with other parameters of the circulation, in the dynamics of the acute period of the PCS of the limbs in dogs.

EXPERIMENTAL METHOD

Experiments were carried out on 13 dogs of both sexes weighing 6-14.6 kg. The CBV was determined by Gregersen's method [14] in the modification of Koziner and Rodionov [7]. The investigation of the CBV in the intial state was carried out twice or three times during adaptation of the animals to the experimental conditions and once immediately before crushing of the limb, 30 min after crushing, and every hour after application and removal of the press. One hind limb was crushed by a press with a force of 1000 kg · wt. for 4 h. The blood pressure in the femoral artery, pulse rate, velocity of the blood flow, and other parameters were measured simultaneously.

EXPERIMENTAL RESULTS

The initial value of the CBV in the dogs varied from 76.9 to 135.6 ml/kg (mean 93.4 ± 2.5 ml/kg), the circulating plasma volume (CPV) from 43.0 to 60.0 ml/kg (mean 55.6 ± 1.8 ml/kg), the circulating erythrocyte volume (CEV) from 28.5 to 48.5 ml/kg (mean 37.8 ± 1.7 ml/kg); no difference was found from the mean values obtained by other workers [5, 7] for dogs under normal conditions.

Crushing the limb was accompanied by intense excitation of the animals and violent attempts to free themselves from the press. The violent motor response was accompanied by barking and yelping and sometimes by involuntary micturition and defecation. These manifestations of excitation gradually subsided and 1-2 h after trauma most of the dogs exhibited only a weak defensive reaction; a state of general inhibition

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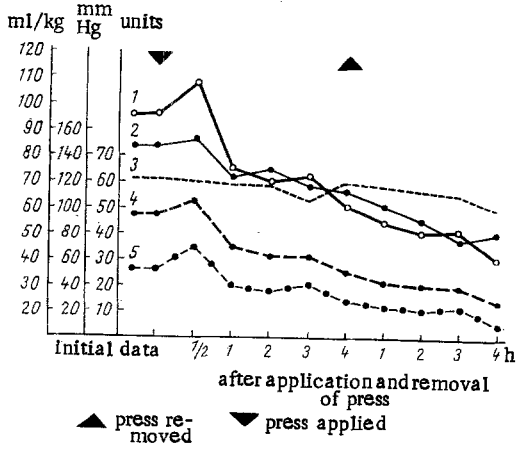

Fig. 1. Changes in volume of circulating blood and its components during development of the acute stage of the prolonged crush syndrome of the limbs in dogs (M ± m): 1) CBV (in ml/kg); 2) arterial pressure (in mm Hg); 3) hematocrit index (in units); 4) CPV (in ml/kg); CEV (in ml/kg).

developed progressively in some of them. These phenomena developed more rapidly in some of the animals, and after 20-45 min no signs of resistance were shown.

Immediately after the beginning of crushing of the limb a sharp rise of arterial pressure with an increase in amplitude of the pulse waves was observed in all animals without exception. This increase was short-lived and unstable; it fluctuated over a wide range. In the initial state the mean arterial pressure was 149.4 ± 1.4 mm Hg. Immediately after application of the press it rose on the average to 199.8 ± 5.3 mm Hg ($P < 0.01$). Then the arterial pressure continued to fall either gradually or catastrophically quickly. The initial increase in arterial pressure was accompanied by an increase in the velocity of the blood flow and vice versa. However, the disturbance of the velocity of the blood flow did not depend directly on the level of the arterial pressure and it did not determine the degree by which the pressure was lowered.

An increase in CBV was observed 30 min after application of the press, and this was followed by a marked decrease in both CBV and its components. After 1 h CBV was reduced on the average by 21.7% below the initial level, except in one dog in which it was increased by 19.1%.

The fall of CBV continued later and its difference from the initial level was 25.2% after 2 h, 21.9% after 3 h, and 35.1% after 4 h (Fig. 1). The arterial pressure fell simultaneously with it: the maximal decrease (by 19.7%) was observed 4 h after application of the press.

The hematocrit index showed no significant changes throughout the period of crushing and remained within the original limits.

Analysis of the results showed that in 12 animals hypovolemia was accompanied both by a decrease in the cell volume and a decrease in the CPV, with a relatively normal hematocrit index. A statistically significant decrease in CBV was accompanied by variations of the arterial pressure within the original limits and a decrease in the velocity of the blood flow by 1.5-2 times.

In one experiment hypervolemia on account of an increase in the cell volume but a relatively normal CPV was observed for 3 h after application of the press.

After removal of the press the arterial pressure, velocity of the blood flow, and CBV continued to fall. The CBV deficit 1 h after removal of the press was 32.6 ml/kg while at the same time CPV was reduced by 41.6% and CEV by 35.8% below the initial level ($P < 0.01$). The arterial pressure fell by this time to 105.6 ± 0.7 mm Hg, and the velocity of the blood flow was more than halved. A similar picture of change in the hemodynamic parameters was observed 2 and 3 h after removal of the press. Nine of the 13 animals (69.2%) had died 4 h after its removal and two dogs were in a preagonal state and died 10 h after trauma while another two, with a relatively normal arterial pressure and a decrease in CBV (to 42.5 and 48.2 ml/kg) and with a reduced hematocrit index (by 8.3 and 7.5 units), died toward the end of the first day after trauma.

Examination of the changes in the hemodynamic parameters clearly revealed a direct relationship between the changes in CBV, arterial pressure, velocity of the blood flow, and the severity of the animals' general condition. A direct relationship between these parameters has often been described [5, 12, 15] although some investigators [3, 4] deny it. The results of the present experiments confirm a direct relationship not only between CBV, the arterial pressure, and the severity of the shock but also between CBV, the velocity of the blood flow, and the pulse rate: with a decrease in CBV the pulse rate invariaby rises.

The investigation of CBV and the other hemodynamic parameters at frequent intervals of time revealed a short phase during the changes in CBV in the acute stage of the PCS when hypervolemia was accompanied by an increase in CEV and CPV, and increase in the arterial pressure and an increase in the velocity of the blood flow, followed immediately by marked and prolonged hypovolemia. The hypervolemia

was based on a reflex mechanism aimed at mobilizing the blood reserves (spasm of the vessels) and the department of hemodilution [2, 6], leading to an improvement in the blood supply to the brain, the heart muscle, and the other internal organs.

Later, with the development of the torpid phase of shock from compression pathological retention of blood took place, and the neurogenic spasm of the vessels was replaced by dilatation. An important role in this process was played by the skeletal muscles, skin, and some internal organs [4, 5, 12], in which dilatation of the capillaries led to slowing of the blood flow and to the development of stasis, with a change in the direction of the blood flow in the capillaries. At this time the many arteriovenous shunts in the intestine and skeletal muscles and also, to a lesser degree, in the brain, liver, and myocardium [9], began to function. With deepening of the torpid phase of shock the volume of blood in the spleen, liver, and lungs and, in particular, in the blood vessels of the muscles increased. These structures are the main pathological blood depots in second to third degree shock [5]. All these changes lead to a gradual or rapid decrease in the mass of blood in active circulation, with a consequent decrease in the return of venous blood to the heart and a decrease in the stroke volume of the heart [16] with the development of circulatory hypoxia [1].

In the later period of PCS, because of the pathological blood retention, the slowing of the blood flow, and the stasis in the capillaries and small veins the permeability of these vessels increased sharply. This phenomenon is regarded as being of paramount importance in the mechanism of development of shock by some authorities [1, 13].

The second, more prolonged period of PCS is thus characterized by the development of hypovolemia and hypotension, by a decrease in the velocity of the blood flow and, judging from data in the literature, by hypoxia. The changes observed at this stage of PCS of the limbs are decisive, and they determine the subsequent course of the traumatic shock.

The nervous system has some influence and some part to play during this period of trauma, but its relative importance is considerably reduced, if only by the fact that much of it is affected by profound inhibition which, at this stage of development of traumatic shock, plays a negative role.

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