BASIC FEATURES OF THE MICROCIRCULATION IN TRAUMATIC SHOCK AS REVEALED BY INTRAVITAL MICROSCOPY OF THE RAT MESOAPPENDIX

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Traumatic shock in rats is accompanied by slowing of the blood flow in the capillaries of the mesoappendix, activation of arteriovenous anastomoses, and the formation of aggregates of erythrocytes in the capillaries and small venules.

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To understand the pathogenesis of shock it is helpful to compare changes in the microcirculation and hemodynamics [15, 16, 18]. Most data on the microcirculation in shock have been obtained in experiments with hemorrhagic shock, and only a few in traumatic shock [6, 12, 20].

The blood flow in the vessels of the rat mesoappendix was studied in relation to the dynamics of shock. In the analysis of the experiments guidance was obtained from results described by other workers [1, 8, 20] for the structure and physiology of the terminal vessels.

EXPERIMENTAL METHOD

Altogether 45 experiments were performed on rats weighing 150-200 g lightly anesthetized with thiopental (10 mg/100 g body weight, 5% solution, intramuscularly). Like other barbiturates, thiopental causes only slight disturbance of the microcirculation, and reduces spontaneous vasomotor activity only to a small degree. Intravital microscopy of the mesenteric vessels and microphotography were carried out with the FMN-3 apparatus and the MBI-3 microscope (objective 10, ocular 7). Photographs were taken with a Zenit-3M camera on A-2 film (180 GOST units) with an exposure of 30 sec. The film was sensitized by treatment with phenidone developer [2]. The rats were photographed on a special methyl methacrylate vessel, and the mesentery, irrigated with Ringer's solution (37°), was placed in a compartment. The solution was passed through a heat-exchanger fitted with an electric heater. Shock was produced by Cannon's method after the narcotic action of the thiopental had begun to diminish. To assess the shock, kymographic recordings were made of the arterial pressure (in the carotid artery), the pulse, and respiration. Phases and periods analogous to those described for other animals were observed during the development of traumatic shock in the rat [3, 4].

EXPERIMENTAL RESULTS

The properties of the terminal vessels of the mesentery and of the circulation in them were determined in 8 control experiments. The following vessels were distinguished: arterioles (of the 1st, 2nd, and 3rd order), venules of different diameters (from 17 to 55), the network of capillaries, and arteriovenous anastomoses (Fig. 1). The latter were infrequent. The blood flow in the arterioles and venules was rapid during observations lasting 2-2.5 h, and it remained laminar. Usually 12-18 capillaries functioned in a field of vision. Individual erythrocytes in the capillaries moved between columns of plasma [10, 13]. In some experiments an attempt was made to assess the linear velocity of the blood flow from the movement of the erythrocytes. In venules 20-25μ in diameter this velocity was 0.5 mm/sec.

The microcirculation at different periods of development of traumatic shock was studied in 22 rats immediately after injury. The fall of arterial pressure (from 120 ± 5.4 to 47 ± 4.2 mm Hg) was accompanied by marked changes in the blood flow in all mesenteric vessels (Fig. 2). In the arterioles and venules

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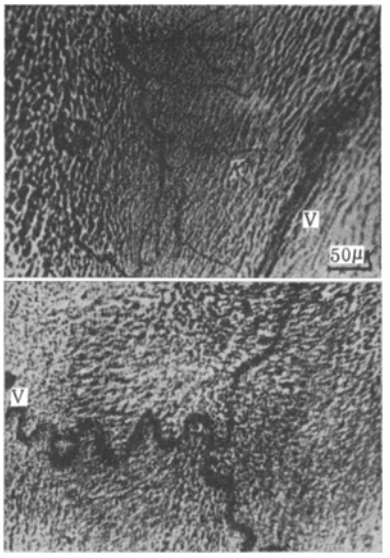

Fig. 1. Vascular system of the mesoappendix of a rat before shock. Above: arteriole (A), venule (V), and capillaries (K); below: venules of the 1st and 2nd order (V).

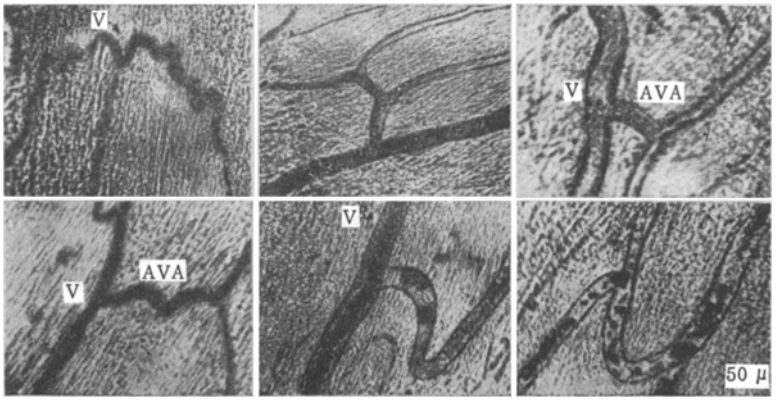

Fig. 2. Blood vessels of the rat mesoappendix in various periods of traumatic shock. On the left, beginning of the torpid phase: above—venules of the 2nd and 3rd order (V), below—arteriovenous anastomosis (AVA); in the center, period of stabilization of the torpid phase: above, vessels filled with erythrocytes and others containing plasma can be seen, below—initial stages of aggregation of erythrocytes in venule (V); on the right—end of the torpid phase: above—arteriovenous anastomosis (AVA), below—venule filled with aggregates of blood cells.

the laminar blood flow was replaced by scattered movement of the blood cells. Pendulum-like and sometimes retrograde movements of the blood were found in the venules, and the velocity of the blood flow was slowed to 0.01-0.03 mm/sec. In arterioles more than 25μ in diameter the blood flow remained fairly rapid, but as a rule in the arterioles of the 3rd order and the metarterioles it stopped altogether.

The change in velocity of the blood flow was evidently associated not only with a decrease in the pressure gradients between the arterial and venous divisions of the vascular system, but also with changes in viscosity of the blood resulting from the increased concentration of cells in the terminal vessels, although the definite hemoconcentration in this phase of shock was not yet accompanied by aggregation of erythrocytes.

Functioning capillaries disappeared after trauma: some collapsed, while others were blocked by cells. The capillary blood flow had not recovered by the time that the animals died. In most experiments a few minutes after injury an increase in activity of the arteriovenous anastomoses was observed: anasto-

moses of different diameters (from 20 to 40μ) were found, and the circulation in the mesoappendix at this stage of shock was carried on mainly through them. The possibility of activation of arteriovenous shunts when the capillary blood flow is disturbed during shock was postulated originally by Blalock [5] and subsequently confirmed by other investigators [12].

In the period of stabilization of the torpid phase of shock, when the arterial pressure was about 75 mm Hg, gradual recovery of the microcirculation was observed in some of the animals in which the manifestations of shock had disappeared. In most animals, however, despite some increase in the velocity of the blood in the 1st and 2rd order arterioles and venules 30-40 μ in diameter, further disturbances of the blood took place in the smaller arterioles and venules, and capillary stasis developed. Aggregation of erythrocytes in the venous division of the vascular system was discovered at this time, and the number of capillaries and small venules containing only plasma was increased.

Intravascular aggregation of erythrocytes in shock has been observed by several workers [7, 12, 19], who have regarded it as the cause of the decrease in the circulating blood volume, the lowering of the arterial pressure, and the anemia which are all characteristic of shock.

With a further increase in the severity of shock, the blood flow became still slower in all the mesenteric vessels, particularly in the venous portion: even in venules up to 20-30μ in diameter a pendulum-like movement of blood was observed, gradually changing to retrograde, and finally to stasis. Although the blood flow continued for longer in the arterioles, it was nevertheless much slower. When the systemic arterial pressure had fallen to 20-25 mm Hg, sometimes retrograde movement of blood was observed in the arterioles also. The blood flow at this time was maintained only in vessels over 60μ in diameter, as a result of the large arteriovenous anastomoses.

The late periods of shock were characterized by increased aggregation of erythrocytes, an increase in the size of the aggregates in the blood vessels, and by their condensation and their appearance not only in the venous, but also in the arterial portion of the vascular system.

In the terminal phase of shock stasis was found in all blood vessels of the mesoappendix.

Traumatic shock is thus characterized by marked disturbances of the circulation of the blood in the terminal vessels of the mesentery, appearing immediately after trauma. The most typical changes are a slowing of the velocity of the blood flow, an increase in the number of cells in the blood in the capillaries and small veins, an increase in viscosity of the blood, aggregation of erythrocytes, and, finally, complete cessation of the capillary blood flow.

There is good reason for interest shown in the use of low-molecular weight dextrans in shock. These substances have a de-aggregating action and thereby facilitate an increase in the volume of blood in active circulation [9, 14, 17]. In the next series of experiments changes in the microcirculation in the rat mesoappendix were studied after injection of low-molecular weight polyvinol at the end of the torpid phase of shock. Polyvinol with a molecular weight of 10,000 (batch No. 27, Leningrad Blood Transfusion Institute) was injected by intravenous drip at the rate of 1 ml/100 g body weight.

These experiments showed that while the polyvinol was being administered the velocity of the blood flow in the terminal vessels increased, the number of aggregates of erythrocytes decreased, and in some cases they disappeared altogether: the capillary blood flow began to recover. However, 15-20 min after the end of polyvinol administration, aggregates of erythrocytes reappeared in the small venules, and later in the arterioles, and the capillary blood flow stopped. These phenomena were accompanied by a secondary decrease in arterial pressure, worsening of the general condition, and death of the animals.

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