MICROCIRCULATION AND PARTIAL OXYGEN PRESSURE IN THE CEREBRAL CORTEX OF RATS WITH HEMORRHAGIC SHOCK

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KEY WORDS: shock; blood loss; microcirculation; partial pressure of oxygen; compensation.

The writers showed previously [1] that changes in the microcirculation in hemorrhagic shock in a vitally important organ such as the liver differ significantly from those in organs not vitally important (the mesentery, skeletal muscles). The blood flow in the microcirculation of the liver falls only slightly and was maintained at a subnormal level for a long time. In the mesentery and skeletal muscles this phase of compensation was totally absent, and the blood flow in the microvessels of these organs fell progressively until it ceased altogether. The character of changes in the microcirculation corresponded exactly to the dynamics of changes in the partial pressure of oxygen in these organs.

In this investigation, a continuation of those conducted previously, the microcirculation was studied in the pia mater and the partial pressure of oxygen in the parietal cortex.

EXPERIMENTAL METHOD

Experiments were carried out on 20 male Wistar rats weighing 340-360 g under pentobarbital anesthesia (4.5 mg/100 g body weight). Before the experiment began the animals were given an intravenous injection of heparin in a dose of 150 i.u./100 g body weight. Hemorrhagic shock was induced by the method of Wiggers and Fein [2]. The microcirculation was studied by contact luminescence biomicroscopy with the LUMAM I-3 microscope. The partial pressure of oxygen was recorded in the course of shock on the LP7e polarograph.

EXPERIMENTAL RESULTS

The microcirculation of the pia mater of the rat brain has a reticular type of structure: The venules form tree-like ramifications whereas arterioles, when they branch, form numerous arteriolo-arteriolar anastomoses. Various combinations of these formations endow the microcirculation of the pia mater with the character of a single-layered vascular network, constructed without any definite plan.

Under normal conditions the blood flow in the microvessels of the pia mater was characterized by a high velocity, homogeneous structure, and the presence of an axial flow of erythrocytes (Fig. 1a). A sharp fall in the velocity of the blood flow which in some cases became jerking in character, was observed 2-3 min after the beginning of blood loss, when the systemic arterial pressure (BP) had fallen to 35 mm Hg. Considerable vasoconstriction and tissue ischemia were observed. The blood flow became granular in character and the axial flow of the erythrocytes disappeared (Fig. 1b). During the very first minutes of development of shock a marked response of the microcirculation to blood loss thus appeared. This led to a considerable fall in the partial pressure of oxygen in the cerebral cortex (Table 1).

During the next 4-5 h the blood flow in the microvessels remained quite high, stable, and homogeneous and it had an ill-defined granular structure. Slight hemodilution was observed. This indicated the onset of a phase of compensation. A general estimate of the blood flow at this time gave a value of 70-80% (at the beginning of the compensation phase) to 40-50% of the initial level (at the end of the compensation phase). The onset of the phase of compensation

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TABLE 1. Partial Pressure of Oxygen in Cerebral Cortex of Rats at Different Periods of Hemorrhagic Shock (M ± m)

Time of measurement	pO_2, mm Hg
Initial value	38,2±2,7
After blood loss:	
30 min	11,5±1,5
1 h	14,5±1,7
2 h	13,8±2,1
3 h	12,5±1,8
4 h	8,4±0,7
5 h	6,4±0,5
6 h	5,7±0,6
7 h	4,3±0,3

Legend. P < 0.001 compared with initial level.

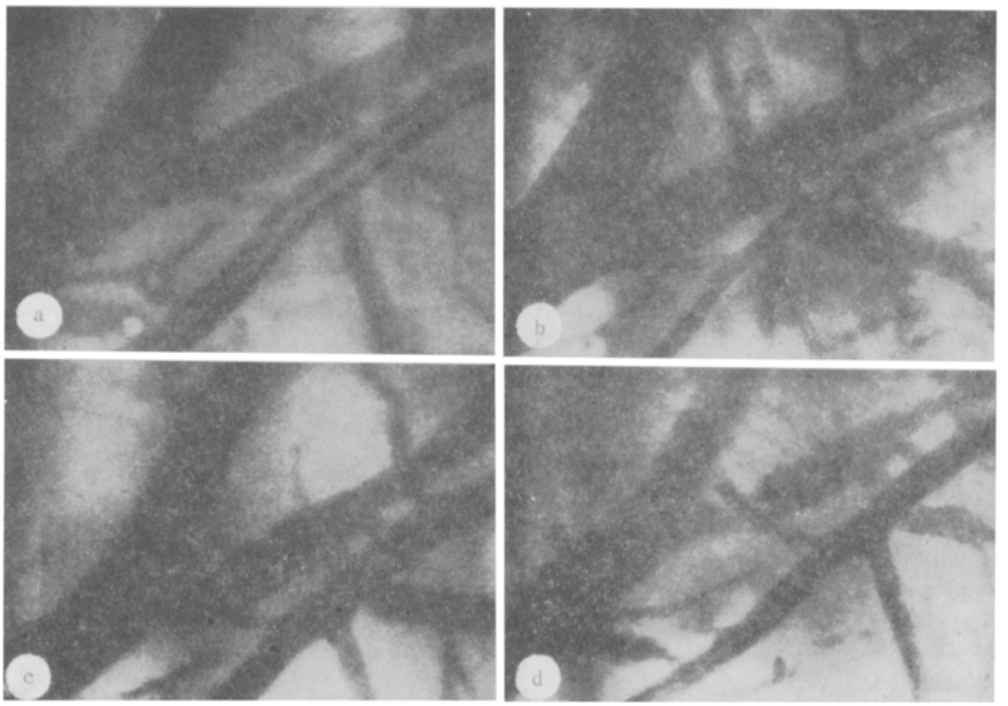

Fig. 1. Photomicrograph of vessels of rat pia mater: a) before blood loss, b) 5 min, c) 30 min, and d) 6 h after blood loss (1 h before animal's death). Magnification 80 ×.

of the circulation at the microcirculatory level led, as already mentioned, to stabilization of the partial pressure of oxygen at a subnormal level. Not until 1-2 h before death of the animals, when shock entered on the terminal phase of its development, was a sharp deterioration of the blood flow observed. Its velocity fell progressively, the structure of the blood flow became increasingly granular in character, and aggregation of the erythrocytes developed. Moveoment of the blood along the microvessels became jerking, and later, pendulum-like. Stasis developed in many vessels (Fig. 1d). Disturbance of the microcirculation was accompanied by a fall in the partial pressure of oxygen to zero, and the animals died.

This investigation confirmed the previous view [1] that there are two types of response of the microcirculation to blood loss. The first type is observed in organs that are not vitally important and it is characterized by a rapid and progressive fall of the blood flow to zero without stabilization at the subnormal level. In other words, the phase of compensation of the circulation is not present in these organs. The second type is observed in vitally important organs essential to survival of the organism, and changes in the microcirculation

in them are not so dramatic in character: Between 30 and 60 min after the initial impairment of the blood flow in response to blood loss a phase of compensation arises and the blood flow stabilizes at a subnormal level, which continues until the onset of shock in the terminal phase of its development.

The authors emphasize the relativity of the concept of "compensation phase" for different organs in hemorrhagic shock. When we speak of a phase of compensation of the circulation, we mean that this concept is applicable, not to the circulatory system as a whole, but only to individual organs with a primary role in the survival of the organism under conditions of hemorrhagic shock.

LITERATURE CITED

1. V. I. Udovichenko and Yu. M. Shtykhno, Anest. Reanimatol., No. 4, 36 (1981).
2. C. J. Wiggers, Physiology of Shock, London (1950).

STRUCTURAL CHANGES IN NEOCORTICAL SYNAPSES AFTER RESUSCITATION

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KEY WORDS: brain; terminal state; postresuscitation period.

The recovery of brain functions after resuscitation from the terminal state is determined by the degree of damage to its structure and the development of compensatory and repair processes in the brain after resuscitation [2]. The restoration and formation of stable structural-functional interrelations and the organization of assemblages of functionally connected neurons after damage depend on the state of the synaptic apparatus [1]. Data on interneuronal contacts in the postresuscitation period are fragmentary and do not reflect the whole complexity and importance of structural damage and compensatory reconstruction of synapses in the pathogenesis of postresuscitation encephalopathy and mechanisms of rehabilitation of the brain after resuscitation [3].

The aim of the present investigation was accordingly to study the number and ultrastructure of synapses in the cerebral cortex during the course of the postresuscitation period.

EXPERIMENTAL METHOD

Male albino rats weighing 170-230 g were anesthetized with ether. Three intact animals served as the control, 16 rats were resuscitated by Negovskii's method after total mechanical asphyxia caused by clamping the intubation tube for 6 min [2]. At the end of the experimental period the brain was perfused with a mixture of 4% paraform and 1% glutaraldehyde in phosphate buffer, pH 7.4. Material was taken 6 h and 1, 3-4, and 6-7 days after resuscitation. Wedges of neocortex were oriented so that the outer layers of the cortex were at the base of the wedge and the boundary with the white matter was at the apex. Some material was processed in the ordinary way for survey ultramicroscopy. The rest was not fixed with osmium, but was stained in the dehydration stage in a 5% solution of phosphotungstic acid (PTA) in absolute alcohol for 3 h. The oriented specimens were embedded in a mixture of Epon and Araldite. Blocks were cut in the tangential plane from the side of the base of the wedge. Ultrathin sections were examined and synapses photographed in the first layer of the sensomotor cortex on the EMV-100LM electron microscope. In sections from each animal stained with PTA 10 randomly chosen fields of vision were photographed under a standard magnification of 15,000 ×. Quantitative analysis was carried out on the negative enlarged four times. The total number of PTA-positive contacts and the numbers of identified and unidentified, straight and curved, symmetrical, asymmetrical, and mixed contacts with respect to paramembranous condensation were

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