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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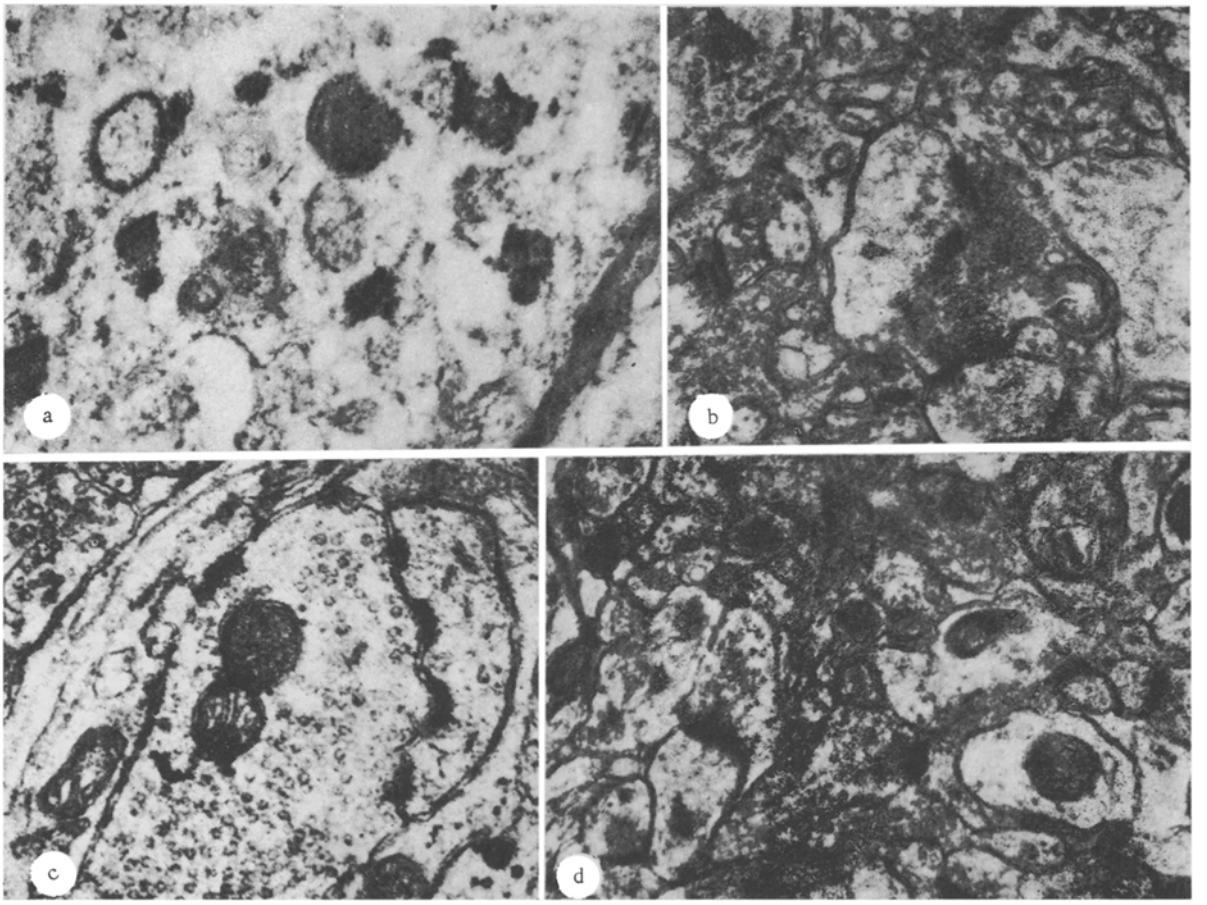

Fig. 1. Interneuronal contacts in neocortex in postresuscitation period. a) Neuropil in neocortex of control animals (PTA, 20,000 ×); b) focal type of destruction (25,000 ×); c) hypertrophy of presynaptic zone (35,000 ×); d) increase in number of small synapses (20,000 ×).

studied; the extensiveness of the synaptic contacts was calculated in conventional units from the number of intersections with a control grid with a 3-mm step. The numerical results were subjected to statistical analysis.

EXPERIMENTAL RESULTS

Mainly synapses with an asymmetrical composition of the contact were found in the neocortex of the control animals, and straight synaptic contacts with three or four intersections with the control grid were most frequently found (Fig. 1a). Among the identified synaptic contacts a mixed composition was rarely seen. Most interneuronal junctions were formed by synapses with one or two active zones.

Destructive changes in the neocortex 6 h after resuscitation led to a decrease of 41.9% ($P < 0.05$) in the total number of contacts, the number of small synapses was sharply reduced, and curved synapses predominated. The ratio between symmetrical and asymmetrical contacts was shifted in favor of the latter. A tendency was noted for an increase in the number of larger synapses — with 5-6, 7-8, or more intersections. During the 1st day the destructive changes intensified: The total number of contacts fell by 51.3% ($P < 0.05$) on account of all types. The synapses underwent predominantly a pale type of destruction, and only rarely was a focal type observed (Fig. 1b).

On the 3rd day the tendency for the number of synapses with 5-6 intersections to increase was more marked. The total number of synapses was increased a little to 63.2% ($P < 0.001$) of the control . Synapses with two or three active zones predominated. A study of material stained by the usual method showed that the structurally changed synapses could be divided into two groups: those with a hypertrophied presynaptic zone and with an increased number of active zones, mitochondria, and synaptic vesicles (Fig. 1c) and synapses undergoing pale and focal types of destruction at different stages of the pathological process.

On the 6th-7th day the deficit of synapses was reduced to 34.1% ($P < 0.05$). The increase in the number of synapses took place on account of small synapses with 1-2 and 3-4 intersections with the control grid (Fig. 1d); the number of large synapses was a little reduced. The number of curved contacts increased. In this series of experiments, just as on the 1st-4th day, synapses with asymmetrical composition predominated but there was an appreciable increase in the number of symmetrical contacts. Medium-sized synapses predominated although there was a fair number of large contacts. The number of curved synapses increased to 4.9 ± 0.34 ($P < 0.01$) per field of vision (106.5% of the control), and the number of straight contacts in this period amounted to 57.1% of the control ($P < 0.05$). The appearance of small synapses and the increase in the number of contacts with symmetrical composition are evidence of the activation of synaptogenesis [1, 4].

Rapidly developing generalized changes in the ultrastructure of synapses and their death were thus observed in the cortex and these changes play an important role in the pathogenesis of postresuscitation encephalopathy. Meanwhile the plastic properties of the synaptic apparatus develop rapidly and become apparent in the neocortex after resuscitation. During the first 3 days mainly compensatory hypertrophy of interneuronal contacts is found, but on the 6th-7th day synaptogenesis is well marked. Structural changes in the synaptic apparatus reflect compensatory and reparative changes in neocortical neurons and constitute an important mechanism leading to restoration of brain functions in the postresuscitation period.

LITERATURE CITED

1. N. N. Bogolepov, Ultrastructure of Synapses under Normal and Pathological Conditions [in Russian], Moscow (1975).
2. V. A. Negovskii, A. M. Gurvich, and E. S. Zolotokrylina, Postresuscitation Sickness [in Russian], Moscow (1979).
3. V. V. Semchenko, in: Active Functions of the Brain [in Russian], Baku (1980), p. 167.
4. S. E. Dyson et al., Brain Res., 114, 365 (1976).

EFFECT OF PROSTAGLANDINS E_1 AND E_2 ON PLATELET SHAPE AND AGGREGATION

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In the process of platelet aggregation an essential role is played by prostaglandins (PG). It has been shown that PGE_1 inhibits [6, 8] whereas PGE_2 stimulates platelet aggregation induced by ADP [6]. The mechanism of this effect has not yet been fully explained. The work of Born has shown the importance of changes in the shape of these cells for the development of aggregation [1]. Changes in platelet shape have been shown to lead to marked aggregation [8].

With these facts in mind it was decided to study the effect of PGE_1 and PGE_2 on the shape of platelets, including platelets activated by exogenous ADP and changes in pH.

EXPERIMENTAL METHOD

Platelets were obtained from rat blood taken from the abdominal aorta. A 3.8% solution of sodium citrate was used as anticoagulant (ratio of blood to anticoagulant 9:1). Platelet-rich plasma was obtained from citrated blood by centrifugation at 250g for 6 min at room temperature. Platelet-rich plasma diluted 1:6 with Tyrode's solution (without magnesium), pH 7.4, was used in the experiments. Fluorescence and transmittance of the platelet suspension were

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