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PARAMEMBRANOUS NEUROFILAMENTOUS STRUCTURES OF CEREBRAL CORTICAL SYNAPSES DURING ISCHEMIA AND THE EARLY POSTISCHEMIC PERIOD

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KEY WORDS: cerebral cortex, synapses, ischemia, postischemic period.

Dense projections (DP) of the presynaptic grid (PG), the substance of the synaptic space, and the postsynaptic condensation (PC) constitute the paramembranous neurofilamentous system of subsynaptic units (SSU) of interneuronal junctions and maintain the integrative function of the brain [7, 9, 11]. Being highly labile formations, the structural components of SSU undergo marked changes during embryonic and postnatal development and during exposure to various factors [3, 8, 10]. It has been shown that in total ischemia and in the period of recirculation, interneuronal integration is disturbed [2]. A definite role in the development of this process may perhaps be played by changes in SSU of interneuronal connections.

The aim of this investigation was to study structural changes in SSU of interneuronal junctions in the cerebral cortex during short-term total ischemia and in the early postischemic period.

EXPERIMENTAL PERIOD

Experiments were carried out on 16 male albino rats weighing 190-210 g under ether anesthesia. Total ischemia and the postischemic period were simulated by inducing clinical death for 5 min from blood loss, followed by resuscitation [6]. The brain was fixed by perfusion with a mixture of 4% paraformaldehyde and 1% glutaraldehyde in phosphate buffer (pH 7.4) with sucrose at the end of ischemia, and after 5, 30, and 90 min of the postischemic period. Oriented pieces of sensomotor cortex were stained while being taken through the 100% ethanol stage in a 5% solution of phosphotungstic acid (PTA) and embedded in plane-parallel order in Araldite. Tangential ultrathin sections were cut through the molecular layer of the neocortex and 15 random fields of neuropil were photographed from one animal under magnification of 15,000 of the ÉVM-100LM microscope. The total number of PTA-positive junctions and the number of indefinite and definite synapses were counted per 100 μ^2 of neuropil from negative under a final magnification of 30,000×. Among the definite synapses, asymmetrical junctions of the A, B, and C type and symmetrical junctions of the D type were distinguished by the degree of definiteness of shape of the PG and the height of DP [8]. The data were subjected to statistical analysis.

EXPERIMENTAL RESULTS

The total number of PTA-positive junctions and the number of indefinite and definite symmetrical synapses during ischemia and in the early postischemic period did not differ from their number in the cerebral cortex of the control animals (Table 1). The total number of definite junctions was reduced only after recirculation for 90 min, due to reduction of the

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TABLE 1. Number of PTA-Positive Junctions in Neuropil of Molecular Layer of Rat Cerebral Cortex in Postischemic Period ($\bar{X} + S_{\bar{X}}$)

Parameter	Number of junctions per 100 μ^2 of neuropil				
	control	ischemia	postischemic period		
			5 min	30 min	90 min
Total number of junctions	39,1±4,4	37,8±4,0	37,7±3,6	37,8±2,8	33,1±2,4
Indefinite junctions	15,2±3,6	15,3±0,9	14,6±1,1	13,7±1,0	15,3±0,4
Definite junctions: asymmetrical	23,9±1,7	22,5±3,4	23,1±4,2	24,1±3,4	17,8±0,8*
	18,4±0,6	17,2±1,7	18,3±2,5	19,1±2,5	13,4±1,1*
A	6,7±0,7	4,8±0,5*	5,8±0,7	6,1±0,5	6,7±0,8
B	6,7±0,8	9,5±0,7*	9,7±0,8*	9,9±0,9*	4,8±0,2*
C	5,0±0,4	2,9±0,3*	2,8±0,4*	3,1±0,3*	1,9±0,2*
symmetrical					
D	5,5±0,4	5,3±0,8	4,8±0,7	5,0±0,7	4,4±0,8

Legend. Significance of differences between parameters calculated relative to control; *P < 0.05.

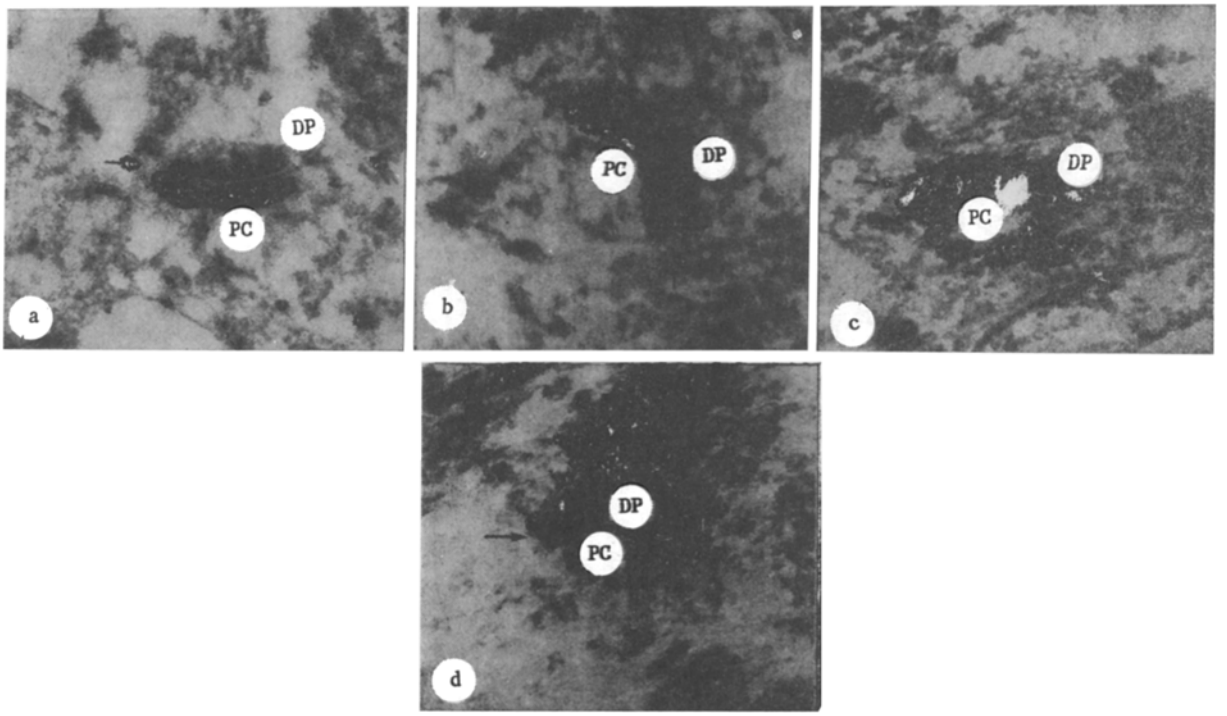

Fig. 1. Paramembranous neurofilamentous structures (SSU) of synapses in molecular layer of rat cerebral cortex in postischemic period: a) Clearly outlined, maximally developed structures of SSU of a type A synapse (control); b) decrease in clarity of outlines of dense projections of presynaptic grid of type A synapse (30 min); c, d) decrease in clarity of outlines of structures of SSU in synapses of types B and C, respectively (90 min). Arrow indicates substance of synaptic space. Stained with PTA, 58,000 ×.

number of asymmetrical synapses. However, in the period of ischemia and recirculation considerable changes were observed in the number of the various types of asymmetrical junctions.

In ischemia the number of junctions with the most fully developed SSU (type A; Fig. 1a) — functionally mature [8] — was reduced by 28.4%. There was an even greater decrease in the number of asymmetrical synapses with minimally developed SSU (type C). Meanwhile the number of more mature intermediate junctions (type B) was considerably increased (by 41.8%). Qualitative analysis of the electron micrographs revealed a decrease in clarity of the outlines of DP of the presynaptic grid of only individual junctions. The structure of PC and of the substance of the synaptic space of the interneuronal synapses showed no significant change.

The electron-cytochemical and morphometric characteristics of the synaptic pool were very similar after recirculation for 5 and 30 min. By contrast with the period of ischemia, on resumption of the cerebral circulation the number of type A junctions was restored to the lower limits of the value of this parameter in the control. However, the number of type C junctions was just as low as in ischemia, and the number of type B synapses was just as high. A small proportion of interneuronal junctions was observed with indistinct homogeneous staining of DP of the presynaptic grid (Fig. 1b). The structure of the substance of the synaptic chain and of PC in most interneuronal junctions was indistinguishable from the control.

The marked reduction (by 25.4%) in the number of definite junctions after 90 min of the postischemic period was due mainly to reduction (by 27.2%) in the number of asymmetrical synapses, and to a much lesser degree, on account of symmetrical junctions, for the latter showed only a tendency for their number to decrease. Changes in the number of the different types of asymmetrical junctions likewise varied. By comparison with the previous period the number of type B junctions was reduced by more than half (Fig. 1c). Under these circumstances it fell below the control level. The degree of reduction of the number of type C synapses was enhanced (Fig. 1d). However, the number of type A junctions was the same as in the control. The fraction of synapses with reduced clarity of outlines of their SSU increased. A reduction in the height of DP and PC of the interneuronal junctions was found, together with an increase in width of synaptic space in the junctions with destructively changed DP.

According to the results of this investigation, indefinite and symmetrical, functionally immature [8] synapses are most resistant to ischemia and to postischemic exposure to various factors. In the period of ischemia reorganization of the asymmetrical synapses begins. Since their total number remained unchanged, the increase in number of type B junctions at this time was due to transformation of synapses of A and C types into type B. This process is linked above all with reorganization of SSU of the interneuronal junctions, the inducing mechanism of which is evidently a sharp increase in the intensity of lipid peroxidation processes in membranous structures and conformational changes in proteins of paramembranous neurofilaments of interneuronal junctions [5]. Partial restoration of the number of synapses with maximally developed DT during the first 30 min took place as a result of reorganization of SSU belonging to the fraction of symmetrical junctions. The possibility of rapid reorganization of the paramembranous neurofilamentous structures of synapses in the CNS is confirmed by data in the literature [11].

The absence of any marked and widespread ultrastructural and electron-cytochemical damage to the structure of the synapses suggests that the predominant factor in the mechanism of disturbance of the integrative function of the neocortex in ischemia and in the first 30 min of recirculation is sudden autointoxication [4], which causes inhibition of the enzyme systems of the synapses.

The marked reduction in the number of definite PTA-positive junctions after 90 min of ischemia is due to loss of the typical structure of the pre- and postsynaptic paramembranous condensations by a group of interneuronal junctions. According to the electrophysiological data, from the 30th to the 90th minute of recirculation specific activity of cortical neurons is rapidly restored [1]. Consequently, this process is accompanied by destruction of the protein component of the paramembranous condensations of a considerable number of synapses, evidently with their subsequent exclusion from the integrative function. The increase in the number of interneuronal junctions with indistinct homogeneous staining of SSU indicates that the comparatively rapid vulnerability of the paramembranous neurofilaments in the synapses is connected with the hydrolytic action of acid proteases on the loosely packed protein structure of these formations. The latter is facilitated by preliminary peroxidation of the synaptic membranes and disturbance of their connection with the neurofilaments of the cytoskeleton [5]. Meanwhile, maintenance of the number of type A junctions at the control level as a result of the conversion of a proportion of the functionally less mature junctions into them, as the results of electrocorticography show [1], leads to considerable restoration of the integrative function of the cerebral cortex toward the 90th minute after ischemia. Compensatory reorganization of the asymmetrical synapses in this period is also based on changes in the density of paramembranous neurofilaments of interneuronal synapses.

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EFFECT OF LOW-FREQUENCY ULTRASOUND ON THE PLEURA AND ADJACENT LUNG TISSUE

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The use of low-frequency ultrasound (US) in clinical practice has expanded widely in the last decade. To treat open suppurative lesions US is used with an energy that has the property of actively moving fluid and creating cavitation, and so on [3, 6]. The bactericidal action of low-frequency US has been confirmed by clinical and experimental research [5, 7]. Under the influence of ultrasound waves, intracellular metabolism is accelerated and energy processes stimulated [3, 7]. Low-frequency US is used to treat biological tissues, during pleurectomy, and also to prevent suppurative complications and for the treatment of empyema [1, 4, 9].

For the reasons given above there is good reason to use the energy of low-frequency US for the treatment of chronic dieases of the pleura. However, the possibility that ultrasonic radiation may have a harmful action on the serous and mucous membranes must be taken into account. It has been shown that if low-frequency US is passed through solutions in the abdominal cavity of rabbits, foci of necrosis and hemorrhage are found in the zone of exposure, especially if the wave guide is in contact with the abdominal wall [8]. Low-frequency US also acts on the bronchopulmonary system when applied by the endobronchial route [2]. It has been shown experimentally that a single or multiple exposure to ultrasound for 30 sec causes virtually no pathological changes in the bronchopulmonary tissue, but lengthening the exposure is accompanied by destruction and desquamation of the bronchial epithelium, by necrosis of the cells of the mucous membrane, and by the development of an inflammatory reaction in the zone of exposure.

There is no information in the literature on the effect of low-frequency US on tissues or on its use in closed cavities such as the pleural cavity.

The aim of this investigation was to study morphological changes in the pleura and adjacent lung parenchyma and also to determine optical therapeutic schedules for experimental exposure to low-frequency US.

EXPERIMENTAL METHOD

Experiments were carried out on 58 male rabbits aged 4-5 months and weighing from 2.5 to 3 kg. The animals were divided into two groups: group 1) control, consisting of eight intact animals; group 2) 50 animals, in which the harmful action of US on the pleura and lung tissue was investigated. The control and experimental animals were kept under identical conditions.

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