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SUBCELLULAR MECHANISMS OF THE ACTION OF WEAK SUPERFLOW-FREQUENCY ELECTROMAGNETIC FIELDS ON THE CEREBRAL CORTEX

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Much interest is currently being displayed in the study of the action of electromagnetic fields (EMF) with parameters close to those of EMF of natural geophysical origin. Advantages of exposure to EMF include their high penetrating capacity, a no-contact method of application, and the possibility of regulating their parameters and conditions within wide limits. Accordingly, research into the action of EMF on subcellular changes in brain structures lying at the basis of changes in functional activity and the state of the CNS assumes particular importance.

We know that EMF can give rise to morphological changes in brain structures [3, 7-12]. However, data in the literature are clearly insufficient to explain the actual mechanism of the ultrastructural changes arising in the brain under the influence of EMF in general, and of weak superlow-frequency EMF (WSLEMF) in particular. Preference in research is often awarded to the study of one particular component of nerve tissue or a neuron, and the majority of investigations have been conducted at the light-microscopic level. The results have been interpreted variously, they are often contradictory, and this is evidence of the complexity of the problem, the fine nonlinear dependence on a multitude of parameters, the different orientation adopted toward the elucidation of morphological responses of the CNS, and different techniques of approach.

Moreover, the electromagnetic fields used by investigators differ sharply in their parameters from EMF of geophysical origin. The aim of this investigation was to study the effect of WSLEMF on the ultrastructure of the cerebral cortex.

EXPERIMENTAL METHODS

The brain of rabbits and rats, previously subjected to single (for 4 h) and repeated (for 4 h daily for 5 days) exposure to a square-pulse WSLEMF, with calculated induction of

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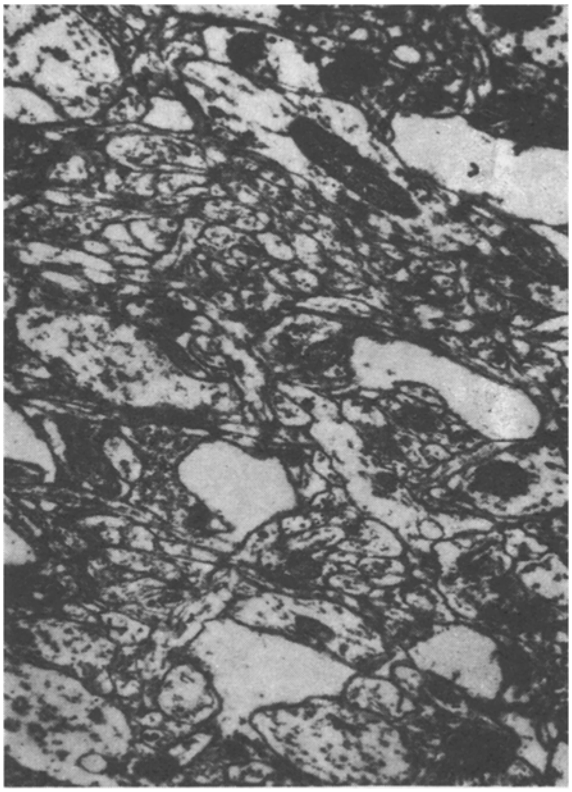

Fig. 1. Ultrastructural changes in glia after a single exposure to WSLEMF. Swelling and translucency of matrix of astrocytic processes (AP) in middle layers of rabbit sensomotor cortex. Magnification 10,000×.

a uniform magnetic field of 0.5 mT, in a volume of 10^{-3} M^3, and with a fixed frequency of 3.12 Hz, was used for morphological analysis. (The animals were irradiated with a WSLEMF by A. Malinin.)

Altogether 38 animals were used, 34 of them experimental and four control. Pieces of nerve tissue were taken from the sensomotor and auditory cortex for electron-microscopic investigation 1 h and 3, 8, 13, and 15 days after the last exposure to the EMF. Material was fixed by perfusion of the brain vessels with a mixture of 1% paraformaldehyde and 1% glutaraldehyde in 0.1 M phosphate buffer, pH 7.2-7.4, followed by postfixation with a 1% solution of osmium tetroxide in the same buffer. The material was dehydrated and stained by the usual method. Identification of the layers was monitored by light microscopy of semithin sections, stained with toluidine blue. Ultrathin sections were examined in the IEM-100B microscope with accelerating voltage of 80 kV.

EXPERIMENTAL RESULTS

The results showed that different structures of cerebral cortical nerve tissue differ in their sensitivity to WSLEMF with different time schedules. For instance, ultrastructural analysis showed that a single exposure to WSLEMF induced only a glial reaction in cerebral cortical nerve tissue, and ultrastructural changes were observed only in astrocytes and their processes (Fig. 1). Swelling and translucency of the matrix of the astrocytic processes, the appearance of vacuole-like outlines in them, and a decrease in the number of organelles in the cytoplasm of the astrocytes were observed. With an increase in the duration of exposure to the EMF with repeated irradiation, besides the response of the glial cells, submicroscopic structural changes also were found in the neuron bodies in the cortex: multiple invaginations of the nuclear membrane, ectopia of the nucleolus, enlargement of vacuoles and lysosomes, and dilatation of the channels of the endoplasmic reticulum (Fig. 2). These structural changes are evidence of changes in the nucleo-cytoplasmic relations that lie at the basis of biosynthetic processes. WSLEMF thus induces ultrastructural changes in the cerebral cortex whose intensity in different elements of nerve tissue depends on the duration of ex-

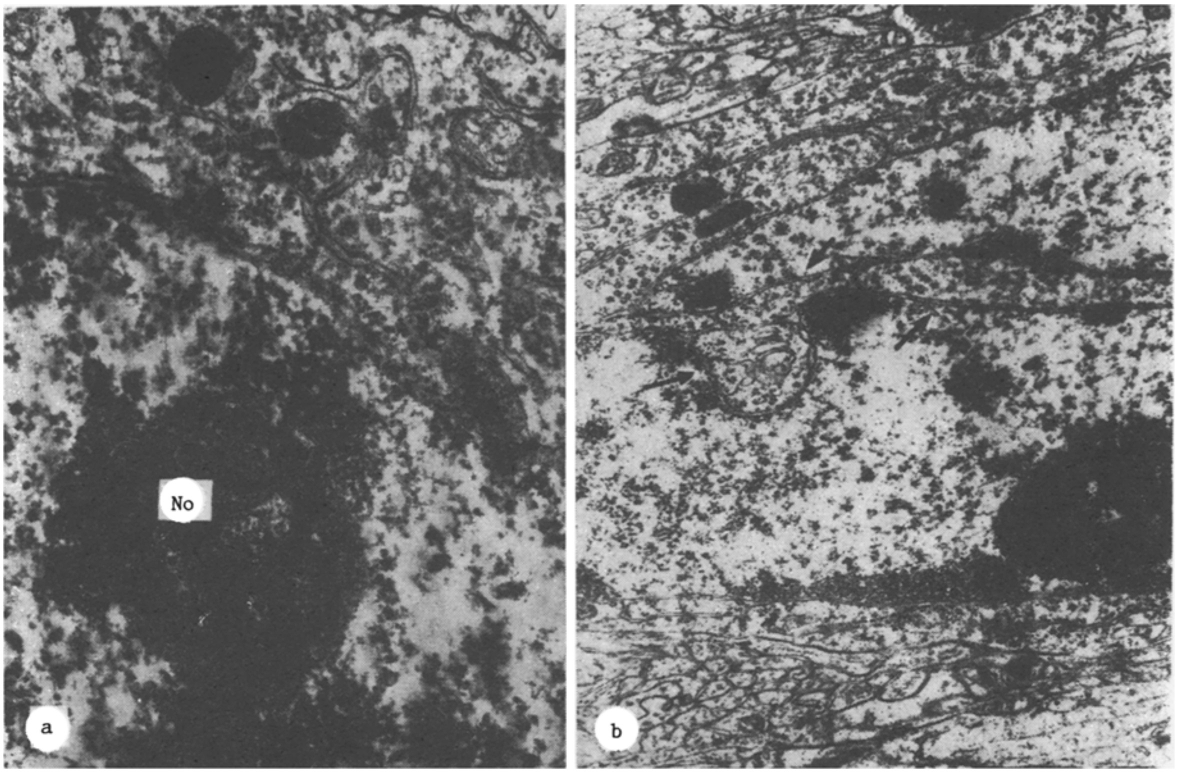

Fig. 2. Ultrastructural changes in neurons in middle layers of rat sensomotor cortex after repeated exposure to WSLEMF. a, b) Ectopia of nucleolus (No). Magnification 22,500×; b) multiple invaginations of nuclear membrane (arrows). Magnification 16,300×.

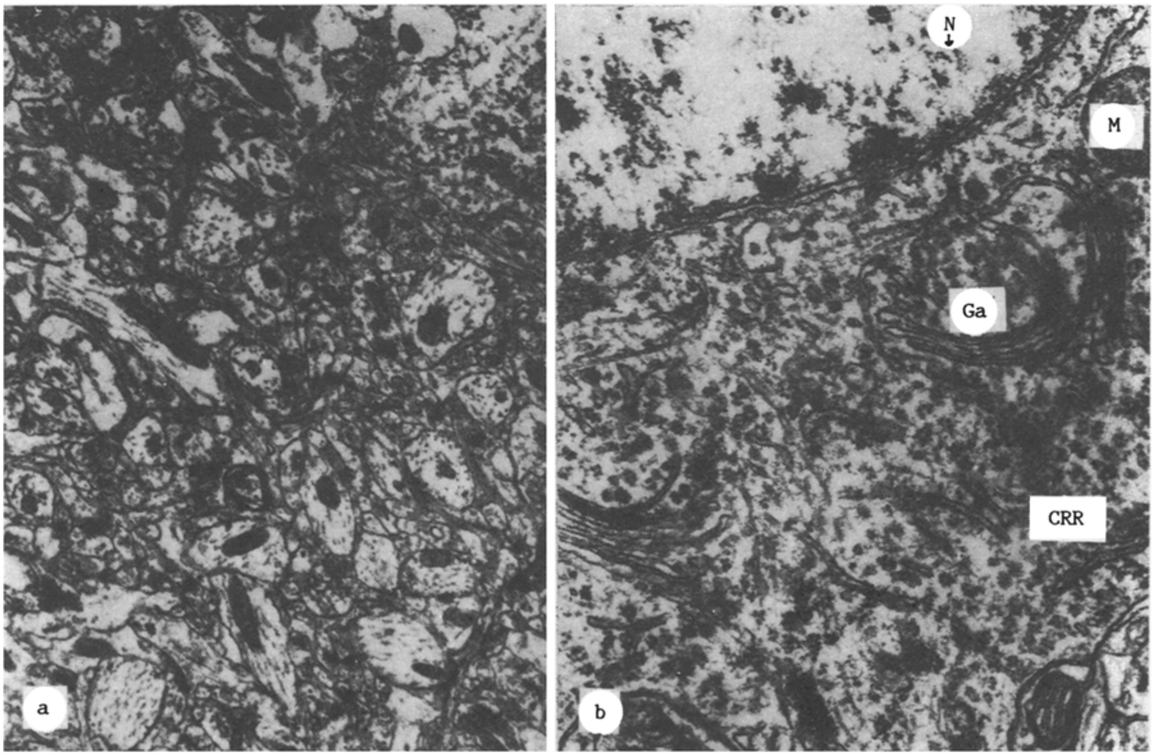

Fig. 3. Restoration of nerve tissue after exposure to WSLEMF. a) Absence of glial response in neuropil in middle layers of rabbit sensomotor cortex 3 days after single exposure to WSLEMF. Magnification 5500×; b) normalization of neuronal ultrastructure in middle layers of rat sensomotor cortex 15 days after repeated exposure to WSLEMF. Magnification 16,300×. N) Nucleus, Ga) Golgi apparatus, CRR) cisterns of rough endoplasmic reticulum, M) mitochondrion.

posure. The glia responds most intensively followed by neurons. WSLEMF has a distinct effect on metabolism of the cortical nerve cells and on functional activity of the neuroglial complex [4].

The study of the time course of the ultrastructural changes after the end of single and repeated exposure to WSLEMF showed that 3 days after single and 15 days after repeated exposure the normal ultrastructure of the nerve tissue was completely restored (Fig. 3). Active intracellular repair processes took the form of a complex series of changes involving subcellular structures of both neurons and glia. No destructive or pathological changes were found in the cortical tissue, evidence that the responses evoked are functional and structural in character. No structural changes likewise were found in the synapses; the ultrastructures of the pre- and postsynaptic components of the synapses were indistinguishable from normal. The resistance of the synapses can be explained by the absence of their response to WSLEMF of the parameters used. As a result of exposure to a steady EMF with inductance of 60 mT, under more stringent conditions, marked changes were found in the synaptic ultrastructure of the cerebral cortex [8].

Dependence of the frequency and severity of the ultrastructural changes in nerve tissue and also of the time course of repair processes on the parameters of the EMF to which the animals were exposed has been noted by several investigators [7, 8, 14]. Some of them [7] have concluded that the action of the EMF is specific. Others consider that changes in the glia and glio-neuronal relations found in the brain under the light microscope are a manifestation of a single nonspecific response of adaptive character [2, 13], connected with increased permeability of the blood-brain barrier [15]. The primary response of ultrastructures of glial elements in the brain is known to be induced by other biophysical agents also. For instance, weak direct electric currents, depending on the parameters of their action on the brain, induce functional ultrastructural changes initially in glial cells, but later in neurons; the response of the nerve tissue, moreover, is reversible [1, 5, 6].

Our electron-microscopic data are evidence that structural changes in the neuroglial complex are similar in character in the case of exposure to WSLEMF and to weak direct electric currents. It can be tentatively suggested that under the influence of functionally adequate biophysical factors, a single subcellular mechanism of nonspecific adaptive reactions is activated. There is therefore reason to suppose that WSLEMF can be used to modulate the functional state of the brain in experimental and clinical-physiological investigations.

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