ACTION OF A MODULATED ELECTROMAGNETIC FIELD ON EXPERIMENTALLY EVOKED EPILEPTIFORM BRAIN ACTIVITY IN RATS

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The effect of a modulated electromagnetic field (MEMF) on experimentally evoked epileptiform activity of brain structures was studied in rats. Exposure to an MEMF with modulation frequencies of 2-30 Hz was shown to induce depression of paroxysmal brain electrical activity in 41% of experiments. Marked weakening of epileptiform activity was observed in 23% of experiments and potentiation in 10.1%. In 25% of experiments exposure to an MEMF did not significantly alter the character of evoked paroxysmal brain activity.

KEY WORDS: epilepsy; electromagnetic field; electroencephalogram; modulating frequencies.

Investigations by Bekhtereva and co-workers [1, 2] have shown that intracerebral electrical stimulation of certain deep brain structures leads to inhibition of paroxysmal electrical activity in patients.

At the same time, it has recently been shown to be possible to produce oriented changes in brain electrical activity by exposure to electromagnetic fields of radio frequencies, modulated by rhythms coinciding with the frequency characteristics of the EEG [3, 4].

In view of the urgency of the search for new methods of treatment of epilepsy, it was decided to undertake special experiments in order to study the possibility of suppressing experimentally evoked epileptiform activity of brain structures by means of a modulated electromagnetic field (MEMF).

EXPERIMENTAL METHOD

Experiments were carried out on 58 noninbred albino rats of both sexes. The animals were first tested for sensitivity to intensive acoustic stimulation in a special chamber. Animals in which acoustic stimulation evoked a violent motor response (irrelevant running, jumping), often terminating in tonic or clonico-tonic convulsions, were chosen for the experiments.

Electrodes were implanted into deep brain structures of the rats (hippocampus, amygdala, mesencephalic reticular formation, caudate nucleus, thalamic and hypothalamic nuclei), and into different regions of the cerebral cortex. Electrical activity was recorded on a BST-1 8-channel electroencephalograph and simultaneously on an "Orion" EEG analyzer. The epoch of analysis was 30 sec. In some experiments persistent epileptiform activity was obtained in the EEG of the animals by means of short-term electrical stimulation of limbic structures of the brain (hippocampus, amygdala) immediately before acoustic stimulation.

The animals were exposed to the MEMF against the background of marked epileptiform activity on the EEG. The parameters of exposure were: carrier frequency 40 MHz, field intensity 10-20 W/m, depth of modulation 80-100%, modulating frequencies 2-30 Hz. The duration of exposure varied in different experiments from 5 to 60 min.

EXPERIMENTAL RESULTS

The experiments showed that intensive acoustic stimulation evoked marked epileptiform activity in the EEG of animals predisposed to audiogenic epilepsy or in rats previously "sensitized" by electrical stimulation

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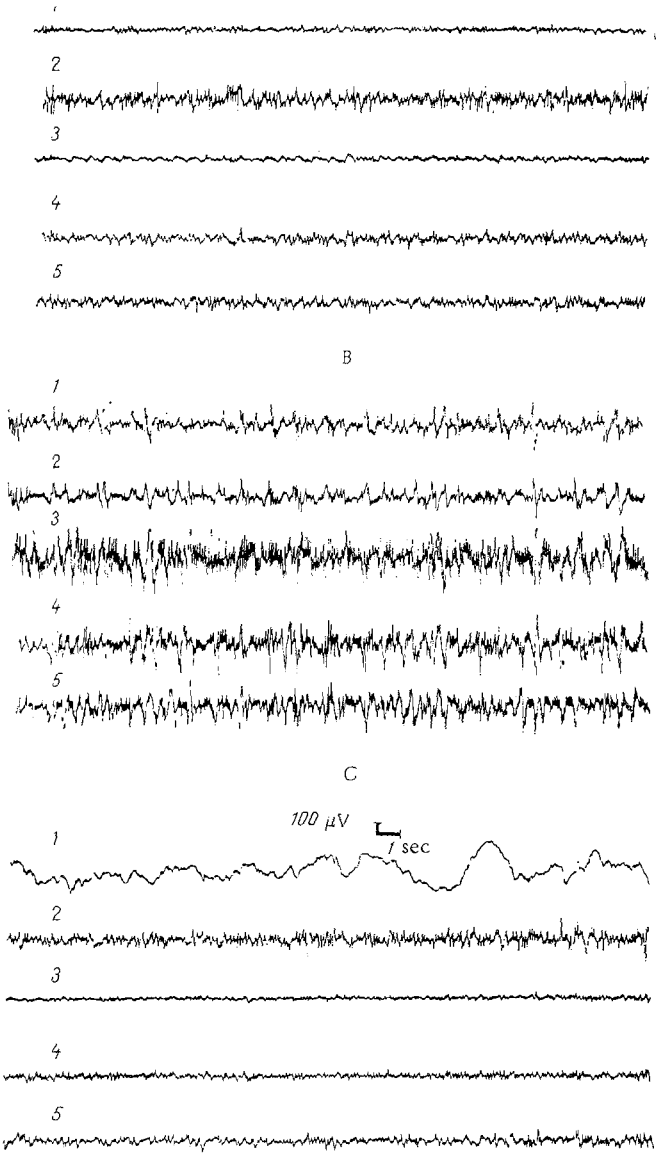

Fig. 1. Dynamics of changes in brain electrical activity of rat during acoustic stimulation and exposure to MEMF. A) Spontaneous brain electrical activity of intact rat; B) development of epileptiform activity at 6th minute of exposure to acoustic stimulus; C) suppression of epileptiform activity at 3rd minute of action of MEMF with modulation frequency of 3 Hz. 1) Sensomotor cortex, 2) dorsal hippocampus, 3) caudate nucleus, 4) mesencephalic reticular formation, 5) occipital cortex.

of the limbic structures of the brain. The activity was of two types: 1) activity of spike-wave type with different degrees of regularity, with frequencies of 1-3 Hz, and an amplitude of up to 500 μV; 2) regular hypersynchronized activity with a frequency of 4-9 Hz and an amplitude of up to 400 μV; 3) periodic bursts of paroxysmal high-amplitude waves and pointed discharges with different repetition frequencies and with an amplitude of 250-400 μV.

In 50% of experiments acoustic stimulation evoked generalized seizure activity, in 25% epileptiform activity was recorded mainly in various deep brain structures; in the remaining 25% of experiments paroxysmal activity was most marked in cortical derivations.

Exposure to MEMF against the background of continued action of the acoustic stimulus led to disappearance of the seizure activity in 41.6% of experiments. In 23.3% of experiments marked weakening of epileptiform

TABLE 1. Energy of Principal EEG Rhythms (numerical values correspond to integrator readings, epoch of integration 30 sec)

Structure	Rhythm	Background	Acoustic stimulation	Exposure to MEMF
Hippocampus	Δ	177±9,5	445±14,6	153±7,6
	θ	203±11,2	313±9,8	219±10,1
	α	201±7,7	212±12,9	171±6,7
	β	168±8,9	170±14,6	156±8,4
Reticular formation	Δ	105±9,7	386±11,1	92±5,6
	θ	100±4,2	217±7,7	86±8,3
	α	78±8,2	112±9,5	68±5,9
	β	111±6,1	110±8,7	93±7,2

activity was observed, as shown in particular by the fact that the seizure activity became irregular, the amplitude of the paroxysmal waves decreased, and the duration of its appearance was considerably shortened. In 25% of experiments exposure to MEMF caused no significant changes in epileptiform activity on the EEG. In 10.1% of experiments exposure to MEMF led to strengthening of the paroxysmal electrical activity.

The latent periods from the beginning of exposure to MEMF to the appearance of the above-mentioned changes on the EEG were characterized by considerable variability in different animals and ranged from 20-30 sec to 15-20 min. The blocking action of MEMF was most effective in cases when the animals' EEG was dominated by generalized seizure activity. Conversely, when local seizure activity was recorded in the cortex or deep brain structures on the animals' EEG, the antiepileptic action of MEMF was less marked; in some cases generalization of seizure activity was observed under these circumstances.

A typical example of the appearance of epileptiform activity in the rat brain in response to acoustic stimulation and its suppression by exposure to an MEMF during continued application of the acoustic stimulus is shown in Fig. 1.

To assess the action of MEMF, changes in the energy of the principal EEG rhythms were determined in this same animal (Table 1).

These data show that the development of epileptiform activity in the brain structures was accompanied by a selective increase in energy in bands corresponding to Δ- and θ-rhythms. Exposure to MEMF led to a decrease in the energy of these rhythms and, in some cases, the values recorded were actually lower than those in the background.

After the end of exposure to MEMF and with continued acoustic stimulation, seizure discharges appeared in the brain structures during the first 5 min in 50% of experiments. As a rule, in such cases, the abundance of seizure activity, based on indices such as amplitude, duration, and degree of generalization, was considerably less than before exposure to the MEMF.

Characteristically a marked antiepileptic effect was observed in different experiments after exposure to MEMF with different modulation frequencies ranging from 2 to 30 Hz, when the frequency remained fixed throughout the experiment. In other cases suppression of seizure activity took place when the modulating frequency was changed actually in the course of the experiment (for example, 16, 7, and 30 Hz). These results point to the need for individual selection of the effective parameters of MEMF in order to obtain an oriented effect on the epileptic response.

These experiments thus point to a distinct antiepileptic action of MEMF on the electroencephalographic manifestations of audiogenic epilepsy in rats. In most experiments, moreover, an antiepileptic effect of MEMF was observed, in the form of a considerable reduction or even the total disappearance of evoked epileptiform activity on the animals' EEG.

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