CHANGES IN BRAIN-EVOKED POTENTIALS UNDER THE INFLUENCE OF A PERMANENT MAGNETIC FIELD

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The effect of a permanent magnetic field on evoked potentials in the cerebral and cerebellar cortex arising in response to sciatic nerve stimulation was investigated in experiments on rats. During exposure to a permanent magnetic field an increase in amplitude of the evoked potential was found and additional waves appeared in its structure. The effect increased with an increase in field intensity over the range 500-4000 Oe.

KEY WORDS: *Evoked potentials; brain; magnetic field.*

A permanent magnetic field (PMF) is a physical factor of the external environment with which modern man is increasingly being brought into contact because of the rapid development of technology. Investigations by Kholodov [13, 14] have demonstrated the high sensitivity of the CNS to the action of magnetic fields. An important stage in the integrative activity of the brain is the generation of the bioelectrical response to the afferent volley.

The object of this investigation was to study the formation of evoked potentials (EPs) in the cerebral and cerebellar cortex during exposure to a high-intensity PMF.

EXPERIMENTAL METHOD

Experiments were carried out on albino rats anesthetized with pentobarbital (40 mg/kg, intraperitoneally), but in some experiments the animals were immobilized with myorelaxin (4 mg/kg, intraperitoneally) and artificially ventilated. EPs arising to above-threshold stimulation of the sciatic nerve (square pulses, 0.5 msec in duration, from an MSÉ-40 stimulator) were derived from the sensomotor area of the cerebral cortex and the cortex of the anterior zones of the vermis cerebelli by unipolar electrodes and recorded on an oscilloscope. Because of the need for rigid fixation of the electrode to exclude induction of an EMF in the magnetic field, in each region two silver electrodes were implanted through the bone and fixed with acrylic glue; the electrode from which the most standard EP was recorded was used in the work.

An SP-15A electromagnet with flat parallel tips measuring 400×300 mm was used for the experiment. The distance between the tips was 100 mm and the south pole was on the upper tip. The magnetic field was virtually homogeneous in the central part of the interpolar space, measuring 300×200 mm, and the drop in intensity in the rest of the field did not exceed 15-20% of the value in the center. The pulsation of intensity was 1.8%. The rats were placed in the gap of the magnet and fixed in the prone position to a frame. The animals were thus exposed totally to the action of the PMF with lines of force running in the vertical direction. The intensity of the PMF used varied from 500 to 4000 Oe and the exposure lasted 10-20 min. EPs were recorded from each rat before activation of the electromagnet, in a magnetic field of assigned intensity, and after switching off the electromagnet.

EXPERIMENTAL RESULTS

Before exposure to the magnetic field the cerebral cortical EPs of the rats consisted as a rule of biphasic waves with a mean latent period of 15.0±1.2 msec, a little longer than

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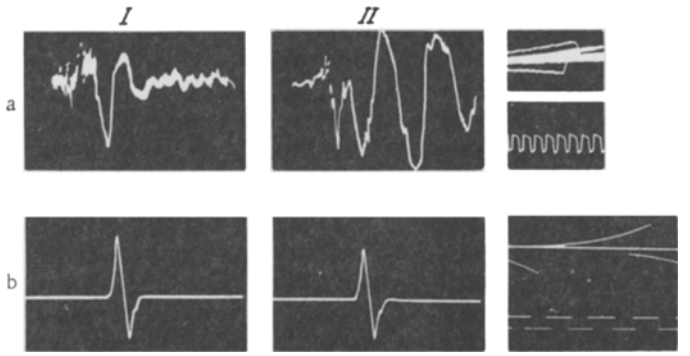

Fig. 1. Effect of PMF (4000 Oe) on EP of sensomotor cortex (a) and action potential of gastrocnemius muscle (b). I) Before exposure; II) during exposure. Calibration of amplification and time for EP 50 μV, 10 msec, for action potential 500 μV, 10 msec.

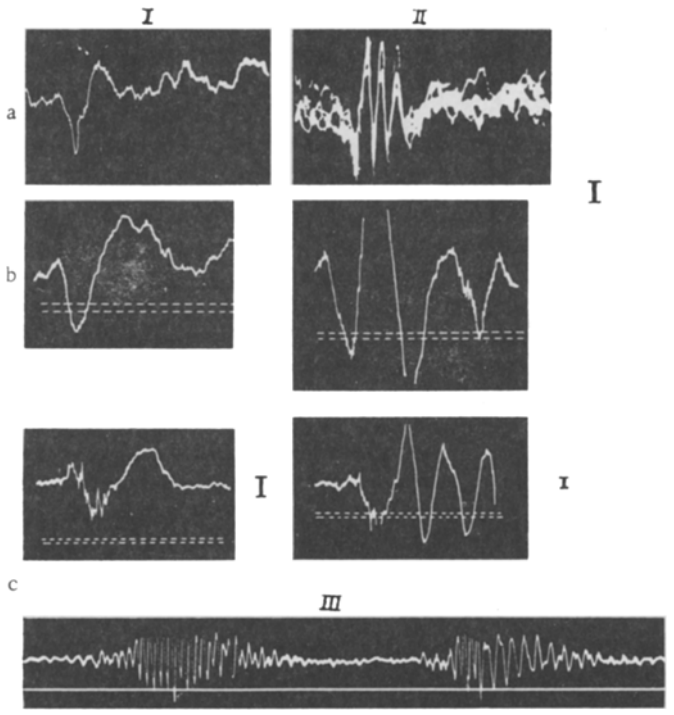

Fig. 2. Effect of PMF (4000 Oe) on cerebellar cortical EP: a, b, c) individual experiments on different rats. I) Before, II) during exposure. Calibration of amplification 50 μV, time 10 msec; III) continuous recording at speed of 5 cm/sec during exposure, experiment c.

the latency of cortical primary responses (PRs) recorded strictly at the focus of maximal activity, but as regards their other characteristics they corresponded completely to the classical EP [8, 10]. The form and parameters of the EPs recorded from the cortex of the vermis cerebelli, where somatotopical organization is ill-defined, corresponded to the characteristics described in the literature [1, 3, 12].

The investigation showed that exposure to a PMF leads to complex changes in EPs in both the cerebral and cerebellar cortex. Changes in EPs in both structures were of the same type: The amplitude of the potential increased and its shape became more complex. It will be clear

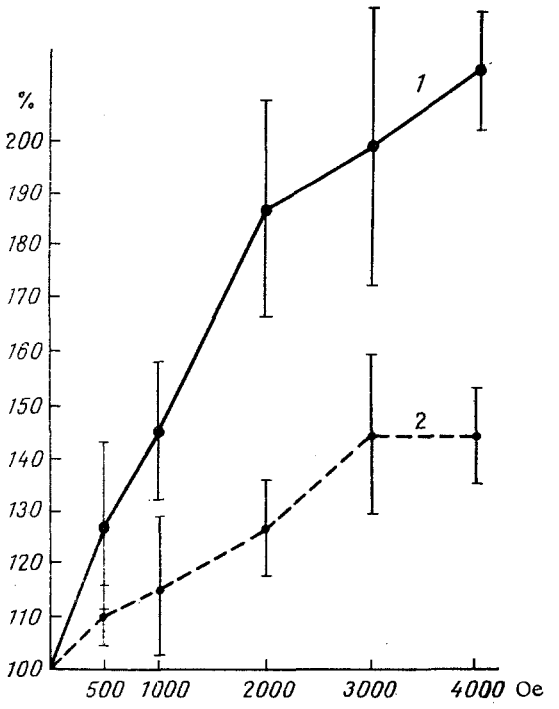

Fig. 3. Changes in EP amplitude as a function of field intensity: 1) cerebellar cortex, 2) cerebral cortex. Ordinate, amplitude of EPs (in % of initial); abscissa, field intensity (in Oe).

from Fig. 1a that instead of the biphasic PR recorded in the sensomotor cortex before exposure, during exposure to a PMF with an intensity of 4000 Oe a monophasic high-amplitude EP appeared. The action potential of the gastrocnemius muscle of a frog in response to stimulation of the sciatic nerve (experiment on a nerve–muscle preparation) is given in Fig. 1b. During the action of the PMF (4000 Oe) the shape of the action potentials was unchanged. Consequently, the PMF acts differently on the electrical potential generated in a relatively simple object such as the nerve–muscle preparation and on the evoked potential generated in central nervous structures in whose formation neurons with a complex synaptic organization participate. As a rule, the first phase of the primary response was preserved in the structure of the multiphasic evoked potential recorded in the animal exposed to the PMF. This applies to EPs of both the cerebral (Fig. 1a) and cerebellar (Fig. 2) cortex. The first phase had the same latent period, shape, and polarity, but its duration was usually reduced, and its amplitude was unchanged (compare Figs. 1a and 2a) or increased (Fig. 2b, c). Instead of the second phase of the PR either a faster high-amplitude wave of the same polarity, merging into additional waves (Fig. 2a, b, c) was recorded or it was practically completely absent, in which case the regular additional waves followed immediately after the first phase of PR (Fig. 1a). In most experiments during exposure to the PMF (4000 Oe) the number of phases in EP reached 4-6 and the duration of each phase was 20-40 msec, so that the total duration of the response was increased by 2-3 times. In individual cases faster additional waves were observed, and in that case the total duration of the multiphasic potential did not exceed the duration of the EP recorded before exposure to the PMF (Fig. 2a). In other experiments a sharper increase was observed in the number of phases and, correspondingly, in the total duration of the bioelectrical response. An experiment in which before exposure to the PMF an EP consisting of a biphasic wave with a total duration of 95 msec was recorded in the cerebellar cortex is illustrated in Fig. 2c. During exposure to the PMF a multiphasic potential appeared. As will be clear from Fig. 2c with a reduction in the sweep (bottom curve) the EP had the shape of a spindle 600 msec in duration arising in response to each stimulus during sciatic nerve stimulation with a frequency of once per second.

To judge from the mean data the duration of the latent periods of EPs in both brain formations was unchanged in the PMF (4000 Oe); the amplitude of the first phase of PR was in-

TABLE 1. Effect of PMF with Intensity of 4000 Oe on Amplitude (in μV) and Temporal Parameters (in msec) of Brain EP (M±m)

Brain structure	No. of rats	Time of investigation	Latent period	First phase of PR		No. of phases	Maximal amplitude
				amplitude	duration		
Cerebral cortex	30	Before exposure	15,0±1,2	155,9±12,5	22,0±0,8	2,0±0,09	239,8±18,1
		In PMF					
		1—3 min	16,5±1,7	193,8±19,0	20,5±1,5	4,5±0,3*	332,9±21,1*
		8—10 min	12,5±1,8	197,4±28,5	19,0±1,5	4,9±0,4*	364,9±29,8*
		After exposure	14,0±1,2	164,7±17,0	23,0±1,0	1,9±0,1	262,9±19,0
Cerebellar cortex	35	Before exposure	15,5±1,2	123,4±7,2	26,5±1,0	1,5±0,04	144,6±7,7
		In PMF					
		1—3 min	15,5±1,0	162,4±8,4*	23,0±1,0*	5,3±0,4*	318,3±20,9*
		8—10 min	17,0±1,6	171,6±12,0*	22,0±1,2*	5,2±0,4*	336,8±26,7*
		After exposure	17,0±1,8	123,6±5,5	26,0±1,1	1,3±0,06	138,7±6,7

*P<0.05.

creased and its duration slightly reduced. The number of phases in EP and the maximal amplitude measured from spike to spike in the highest voltage part of the EP increased significantly. The changes in EP remained as a rule throughout the period of exposure. After switching off the electromagnet the magnitude and shape of the responses returned quickly to normal (Table 1).

The degree of changes in the cerebral and cerebellar cortical EPs increased with an increase in the intensity of the PMF (Fig. 3). In the cerebellar cortex significant changes in the parameters of the EP as reflected in the mean data were found in a PMF with an intensity of 1000 Oe, whereas in the cerebral cortex they were found in a field with an intensity of 2000 Oe. It will be noted that in the cerebellar cortex changes in EP arose in fields of lower intensity and were more marked in degree than in the cerebral cortex. These differences in the response may be considered to be due to differences in the cytoarchitectonics and electrogenesis of EPs in the cerebellar and cerebral cortex [7].

The size and shape of EPs can change regularly depending on the functional state of the neurons and the intensity of stimulation. An increase in the complexity of the shape of the response on account of additional waves and the appearance of multicomponent EPs have been observed with an increase in the level of awakening of the animal or in the strength of stimulation [8, 10, 11]. The conversion of monobiphasic EPs into polyphasic is regarded as intensification of the response through activation of additional structures [4]. Changes in the size and shape of EPs observed in the present experiments can tentatively be attributed to the creation of conditions in the PMF facilitating the onset and spread of excitation in neuronal ensembles generating the electrical response to afferent stimulation. One possible mechanism lying at the basis of this phenomenon may be a change in the state of the membrane structures. A certain tendency toward a depolarizing effect of the PMF has been found by measuring the membrane potential of cells of various objects: *Nitella* [9], smooth-muscle cells of the frog stomach [2], giant neurons of the nerve ganglion of *Helix pomatia* [5], and the molluscan nerve ganglion [15]. A decrease in the impedance of the rabbit brain during exposure to a PMF has been reported [6]. Important results have been obtained on the pigeon cerebellar cortex. During exposure to a PMF with an intensity of 200-500 Oe a change in the steady potential in the layer of Purkinje cells and a simultaneous increase in the strength of the bioelectrical response of the flocculonodular lobe to vestibular stimulation, expressed mainly as an increase in the amplitude and duration of postrotational spindle-shaped discharges, have been found. These results are interpreted as facilitation and they are linked with changes in transmembrane ionic currents [16, 17].

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MECHANISM OF THE INFLUENCE OF SYMPATHETIC NERVES ON KIDNEY FUNCTION

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In dogs receiving large doses of reserpine hyponatriemia, hypokaliemia, oliguria, a reduction in the renal blood flow and in the sodium and potassium excretion, together with abolition of the inhibitory effect of the splanchnic nerve on the function of the glomerular and tubular portions of the nephron are observed as the result of functional insufficiency of the sympathetic innervation of the kidneys through the development of catecholamine deficiency.

KEY WORDS: *Sympathetic nerves; reserpine; kidney function.*

Previous investigations established depression of the function of the glomerular and tubular portions of the nephron of the kidney on the side of splanchnic nerve stimulation [1]. To confirm that the inhibition of kidney function was due to activity of adrenergic fibers it was decided to use reserpine to block the action of the sympathetic innervation.

EXPERIMENTAL METHOD

Chronic experiments were performed on 12 dogs with separately exteriorized ureters. Altogether 18 experiments were performed on six intact animals (control) and 12 experiments on

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