complex after blocking of the hyperactive DDS, when the DS become "autonomous" generators, also reveals their role as functionally independent structures.

These investigations show that not every focus of excitation acquires the function of a DDS; only a structure which forms a functional volley of sufficient intensity can do so. Results of the investigation reveal evident differences between the DDS and the dominant focus: Unlike the DDS, the dominant does not potentiate excitation in other foci and does not determine their behavior, but depresses their activity [6]. Coordinated inhibition of activity of other centers and of other reactions is an essential feature of the dominant both as a focus of excitation and as a principle of nervous activity. When defining the leading focus in epilepsy it is thus desirable and more correct to speak of a "determinant focus" and not of a "dominant focus." The investigations described above may help to shed light on the pathogenic structure of many forms of CNS pathology and, in particular, of epilepsy.

LITERATURE CITED

1. G. N. Kryzhanovskii, Pat. Fiziol., No. 4, 3 (1973).
2. G. N. Kryzhanovskii, in: Simulation of Diseases [in Russian], Moscow (1973), p. 120.
3. G. N. Kryzhanovskii et al., Byull. Éksp. Biol. Med., No. 7, 15 (1974).
4. G. N. Kryzhanovskii et al., Byull. Éksp. Biol. Med., No. 1, 23 (1976).
5. G. N. Kryzhanovskii et al., Byull. Éksp. Biol. Med., No. 2, 147 (1976).
6. A. A. Ukhtomskii, Collected Works [in Russian], Vol. 1, Leningrad (1950).
7. A. A. Shandra, Fiziol. Zh. SSSR, No. 11, 1597 (1975).
8. J. Bancaud et al., Electroenceph. Clin. Neurophysiol., 37, 275 (1974).
9. W. J. H. Nauta, J. Psychiat. Res., 8, 167 (1971).
10. D. N. Pandya et al., Brain Res., 13, 13 (1969).
11. W. Penfield and H. Jasper, Epilepsy and the Functional Anatomy of the Human Brain, Little, Brown and Co., Boston (1954).

BRAIN ELECTRICAL ACTIVITY OF RABBITS WITH EXPERIMENTAL HERPETIC ENCEPHALITIS

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UDC 616.831-002-022.6-02:616.523]-092.9-073.97

A clinical and electrophysiological investigation on 33 rabbits with experimental herpetic encephalitis revealed changes in brain electrical activity correlating with the clinical picture of the disease. In the acute period of encephalitis diffuse slowing of the brain potentials was accompanied by paroxysmal activity of two types: paroxysmal periodic complexes and "spike + slow wave" complexes at the rate of three per second.

KEY WORDS: herpes; experimental infection; brain potentials.

Valuable data characterizing the dynamics of the pathological process in the CNS can be obtained by the use of experimental models of herpetic encephalitis. Work so far published deals mainly with the investigation of clinical and morphological changes in animals with herpetic encephalitis [2, 9] and there are only isolated references to the study of brain electrical activity [6].

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(Presented by Academician V. N. Chernigovskii.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 83, No. 1, pp. 10-12, January, 1977. Original article submitted March 16, 1976.

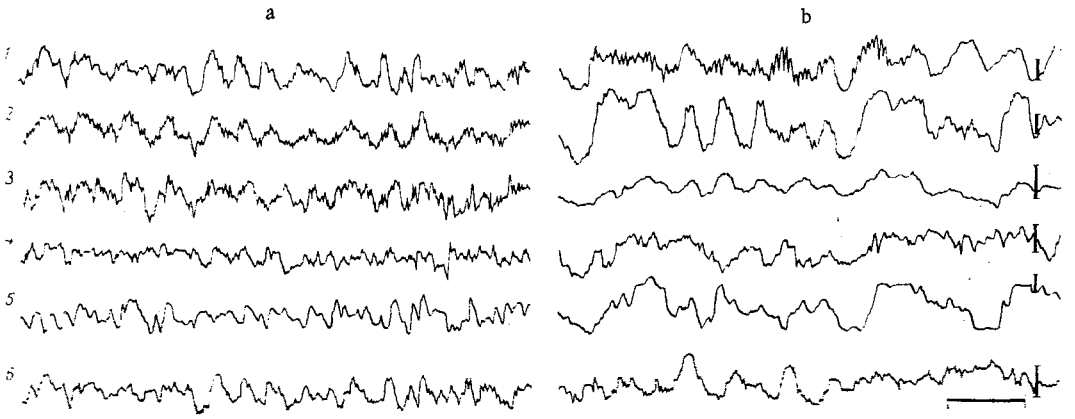

Fig. 1. Brain electrical activity of rabbit No. 11 under normal conditions (a) and in acute stage of herpetic encephalitis (b). Derivations: 1) sensomotor cortex; 2) posterior hypothalamic nucleus; 3) reticular formation; 4) visual cortex; 5) lateral geniculate body; 6) globus pallidus. Calibration: 1 sec, 100 μV.

Data on the dynamics of electrical activity of the cerebral cortex and deep brain structures of rabbits with experimental herpetic encephalitis are given in this paper.

EXPERIMENTAL METHOD

Experiments were carried out on 33 chinchilla rabbits weighing 2.5-3 kg. Glazed manganin electrodes, 200 μ in diameter, were inserted by means of a stereotaxic apparatus, using coordinates from the atlas of Fifkova and Maršala [5], into the visual and sensomotor cortex, mesencephalic reticular formation, posterior hypothalamic nucleus, globus pallidus, and lateral geniculate body. The reference electrode during the investigation was fixed to the animal's ear.

Experimental encephalitis was induced by intracerebral injection of herpes simplex virus (Tolstoi strain). The skull was trephined and 0.25 ml of a suspension in a dilution of 10^{-3} was injected into the surface layer of the occipital cortex. Twelve control rabbits received an injection of 0.25 ml physiological saline under similar conditions. Potentials were recorded on an eight-channel electroencephalograph (Schwarzer) by a monopolar technique before infection, in the incubation period, and in the period of acute encephalitis.

EXPERIMENTAL RESULTS

The brain electrical activity of the rabbits before infection, recorded in the cortex and deep structures, was characterized by polyrhythmic activity with predominance of waves with a frequency of 4-8/sec and an amplitude of 100-150 μV. In addition, irregular slow waves with a frequency of 2-3/sec and an amplitude of 140-150 μV were recorded in the cortex (Figs. 1a, 2a, and 3a). The respiration rate averaged 125 ± 10.3/min.

During the incubation period, the average duration of which was 5 ± 2.3 days, the character of the potentials remained unchanged.

The first signs of encephalitis were loss of appetite, lethargy, and disturbances of movement coordination. During this period the recorded brain electrical activity showed a mildly asynchronous pattern of the waves and a rather more pronounced stress rhythm than normal. This was followed by the acute stage of the disease, which lasted for between 12 and 48 h. The symptoms of encephalitis increased rapidly: tonic-clonic convulsions and partial or complete paralysis of the limbs developed. The respiration rate was not significantly changed (130 ± 13.7/min). During this period a considerable and diffuse slowing of the brain potentials was observed in 11 rabbits. Records from the cortex, subcortex, and brain stem began to be dominated by polymorphic waves with a frequency of 0.5-3/sec and an amplitude of 200 μV (Fig. 1b). Diffuse pathological activity in the form of "spike + slow waves" complexes with a frequency of three per second (Fig. 2b) and paroxysmal periodic complexes (PPC) illustrated in Fig. 3b, was recorded in 22 rabbits. PPC were recorded constantly with an interval of 2-4 sec and consisted of three phases: the first negative phase lasting 140 msec, the second positive phase of 320 msec, and the third negative phase of 610 msec. These forms of paroxysmal activity were recorded from the beginning of the acute period of encephalitis until the terminal phase; they were observed both in the presence and in the absence of clinically demonstrable convulsions. In the terminal

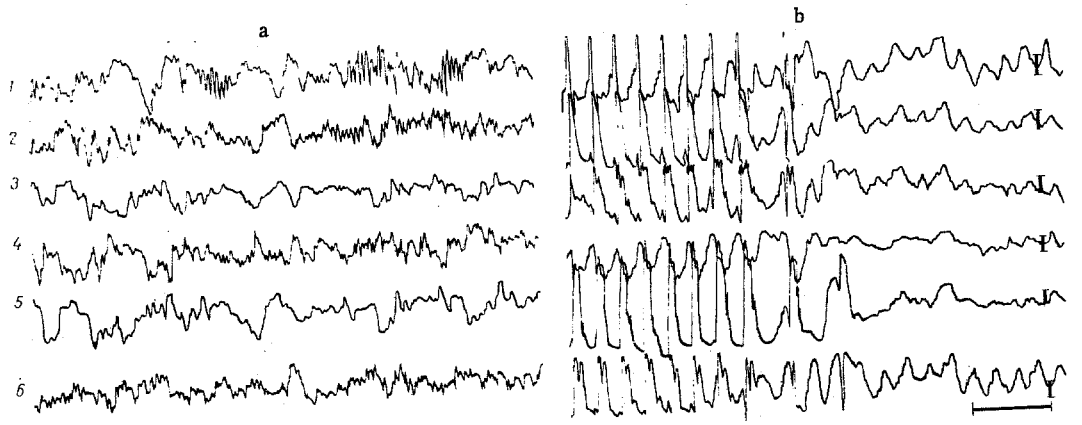

Fig. 2. Brain electrical activity of rabbit No. 6 under normal conditions and in acute stage of herpetic encephalitis. Legend as in Fig. 1.

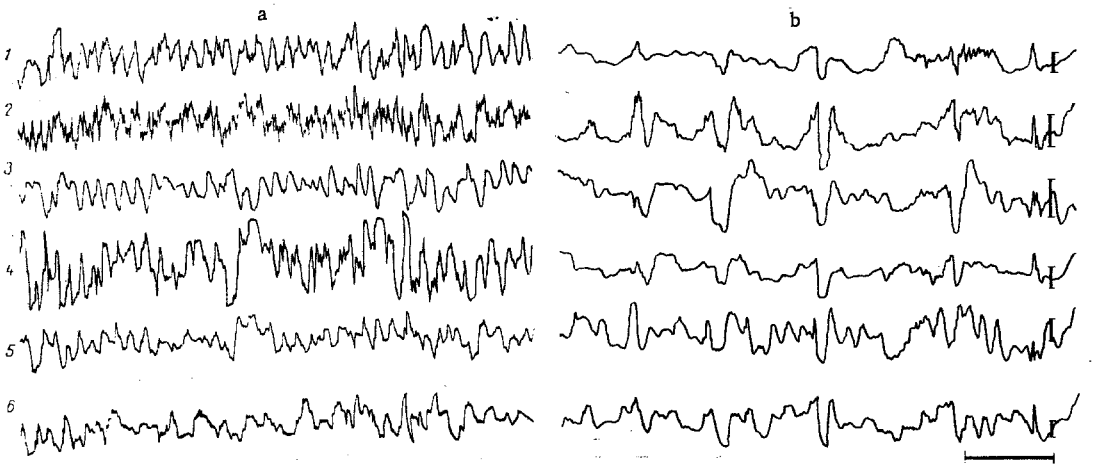

Fig. 3. Brain electrical activity of rabbit No. 20 under normal conditions and in acute stage of herpetic encephalitis. Legend as in Fig. 1.

period of the disease the amplitude of the waves fell, rhythmic activity was depressed, and the curves became flattened. All the infected animals died after 6 ± 1.1 days with signs of general paralysis. The presence of herpetic encephalitis was confirmed at autopsy.

In the rabbits of the control group, after injection of physiological saline no evidence of disease and no changes in the pattern of the brain potentials were found.

The electrophysiological findings, characterizing acute herpetic encephalitis in rabbits, were similar to those described in a previous investigation [6] in which only cortical electrodes were used.

In a discussion of the possible mechanisms of these disturbances of brain electrical activity found in rabbits with herpetic encephalitis, and taking into account the pathological data [2, 3], the sharp increase in slow polymorphic waves in the Δ-rhythm band may be explained by the development of hypoxia of the brain tissue, chiefly through edema and disturbances of the circulation of blood and cerebrospinal fluid in the brain. According to some workers [1], a similar type of slow activity is characteristic of cerebral hypoxia caused by various factors. Disturbances of the cerebral hemodynamics evidently also contribute to the development of paroxysmal activity [7], but in the writers' opinion the chief role is played by changes in cell metabolism arising through damage to the nerve cells caused by the herpetic virus.

Slowing of electrical activity and also PPC have been found on investigation of the surface EEG in patients with herpetic encephalitis [4, 8]. The results now obtained in rabbits with experimental herpetic encephalitis fill in additional details of these observations and show that disturbances of electrogenesis take place not only in the cortex, but also in deep brain structures.

LITERATURE CITED

1. A. M. Gurvich, Electrical Activity of the Dying and Reviving Brain [in Russian], Leningrad (1966).
2. M. B. Tsuker, Zh. Nevropat. Psikhiat., No. 10, 1441 (1974).
3. A. K. Shubladze and T. M. Maevskaya, Herpes [in Russian], Moscow (1971).
4. C. J. Breeden, T. C. Hall, et al., Ann. Intern. Med., 65, 1050 (1966).
5. E. Fifková and I. Maršala, in: J. Bureš et al., Electrophysiological Methods in Biological Research, Academic Press, New York (1967), p. 653.
6. I. F. Griffith, S. Kibrick, P. R. Dodge, et al., Electroenceph. Clin. Neurophysiol., 23, 263 (1967).
7. P. F. A. Hoeffer, R. A. Napoli, and S. Lesse, Arch. Neurol., 9, 112 (1963).
8. L. S. Illis and F. M. Taylor, Lancet, 1, 718 (1972).
9. C. Lévaditi, Ectodermoses Neurotropes. Poliomyélite, Encéphalite, Herpès, Paris (1922).

DETECTION OF CONDUCTION ALONG EFFERENT SPINAL TRACTS BY LOCAL ELECTROMYOGRAPHY AFTER SPINAL CORD INJURIES IN MAN

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UDC 616.832-001-07:616.74-073.97

Electrical activity of the gastrocnemius and tibialis anterior muscles during an active effort to extend or flex the ankle was investigated in patients with spinal cord injuries. In some such patients action potentials of motor units (MU) were recorded during voluntary efforts to flex and extend the ankle despite the absence of an interference electromyogram (EMG). Voluntary activation of MU led to the conclusion that efferent conduction along spinal tract which is partially preserved after spinal trauma can be detected by local electromyography.

In some patients after spinal cord trauma, despite the anatomical integrity of that structure, voluntary movement of the lower limbs is absent. This does not mean, however, the complete cessation of conduction of excitation along the spinal tract. The difficulty of assessing efferent conduction along spinal tracts during neurological investigation is made greater by the fact that most such patients develop spasticity of the muscles, which marks their reflex reactions. The method of interference electromyography is insufficiently sensitive to detect the electrical activity of individual motor units that still remain functionally connected with the higher levels of the CNS after spinal cord injury. Nowadays, the method of local electromyography [6] is extensively used in clinical practice for the diagnosis of peripheral nerve injuries [1], muscular diseases [2], and circulatory disturbances in the muscles of the lower limbs [4], and for the detection of disturbances of muscular synaptic transmission of excitation [3].

It was accordingly decided to use the method of local electromyography to assess efferent conduction along the corticospinal tract after traumatic injury to the spinal cord.

EXPERIMENTAL METHOD

Concentric needle electrodes (treated with alcohol) and a two-channel Medicor electromyograph were used. The electrodes were inserted into the belly of the muscle to be tested, perpendicularly to the surface. Potentials of motor units (MU) were recorded from the tibialis anterior and gastrocnemius muscles during passive flexion and extension of the ankle and during voluntary efforts to flex or extend the ankle. To detect

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(Presented by Academician of the Academy of Medical Sciences of the USSR M. I. Kuzin.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 83, No. 1, pp. 13-15, January, 1977. Original article submitted June 10, 1976.