ELECTRICAL ACTIVITY OF THE BRAIN IN ALBINO RATS DURING FREE MOVEMENT

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The EEG was recorded from the neocortex and dorsal hippocampus in 22 albino rats allowed to behave freely. Spontaneous manifestations of the hippocampal θ-rhythm occurred during behavioral activity, and also in the phase of "fast" sleep, in which synchronization of θ-activity reached its maximum. The phase of "slow" sleep consisted of relatively short cycles of successive EEG changes with a gradual increase in synchronization, separted from each other by short periods of desynchronization or concluding by the development of a phase of "fast" sleep. The application of unexpected stimuli to a tranquil, waking animal could evoke either θ-synchronization or desynchronization of hippocampal electrical activity against the background of desynchronization of the neocortical EEG, accompanied by various behavioral phenomena.

Many "behavioral" investigations have been carried out on albino rats. However, the dynamics of brain electrical activity of these animals and its connection with other indices of integral brain activity during free movement, despite some attention in the past [2, 3, 8-11], still remains inadequately studied.

In the present investigation, a detailed analysis was therefore made of the hippocampal and neocortical EEG in natural states such as waking, "slow" sleep, and "fast" (paradoxical) sleep, and also during the application of unexpected stimuli.

EXPERIMENTAL METHOD

Chronic experiments were performed on 22 albino rats into which single electrodes had previously been implanted into the frontal and occipital regions of the neocortex and into the dorsal hippocampus, and, in some animals, also into the hypothalamus and mesencephalic reticular formation. The electrodes were introduced under stereotaxic control in accordance with the atlas of Fifkova and Marsala [4]. The outer ends of the electrodes were soldered to miniature 6 and 7 contact receptacles fixed to the skull surface with phosphate cement and acrylate. Mainly monopolar and bipolar leads were used with a large interelectrode distance. Some animals also had silver wire electrodes implanted into the occipital muscles to record their tone. The animals were observed under conditions of free behavior. The EEG was recorded on an 8-channel Alvar electroencephalograph or 17-channel "Nihon Koden" polygraph with frequency analyzer. The record with the strongest (under the corresponding conditions), regular θ-rhythm was subjected to frequency analysis. At the end of the experiments, the positions of the electrodes in the animals' brain were verified histologically.

EXPERIMENTAL RESULTS

Analysis of the EEG leads showed that a well-marked θ-rhythm of hippocampal origin could be recorded not only in the hippocampus itself, but also in other brain areas: the posterior areas of the neocor-

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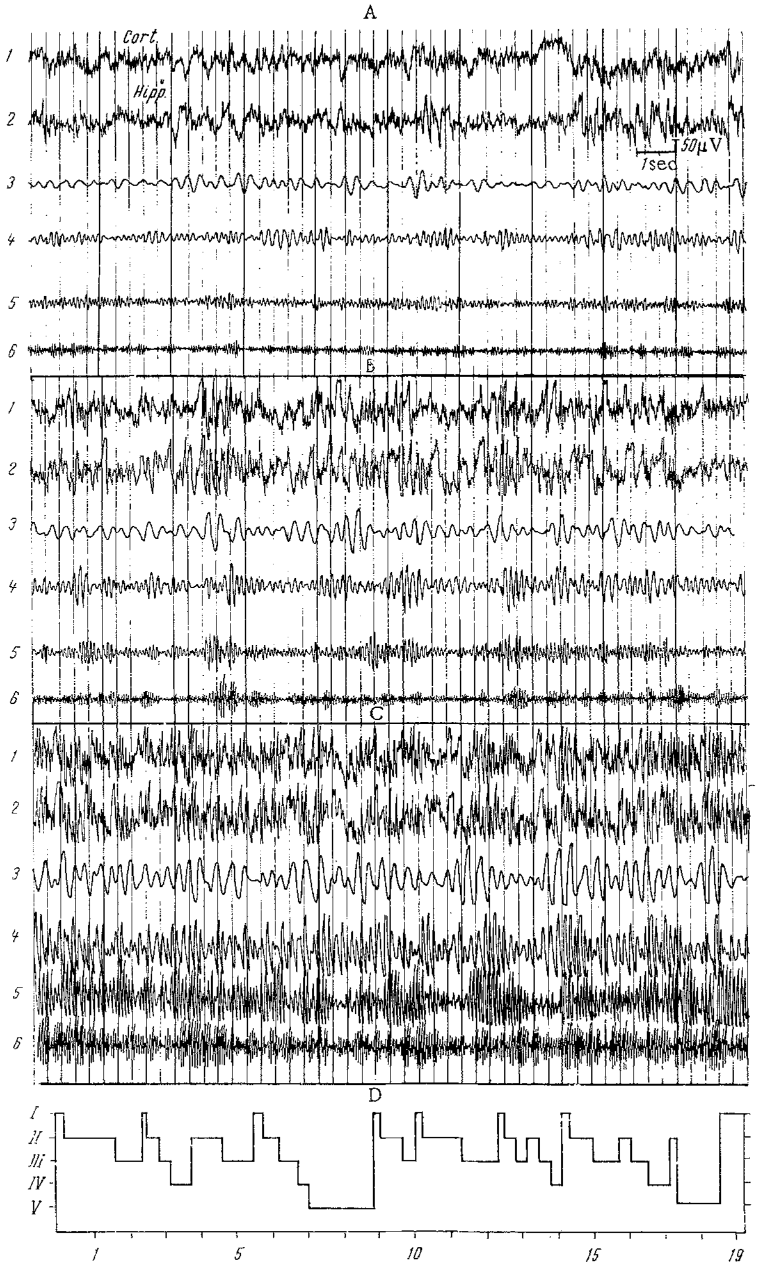

Fig. 1. Successive changes in EEG of rat during development of phase of "slow" sleep. A) Rest; B) slow sleep; C) completion of slow sleep; D) minor and major cycles of sleep (rat). From top to bottom; 1, 2) EEG of neocortex (fronto-occipital lead) and hippocampus; 3-6) frequency analysis of hippocampal EEG in Δ-, θ-, α-, and low β-wavebands (amplitudes are 6 dB higher in C than in A and B). In D, ordinate: I) active waking (see EEG in Fig. 3C); II) tranquil waking and beginning of slow sleep (corresponds to A); III) stage of fully developed slow sleep (corresponds to B); IV) stage of maximal synchronization, completing cycle of slow sleep (corresponds to C); V) phase of fast sleep (see EEG in Fig. 2); abscissa, time (in min).

tex, the hypothalamus, and mesencephalic reticular formation, evidently because of functional connections of the hypocampus and also because of the properties of the brain as a volume conductor.

In a tranquil waking state (lying in the prone position, eyes open) the neocortical and hippocampal EEG shows irregular activity in the form of uneven slow waves, single or in groups, with a frequency of

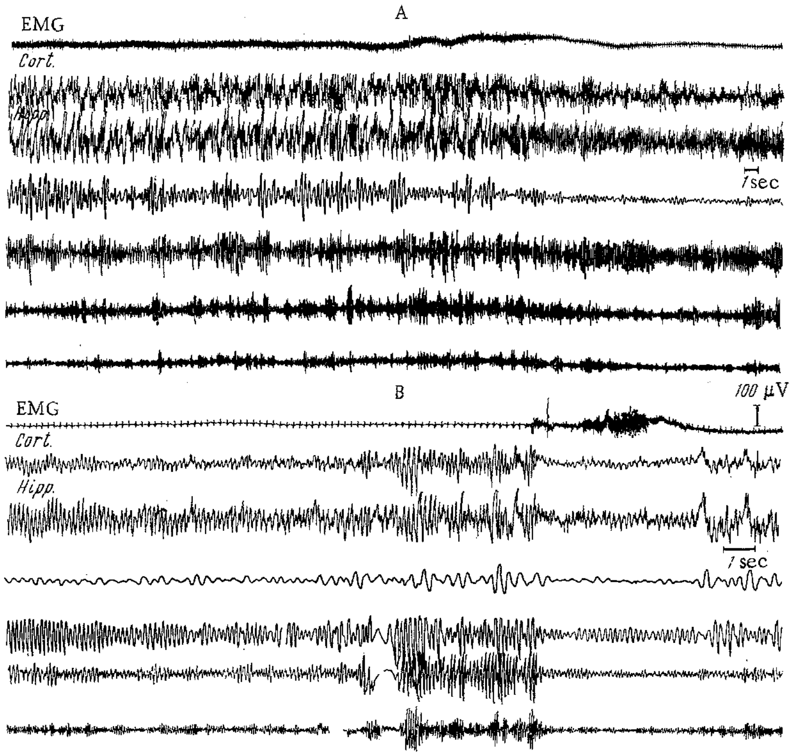

Fig. 2. Phase of fast sleep in a rat: A) final stage of slow sleep and beginning of fast sleep; B) final stage of fast sleep. From top to bottom: EMG of occipital muscles (ECG potentials can be seen), thereafter as in Fig. 1 (A, B).

3-6/sec and relatively low amplitude (50-100 μV), against the background of desynchronization, and also of fast waves sometimes grouped into spindle-shaped bursts (Fig. 1A). Spontaneous behavioral activation of the animal (washing, scratching, investigative behavior) led to the appearance of a distinct hippocampal θ-rhythm (6-7/sec) together with desynchronization in the frontal areas of the neocortex (Fig. 3C).

With the change to the phase of slow sleep, the EEG pattern became similar to that during tranquil waking, with gradual strengthening of the high-amplitude slow Δ- and θ-waves and a more regular formation of spindles with frequencies of 8-10 and 12-15/sec, which could appear separately or in combination with each other. As sleep developed, the spindles appeared increasingly frequently, with a simultaneous increase in the amplitude of the waves within the burst and with increasing complexity of its frequency structure: higher frequencies at the beginning and (or) end of the burst with slowing in the middle, sometimes down to 5-6/sec (Fig. 1B). Finally, the slow waves were masked by confluent spindles, and a continuous flow of high-voltage activity with a predominant frequency of 8-15/sec was recorded (Fig. 1C), followed by temporary desynchronization of brain electrical activity, after which the whole cycle was repeated. Maximum synchronization of the EEG, with which the cycle ended, could also be manifested differently, when the continuous flow of relatively fast activity was preceded by a period of reduction in amplitude of the spindles, and their replacement by continuous high-voltage Δ-activity (Fig. 2A). The duration of this type of cycle (a "minor" cycle of slow sleep) varied from 40-150 sec (sometimes at the beginning of sleep to 400 sec). As the cycles were repeated, the relative number of periods of marked high-amplitude spindles (separate and confluent) and of Δ-waves increased and they began to predominate. Finally, one of the "minor" cycles ended (after the period of maximum synchronization) by changing into the phase of fast sleep (Fig. 2). This completed a "major" cycle of sleep. The number of minor cycles of slow sleep in one major cycle varied from 1 to 8-10. These results demonstrate the complex, polymorphic character of the phase of slow sleep in albino rats. Each cycle of slow sleep consists of a definite and regular sequence of EEG manifestations with a gradual increase in EEG synchronization, resembling the final stages of slow sleep in other animals [11] and man. The precise reproducibility of the minor cycles within the major cycles is of particular interest, for the "microcycles" of human sleep are evidently vestiges of it [1].

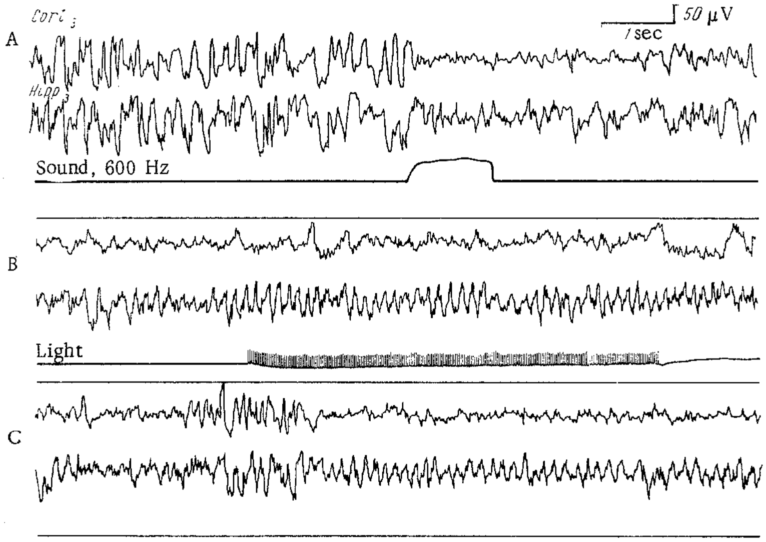

Fig. 3. Two types of activation on rat EEG: A) desynchronization in sensomotor cortex and hippocampus in response to acoustic stimulation (tone of 600 Hz); B) desynchronization in sensomotor cortex and well-defined, regular θ-rhythm in hippocampus in response to repetitive photic stimulation (45 Hz); C) spontaneous activation, reflected in desynchronization in sensomotor cortex and θ-rhythm in hippocampus during investigative behavior. From top to bottom: EEG of sensomotor cortex and hippocampus, marker of stimulation.

The onset of the phase of fast sleep is determined by a number of features. Electrographically it is manifested by the appearance of a very distinct, regular (like a machine) and high-amplitude θ-rhythm (7-8/sec, 200-500 μV) in the hippocampus and posterior areas of the neocortex, with simultaneous depression of all EEG waves in the anterior areas of the neocortex. If the phase of fast sleep is long enough, individual periods of a slight increase in amplitude and frequency of the θ-rhythm, followed by a decrease, can be distinguished in it. The whole period of fast sleep is marked by absence of electrical activity of the neck muscles. Atony usually leads to the animal lying on the floor with its limbs spread out. Twitching of the limbs and vibrissae were observed. The end of the phase of fast sleep was characterized by marked drop in amplitude and frequency of the θ-rhythm (sometimes there was a sudden increase in amplitude of the θ-waves in the EEG before this), combined with a burst of activity on the EEG and characteristic movement of the head (Fig. 2). The animal then either resumed its sleeping posture and a new major cycle began, or behavioral awakening occurred after a few seconds. Awakening the animal artificially in the stage of fast sleep, by means of a powerful external stimulus, led to rapid disappearance of the θ-rhythm with desynchronization in the EEG, the appearance of muscle tone, and a change in posture.

Application of expected stimuli while the animal was in a tranquil, waking state could give rise to the following types of EEG responses.

1. Slight additional desynchronization in the neocortex with no change in the hippocampal EEG (indeterminate rhythm) is evidently a weak manifestation of the activation response and is not accompanied by postural or behavioral responses.

2. Desynchronization in the neocortex, combined with the appearance of a distinct hippocampal θ-rhythm of 6-8/sec, takes place in albino rats only when activation of the animal's behavior response is observed; this behavioral response could take the form of attention, searching, looking around, investigative behavior, movement toward the source of the stimulus, and so on (Fig. 3B).

3. Marked depression of all rhythms in the neocortex and hippocampus (desynchronization of the cortical and hippocampal EEG) was sometimes seen after administration of loud acoustic stimuli, touching the animal, and so on. The animal shivered, arched its back, froze or jumped, and sometimes defecated, evidently showing fright (Fig. 3A).

4. Absence of the EEG and of behavioral responses is either a manifestation of extinction of the orienting reaction (habituation) or, if the stimulus is presented for the first time, it indicates a state of indifference of the animal toward that particular stimulus.

Two different types of EEG response to application of unexpected stimuli (the orienting reaction) were thus revealed. They are evidently connected with activity of different brain mechanisms. The existence of two mechanisms of activation of the limbic brain structures by reticular and hypothalamic structures is indicated by the results of other investigations [5, 12]. Judging from the behavioral manifestations, the first of these mechanisms in connected with the animal's active, investigative attitude toward the change in its situation, whereas the second rather suggests a passive (negative) response of the animal. It is impossible to attribute these changes simply to differences in the degree of elevation of the level of waking. A more adequate explanation is to attribute these EEG phenomena to qualitative difference in activation associated with the activation of different mechanisms of motivation. This hypothesis is confirmed by the results of experiments on cats [5]. EEG recordings in albino rats during stimulation of positive and negative motivational zones and during self-stimulation, on the other hand, have not yet provided decisive evidence in support of this hypothesis [6, 7].

LITERATURE CITED

1. L. P. Latash, The Hypothalamus, Adaptive Activity, and the Electroencephalogram [in Russian], Moscow (1968).
2. N. G. Mikhailova, Transactions of the Institute of Higher Nervous Activity. Pathophysiological Series [in Russian], Vol. 10, Moscow (1962), p. 116.
3. J. F. Brugge, Electroenceph. Clin. Neurophysiol., 18, 36 (1965).
4. J. Bures, M. Petran, and J. Zachar, Electrophysiological Methods in Biological Research, Prague (1967).
5. E. Grastyan, G. Karmos, L. Angyan, et al., Gagrskie Besedy (Moscow), 5, 159 (1968).
6. M. Ito, J. Physiol. Soc. Japan, 26, 461 (1964).
7. M. Ito, Electroenceph. Clin. Neurophysiol., 21, 261 (1966).
8. F. Michel, M. Klein, D. Jouvet, et al., C. R. Soc. Biol., 155, 2389 (1961).
9. E. Roldan, T. Weiss, and E. Fifkova, Electroenceph. Clin. Neurophysiol., 15, 775 (1963).
10. J. E. Swisher, Science, 138, 1110 (1962).
11. R. Ursin, Brain Res., 11, 247 (1968).
12. T. Weiss, Z. Bogdanecky, E. Roldan, et al., Gagrskie Besedy (Moscow), 5, 374 (1968).