ROLE OF THE POSTERIOR HYPOTHALAMUS IN ACTIVITY OF THE ASCENDING ACTIVATING SYSTEM

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In chronic experiments on cats after premesencephalic section of the brain stem electrical stimulation of the posterior hypothalamus evoked desynchronization of neocortical electrical activity. After isolated injury to the posterior hypothalamus, moderate electrical stimulation of the medial part of the mesencephalic reticular formation did not evoke any marked activation of the neocortex. The results point to the important role of the hypothalamus in the activity of the ascending activating system.

KEY WORDS: *electroencephalography; hypothalamus; brain-stem reticular formation.*

The hypothalamus plays an important role in the regulation of sleep and waking [13, 15]. Excitation of the posterior hypothalamus is accompanied by activation of the neocortex [2, 9]. This part of the hypothalamus, moreover, is regarded by many workers as the diencephalic end of the activating system [1, 3, 4, 8, 11]. However, activation of the neocortex during stimulation of the posterior hypothalamus is at present explained by accompanying excitation of the mesencephalic reticular formation [5, 10].

The possibility of bioelectrical cortical activation both after complete blocking of the mesencephalic reticular formation and after isolated injury to the posterior hypothalamus was investigated.

EXPERIMENTAL METHOD

In chronic experiments on eight adult cats all commissures connecting the cerebral hemispheres were divided under pentobarbital anesthesia (35-40 mg/kg). Next, in one series of experiments (4 cats) the brain stem was divided unilaterally at the level of the anterior border of the superior colliculi, and in another series, after spontaneous electrical activity had been established, the posterior hypothalamus was injured unilaterally by electrocoagulation (4 cats). Electrical activity of different parts of the neocortex was recorded by a 17-channel ink-writing electroencephalograph. Electrical stimulation of the posterior hypothalamus and mesencephalic reticular formation was by square pulses. At the end of the experiments the level of division of the brain stem and injury to the posterior hypothalamus, as well as the location of the uninsulated tips of the stimulating electrodes, were verified morphologically.

EXPERIMENTAL RESULTS

In cats with division of all interhemispheric connections and unilateral division

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of the brain stem high-frequency electrical stimulation (2.7 V, 300 Hz, 0.1 msec) of the posterior hypothalamus of the injured hemisphere on the 3rd day after the operation evoked desynchronization of electrical activity in the ipsilateral neocortex (Fig. 1). The effect of stimulation of the posterior hypothalamus was most clearly revealed in the frontal areas of the neocortex. After stimulation of this kind no change was observed in spontaneous electrical activity in the contralateral, intact hemisphere. The same picture, but with a longer aftereffect, was observed to stimulation of the posterior hypothalamus and 20-30 days after anterior division of the brain stem. Since terminal degeneration is complete by this time [14], the possibility of desynchronization of cortical electrical activity through excitation of ascending reticular fibers running in the region of the posterior hypothalamus during electrical stimulation was ruled out in this case.

Desynchronization of cortical electrical activity under these experimental conditions indicates the possibility of excitation of the posterior hypothalamic neurons themselves independently of the mesencephalic reticular formation.

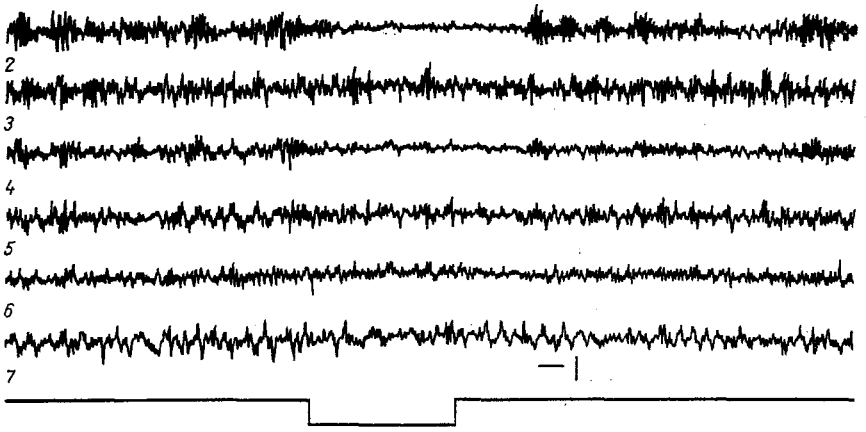

Fig. 1. Effect of stimulation of posterior hypothalamus (L) on neocortical activity (explanation in text). Records: 1) sensomotor cortex (L), 2) sensomotor cortex (R), 3) anterolateral association cortex (L), 4) anterolateral association cortex (R), 5) visual cortex (L), 6) visual cortex (R), 7) marker of electrical stimulation. Calibration: 2 sec, 200 μV.

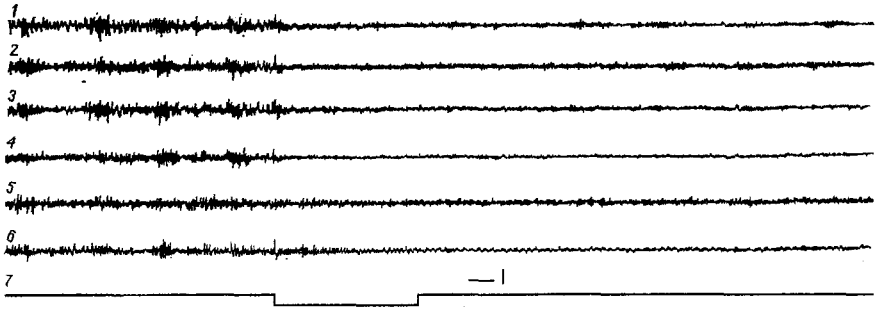

Fig. 2. Effect of stimulation of medial part of mesencephalic reticular formation on neocortical activity (explanation in text). Records as in Fig. 1. Calibration: 2 sec, 300 μV.

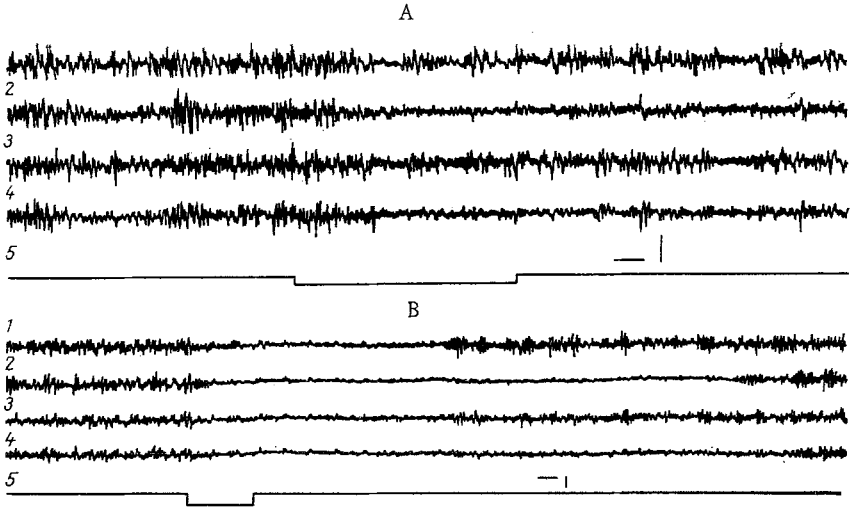

Fig. 3. Effect of weak (A) and strong (B) stimulation of mesencephalic reticular formation after unilateral injury to posterior hypothalamus (explanation in text). Records as in Fig. 1. 5) Marker of electrical stimulation. Calibration for A: 3 sec, 200 μV; for B: 3 sec, 300 μV.

In the experiments of series II on cats after preliminary division of all interhemispheric connections, electrical stimulation (2.8 V, 300 Hz, 0.1 msec) of the medial part of the mesencephalic reticular formation evoked desynchronization of activity equally in the cortex of both hemispheres (Fig. 2). However, after unilateral injury to the posterior hypothalamus, electrical stimulation (2.8 V, 300 Hz, 0.1 msec) of the same area of the reticular formation as in the previous series of experiments evoked cortical activation with a well-marked aftereffect only in the intact hemisphere. In the cortex of the injured hemisphere no significant changes in spontaneous electrical activity were observed (Fig. 3A). In response to stronger electrical stimulation (4 V, 300 Hz, 0.1 msec) of the mesencephalic reticular formation cortical activation was observed in the injured hemisphere also, with a short aftereffect (Fig. 3B). This fact points to the ability of the mesencephalic reticular formation to activate the cortex without involvement of the posterior hypothalamus in this process. Hence it follows that neocortical activation depends not only on pathways running through the hypothalamic region. In this respect the results of these experiments fully support the views of other workers [7].

The results thus indicate an important role of the posterior hypothalamus in neocortical activation. Considering also data in the literature indicating that the neocortex may be activated after total blocking of the mesencephalic reticular formation [6, 12, 16, 17], it must be assumed that the posterior hypothalamus is an important part of the ascending activating system.

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