THE EFFECT OF DIENCEPHALIC AND MESENCEPHALIC DAMAGE ON CORTICAL ELECTRICAL ACTIVITY IN PIGEONS

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Many authors have pointed out the influence of the sympathetic nervous system on the conditioned-reflex and electrical activity of different parts of the brain [1, 3, 6-10, 12, 22 and others]. In most cases the effects on the peripheral parts of the sympathetic nervous system were studied. It is known that the higher autonomic and particularly and higher sympathetic nervous centers are situated in the hypothalamic region [19-21, 24]. It has been shown that experimental damage to this region disturbs the normal activity of the higher centers of the central nervous system [4, 5], and alters the electrical activity of the cerebral hemispheres [23, 25,28 and others]. Stimulation of the hypothalamic region enhances the activity of the cerebral hemispheres [11, 14-18, 27].

The work here described is a study of the formation of hypothalamocortical connections during the process of evolution.

METHOD

Chronic experiments were carried out on 12 pigeons. After the animals had become acquainted with the conditions within a soundproof room, by means of either attached or implanted needle electrodes records were made of the electroencephalogram from symmetrically opposite regions of the occipital and parietal regions of the hemispheres. We investigated the orientating reactions in response to a 100 w lamp, and to the sound from an 800 cycle audio-oscillator. A nichrome wire unipolar electrode measuring 0.15-0.2 mm and insulated with bakelite varnish was introduced into the diencephalon or midbrain.

When squarewave stimuli of 1-5 v at a frequency of 30-250 per second were applied to the region under the electrode, a constriction or dilatation of the ipsilateral pupil could be observed. In 3 birds the pupillary reaction occurred on both sides, but was better shown on the side of the stimulus. In most of the birds in addition the eye turned inwards, the nictitating membrane relaxed, the eyelids closed, and there were various other motor responses including rotation of the head, raising of the tail, or general restless movements.

After the normal background of electrical activity of the cerebral hemispheres had been established, we destroyed the tissue beneath the electrode by means of high-frequency or direct current applied for from 20 seconds to 2 minutes. Usually the duration of the current was controlled during the destruction in terms of the observed pupillary symptoms, which were the opposite of those observed with stimulation.

From the first day after the destruction onwards we studied the spontaneous electrical activity of the cerebral hemispheres, and the orientating reactions in response to light and sound.

After these investigations the brain was fixed in 10% formol, and then examined histologically. The region damaged was identified in sections of the brain 15-30µ thick stained in Nissl.

RESULTS

The normal electroencephalogram (EEG) taken from symmetrically opposite regions of the cerebral hemispheres shows well-marked slow waves at a frequency of from 2 to 5 per second and a voltage of 30-150 $\mu\nu_e$ and superimposed on these waves there is a high-frequency rhythm of amplitude 5-20 $\mu\nu_e$

In some of the pigeons, 5-10 days after implantation of the sub-cortical electrode, a tendency was noticed to asymmetry of the EEG of the two sides due to a change on the ipsilateral side. Evidently, the actual proceduse of implanting the electrode into such a small animal as a pigeon may at some points creet a functional effect on the central nervous system.

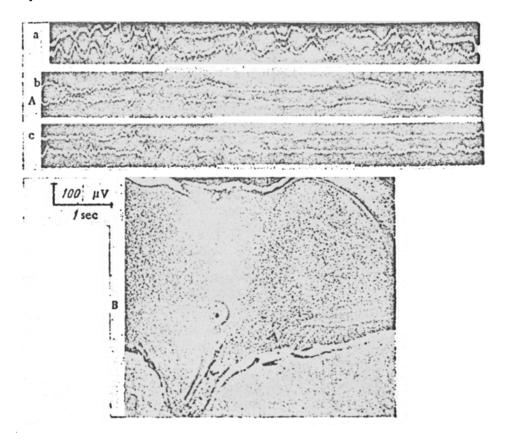


Fig. 1. Change in the electrical activity, and in the orientating response to light in pigeon No. 9. The traces shown in \underline{A} show the influence of left-sided damage to the diencephalon. The damage itself is illustrated in Fig. B. a) EEG and orientating reaction in response to light before destruction of the tissue beneath the electrode; b) ditto 4 days after the destruction; c) ditto after 16 days. The curves in each oscillogram, from above downwards, are: EEG on the left; EEG on the right; time marker (0.02 seconds), and the same trace also indicates the stimulus.

The orientating responses to light and sound were normally constant, and appeared as a typical desynchronization; they varied from one animal and one experiment to another only in the degree of the effect, and in the duration of the response and of its after-action. Extinction of the orientating reaction, produced during an experiment in which the stimulus was applied at 30-second intervals, was not maintained. Usually partial extinction occurred by the 20-25th application of the stimulus.

From the results obtained by destroying the tissue beneath the electrode, we could distinguish two groups of pigeons.

In the first group of 5 animals, after the destruction there was a considerable depression of cerebral electrical activity which was shown either as a reduction, complete disappearance, or irregularity of the slow waves, and as a reduction in the amplitude of the high-frequency waves (Fig. 1A). There was less change in the orientating reactions to light and sound. As a rule they disappeared or else were poorly shown on the first day after the infliction of the damage, and then recovered, despite the absence of the normal background electrical activity. All the EEG changes were better shown on the ipsilateral side. Suppression of the spontaneous electrical activity was prolonged, and in some birds it did not wear off until the end of the period of observation, which was continued for from 2 weeks to 1 month. Recovery was usually incomplete, and occurred chiefly in the orientating reactions. After recovery, the reactions were variable, and were better shown in response to the repeated stimuli than to the first stimuli, and they showed no tendency to extinction.

In all the birds of this group, the damage was located chiefly in the diencephalon (thalamus and hypothalamus, Fig. 18), and was less extensive in the midbrain, where it affected chiefly the basal regions abutting below on the optic tract, and on the medial side on the hypothalamic region. In birds, the ganglion opticum basale is situated here, which some authors classify as part of the hypothalamus. According to the site of the damage, the motor impairment of this group of birds was negligible, and consisted only in a reduction of motor activity. Some of them developed a tremot, and the feathers were ruffled; these changes were symptoms of damage to the diencephalon.

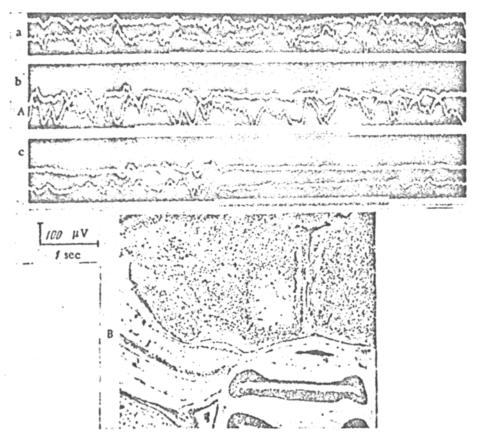


Fig. 2. Change of electrical activity and orientating reactions in response to light in pigeon No. 4 induced by damage to the left side of the mid-brain (B). a) EEG and orientating reaction in response to light before damage of the tissue beneath the electrode; b) ditto one day after infliction of the damage; c) ditto 3 days after the damage. Curves as in Fig. 1.

In the second group of 7 pigeons the changes in the EEG after the destruction comprised a marked increase in the amplitude of the slow waves, changes in their shape, and the appearance of extremely slow high amplitude waves at 1-2 per second, and a suppression of the high-frequency rhythm. The orientating reactions to light and sound disappeared in most cases. In this group of pigeons, the asymmetry of the EEG was very marked after the destruction, and all of them appeared on the ipsilateral side, and sometimes there were also changes on the contralateral side. Although the changes were extremely marked, they disappeared after 3-10 days (Fig. 2A). When the damage was central, the EEG changes occurred bilaterally.

In all the animals of this group, the damage was localized mainly in the midbrain – not in its basal region adjoining the hypothalamic area but in its central and dorsal portions, with massive injury to n. ruber (Fig. 2, B). Although after the damage the pupillary systems were similar to those found in the first group,—the difference was that there was considerable motor impairment. It was shown as a paralysis of the foot, rotation of the neck and head so that the apex faced downwards, awkward movements, and sometimes complete inability to stand or maintain the normal posture.

There is therefore a definite relationship between the nature of the EEG changes and the site of the damage in the midbrain or diencephalon. These results agree on the one hand with those of Obrador [28] and Kennard [25], who

after extensive destruction of the hypothalamus in cats and monkeys found a marked reduction in the amplitude and frequency of the brain waves, and on the other with the results of several authors [13, 26] who found that after damage to the pontomesenchephalic region of the reticular formation, slow high amplitude activity, characteristic of sleep, developed in the EEG. Cerebral electrical changes resembling those found in sleep and the increase of the threshold of the desynchronization response to stimulation have also been observed by some authors to follow damage of the hypothalamus [23, 26].

Both kinds of changes observed after damage to the hypothalamus, and particularly to its posterior portion represent gross deviations from the normal. Therefore most investigators assume the presence in the hypothalamus of a system which activates and potentiates the activity of the cerebral hemispheres.

At present there is not sufficient information for the problem to be assessed. From the results we have obtained we may propose provisionally that in birds, the diencephalon, and particularly the hypothalamus, does form part of such an activating system.

Our investigations have shown that unilateral damage to the diencephalon and midbrain of pigeons causes marked changes in the electrical activity of the cerebral hemispheres. The EEG changes depend on the site of the damage: when the diencephalon is chiefly affected both the slow and rapid rhythms of the cerebral hemispheres are depressed. Changes in the EEG induced by damage to the diencephalon are compensated more slowly than those resulting from damage to the midbrain.

SUMMARY

Chronic experiments were performed on pigeons. Damage to the diencephalon and midbrain induced distinct changes of the electrical activity of the cerebral hemispheres. There was a definite relationship between the nature of the EEG changes and the site of the injury; when chiefly the diencephalon and especially the hypothalamus were affected, both the slow and rapid waves in the cerebral hemispheres were depressed; when the damage affected principally the midbrain, and particularly its dorsal portion, high amplitude slow waves appeared in the EEG, and the rapid waves were depressed. Compensation of the EEG changes due to diencephalic damage was slower than it was for changes induced by midbrain injuries.

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