MECHANISMS OF THE SPREAD OF GENERALIZED ACTIVATION OF THE CORTEX AFTER MECHANICAL TRAUMA

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The problem of the generalization of excitation in the cerebral cortex is an important aspect of the wider question of cortico-subcortical relationships. However, the physiological mechanisms of this process are not completely clear. This applies equally to the mechanisms of generalization of the excitation arising at any pathologically changed point of the cortex (tumor, trauma).

Until recently it was widely believed by physiologists that a pathological focus may exert an influence on remote cortical zones mainly through intracortical connections, i.e., on the basis of the "horizontal" spread of excitation. The results of investigations carried out in the last few years have shown that this view takes account of only one of the possible mechanisms of spread of primary cortical excitation over the cerebral cortex.

It has been shown in P. K. Anokhin's laboratory that the generalization of excitation in the cerebral hemispheres may take place also through cyclical cortex—subcortex—cortex systems, i.e., in a "vertical" manner. This particular mechanism has been proved in the case of generalization of paroxysmal excitation arising in the cortex following local application of strychnine [4-6], and also in the case of the generalization of excitation arising in the cortex in response to its local injury [3].

The object of the present investigation was to continue the study of the mechanisms of spread of excitation throughout the cerebral cortex following local mechanical trauma to the cortex.

EXPERIMENTAL METHOD

Experiments were carried out on 60 rabbits anesthetized with urethane (1.5 g/kg) or Nembutal (40 mg/kg).

As an index of the reaction of the cerebral cortex to trauma, changes in the electrical activity of various regions of the cortex were recorded. The bioelectrical activity of the cerebral cortex was recorded on an ink-writing electroencephalograph. Bipolar needle electrodes, anchored to the cranial bones, or cotton wicks placed on the exposed surface of the cortex, were used to detect the potentials. The recording electrodes were situated at different regions of both hemispheres, so that one pair of electrodes was alongside the injured area at a distance of 3-5 mm away from it.

A traumatic focus was created at a certain point of the cortex by burying a bundle of fine needles in the brain. Usually a mild traumatic focus was created by puncture with one needle, while a powerful focus was produced by means of a bundle of needles, 4.5-5 mm in diameter, which was inserted into the brain tissue to a depth of 1.5-2 mm.

EXPERIMENTAL RESULTS

During mechanical stimulation of a localized region of the cerebral cortex a sudden, generalized change in the electrical activity of the whole cortex characteristic of urethane anesthesia takes place. Slow, synchronized waves with a frequency of 3-5 per second and an amplitude of 200-250 μV were replaced by waves with a frequency of 25-30 per second and an amplitude of 20-25 μV. The latent period of this reaction was 1.0-1.5 sec. Restoration

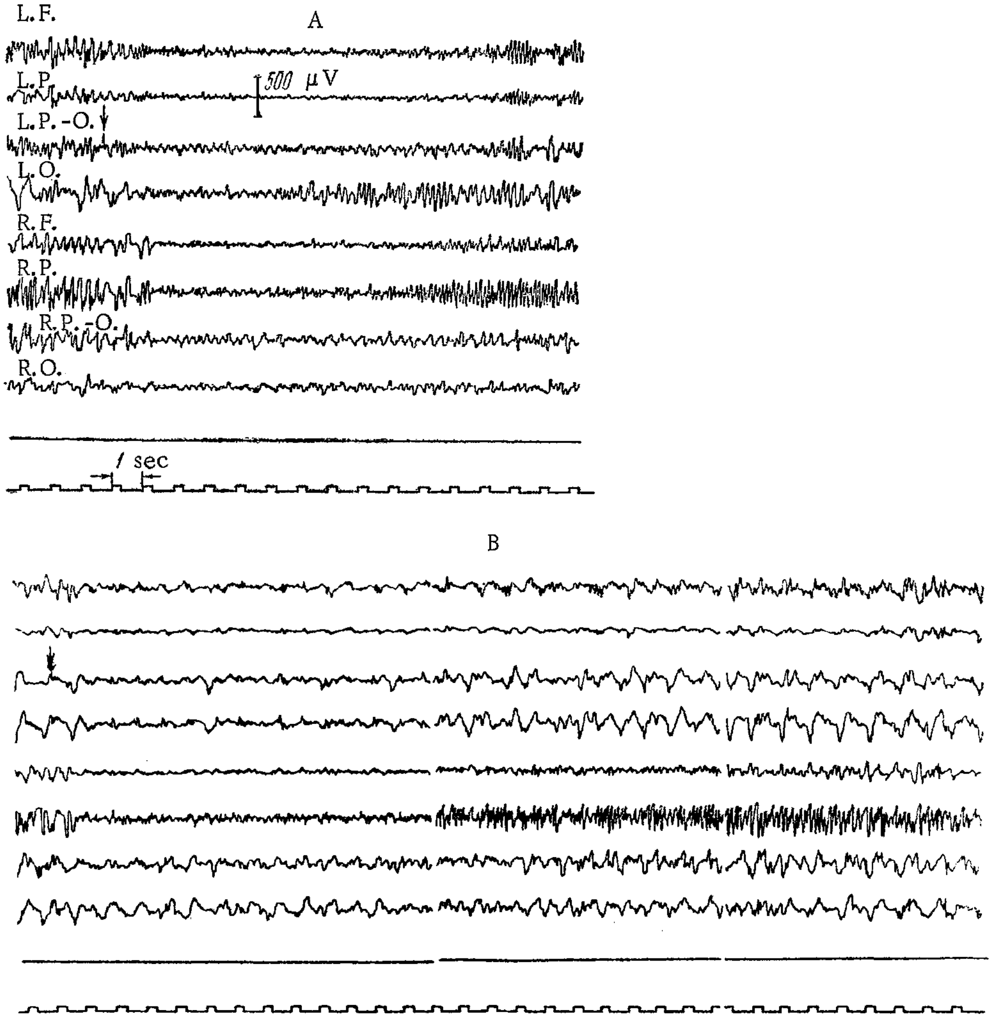

Fig. 1. Generalized change in the electrical activity of the cerebral cortex following mild (A) and strong (B) local mechanical trauma. Here and in Figs. 2 and 3 the arrow denotes infliction of trauma.

of the initial background of electrical activity began 1-3 min after infliction of the trauma, and the recovery was complete after 10-15 min. The degree of the changes in the EEG depended on the size of the traumatic focus. With weak trauma (puncture with one needle) the electrical activity of the cortex also changed, but the changes were of short duration, and the EEG regained its original appearance after 1-2 min. Strong trauma (puncture with a bundle of needles) led to the more prolonged, generalized change in electrical activity described above (Fig. 1, A and B). After restoration of the original background of the EEG the reaction could be obtained again.

A generalized change in the EEG in response to local mechanical trauma was observed in the animals anesthetized both with urethane and with Nembutal, but in the case of Nembutal this reaction developed only in response to strong trauma. The latent period of the reaction in Nembutal anesthesia rose to 3-5 sec and the changes in the EEG, although generalized in character, were less enduring than in the case of urethane anesthesia.

How does this generalization take place? Is it due entirely to intra- and intercortical connections, or do the generalizing structures of the subcortex play a part in its causation? To answer these questions it was also necessary to carry out experiments in which the spread of excitation along the "horizontal" connections of the cortex was prevented. For this purpose the corpus callosum was divided and the changes in the electrical activity were then studied in the ipsilateral and contralateral hemispheres following local trauma to one hemisphere.

The results of these experiments showed that after division of the corpus callosum the changes in the electrical activity of the cerebral cortex in response to local trauma to the cortex were generalized as before, and were detected at different points of the stimulated and the opposite hemispheres (Fig. 2).

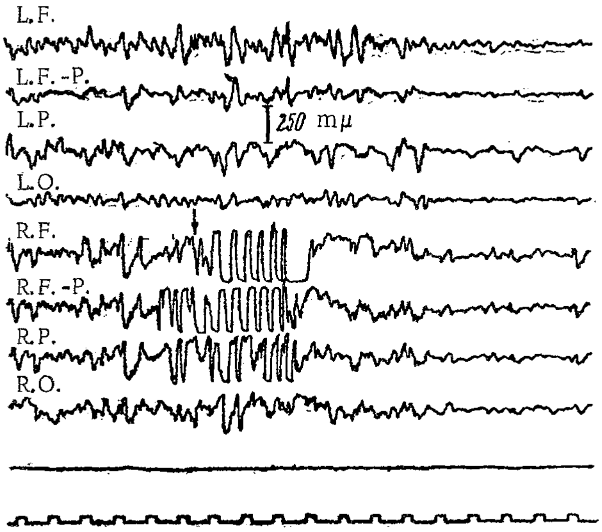

Fig. 2. Generalized changes in cortical electrical activity in response to strong local trauma after division of the corpus callosum.

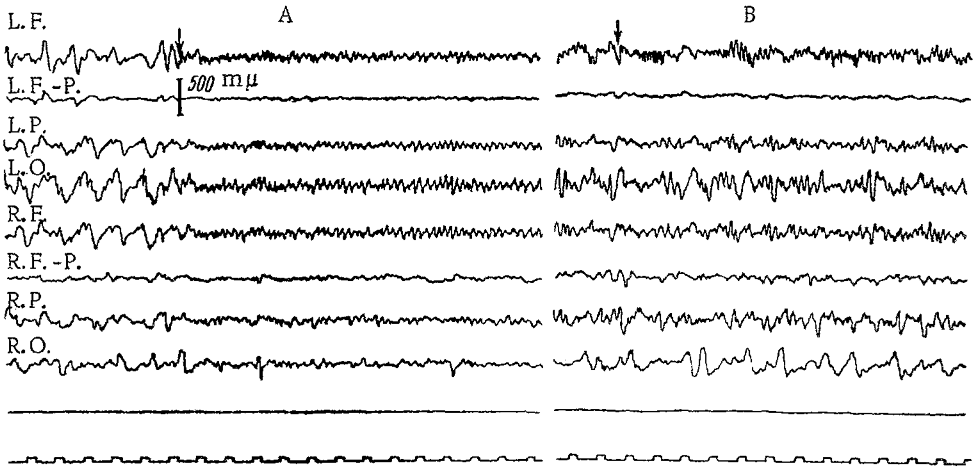

Fig. 3. Generalized changes in electrical activity of the cerebral cortex in response to strong local mechanical trauma before administration of chlorpromazine (A) and absence of changes in the electrical activity of the cerebral cortex in response to strong trauma after administration of chlorpromazine (B).

It was concluded from these experiments that the influence of the traumatic focus on the various parts of the cerebral cortex is effected not only through the intracortical connections, but also through the inclusion of certain subcortical formations in this activity. The generalized character of the changes in the EEG in response to local trauma to the cortex, and the simultaneous appearance of these changes in different cortical zones – all these signs suggested that they are associated with the involvement of the generalizing structures of the subcortex and, in particular, the reticular formation of the brain stem and thalamus. However, this hypothesis required experimental confirmation.

In the first place an attempt was made to block the generalizing reticular apparatuses with chlorpromazine. Chlorpromazine is known to cause selective blocking of the adrenergic structures of the reticular formation and to block the conduction of excitation through them to the cerebral cortex [1, 2]. The results of these experiments showed that after administration of chlorpromazine (5 mg/kg) local stimulation of the cortex no longer gave rise to generalized changes in the EEG. The changes in the EEG were limited to the stimulated hemisphere (Fig. 3).

Further proof of the participation of the reticular mechanisms in the generalization of "traumatic" excitation was obtained in experiments in which the biopotentials were recorded directly from the reticular formation of the brain stem and thalamus. Guidance for insertion of the electrodes was obtained from the works of A. B. Kogan (1949, 1952), and of Gangloff and Monnier (1956).

The results of these experiments showed that the electrical activity of the subcortical structures, like the activity of the cerebral cortex, changed in response to local trauma. The characteristic feature of the reaction of the subcortical structures was the appearance of synchronized waves with a frequency of 4-6 per second. In some of these experiments the changes in electrical activity in response to cortical trauma developed in the subcortical structures on the side of stimulation sooner than in the contralateral hemisphere.

The results obtained thus indicate that the generalizing structures of the subcortex play an important role in the process of generalization of excitation from a local pathological focus over the cortex. It might be considered that the changes in electrical activity in the present experiments were related to "Leao's spreading depression" [7]. However this can hardly be true. Leao's depression develops after a longer latent period, spreads much more slowly, and gradually extends to the adjacent areas of the cortex. In the present experiments the changes in electrical activity developed rapidly and, more important, they appeared simultaneously in different parts of both hemispheres.

SUMMARY

Experiments on anesthesized rabbits were used to study the mechanisms of stimulation transmission during a local mechanical injury to the cortex of the cerebral hemispheres. It was shown that a generalized change in the electrical activity of the brain cortex occurs in response to a local mechanical injury to the latter.

In the process of generalization of this stimulation not only the brain cortex but also the generalizing subcortical structures – the reticular formation of the brain stem and thalamus – take part.

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