CONNECTIONS BETWEEN THE ROSTRAL DIVISION OF THE RETICULAR FORMATION AND THE SMALL INTESTINE

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If the reticular formation, topographically connected with the respiratory, vasomotor, and visceromotor centers, is stimulated functional changes are observed in the internal organs [5]. Meanwhile interoceptive impulses, especially impulses from the carotid sinus and aorta [4], increase the activity of the reticular formation. These findings have been confirmed morphologically [2]. However, the extent to which the rostral (mesencephalic) portion of the reticular formation, which activates somatic function, is concerned in this interaction with the autonomically innervated organs has not yet been studied.

The object of the present investigation was to obtain more precise information concerning the connections between the rostral portion of the reticular formation and the small intestine. We studied the effect of stimulation of the rostral division of the reticular formation on the motor function of the small intestine, and the effect of stimulation of the stretch receptors of the small intestine on the reticular formation.

EXPERIMENTAL METHOD

Experiments were carried out on two dogs with Thiry-Vella loops of small intestine and with electrodes permanently implanted into the rostral portion of the reticular formation and into various parts of the cerebral cortex. The electrodes were implanted and their position verified by A. M. Marits's method [3]. The motor activity of the isolated loop of small intestine was recorded by the usual water-air transmission. The EEG was recorded on a four-channel ink-writing electroencephalograph having a frequency range of from 3 to 50 cps. Bipolar leads were used to pick up the potentials. The animals were screened. The rostral portion of the reticular formation was stimulated by means of a type ÉI-1 pulse generator, through the implanted electrodes, and the stretch receptors of the small intestine were stimulated by inflating a rubber balloon, inserted into the isolated loop of intestine, to the required size verified by means of a manometer.

EXPERIMENTAL RESULTS

The experiments showed that stimulation of the rostral portion of the reticular formation produced an effect on the motor function of the small intestine which depended on the intensity of the applied stimulus. After moderate stimulation of the rostral portion, causing a characteristic alerting reaction in the dog (which raised its head, pricked up its ears, and looked around), the motor function of the isolated intestine showed little or no change (Fig. 1, a, e). After stronger stimulation, causing an aggressive reaction in the dog (which tried to break out of its harness, growled, and gnawed furiously at things lying close by), the motor activity of the isolated loop of bowel was inhibited – the stronger the stimulation, the greater the degree of inhibition (Fig. 1, b, c).

The intensities of the moderate and strong stimuli varied in different animals, not only on account of individual differences, but also because of the degree of encapsulation of the electrodes with connective tissue. In the dog Laika, for instance, in most experiments the effects of strong stimulation were observed in response to a current of 2.5 A and frequency 100 cps (see Fig. 1, b), whereas in Ryzhik these effects were observed in response to a current of strength 0.5 A and frequency 100 cps (see Fig. 1, d). Meanwhile, in the same dog, but on different days, the effects of stimulation of different strength varied, evidently with the state of tonus of the reticular formation. However, irrespective of the excitability of the reticular formation on a particular day, it was always possible to choose an intensity of stimulation at which the motor activity of the intestine was inhibited, and this inhibition, moreover, always coincided with the appearance of the aggressive reaction.

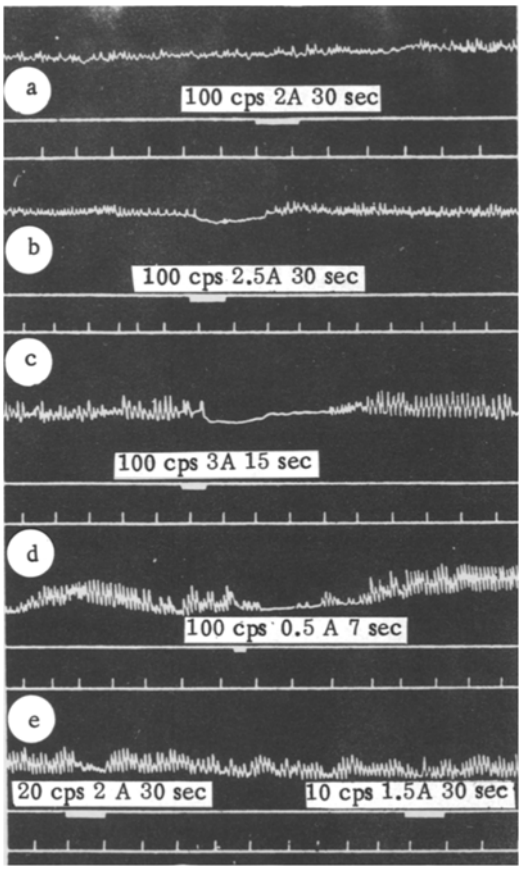

Fig. 1. Effect of stimulation of the rostral division of the reticular formation of different intensity on the motor activity of the isolated loop of small intestine of the dogs Laika (a, b, c) and Ryzhik (d, e).

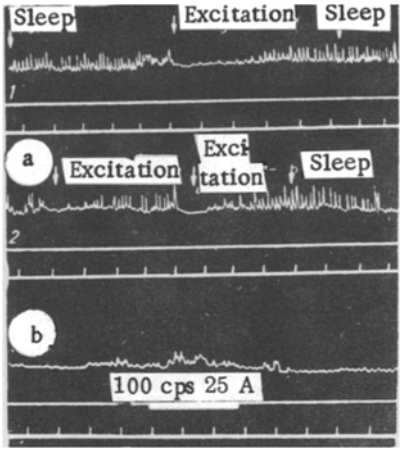

Fig. 2. Effect of chlorpromazine (a, 1, 2) and stimulation of the rostral portion of the reticular formation after administration of the drug (b) on the motor activity of the isolated loop of small intestine.

As an additional method of investigation, the reticular formation was blocked with chlorpromazine (injected intravenously in a dose of 1-2 mg/kg body weight). Stimulation of the rostral portion of the reticular formation in these conditions caused no change either in the animal's behavior or in the motor activity of the isolated loop of bowel (Fig. 2, b). For a few hours after its administration, chlorpromazine itself produced periodic changes in the motor activity of the intestine, with alternation of phases of inhibition and stimulation. A phase of increased motor activity was invariably accompanied by profound sleep in the dog, which hung in its harness, while the phase of inhibition of motor activity was accompanied by waking and aggressive behavior (see Fig. 2, a, b).

On the basis of the principle of the universality of back couplings, it could be postulated not only that the motor function of the small intestine is dependent on the rostral portion of the reticular formation, but also that the rostral portion is dependent on the flow of afferent impulses arriving from the small intestine. This hypothesis was tested in the same dogs in experiments in which the balloon inserted into the isolated loop of small intestine was inflated to give a pressure of 40-120 mm Hg. The changes in the EEG of the rostral portion of the reticular formation were then recorded.

Stimulation of the stretch receptors of the small intestine caused large, slow waves to appear on the EEG of the reticular formation, and the amplitude and frequency of the fast waves were increased. This effect gradually wore off. When the stimulation of the reticular formation ceased, it caused a transient repetition of the same effects (Fig. 3).

This effect of stimulation of the stretch receptors of the intestine on the electrical activity of the rostral portion of the reticular formation was seen especially clearly in those cases in which distension of the loop of bowel was carried out immediately after moderate stimulation of the rostral portion itself, through the electrodes implanted in it (Fig. 3). Activation of the rostral portion of the reticular formation by stimulation of the stretch receptors of the small intestine caused, in turn, an increase in the electrical activity of the cerebral cortex.

The previous results showing the role of the reticular formation in the regulation of the autonomic functions [1] were thus confirmed. At the same time, the results of our experiments showed that it is not only the bulbar portion of the reticular formation with its autonomic centers, but also the rostral portion, regarded as an activator of the somatic functions, which take part in the regulation of autonomic functions.

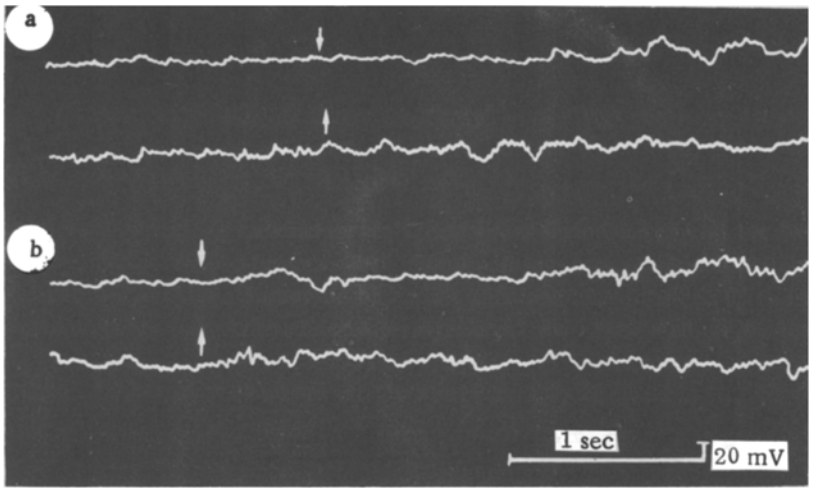

Fig. 3. Effect of inflating a balloon (80 mm Hg) in the isolated loop of small intestine on the EEG of the reticular formation (a); effect of the same inflation, but carried out 30 sec after stimulation of the reticular formation (b). Arrow pointing downward – beginning of inflation; arrow pointing upward – end of inflation.

SUMMARY

Experiments were staged on dogs with electrodes chronically implanted into the rostral portion of reticular formation and various areas of the cerebral cortex. An inquiry was made into the effect produced by stimulating the rostral portion of reticular formation on the motor function of the small intestine and by stimulating the baroceptors of the small intestine on reticular EEG. Whereas moderate stimulation of reticular formation caused no significant changes in the motor function of the small intestine, strong stimulation inhibited it. Stimulation of the baroceptors of the small intestine by means of a rubber bulb, introduced into the isolated portion of the small intestine, caused changes in reticular, and later in the cortical EEG.

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