EFFECT OF STIMULATION OF THE SENSORIMOTOR CORTEX ON UNIT ACTIVITY IN BULBAR PRESSOR STRUCTURES

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Activity of 58 bulbar "vasomotor" neurons was investigated in experiments on cats. Cortical stimulation evoked excitation in 25 neurons, initial excitation followed by inhibition in 7, and inhibition alone in 18 neurons. Excitation developing in neurons during stimulation of the tibial nerve can be increased or decreased by cortical stimulation.

A link between the cerebral cortex and bulbar reticular formation has been demonstrated morphologically [12,15] and by the neuronographic method [4,6,7,11]. Baumgarten and Mollica [5] and Scheibel and coworkers [17] investigated responses of single units in the bulbar medial reticular formation to stimulation of the sensorimotor cortex. They investigated neurons responding to polarization of the cerebellum. They concluded that cortical links with this type of bulbar reticular neurons are ill-defined.

The object of this investigation was to study the influence of the sensorimotor cortex on activity of reticular neurons located in bulbar pressor structures and responding in a particular manner to an increase and decrease in the flow of afferent impulses from vascular mechanoreceptors during fluctuations of arterial pressure: by inhibition of activity when the pressure rises and an increase in activity when it falls (Fig. 1C).

EXPERIMENTAL METHOD

Experiments were carried out on 35 cats weighing 2-3 kg lightly anesthetized with ether or immobilized with muscle relaxants. The main methods used have been described previously [1,2]. Action potentials of the neurons were recorded extracellularly by means of steel microelectrodes, insulated with varnish throughout their length except at the end, and having a tip 8-15 μ in diameter, inserted into the lateral and parvocellular nuclei of the bulbar reticular formation in accordance with the coordinates of Szentagothai's atlas. The potentials were recorded on a "Disa" CRO. Altogether 58 units were studied. When the determination of unit activity was complete, the same points were stimulated (5-10 V, 30/sec, 3 msec) through the same microelectrode that had been used to record the potentials.

The cortex was stimulated in the medial and lateral parts of the posterior sigmoid gyrus with square pulses through bipolar needle electrodes inserted into the cortex to a depth of 1.5-2 mm [13].. The parameters of stimulation for cats lightly anesthetized with ether were below the threshold of motor response (3-5 V, 20/sec, 0.1 msec, and for cats immobilized with muscle relaxants 5-15 V, 20/sec, 0.1 msec). The duration of stimulation varied from 10 to 20 msec.

EXPERIMENTAL RESULTS

Stimulation of parts of the sensorimotor cortex led to transient elevation of the arterial pressure (from 8 to 40 mm Hg), sometimes accompanied by fluctuations of its level (Fig. 1D). At the end of stimulation, fluctuations of arterial pressure were frequently observed.

Activity of most neurons was modified during cortical stimulation, and only eight neurons gave no response. Three types of unit responses were observed: 1 neuron (25) reacted by excitation, by an increase in discharge frequency (Fig. 1A), rising gradually and continuing throughout the period of stimulation. The increase in discharge frequency began before elevation of the arterial pressure, and at the maximum of arterial pressure the discharge frequency was sometimes slightly reduced, although remaining above its initial level. This decrease in discharge frequency may have been due to secondary influences from vascular

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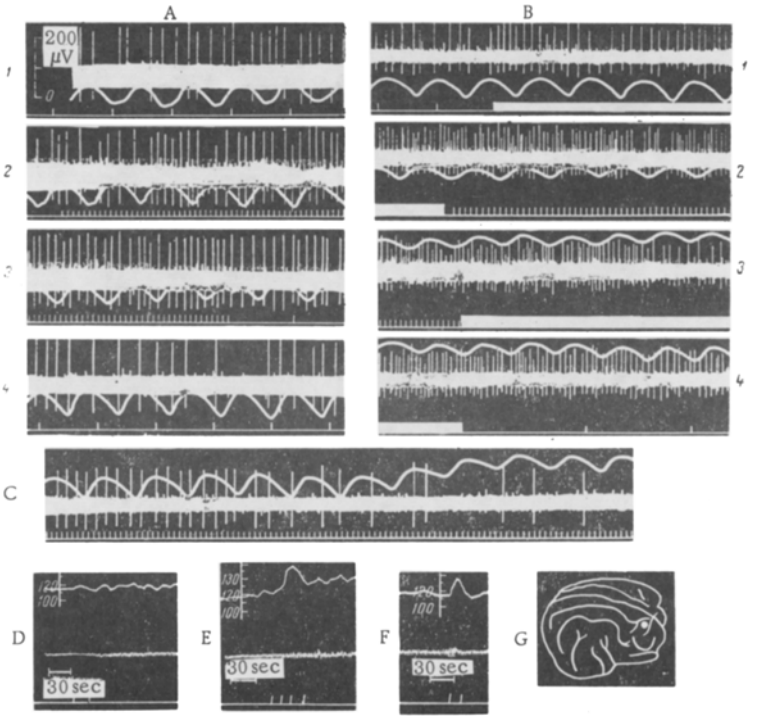

Fig. 1. Increase in unit response during combined stimulation of tibial nerve and sensorimotor cortex. A) Stimulation of cortex (3 V, 20/sec, 0.1 msec) for 15 sec: 1) initial activity, 2) beginning of cortical stimulation, 3) end of stimulation, 4) 1 min later; B) combined stimulation: 1) initial activity and beginning of stimulation of tibial nerve (white line), 2) stimulation of cortex added 1 sec later, 3) end of cortical stimulation while nerve stimulation continues, 4) end of stimulation of nerve; C) effect of mechanical lowering and raising of arterial pressure; D) effect of cortical stimulation on blood pressure and respiration; E) effect of combined stimulation; F) stimulation of "point"; G) diagram showing location of electrodes in cortex. Traces on oscillogram from top to bottom: discharges of neurons, arterial pressure, time marker (0.5 sec), and marker of stimulation; on kymogram: (for all figures) arterial pressure, respiration, marker of stimulation.

mechanoreceptors [2]. At the end of cortical stimulation, a gradual return to the initial discharge frequency took place (Fig. 1A).

Other neurons (7) responded to cortical stimulation by an initial, transient (1.5-3 sec) burst of frequent discharges followed by a decrease in frequency, and sometimes by their total disappearance (Fig. 2A). Finally, a third group of neurons (18) responded only by a decrease in discharge frequency (Fig. 2B). The decrease in frequency took place before the arterial pressure began to rise, it varied in degree sometimes amounting to the complete disappearance of the discharges, and it lasted throughout the period of stimulation and for 8-10 sec after its end, when a gradual return to the initial frequency took place.

The responses described were bilateral and were observed both in animals anesthetized with ether and in animals immobilized with muscle relaxants. This rules out the possibility that the changes in unit activity were the result of proprioceptive impulses associated with possibly unnoticed muscle contractions. It must be considered that the observed responses of these neurons were the result of the action of impulses from the cortex on them.

The effect of stimulation of part of the sensorimotor cortex on unit responses during the action of afferent impulses from the tibial nerve on them was investigated in 13 neurons [2]. Most neurons responding to

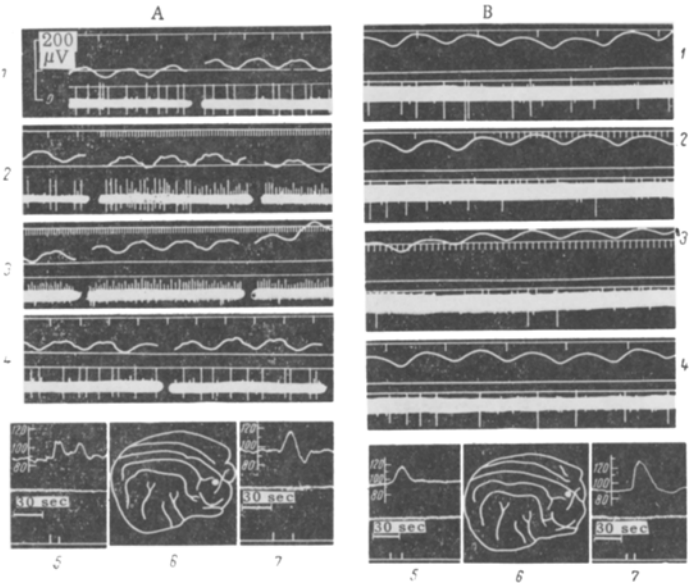

Fig. 2. Unit responses to cortical stimulation. A) Response of a neuron with an initial burst of excitation followed by inhibition (microelectrode 15 μ in diameter, in lateral nucleus; tubocurarine): 1) initial activity, 2) beginning of cortical stimulation (15 V; 40/sec; 0.1 msec), stimulation artefacts visible, 3) 6 sec later, only stimulation artefacts visible, 4) 1 min after end of stimulation, 5) effect of cortical stimulation on AP, 6) diagram showing position of electrodes on cortex, 7) effect of stimulating "point." Traces on oscillogram from top to bottom: time marker, 0.5 sec, and marker of stimulation, arterial pressure, unit discharges; B) response of neuron by inhibition of activity (microelectrode, 10 μ in diameter, in parvocellular nucleus; tubocurarine): 1) initial activity, 2) beginning of cortical stimulation (15 V; 20/sec; 0.1 msec), 3) 7 sec after beginning of stimulation on arterial pressure and respiration, 6) diagram showing position of electrode on cortex, 7) effect of stimulating "point." Order of traces on oscillogram and kymogram the same as in A.

cortical stimulation also responded to stimulation of the tibial nerve. The response of some neurons was uniform in direction: during both types of stimulation either an increase or inhibition of unit activity took place. Some neurons, however, responded to one type of stimulation by an increase, and to the other by a decrease in activity, i.e., the response was varied in direction. In the cases of a unidirectional response, an increase in the effect was observed during combined stimulation. It can be seen in Fig. 1 that the neuron responded by an increase in discharge frequency to stimulation both of the cortex (Fig. 1A) and of the nerve (Fig. 1B, 1). When stimulation of the cortex was added at a time of a developing increase in discharge frequency in response to stimulation of the nerve (Fig. 1B, 2),it produced a still greater increase in discharge frequency. After cortical stimulation had ended (Fig. 1B, 3) but stimulation of the nerve continued, the discharge frequency was not reduced. A gradual decrease in frequency began only after the end of nerve stimulation (Fig. 1B, 4). This suggests that impulses from the cortex caused an increase in reactivity of that particular neuron to impulses from the tibial nerve. On the other hand, when the responses occurred in different directions, the effects of combined stimulation were evidently dependent on the magnitude of the unit responses to stimulation of cortex and nerve separately. The response of a neuron inhibited by cortical stimulation (Fig. 2B) and excited by stimulation of the tibial nerve (Fig. 3A) is demonstrated in Fig. 3. Addition of cortical stimulation while the discharge frequency was increasing in response to stimulation of the tibial nerve caused inhibition of unit activity, which continued after cortical stimulation had been discontinued.

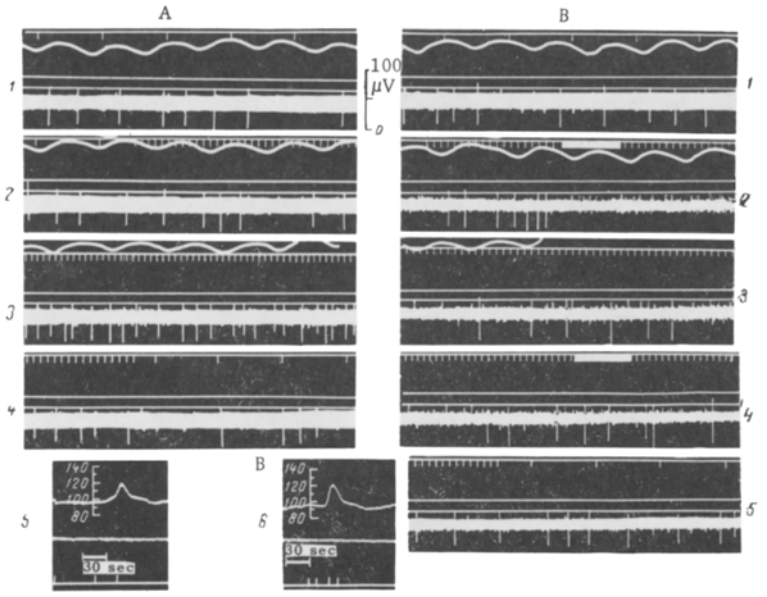

Fig. 3. Decrease in unit response during combined stimulation (the same neuron as in Fig. 2B). A) Effect of stimulation of tibial nerve: 1) initial activity, 2) beginning of stimulation of nerve, 3) 15 sec later, 4) end of stimulation of nerve (35 sec after beginning of stimulation), 5) effect of stimulation of nerve on arterial pressure and respiration; B) combined stimulation: 1) initial activity, 2) left portion – beginning of stimulation of nerve, right portion – 10 sec later, beginning of stimulation of cortex (–) against the background of nerve stimulation, 3) 15 sec after beginning of nerve stimulation and 5 sec after beginning of cortical stimulation, 4) end of cortical stimulation (–) while nerve stimulation continues, 5) end of nerve stimulation, 6) effect of combined stimulation. Order of traces on oscillogram and kymogram the same as in Fig. 2B.

Absence of response to afferent impulses from the tibial nerve during cortical stimulation may be the result of interference between two types of incoming afferent impulses. However, the possibility is not ruled out that the cortical impulses may have an inhibitory effect on afferent relays [8,9]. The absence of response to stimulation of the tibial nerve after the end of cortical stimulation suggests that impulses from the cortex lower the reactivity of that particular neuron to impulses from the tibial nerve.

Our own findings [1,2] and data in the literature [11,14,16] indicate that neurons responding to changes in the flow of afferent impulses from mechanoreceptors of the carotid sinus are present in bulbar pressor structures. An increase in this flow leads to inhibition of activity of these neurons [2,11,14,16], accompanied by a decrease in the flow of impulses in sympathetic nerves [2,3,14,18] and by a fall of arterial pressure. A decrease in this flow produces the opposite effects. After denervation of the carotid sinus and aortic zone zones, these responses did not arise [11,14,18]. These results suggest that the neurons studied in the present investigation are probably vasomotor in function. The experimental results show that cortical influences on these neurons are well-marked. Excitation of the cortex may produce either an increase or a decrease in activity of these neurons and modify their reactivity to peripheral afferent impulses.

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