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MODULATING EFFECT OF THE SECOND SOMATOSENSORY AREA OF THE CORTEX ON UNIT ACTIVITY IN SPECIFIC AND NONSPECIFIC THALAMIC NUCLEI DURING ELECTROACUPUNCTURE

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KEY WORDS: cerebral cortex; electroacupuncture; thalamus

Much attention has recently been paid to the study of the neurophysiological mechanisms of pain and the search for the most effective methods of its relief [1-3, 5, 6, 10].

The second somatosensory area of the cortex (SII) is not only one of the cortical areas containing the mechanism of primary analysis and screening of incoming information into the CNS, but it also participates in the evaluation of extremal, including nociceptive, stimuli [3]. On the basis of the fact that both the facilitatory and inhibitory influences of the cerebral cortex play an important role in determination of the functional state of deep brain structures [4], it has been suggested that blocking the conduction of nociceptive impulses by electroacupuncture (EAP) stimulation may be largely determined by a change in the character of cortico-subcortical interaction [8, 9], in particular in the specific and nonspecific thalamic nuclei [7].

In connection with this problem the aim of the present investigation was to study the modulating influence of area SII on the character of electrical responses of single neurons in the specific and nonspecific thalamic nuclei evoked by nociceptive and nonnociceptive stimulation against the background of EAP stimulation.

## EXPERIMENTAL METHOD

Acute experiments were carried out on 16 cats anesthetized with thiopental sodium (25 mg/kg, intraperitoneally), immobilized with suxamethonium, and artificially ventilated. After fixation of the animal in a stereotaxic apparatus all regions of operations were infiltrated with 0.5% procaine solution and the skull was trephined. Unit activity in the posterior ventromedial nucleus (VPM) and parafascicular complex (PFC) of the thalamus was recorded

Central Research Institute of Reflex Therapy, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR A. V. Val'dman.) Translated from Byulleten' Eksperimental'noi Biologii i Meditsiny, Vol. 95, No. 6, pp. 12-14, June 1983. Original article submitted December 17, 1982.

TABLE 1. Functional Distribution of Neurons in Thalamic Nuclei

Structure studied	perij numl	acter imula oheral oer of ed cel	regio	ponses of neurons f SII and of ns number of ac- tivated cells			lls activated y by stimu- ion of SII
	HT	МТ	LT	HT	MT	LT	_ a d Ce
VPM PFC	2	2	1	2	5 5	3 2	7 5

Legend. HT) High-threshold nociceptive neurons; MT) neurons of mixed type, receiving high-threshold and low-threshold afferents; LT) low-threshold nonnociceptive neurons.

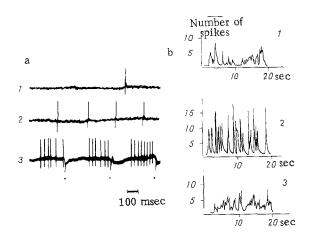


Fig. 1. Changes in unit activity in VPM and PFC during EAP stimulation and electrical stimulation of SII. a) Traces of spike activity of a neuron in PFC: 1) spontaneous activity, 2) increase in discharge frequency of neuron during EAP stimulation, 3) inhibition of discharges of neurons during single stimulation of SII; b) histograms of unit activity in VPM: 1) spontaneous activity, 2) increase in discharge frequency of neuron during EAP stimulation, 3) inhibition of discharges of neuron after tonic stimulation of SII.

extracellularly (not earlier than 3-4 h after injection of thiopental sodium) by glass microelectrodes with a tip  $1-3\,\mu$  in diameter, filled with 2 M KCl solution. The coordinates of the structures to be investigated were determined from a stereotaxic atlas of the cat's brain [11]. Unit activity was analyzed by means of an ANOPS-10l analyzer and led to an automatic x-y writer.

Single peripheral stimulation of nociceptive and nonnociceptive character was applied to the contralateral forelimb, the lower lip, and the pulp of a lower canine tooth, with square pulses of current (0.1-3 msec, from 0.5 to 20 mA).

EAP stimulation was given by means of acupuncture needles, inserted into the concha auriculae, to which a pulsed current was applied (0.1 msec, up to 10 mA in strength, frequency 1-3 Hz, duration 1-3 min). Low-frequency cortical stimulation in area SII was given through a bipolar electrode (interelectrode distance 1.5 mm), with square pulses (0.1 msec, 3-5 Hz, up to 500  $\mu$ A).

To monitor the animal's functional state the electrocardiogram, arterial pressure, and electroencephalogram were recorded. The body temperature was kept at 38°C. The location of the recording electrode was determined histologically.

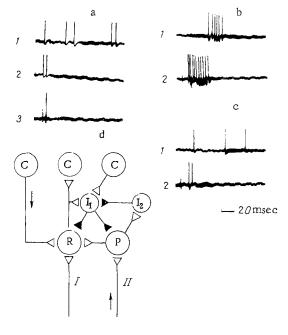


Fig. 2. Changes in spontaneous and evoked activity of a high-threshold VPM neuron in response to peripheral and central stimulation accompanied by EAP stimulation. 1) Spontaneous activity, 2) response of neuron to stimulation of contralateral forelimb, 3) response of neuron to stimulation of SII; b) change in spontaneous and evoked unit activity after EAP stimulation; 1) spontaneous activity; 2) response of neuron to stimulation of contralateral forelimb; c) restoration of spontaneous and evoked unit activity after tonic stimulation of SII: 1) spontaneous activity, 2) response of neuron to stimulation of contralateral forelimb; d) scheme of hypothetical organization of functional neuronal pool (for explanation, see text).

## EXPERIMENTAL RESULTS

Of 37 neurons investigated, 22 were in VPM. Seven of these cells exhibited phasic responses only to single stimulation of SII; 15 neurons responded by spike discharges both to stimulation of the cortex and to single peripheral stimulation. Responses of the neurons to single stimulation of SII consisted of activation or inhibition reactions. Fifteen neurons were recorded in PFC, of which five cells were activated only during stimulation of SII whereas ten neurons responded to both central and peripheral stimulation by activation and inhibition reactions (Table 1).

A definite pattern in the responses of the cells to stimulation of SII and peripheral regions was established, namely: Inhibition of the spontaneous rhythm after single cortical stimulation was observed as a rule in high-threshold neurons, in neurons which had both a high-threshold and a low-threshold afferent input, and less frequently, in neurons with only a low-threshold afferent input (Table 1).

Investigation of the bioelectrical responses of single neurons which responded to peripheral and (or) central stimulation in the course of EAP stimulation was particularly interesting.

One response of neurons in VPM and PFC observed during EAP stimulation of the concha auriculae and stimulation of SII is illustrated in Fig. 1b. The initial spontaneous firing rate of the VPM neuron rose sharply after EAP stimulation; tonic stimulation of SII led to a decrease in discharge frequency of the neurons practically to its initial level. An inhibitory effect was observed in this case during the first few seconds of stimulation of SII and it developed as stimulation continued. In response to single stimuli applied to SII, an effect of inhibition of spontaneous activity also was observed in eight VPM neurons and three PFC neurons. For instance, it will be clear from Fig. 1a that the initial spontaneous discharge frequency of the PFC neuron increased as early as after the first minute of EAP stimulation of the concha auriculae. The spontaneous discharge frequency of the cell continued

to rise 3 min after the beginning of EAP stimulation. Single stimuli applied at this time to SII caused inhibition of spontaneous activity of the neuron for 150-200 msec, followed by restoration of its activity.

Traces of responses of a high-threshold VPM neuron to single stimulation of the contralateral forelimb and of SII are shown in Fig. 2a.

This particular neuron, with low-frequency spontaneous activity and responding to stimulation of the limb by two spikes, and to stimulation of SII by one spike, changed not only the character of its spontaneous activity after EAP stimulation for 3 min, but also the character of its response to stimulation of the limb; the cell began to discharge with regular volleys of 6-11 spikes (Fig. 2b). After stimulation of SII for 2 min the neuron recovered its original spontaneous activity and the character of its responses to stimulation of the limb (Fig. 2c).

It can be tentatively suggested that EAP stimulation of the concha auriculae changed the activity of the neuron tested by activation of a pacemaker (P) neuron (Fig. 2d), activating a relay neuron (R) and an inhibitory interneuron ( $I_2$ ) which, in turn inhibited a cell of Renshaw type ( $I_1$ ). During tonic stimulation of cortical area SII, the inhibitory cell ( $I_1$ ) was activated by corticofugal impulses, and this inhibited the pacemaker neuron and thus restored the system to its original functional state. The relay neuron (R) began to discharge with infrequent spontaneous spikes and to respond to stimulation of the limb with two spikes.

EAP stimulation thus changes the character of responses of both spontaneously active neurons, not responding to peripheral stimuli, and of neurons responding to afferent stimuli with well-marked volley activity. Under these circumstances a new functional state of the latter neurons arises. Meanwhile changes in the functional state of the cortex (in this case area SII) by single or tonic electrical stimulation lead to inhibition of unit activity in the specific and nonspecific thalamic nuclei, thereby exerting a modulating influence on afferentation arriving from peripheral regions.

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