REFLEX EFFECT OF ELECTROACUPUNCTURE ON SPONTANEOUS AND EVOKED UNIT

ACTIVITY IN THE PARAFASCICULAR COMPLEX OF THE CAT THALAMUS

R. A. Durinyan, E. E. Meizerov, B. T. Chuvin, UDC 616.8-009.7-085.84.4.4]-0929.9 and V. K. Reshetnyak

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The wide experience of the successful use of reflex therapy and, in particular, of electroacupuncture (EAP) stimulation for the prevention or relief of pain syndromes which has accumulated in the last ten years [9, 11] has opened up new prospects for improved methods of analgesia. For this purpose not only the mechanisms of formation of pain sensations [1, 4, 7, 8], but also mechanisms of reflex methods of analgesia are being intensively studied at the present time [2, 3].

It has been shown by the evoked potentials (EP) method and by studying responses of single neurons that neurons of the parafascicular complex (PFC) of the thalamus respond to stimulation of various sensory systems [6], including response to nociceptive stimulation [10].

The writers' previous investigations [5] showed that the amplitude of EP arising in PFC in response to nociceptive stimulation was depressed after EAP stimulation. The amplitude of responses arising during non-nociceptive stimulation was virtually unchanged. This indicates that the neuron population from which EP were recorded consists of different functional groups.

The aim of the present investigation was accordingly to study differences in spontaneous and evoked unit activity in PFC in response to nociceptive and non-nociceptive stimulation and the effect of EAP stimulation on them.

EXPERIMENTAL METHOD

Acute experiments were carried out on 22 cats previously anesthetized with thiopental sodium (20-25 mg/kg, intraperitoneally), immobilized with suxamethonium, under artificial ventilation of the lungs, by a stereotaxic technique. Unit activity in PFC was recorded on the VC-9 electrophysiological system (Nihon Kohden, Japan) 3-4 h after injection of thiopental sodium, by extracellular glass microelectrodes filled with 2M KCl solution. The investigations were thus carried out on virtually unanesthetized animals. Spike discharges were analyzed by the ANOPS-101 analyzer and printed out on an x-y printer. The coordinates of the structure being tested were determined by reference to a stereotaxic atlas of the cat brain [12]. Peripheral single stimulation of nociceptive and non-nociceptive character were applied to the forelimb, lower lip, and dental pulp (lower canine tooth) contralaterally to the region of recording, by square pulses of current (0.1-3 msec, from 0.5 to 20 mA). EAP stimulation was carried out with acupuncture needles inserted into the concha auriculae or the forelimb, to which a pulsed current was applied (0.1 msec, up to 10 mA, 1-3 Hz, for 1-3 min). To monitor the animal's functional state, the ECG, EEG, and BP were recorded. The body temperature was maintained at 38°C. The location of the tip of the recording electrode was determined histologically.

EXPERIMENTAL RESULTS

Spontaneous and evoked activity of 92 PFC neurons was investigated in response to single peripheral stimulation of nociceptive and non-nociceptive character, during EAP stimulation of the concha auriculae or forelimb. Under these circumstances 46.7% of cells constituted a group of neurons which responded by phasic spike discharges to testing peripheral stimulation. Besides phasic responses, tonic changes of evoked activity, lasting from 3 to 40 sec after testing stimulation, were observed in 12% of neurons, forming the second group. In response

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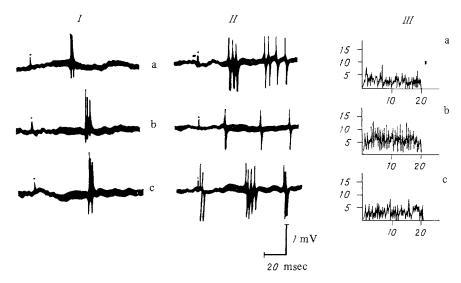


Fig. 1. Changes in spike discharge of a neuron of "mixed" type during EAP stimulation of concha auriculae. Traces of neuronal response before (I) and after EAP stimulation (II): a) response of neuron to stimulation of contralateral lip (1.5 mA, 0.1 msec), b) response of neuron to stimulation of contralateral pulp of lower canine tooth (15 mA, 3 msec), c) response of neuron to stimulation of contralateral forelimb (2 mA, 0.1 msec); III) histograms of spike discharge of same neuron: a) spontaneous unit activity, b) spike discharge after EAP stimulation, c) spike discharge 6.5 min after end of EAP stimulation. Abscissa, time (in sec); ordinate, number of spikes.

to single stimulation of nociceptive and non-nociceptive character 55.4% of the 92 PFC neurons were excited, 3.3% were inhibited, and the remaining 41.3% (38 neurons) did not respond to testing peripheral stimulation, but changed the character of their spontaneous firing pattern under the influence of EAP stimulation. The discharge frequency was reduced in 16 of these cells, but increased in 22 neurons, followed by a return to the initial level during a short time after the end of EAP stimulation.

For a more detailed analysis of unit responses to peripheral testing stimulation, the neurons were divided into groups depending on their response to non-nociceptive and (or) nociceptive stimulation.

The main group, namely 26 of 51 cells (51%), consisted of high-threshold nociceptive neurons excited only by intensive (up to 20 mA, 1-3 msec) electrodermal stimulation or stimulation of the pulp of the canine tooth. Neurons of "mixed" type, responding by excitation to application of peripheral stimulation of both non-nociceptive and nociceptive character, accounted for 35.3% (18 of 51 cells). Comparatively few PFC cells, namely 7 of 51 (13.7%), were excited in response to non-nociceptive electrodermal stimulation (up to 2 mA, 0.1 msec).

The study of changes in spike responses of neurons excited in response to peripheral testing stimulation, under the influence of EAP stimulation, was particularly interesting. This study showed that changes in evoked responses after EAP stimulation showed no definite trend in 7 cells, placed in the group of low-threshold non-nociceptive neurons: 3 cells facilitated their responses, 2 inhibited, and 2 left virtually unchanged the character of their responses. In 9 of the 13 neurons of "mixed" type investigated after EAP stimulation, facilitation of responses to non-nociceptive peripheral stimulation and inhibition of responses to nociceptive stimulation were observed. In four neurons of this group no tendency toward inhibition or facilitation was observed in their responses. Traces of responses of a neuron of "mixed" type in PFC before and after EAP stimulation are given in Fig. 1: I, II. The cell responded to non-nociceptive electrodermal stimulation of the lip and of the forelimb with a latent period of 31 and 44 msec respectively and to nociceptive stimulation of the pulp of the canine tooth with a latent period of 42 msec, by discharges consisting of 2 or 3 spikes (Fig. 1: I, a, b, c).

The character of responses to peripheral stimulation after EAP stimulation of the concha auriculae changed as follows: responses of the neuron to non-nociceptive electrodermal stim-

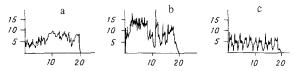


Fig. 2. Histograms of spike discharge of high-threshold nociceptive PFC neuron: a) spontaneous unit activity, b) spike discharge in response to nociceptive stimulation of contralateral lip (15 mA, 3 msec), c) spike discharge after EAP stimulation of concha auriculae in response to nociceptive stimulation of lip. Abscissa, time, (in sec); ordinate, number of spikes.

ulation were facilitated, i.e., the number of discharges in the burst was greater than in responses before EAP stimulation, and secondary bursting discharges appeared; the latent periods also were reduced during stimulation of the lip and forelimb to 23 and 38 msec respectively (Fig. 1: II, a, c). Meanwhile responses of the neuron during stimulation of the pulp of the canine tooth were absent immediately after EAP stimulation and for a short time longer (Fig. 1: II, b); a considerable increase in the frequency of spontaneous activity was observed under these circumstances. Histograms of the discharges of this particular neuron before and after EAP stimulation are shown in Fig. 1: III. Under the influence of EAP stimulation the cell thus changed both its evoked firing pattern and its spontaneous discharge, followed by a return practically to the original level (Fig. 1: III, c).

Neurons with tonic changes in evoked activity in response to testing nociceptive stimulation also changed the character of their responses after EAP stimulation. Six of eight neurons which considerably increased their spike activity in response to nociceptive stimulation did not respond by enhancement of their spontaneous discharge during or for some time after the end of EAP stimulation. Poststimulus histograms of responses of a high-threshold nociceptive PFC neuron to stimulation of the lip, before and after EAP stimulation, are illustrated in Fig. 2b, c. This neuron, which initially exhibited spontaneous activity (Fig. 2a), increased its discharge frequency in response to nociceptive stimulation of the lip (Fig. 2b). After EAP stimulation, nociceptive stimulation of the lip caused no increase in the discharge frequency of this neuron (Fig. 2c).

The results are thus evidence that EAP stimulation, by changing the character of the firing pattern of the neurons in PFC, to which an important role in mechanisms of nociception is ascribed [2, 10], forms a new functional state of the cells; EAP stimulation under these circumstances limits their activation by high-threshold nociceptive afferents. The analgesic effect of EAP stimulation may be produced by changes in unit activity of the deep brain structures [3], including thalamic nuclei [5], which transmit nociceptive information in the ascending direction. It can be postulated that changes in the functional state of neurons under the influence of EAP stimulation modify the architectonics of a functional system which, when formed in the CNS, takes part in the response of the organism to pain.

LITERATURE CITED

- 1. A. V. Val'dman, Vestn. Akad. Med. Nauk SSSR, No. 9, 11 (1980).
- R. A. Durinyan, Vestn. Akad. Med. Nauk SSSR, No. 9, 38 (1980).
- 3. L. V. Kalyuzhnyi, Vestn. Akad. Med. Nauk SSSR, No. 2, 75 (1982).
- 4. G. N. Kryzhanovskii, Vestn. Akad. Med. Nauk SSSR, No. 9, 33 (1980).
- 5. E. E. Meizerov, V. K. Reshetnyak, and R. A. Durinyan, Byull. Éksp. Biol. Med., No. 8, 12 (1981).
- 6. F. N. Serkov and V. N. Kazakov, Neurophysiology of the Thalamus [in Russian], Kiev (1980).
- 7. L. N. Smolin, Neirofiziologiya, No. 6, 621 (1981).
- 8. K. V. Sudakov, Vestn. Akad. Med. Nauk SSSR, No. 9, 17 (1980).
- 9. A. I. Treshchinskii and M. N. Basmanov, Anesteziol. Reanimatol., No. 1, 62 (1977).
- 10. W. K. Dong, H. Ryu, and I. H. Wagman, J. Neurophysiol., 41, 1592 (1978).
- 11. M. B. Eriksson, B. H. Sjolund, and U. Nieizens, Pain, 6, 335 (1979).
- 12. H. H. Jasper and C. Ajmone-Marsan, A. Stereotaxic Atlas of the Diencephalon of the Cat, Ottawa (1954).