spheres with the brain stem. It can be concluded from the experimental results that the cerebral hemispheres exert tonic inhibitory control over interneuronal activity in the spinal cord. This control is disturbed by removal of the sensomotor cortex, from which the pyramidal system arises, and after division of the basis pedunculi, through which its fibers run. However, the same effect also arises after division of the tegmentum mesencephali, through which run fibers from the cortes and tegmental nuclei to the red nucleus, which belongs to the extrapyramidal system. A similar effect was found during an investigation of the effect of division of the tegmentum on conduction of impulses in the nuclei of the dorsal columns [2]. The inhibitory action of the cerebral hemispheres on spinal interneurons is thus linked with functionally different descending systems. It can be tentatively suggested that this action is realized through nuclei of the brain-stem reticular formation, on which fibers from different descending pathways converge widely.

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EFFECT OF ELECTROACUPUNCTURE ON CHANGES IN FIRING PATTERN OF CORTICAL NEURONS OF THE SECOND SOMATOSENSORY AREA

M. L. Kukushkin and V. K. Reshetnyak

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KEY WORDS: somatosensory cortex; nociception; electroacupuncture.

Depression of nociceptive sensitivity during reflex therapy is associated with inhibition of condution of nociceptive impulses in afferent pathways of the CNS due to activation of the brain antinociceptive system [2, 5, 7]. An important role in the development of the analgesic affect arising after electroacupuncture (EAP) is played by the second somatosensory area of the cortex (area SII), which is responsible for modulating activity of the brain antinociceptive system [2, 5]. It has also been shown that the amplitude of evoked potentials in region SII after EAP in response to nociceptive stimulation is reduced, whereas during nonnociceptive stimulation it is increased [6]. These results suggested that EAP has opposite effects on nociceptive and non-nociceptive neurons in area SII.

To confirm this hypothesis directly it was decided to study changes in spontaneous and evoked single unit activity in area SII in response to nociceptive and nonnociceptive stimulation after EAP.

EXPERIMENTAL METHOD

Acute experiments were carried out on 32 adult cats anesthetized beforehand with hexobarbital (25-30 mg/kg, interperitoneally), immobolized sith succinylcholine, and artificial-

Central Scientific-Research Institute of Reflex Therapy, Ministry of Health of the USSR, Moscow.

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ly ventilated. Unit activity in area SII was recorded by a monopolar technique with glass microelectrodes (diameter of tip 1-3 μ), filled with 2 M KCl solution (resistance 4-10 MΩ) Unit activity was recorded on a VC-9 electrophysiological system (Nihon Kohden, Japan) and simultaneously on magnetic tape. The spike discharges were processed by means of an ANOPS-101 phase-amplitude analyzer and electronic computer. The significance of results was determined by Student's t test. To obtain non-nociceptive responses electrical stimulation was applied to the tissues of the super lip and the fore and hind limbs. The intensity of stimulation did not exceed 2 mA for pulses 0.1-0.3 msec in duration, or not more than 3 thresholds from the time of appearance of the primary sensory response. Preliminary investigations of unrestrained animals showed that the nociceptive behavioral responses appeared to stimulate more than 4-5 thresholds higher than that of the primary sensory response. Stimulation of the lump of the upper canine teeth and also of the tissues of the lip and the fore- and hind limbs with a pulsed current (10-15 mA pulse duration 0.1-0.3 msec) was used as nociceptive stimulation. EAP was applied to the concha ariculae or to distal parts of the limbs through implanted steel needles for 5 min. The frequency of stimulation was 1-3 pulses/sec and the intensity of EAP was limited by the appearance of local muscular contractions. Cortical unit activity began to be recorded 3-4 h after injection of the anesthetic. The animals' functional state was monitored, with measurement of the heart rate, blood pressure, and cerebral cortical temperature.

EXPERIMENTAL RESULTS

Altogether 101 neurons were recorded, of which 53 (52.5%) were spontaneously active and responded to no kind of peripheral stimulation, whereas 48 neurons (47.5%) showed evoked responses of excitation in the form of single spikes or burst discharges in response to stimulation. Depending on their responses to peripheral stimuli, the recorded neurons were divided into three groups. The first group consisted of neurons (58.3%) responding to weak electrical stimulation (under 2 mA). They were qualified as non-nociceptive low-threshold neurons (LTN). The second group of neurons (18.8%) responded only to strong electrodermal stimulation or to stimulation of the pulp of the canine teeth, and they consisted of high-threshold nociceptive neurons (HTN). The threshold of excitation of the HTN was 8-12 times higher than that of LTN. The third group consisted of neurons (22.9%) of mixed type (NMT). These neurons began to respond to stimulation of weak intensity, as a rule by one or two spikes with a short latent period. An increase in the strength of stimulation led to the appearance of additional spikes, arising after a longer latent period (from 80 to 350 msec). In most cases all three types of neurons could be recorded during the same insertion of the electrode, if made perpendicularly to the cortical surface. A particular feature of these neurons was the similarity of their receptive fields, confirming the somatotopic principle of organization of area SII [9]. The latent period of the majority of HTN exceeded 15 msec and it was 1.5 times longer than the latent period of the LTN. This is in good agreement with the views on the slower condution of nociceptive impulses spreading along A-δ- and C-fibers [10].

EAP changed the spontaneous discharge frequency of area SII neurons. Most spontaneously active neurons (47.2%) inhibited their activity, 22.6% of neurons were activated, and 30.2% of cells did not change their firing rate, although some neurons changed the character of their spontaneous discharge. Predominant inhibition of unit activity in the sensomotor cortex after EAP also was observed by other workers [5, 8]. A marked inhibitory effect also was noted after microiontophoretic application of morphine, β-endorphin, and enkephalins to cortical neurons [4, 5, 8]. Inhibition of neurons observed after EAP and application of morphine and opioid peptides, unlike their activation, is abolished by naloxone [5, 8]. The results thus suggest that inhibition of spontaneous activity of area SII neurons after EAP is mediated by opioid peptides, whose participation in mechanisms of reflex analgesia has been demonstrated by many workers [5, 7, 11].

Changes in evoked responses of area SII neurons to nociceptive and non-nociceptive stimulation after EAP also had differences. Non-nociceptive LTN did not change the character of their response after EAP and their discharge consisted of one or two spikes with the same latent period as before EAP. Neurons responding to nociceptive stimulation behaved somewhat differently. The majority (six of nine) of nociceptive TN did not change their response after EAP. However, three neurons inhibited their evoked activity. The trace of one of the neurons which responded to nociceptive stimulation (15 mA, 0.1 msec) of the contralateral limb and which inhibited its response after EAP, is illustrated in Fig. 1. Immediately after EAP the neuron did not respond to stimulation. An increase in the intensity of the stimulus likewise

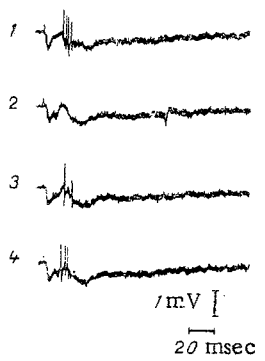

Fig. 1

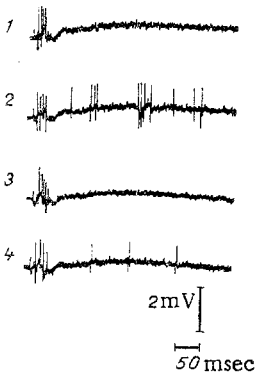

Fig. 2

Fig. 1. Inhibition of evoked response of HTN after EAP: 1) response of neuron before EAP. 2, 4, 5) Responses 1, 5, and 15 min after EAP respectively.

Fig. 2. Inhibition of long-latency component of response of NMT after EAP. 1, 3) Evoked response to non-nociceptive stimulation before and after EAP respectively; 2, 4) evoked response to nociceptive stimulation before and after EAP respectively.

did not induce the neuron to respond. The test neuron began to respond to stimulation 4 min after the end of EAP, but the number of spikes in the response was reduced, the latent period increased, and the neuron did not discharge to every stimulus presented. Not until 15 min after EAP was the character of response of the neuron restored. Evoked responses of two other nociceptive neurons also were similarly suppressed.

An investigation of NMT, responding simultaneously to nociceptive and non-nociceptive stimulation, showed that EAP inhibited mainly late long-latency components of responses to nociceptive stimulation in nine of 11 such neurons, but had no effect on the short-latency response arising to non-nociceptive stimuli (Fig. 2). In other words, during strong and weak stimulation after EAP, NMT responded near-identically, evidencing selective inhibition of the nociceptive input after EAP to NMT. Similar results were obtained by the present writers when studying neurons in the ventrobasal complex of the thalamus [3] and by other investigators in the sensomotor cortex [5, 8]. The results of the present investigation are in good agreement with those of previous studies, which showed that EAP leads to an increase in evoked potentials recorded in area SII in response to non-nociceptive stimulation, and to a decrease in amplitude of evoked potentials to nociceptive stimulation [6]. The presence of nociceptive neurons in area SII and changes in their evoked activity after EAP confirm the important role of area SII in the mechanisms of pain [1] and of reflex analgesia [2, 7]. The data showing marked depression of late nociceptive responses in NMT, responsible for the genesis of the protopathic component of pain, by EAP and the very weak inhibition of activity of specific nociceptive neurons responsible for the formation of the epicritic component of pain, explain clinical observations indicating that morphine and EAP affect mainly protcpathic pain and cause only weak depression of actue epicritic pain [1, 7], and are in good agreement with them.

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DENDRITIC SYNAPSES OF CAT PHRENIC MOTONEURONS

I. A. Keder-Stepanova and V. B. Petryaevskaya

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KEY WORDS: phrenic nucleus; motoneurons; dendrites; spines.

The onset of respiratory rhythmic activity in neurons of the phrenic nucleus in the spinal cord of intact animals is determined by descending pathways from the medullary respiratory center. We know that central respiratory neurons have monosynaptic connections with motoneurons of the diaphragm [4, 6]; all motoneurons, moreover, receive the same synaptic excitatory inflow [2]. As a result of supraspinal influences synchronous activity of phrenic motoneurons takes place, and is manifested as a slow depolarization wave, in phase with inspiration. The problem of the structural basis of this synchronous activation has not yet been solved. It was suggested some time ago that descending respiratory fibers terminate on dendrites of phrenic motoneurons, which form tightly packed bands [1]. If junctions of any kind exist between dendrites, a small number of fibers approaching them may trigger all the motoneurons of the nucleus. The existence of electrical synapses has not previously been discovered in the phrenic nucleus [5].

This paper describes various formations which may be responsible for junctions between dendrites of motoneurons and, in the writers' opinion, which may be the basis for their synchronous activation.

EXPERIMENTAL METHOD

Regions consisting of 4-6 cervical segments were excised after acute experiments on 48 adult cats. Pieces of spinal cord were stained by Golgi's method in the modification of Kopsch or Bubenaite. Frontal, horizontal, and sagittal serial sections 100 μ thick were used.

EXPERIMENTAL RESULTS

Dendrites of phrenic motoneurons can be divided into six types depending on the zones of their terminal ramifications (projections). Most radial dendrites and also rostrocaudal dendrites form bundles or bands. Spines were found on widely different segments of dendrites in all bands. Most frequently projections, located very close together and resembling a comb, could be seen on the surface of the dendrite. Spines resembling long rods without thickenings, or with various kinds of thickenings in the form of simple or more complex plaques also were seen. Regions of interweaving dendrites, with single projections clearly visible on their surface, are shown in Fig. 1a, and well-marked spines resembling rods with plaques at their ends are shown in Fig. 1b, c — in this case one plaque sits on a curved rod (high degree of differentiation). Various types of well-organized spines can be seen in Fig. 1d, between dendrites running into lamina VIII. Projections whose tips are arranged opposite one another can also be seen on dendrites of a ventrolateral band (Fig. 1e). The same picture also is observed in a band running in the ventromedial direction (Fig. 1f); a spine in contact with a neighboring dendrite can be seen here. Often one dendrite could be seen to run toward the neighboring group of cells of the nucleus and, when followed further, to form an *en passant* synapse with the initial segment of the dendrite of a certain motoneuron. These synapses are indicated in Fig. 2a, b by arrows; in Fig. 2a the right dendrite of a large cell forms

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