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MODULATING EFFECT OF THE SECOND SOMATOSENSORY AREA OF THE CORTEX TO ELECTROACUPUNCTURE EFFECTS IN THE TRIGEMINAL NUCLEI

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The cerebral cortex plays an important role in the control of conduction of afferent information at different levels of the CNS [1, 3]. This control is effected also on the trigeminal nuclei [7, 8, 10], which play an important role in the formation of pain syndromes and, in particular, in trigeminal neuralgia [5]. As has been shown, electroacupuncture (EAP) modifies the functional state of several of the trigeminal nuclei [2]. For instance, in the caudal trigeminal nucleus (CTN) it leads to inhibition of nociceptive signals, whereas in the oral trigeminal nucleus (OTN) it facilitates conduction of afferent signals. An important role in the formation of the effects of EAP at different levels of the CNS is ascribed to the second somatosensory area of the cortex (area SII) [3, 4, 6]. However, its direct influence on the effectiveness of EAP at the level of the primary relay nuclei has not yet been studied.

In this investigation the effect of reversible functional blocking of area SII on the conduction of afferent signals through OTN and CTN during EAP was studied.

EXPERIMENTAL METHOD

Acute experiments were carried out on 12 adult cats, anesthetized with hexobarbital (50 mg/kg, intraperitoneally), immobilized with suxamethonium, and artificially ventilated. Afferents of the pulp of the canine tooth (3 msec, up to 20 mA) and of the lip (0.3 msec, 1-2 mA) were stimulated. EAP was applied through steel acupuncture needles, inserted into the lower part of the base of the concha auriculae (where trigeminal nerve endings are located), in the form of square pulses of current 1 and 2 msec in duration, with an intensity of 16 mA, and with a frequency of 1 Hz for 15 min. Reversible functional blocking of contralateral area SII was carried out during EAP by application of cold. The cortical surface temperature in area SII was about 22°C. After the end of EAP and before recording of evoked potentials (EP), the cortex was heated up to the original temperature. Evoked potentials (EP) were recorded by a monopolar method, ipsilaterally in OTN and in the ventromedial border of CTN. The results were averaged by specialized computer on the basis of 10 presentations. The site of recording of EP was verified histologically after microcoagulation.

EXPERIMENTAL RESULTS

When EAP was carried out with area SII intact, changes in evoked activity were observed in nuclei of the trigeminal complex. In CTN the EP arising to stimulation of the dental pulp were depressed by 50% whereas EP in response to stimulation of the lip in that nucleus were unchanged (Fig. 1). Meanwhile, in OTN, an increase in amplitude of EP in response to stimulation of both dental pulp and lip, by 150-160%, was observed.

One way whereby the cerebral cortex can influence the nuclei of the trigeminal complex is via the pyramidal tract [7, 10]. In the experiments of series I, to discover whether, in

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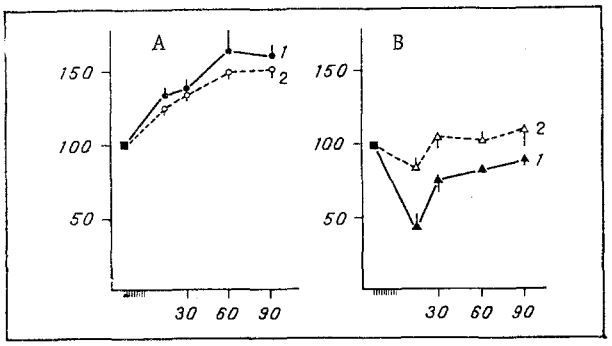

Fig. 1

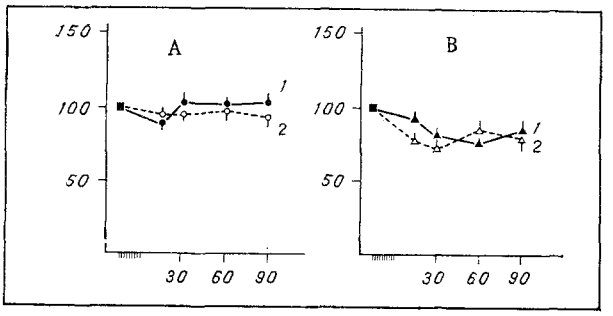

Fig. 2

Fig. 1. Dynamics of changes in amplitude of EP in trigeminal nuclei after EAP. Abscissa, time (in min); ordinate, amplitude of EP (in per cent). A) OTN, B) CTN. Stimulation of: 1) dental pulp, 2) lip.

Fig. 2. Dynamics of changes in EP amplitude in trigeminal nuclei during EAP accompanied by functional blocking of area SII of the cortex. Legend as to Fig. 1.

principle, the cerebral cortex participates in the mechanisms of EAP at the level of the trigeminal complex, the pyramidal tract was destroyed in the zone of the trapezoid body. This led to abolition of the controlling action of EAP on conduction of the signals through the trigeminal nerve. For subsequent more precise analysis of the role of area SII in the mechanisms of EAP, this region was subjected to local cold block, which caused temporary, reversible blocking of its effect on subsequent processes.

Against this background of cold block of area SII, EAP had no modulating effect on signal conduction through the trigeminal nuclei (Fig. 2). In CTN, for instance, the amplitude of EP in response to stimulation of the dental pulp and lip fell by only 20%, and in OTN it was virtually unchanged.

These data indicate that area SII has an important role in the initiation of the mechanisms of change of the state of CNS function under the influence of EAP. Abolition of the effect of EAP, incidentally, is observed simultaneously in two functionally different nuclei of the trigeminal complex, namely in OTN and CTN. Functional inactivity of area SII is responsible for weakening of the effect of inhibition of conduction of nociceptive impulses through CTN during EAP. Meanwhile absence of facilitation of conduction through OTN under these conditions means that EAP is ineffective in mobilizing the reflex protective responses of the body and, in particular, the mouth opening reflex, in which OTN plays the leading role [9]. This confirms the conclusion that area SII of the cerebral cortex has an essential role in the modulation of sensitivity to pain and coordination of a complex of protective reactions of the body in extremal states [3].

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