EFFECT OF ELECTROACUPUNCTURE ON INTEGRATION OF NOCICEPTIVE AND NON-NOCICEPTIVE INFORMATION IN THE SECOND SOMATOSENSORY CORTICAL AREA

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Among methods of nonmedicinal treatment of pain syndromes acupuncture and electroacupuncture (EAP) have become widely used in recent years [9, 10]. The great practical importance of the problem of relief of pain has necessitated experimental and clinical studies of mechanisms of reflex analgesia on a comprehensive scale [4, 6, 9].

In the modern view nociceptive sensation is the integrated response of the CNS invariably involving cortico-subcortical mechanisms of interaction [2, 7, 8]. Animal experiments have shown that the second somatosensory cortical area (SII) plays the leading role in evaluation of the extremal significance of incoming information (including nociceptive) at the cortical level [3]. It has been suggested that a reduction in the intensity of pain sensations associated with EAP may be largely due to a change in the character of cortico-subcortical interactions in the direction of strengthening of the mechanism of cortical inhibition [5].

In the investigation described below changes in evoked potentials (EPs) to nociceptive and non-nociceptive stimulation were studied in SII during EAP.

EXPERIMENTAL METHOD

Acute experiments were carried out on 18 cats anesthetized by intraperitoneal injection of hexobarbital solution in a dose of 40 mg/kg, immobilized with suxamethonium and supported by artificial ventilation of the lungs, by means of a stereotaxic technique. Cortical EPs were recorded by monopolar silver electrodes 0.9 mm in diameter, and in the parafascicular complex of the thalamus (PFC) by steel electrodes with a tip 50-70 μ in diameter, 5 h after injection of the anesthetic.

Nociceptive responses were evoked by single stimulation of the dental pulp (lower canine tooth) by square pulses (1-3 msec, not exceeding 20 mA). Non-nociceptive responses were obtained to stimulation of the lower lip through bipolar needle electrodes with a pulsed current (0.1 msec, not exceeding 5 mA).

EAP stimulation was given through three acupuncture needles, inserted into different points of the concha auriculae of the cat, to which a current was applied (1.2 msec, 3 Hz, not exceeding 14 mA, for 15-30 min). Low-frequency stimulation of SII (1-3 Hz) to modify its modulating effect was carried out through a bipolar electrode (interelectrode distance 1.5 mm) with square pulses (0.1 msec, not exceeding 500 μA).

EPs were assessed after accumulation and averaging over 16 presentations by specialized computer, followed by statistical analysis (M ± m). The surface temperature of the cortex, arterial blood pressure, and ECG were recorded. At the end of the experiment the animal was given a lethal injection of hexobarbital and the brain was removed for histological verification of the location of the recording electrodes.

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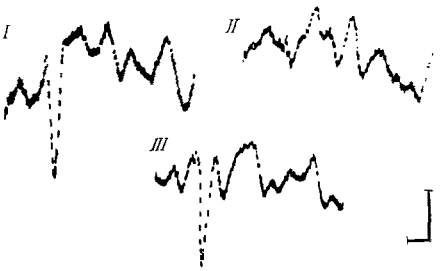

Fig. 1

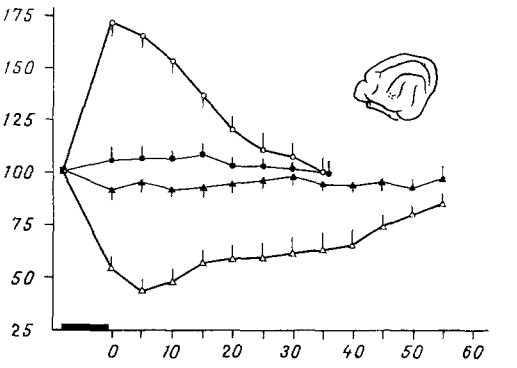

Fig. 2

Fig. 1. Effect of EAP and naloxone on changes in EP amplitude in SII to stimulation of dental pulp. I) Control EP; II) EP in SII after EAP; III) EP in SII after EAP and injection of naloxone. Calibration: time 100 msec, amplitude 50 μV.

Fig. 2. Changes in amplitude of EPs in SII to nociceptive (empty triangles) and non-nociceptive (empty circles) stimulation after EAP. Filled circles and triangles denote changes in amplitude of EP in control respectively. Top right) diagram showing location of recording electrodes in SII. Abscissa, time (in min); ordinate, EP (in %).

EXPERIMENTAL RESULTS

When EPs were recorded in cortical projection areas to stimulation of the pulp of the contralateral tooth, the focus of maximal activity of the potentials was located in the rostral part of the anterior ectosylvian gyrus, i.e., in area SII. The evoked response consisted of a well-marked primary positive wave followed by a negative wave (of low amplitude, appearing irregularly). The mean amplitude of the EP was 150 μV, the duration of the positive wave 40 msec, and its latent period 10.7 msec (Fig. 1, I). When EPs were recorded in other cortical projection areas, the amplitude of the responses was much lower (not exceeding 100 μV), the latent period of the primary response was longer (by 2-5 msec), and the appearance of the positive–negative waves was unstable. Responses to threshold stimulation of the lower lip were recorded at the same points as those to stimulation of the dental pulp.

After auricular EAP a marked decrease was found in the amplitude of EPs to stimulation of the dental pulp — by up to 40% of the control value — followed by recovery 50-60 min later (Fig. 2, lower part). EAP had a directly opposite effect on the character of responses to non-nociceptive stimulation of the lower lip. In this case the amplitude of EPs increased by 75% above the control level. Facilitation of the responses continued for 15-20 min after the end of EAP (Fig. 2, top part). Particular attention is drawn to the fact that the periods of maximal depression and facilitation of the EPs practically coincided in time. This reciprocal relationship between the responses of cortical neurons to the arrival of nociceptive and non-nociceptive impulses, which other workers also have observed [11], is probably the result of the development of a new functional state in the CNS after EAP. As a result both selective facilitation of the conduction of afferent impulses along low-threshold, fast-conducting systems and activation of the cortical neurons receiving efferent signals along specific systems take place. The more prolonged depression of EPs to nociceptive stimulation than their facilitation to non-nociceptive stimulation can evidently be explained by activation of neurochemical mechanisms modulating the conduction of nociceptive impulses during EAP.

To test this hypothesis, naloxone (5 mg/kg), an antagonist of opiates, including endogenous opiates, was injected intravenously at a time when the amplitude of the nociceptive EPs was depressed. After injection of naloxone a more rapid recovery of the amplitude of EP to stimulation of the pulp of the canine tooth was observed practically up to the control level (Fig. 1).

Blocking the conduction of nociceptive afferent impulses during stimulation of the dental pulp was observed at the level of the primary sensory relays during direct cortical stimulation [1]. During stimulation of SII for 1 min the amplitude of nociceptive EPs in the parafascicular complex of the thalamus was depressed for 1.5-3 min (Fig. 3). Incidentally, stimu-

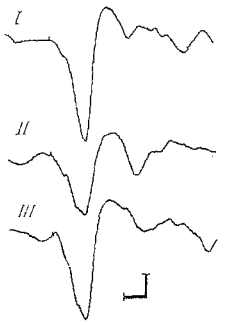

Fig. 3. Changes in EPs in PFC during stimulation of dental pulp after electrical stimulation of SII. I) Control EP; II) EP after stimulation of SII; III) EP 2 min after stimulation of SII. Calibration: time, 100 msec; amplitude, 25 μV.

lation of other cortical areas (SI, motor area, parietal association region) caused a much weaker depressive effect. These observations confirmed the view that SII can exert a modulating influence on the conduction of afferent impulses along specific and nonspecific projection systems [4], both during direct activation of this area and also during EAP [5].

LITERATURE CITED

1. S. M. Butkhuzi, V. G. Verishvili, and E. V. Abzianidze, Soobshch. Akad. Nauk Gruz. SSR, 98, No. 3, 677 (1980).
2. A. V. Val'dman, Vestn. Akad. Med. Nauk SSSR, No. 9, 11 (1980).
3. R. A. Durinyan, Vestn. Akad. Med. Nauk SSSR, No. 9, 38 (1980).
4. R. A. Durinyan, Usp. Fiziol. Nauk, No. 1, 3 (1980).
5. R. A. Durinyan, V. K. Reshetnyak, O. N. Moskovets, et al., in: Abstracts of Proceedings of the 2nd Congress of Physiologists of the Moldavian SSR [in Russian], Kishinev (1980), p. 98.
6. L. V. Kalyuzhnyi and E. V. Golanov, Usp. Fiziol. Nauk, No. 3, 85 (1980).
7. G. N. Kryzhanovskii, Vestn. Akad. Med. Nauk SSSR, No. 9, 33 (1980).
8. K. V. Sudakov, Vestn. Akad. Med. Nauk SSSR, No. 9, 17 (1980).
9. A. I. Treshchinskii and S. N. Basmanov, Anesteziol. Reanimatol., No. 1, 62 (1977).
10. R. Astuti and M. Vannucci, Riv. Ital. Agopunt., 13, 47 (1980).
11. K. Toda, M. Ichioka, H. Suda, et al., Exp. Neurol., 63, 652 (1979).