CHANGES IN CEREBRAL CORTICAL ELECTRICAL ACTIVITY IN RATS WITH COMPRESSION OF THE INFRAORBITAL NERVE

V. G. Dolgikh, V. K. Reshetnyak, and G. N. Kryzhanovskii* UDC 616.833.152-001.35-092.9-07: 616.831.31-073.97

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Compression of the second and third branches of the trigeminal nerve in most cases plays a leading role in the pathogenesis of trigeminal neuralgia (especially in the initial stage of the disease) [7]. Our experiments using rats as the model showed that chronic compression of the infraorbital nerve leads to the development of a pain syndrome, manifested as changes in the animal's behavior, thresholds of pain sensitivity, the microcirculatory system, and stress-sensitive organs [5]. Development of the pain syndrome is associated with the formation of generators of pathologically enhanced excitation (GPEE) in the central structures of the nociceptive system [2].

In the investigation described below changes in spontaneous and evoked electrical activity in the rat cerebral cortex during compression of the infraorbital nerve were studied.

EXPERIMENTAL METHOD

Experiments were carried out on 19 male Wistar rats weighing 350-400 g. The infraorbital nerve was compressed 1.5-2 months before the electrophysiological investigations by applying two incomplete ligatures to the nerve. The operation to prepare the animal for the electrophysiological investigation was performed under ether anesthesia. After trephining of the skull above the somatosensory cortex, the animal was given a muscle relaxant and artificial ventilation of the lungs carried out. The skin was stimulated by square pulses of current (0.1-10 mA, duration 0.3 msec), applied through needle electrodes in symmetrical points of the rat's snout in the region of the infraorbital foramen. Evoked potentials (EP) were derived from the surface of the cerebral cortex by means of silver ball electrodes 1.0 mm in diameter at the focus of maximal activity in symmetrical regions of the right and left cerebral hemispheres. The reference electrode was located in the region of the nasal sinus. To prevent drying of the cortex or shunting of the electrodes, mineral oil was used. Averaged EP were recorded on "Disa" and "Nihon Kohden" oscillographs and on a DFR tape recorder ("Sony") for 10 presentations.

EXPERIMENTAL RESULTS

An increase in amplitude of the first component of the EP in the contralateral cerebral cortex on average by 272 ± 14% was observed 1.5-2 months after compression of the infraorbital nerve in 75% of animals (15 rats) in response to stimulation of the snout on the side of injury to the nerve compared with stimulation of the intact side (Fig. 1). This increase in amplitude of EP was accompanied by lowering of the thresholds of their appearance. With contralateral recording the threshold of appearance of EP during stimulation of the side of nerve injury was 0.29 ± 0.02 mA, compared with 0.50 ± 0.04 mA with stimulation of the intact side ($p < 0.05$). Thresholds of appearance of

*Academician of the Academy of Medical Sciences.

Laboratory of Pathophysiology of Pain and Laboratory of General Pathology of the Nervous System, Research Institute of General Pathology and Pathological Physiology, Academy of Medical Sciences, Moscow. Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 113, No. 5, pp. 458-459, May, 1992. Original article submitted April 16, 1991.

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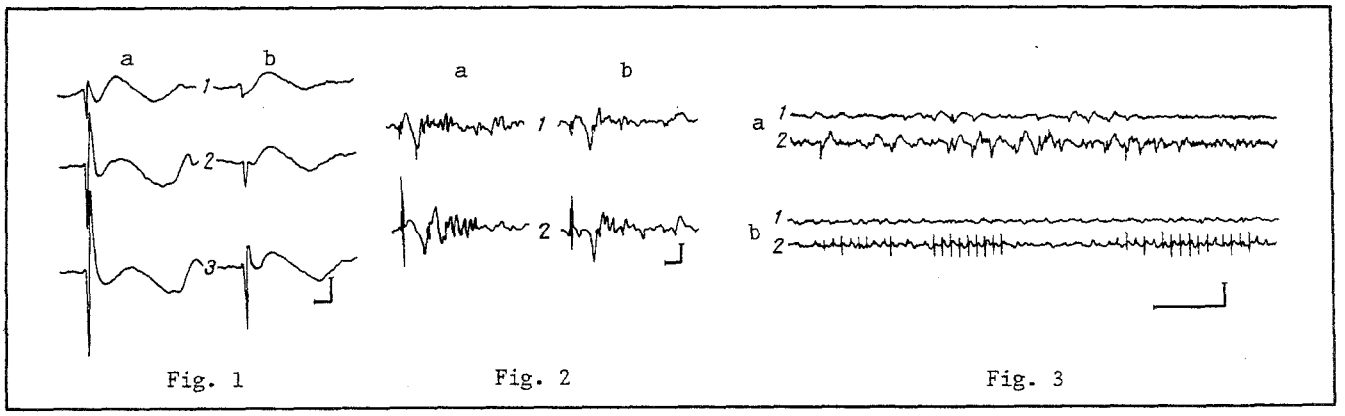

Fig. 1 Fig. 2 Fig. 3

Fig. 1. Evoked potentials in somatosensory cortex of contralateral cerebral hemispheres in rats with compression of infraorbital nerve during stimulation of snout on side of injured (a) and intact (b) nerves at threshold intensity (1) and with stimulation of twice (2) and four times (3) threshold strength. Calibration: 1 mV, 50 msec.

Fig. 2. Late component of evoked potentials and after-discharge in somatosensory cortex of ipsilateral (1) and contralateral (2) cerebral hemispheres in rats with compression of infraorbital nerve and in response to stimulation of snout on side of injured (a) and intact (b) nerves. Calibration: 1 mV, 200 msec.

Fig. 3. Epileptiform activity in somatosensory cortex of cerebral hemispheres in rats with compression of infraorbital nerve: pointed waves (a) and spike-wave complexes (b) with ipsilateral (1) and contralateral recording (2). Calibration: 5 mV, 1 sec.

EP with ipsilateral recording were 0.68 ± 0.06 mA and 1.02 ± 0.04 mA respectively ($p < 0.05$). Changes in the amplitude of EP in the ipsilateral derivation were not significant ($p > 0.01$).

Another distinguishing feature of evoked activity was a late component of EP, with a latency of 150-200 msec. It was recorded most frequently in the hemisphere contralateral to the side of nerve injury: in response to stimulation of the tissues of the snout on the side of nerve injury in 17 animals, and to stimulation of tissues on the intact side of the snout in 16 animals. The late component of EP was recorded somewhat less frequently in the somatosensory cortex of the hemisphere ipsilateral to the side of infraorbital nerve injury: to stimulation of tissues on the intact side of the snout in 13 animals and tissues of the snout on the side of nerve injury in 10 animals.

An after-discharge consisting of 5-7 negative-positive waves with a frequency of 12-17 Hz was recorded (Fig. 2) in three rats with compression of the infraorbital nerve immediately after the late component of EP.

In four animals, epileptiform activity containing pointed waves and spike-wave complexes was recorded in the somatosensory cortex (Fig. 3).

It can be concluded from the results of these investigations that chronic trauma to the infraorbital nerve is accompanied by the appearance in the CNS of a GPEE, whose activity is manifested as changes in EP mentioned above and spontaneous epileptiform activity in the somatosensory cortex. Similar epileptiform changes in spontaneous and evoked activity in the cerebral cortex have been described in patients with trigeminal neuralgia [1, 6].

GPEE formation in the nuclei of the trigeminal complex and, in particular, in the caudal trigeminal nucleus, by means of several different agents [2, 4, 10, 11] is accompanied by the development of a pain syndrome in animals, typical of trigeminal neuralgia. The appearance of hyperactivity in the caudal trigeminal nucleus has been described in patients with damage to the trigeminal nerve [9, 12]. High-frequency neuronal hyperactivity is observed in mesothalamic structures also [8].

Microinjection of procaine into the caudal trigeminal nucleus on the side of infraorbital nerve compression, which was used in some of our experiments, led to a decrease in amplitude of EP by 70% in both hemispheres during stimulation of the animals' snout both on the side of nerve injury and on the intact side. This fact can be explained by weakening of the hyperactivating influences of GPEE in the caudal trigeminal nucleus on higher structures of the CNS and, in particular, of the cerebral cortex.

LITERATURE CITED

1. V. A. Karlov, V. A. Polyantsev, S. E. Petrenko, et al., Zh. Nevropatol. Psikhiat., **83**, No. 4, 692 (1983).
2. G. N. Kryzhanovskii, S. I. Igon'kina, V. N. Grafova, et al., Byull. Éksp. Biol. Med., **78**, No. 11, 16 (1974).
3. G. N. Kryzhanovskii, Determinant Structures in Pathology of the Nervous System [in Russian], Moscow (1980).
4. G. N. Kryzhanovskii, M. P. Gorizontova, S.-I. Igon'kina, et al., Byull. Éksp. Biol. Med., **111**, No. 1, 9 (1991).
5. G. N. Kryzhanovskii, V. K. Reshetnyak, V. G. Dolgikh, et al., Byull. Éksp. Biol. Med., **112**, No. 8, 120 (1991).
6. E. E. Meizerov, V. K. Reshetnyak, A. M. Tauluev, et al., Zh. Nevropatol. Psikhiat., **86**, No. 12, 1795 (1986).
7. O. N. Savitskaya and V. A. Karlov, Zh. Nevropatol. Psikhiat., **89**, No. 4, 3 (1989).
8. O. J. Andy, Pavlov J. Biol. Sci., **22**, No. 4, 132 (1987).
9. R. G. Black, Pain and Suffering: Selected Aspects, Chapter 12, Springfield, Mass. (1970), pp. 119-137.
10. R. G. Black, Adv. Neurol., **4**, 651 (1974).
11. Y. Sakai, Y. Nishijima, N. Mikuni, et al., Pain, **7**, 147 (1979).
12. J. R. Schvarz, Advances in Pain Research and Therapy, Vol. 3, New York (1979), pp. 331-336.

MECHANISM OF THE ANTIARRHYTHMIC EFFECT OF LASER IRRADIATION

S. D. Mikhailova, G. I. Storozhakov, S. Yu. Gukova, and T. M. Semushkina

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The antiarrhythmic effect of intracardiac laser irradiation in myocardial ischemia is associated mainly with reduction of the coagulability and viscosity of the blood, inhibition of lipid peroxidation, and activation of mast cells, releasing vasodilators and improving the blood supply to the myocardium [2, 3]. In the ischemic heart these factors change not only the biochemical composition of the blood, but also excitability of the receptors, and this must inevitably be reflected in the functional state of the afferent innervation of the heart, whose role in the antiarrhythmic action of laser irradiation is not yet clear. Meanwhile, activation of the parasympathetic nervous system is known to lead to an increase in electrical stability of the myocardium [11, 13].

The aim of this investigation was to study the role of afferent information reaching the CNS along fibers of the vagus nerves from the receptor structures of the heart during intracardiac laser irradiation.

EXPERIMENTAL METHOD

Experiments were carried out on 65 male and female cats weighing 2.5-4 kg, under pentobarbital anesthesia (40 mg/kg, intraperitoneally) and artificial respiration. Myocardial ischemia was induced by compression of the circumflex branch of the left coronary artery for up to 15 min. The development of ischemia in the myocardium was recorded during 15 min after compression of the coronary artery and 15 min after release of the guide loop on the coronary artery. The following idioventricular disturbances of the cardiac rhythm were taken into consideration in the analysis: grouped ventricular extrasystoles, ventricular tachycardia, and ventricular fibrillation. Laser irradiation of the right atrium was carried out with light from an LG-75 helium–neon laser (power at the end of the light guide 3-5 mW,

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