However, marked generalized lesions of neuronal membrane formations are evidence of progressive destructive processes in the cells. Profound disturbance of membrane ultrastructure mainly in neurons in the late stages of exposure to hypoxia has been reported in several publications [1, 6]. At this time the harmful action of other factors, and, in particular, of a broad spectrum of lysosomal enzymes, is manifested [3]. Excessive activation of LPO in the neurons during ischemia and in the early stages of recirculation evidently plays a leading role in the increased lysosomal membrane permeability and in the solubilization and decompartmentalization of acid hydrolases, and through peroxidation of membrane structures it creates optimal conditions for realization of the hydrolytic effect of the lysosomal enzymes.

The sharply intensified processes of LPO in brain tissue during ischemia and in the early postischemic period thus cause damage to membrane structures at the molecular level and lysosome formation, which aggravates subsequent destruction of the nerve cells.

LITERATURE CITED

1. N. N. Bogolepov, Ultrastructures of the Brain in Hypoxia [in Russian], Moscow (1979).
2. V. D. Konvai, Patol. Fiziol., No. 5, 30 (1982).
3. V. G. Korpachev and V. V. Semchenko, Patol. Fiziol., No. 6, 62 (1980).
4. V. A. Negovskii, A. M. Gurvich, and E. S. Zolotokrylina, Postresuscitation Sickness [in Russian], Moscow (1979).
5. I. D. Stal'maya and T. G. Garishvili, in: Modern Methods in Biochemistry [in Russian], Moscow (1977), pp. 66-68.
6. V. P. Tumanov and M. D. Malamud, Changes in the Central Nervous System after Thermal, Radiation, and Combined Trauma [in Russian], Kishinev (1977).
7. N. M. Émanuél', Vest. Akad. Nauk SSSR, No. 9, 66 (1981).
8. J. Fridowich, Adv. Neurol., 26, 255 (1979).
9. J. Glaving, Acta Chem. Scand., 17, 1635 (1963).
10. L. Placer, Nahrung, 12, 679 (1968).

CHANGES IN ORBITOFRONTAL AND SOMATOSENSORY CORTICAL ELECTRICAL ACTIVITY DURING ELECTROACUPUNCTURE

V. K. Reshetnyak, M. L. Kukushkin, and R. A. Durinyan

UDC 615.844.4.015.4:612.825.2.014.423

KEY WORDS: electroacupuncture; morphine; evoked potential; cortex.

Considerable attention has been paid to the study of the mechanisms of pain [2, 8, 9, 12, 13]. At the same time, the mechanisms of formation of reflex analgesia are being studied, as one of the most promising methods of treatment of acute and chronic pain syndromes [4, 5]. The present writers have shown [11] that electroacupuncture (EAP) considerably depresses the conduction of nociceptive impulses at the level of the primary relay nuclei without affecting transmission of nonnociceptive tactile afferent impulses. It has been shown that EAP blocks nociceptive impulses in the specific and nonspecific thalamic nuclei. In particular, depression of nociceptive impulses is stronger in the parafascicular complex than in the specific projection nuclei, in the ventromedial nucleus for example [10]. An essential role in the mechanisms of pain perception is played by the orbitofrontal and second somatosensory (SII) areas of the cortex [2, 4].

Central Research Institute of Reflex Therapy, Moscow.

(Presented by Academician of the Academy of Medical Sciences of the USSR A. V. Val'dman.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 96, No. 7, pp. 14-16, July, 1983. Original article submitted June 4, 1982.

0007-4888/83/9607-0894$07.50

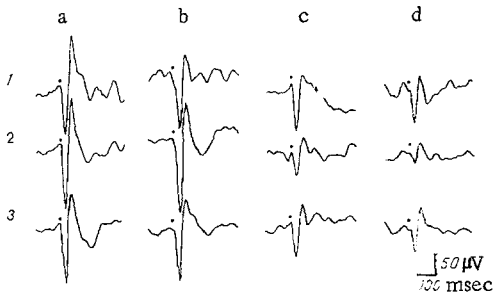

Fig. 1

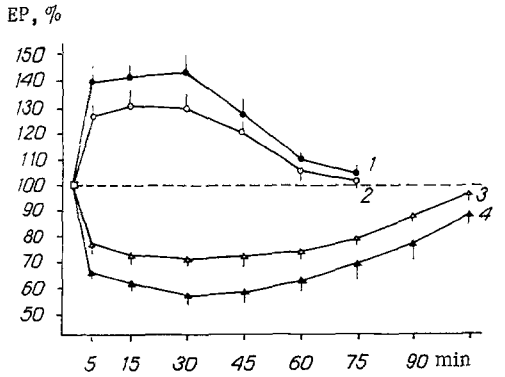

Fig. 2

Fig. 1. Changes in amplitude of EP to nonnociceptive (a, b) and nociceptive (c, d) stimulation in area SII (a, c) and orbital gyrus (b, d) after EAP. 1) Control, 2) 15 min after EAP, 3) (for a and b) 60 min, and (for c, d) 90 min after EAP. Stimulation artefact indicated by a dot.

Fig. 2. Changes in amplitude of EP to nonnociceptive (1, 2) and nociceptive (3, 4) stimulation after EAP. 2, 3) In SII; 1, 4) in orbital gyrus.

In the investigation described below changes in evoked potentials (EP) to nociceptive and nonnociceptive stimulation were studied in the orbitofrontal cortex and in area SII during EAP.

EXPERIMENTAL METHOD

Acute experiments were carried out on adult cats anesthetized with pentobarbital (40 mg/kg, intraperitoneally), curarized, and artificially ventilated. EP were recorded in the cortex of SII and the orbital gyrus by monopolar silver electrodes with a tip 0.8 mm in diameter. The reference electrode was secured in the frontal bone. Investigations began 5-6 h after injection of the anesthetic. Nociceptive responses were evoked by stimulation of the pulp of the upper canine tooth with single square pulses of current 1 msec in duration and up to 10 mA in strength. Nonnociceptive responses were obtained to stimulation of the upper lip through bipolar needle electrodes with pulses of current (0.1 msec, not more than 2 mA). EAP stimulation was applied through needles introduced into the concha auriculae, to which current was applied (1 Hz, 1 msec, not more than 5 mA) for 30 min. EP were evaluated after averaging of 20 presentations on a specialized computer.

EXPERIMENTAL RESULTS

EP arising in the orbitofrontal cortex and in area SII in response to stimulation of the pulp differed both in latent period and in amplitude. Naturally the latent periods of responses in the specific cortical projection area SII were shorter, namely 8.7 ± 2.43 msec. The latent period in the orbitofrontal cortex was 13.1 ± 3.41 msec. The amplitude of EP in SII was about 150 μV, and in the orbitofrontal cortex about 100 μV. Great instability of the positive and, in particular, the negative waves was observed in the orbitofrontal cortex compared with EP recorded in area SII. Responses arising in SII to nonnociceptive stimulation had a latent period of 6.2 ± 1.92 msec and an amplitude of 200 μV. The latent period of responses in the orbitofrontal cortex was 12.7 ± 2.74 msec and their amplitude was 150 μV.

After EAP the amplitude of EP to nociceptive stimulation fell considerably. Depression of EP was more marked in the orbitofrontal cortex than in area SII (Fig. 1) and amounted to 58 and 75%, respectively, of the control level. Investigation of the character of the changes in EP to nonnociceptive stimulation showed that EAP, on the contrary, considerably facilitated these responses (Fig. 1) in the frontal cortex by 45% and in SII by 30%. It should be noted that facilitation of EP to nonnociceptive stimulation in the orbitofrontal cortex not only was more marked than the responses in SII, but it was also more prolonged than in SII (Fig. 2). The magnitude and duration of depression of the amplitude of the responses to nociceptive stimulation also were greater in the orbitofrontal cortex (Fig. 2). It will be noted that periods of maximal depression of responses to nociceptive stimulation and periods of facilitation of responses to nonnociceptive stimulation coincided in time. These data suggest that EAP leads to reorganization of the functional state of efferent systems, by facilitating conduction of nonnociceptive impulses, which travel mainly along the specific lemniscal system, and by blocking con-

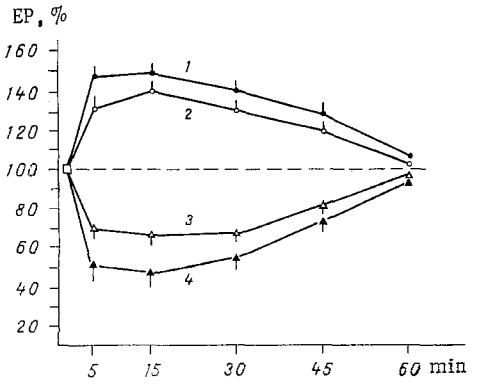

Fig. 3. Changes in amplitude of EP to nonnociceptive (1, 2) and nociceptive (3, 4) stimulation after intravenous injection of morphine. 2, 3) In SII; 1, 4) in orbital gyrus.

duction of nociceptive impulses, traveling along the extralemniscal afferent system. This hypothesis is in good agreement with views on the inhibitory interaction between the lemniscal and extralemniscal systems [3, 9].

In other experiments the action of morphine (5 mg/kg, intravenously) on changes in EP in the frontal cortex and in SII in response to nociceptive and nonnociceptive stimulation was investigated. The character of the change in EP under the influence of morphine was found to be largely similar to the time course of the changes in EP in SII and the orbitofrontal cortex during EAP (Fig. 3). After injection of morphine an increase in EP in response to nonnociceptive stimulation was observed, and in the orbitofrontal cortex, moreover, this increase was more marked than in SII. During nociceptive stimulation the amplitude of EP, just as during EAP, was depressed more in the frontal cortex than in SII.

It has been shown [4, 6, 7] that an essential role in the mechanism of reflex analgesia is played by endogenous opiates and various other neurotransmitters. It is also known that the concentration of opiate receptors on neurons in the frontal cortex is much higher than the density of these receptors on SII neurons [14]. This may probably explain the fact that depression of nociceptive responses in the frontal cortex after EAP is more marked than in SII. This is in good agreement with our views of the role of SII in mechanisms of modulation of antinociceptive systems and reflex analgesia [4]. The increase in amplitude of responses to nonnociceptive stimulation was probably due also to secretion of endogenous opiates and of other neurotransmitters, and was connected with facilitation of the transmission of afferent impulses along the fast-conducting lemniscal projections. The results of the present experiments to study the action of morphine on the amplitude of EP to nociceptive and nonnociceptive stimulation confirms this hypothesis.

It can thus be concluded that EAP, like morphine, leads to an increase in the flow of afferent impulses along the lemniscal system and, correspondingly, it depresses conduction of nociceptive impulses along the nonspecific multisynaptic system, in the work of which an essential role is played by endogenous opiates.

LITERATURE CITED

1. A. V. Val'dman and D. Yu. Ignatov, The Central Mechanisms of Pain [in Russian], Leningrad (1976).
2. A. V. Val'dman, Vest. Akad. Med. Nauk SSSR, No. 9, 11 (1980).
3. R. A. Durinyan, Cortical Control of Nonspecific Brain Systems [in Russian], Moscow (1975).
4. R. A. Durinyan, Vest. Akad. Med. Nauk SSSR, No. 9, 38 (1980).
5. R. A. Durinyan, Usp. Fiziol. Nauk, 11, No. 1, 3 (1980).
6. R. A. Durinyan and E. O. Bragin, Med. Ref. Zh., 9, No. 11, 17 (1979).
7. R. A. Durinyan, V. K. Reshetnyak, and S. M. Zaraiskaya, Med. Ref. Zh., No. 5, 13 (1981).
8. L. V. Kalyuzhnyi, Vest. Akad. Med. Nauk SSSR, No. 2, 75 (1982).
9. G. N. Kryzhanovskii, Vest. Akad. Med. Nauk SSSR, No. 9, 33 (1980).
10. E. E. Meizerov, V. K. Reshetnyak, and R. A. Durinyan, Byull. Éksp. Biol. Med., No. 8, 12 (1981).
11. O. N. Moskovets, V. K. Reshetnyak, and R. A. Durinyan, Byull. Éksp. Biol. Med., No. 1, 7 (1980).
12. K. V. Sudakov, Vest. Akad. Med. Nauk SSSR, No. 9, 17 (1980).
13. V. M. Khayutin, Vest. Akad. Med. Nauk SSSR, No. 9, 26 (1980).
14. R. Simantov and S. H. Snyder, Proc. Natl. Acad. Sci. USA, 75, 2515 (1978).