CHANGES IN THE JAW OPENING REFLEX DURING ANALGESIA AS A RESULT OF AURICULAR ELECTRICAL STIMULATION

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Tests which objectivize analgesia arising as a result of reflex therapy play an important role in the study of mechanisms of pain and endogenous analgesia. Usually evoked brain potentials, reflex limb withdrawal, and behavioral responses are used as parameters. Recording muscular reflexes is often used by investigators as one of the simplest methods of assessment. One such reflex in the case of stimulation of structures in the mouth is the jaw opening reflex (JOR). Some investigators [9, 11], consider that it is the motor manifestation of the nociceptive response. However, the study of the effect of electroacupuncture on JOR has given contradictory results [9, 15]. In our opinion this is due to differences in the level of anesthesia of the animals.

The aim of this investigation was to study the effect of auricular electrical stimulation (AES) on JOR in unrestrained animals and its correlation with changes in response of the animals to pain.

EXPERIMENTAL METHOD

Experiments were carried out on 10 adult unrestrained cats. As a preliminary stage, electrodes for stimulating the lip were inserted under pentabarbital anesthesia (40 mg/kg, intraperitoneally). The electrode socket was fixed to the skull. The animals were tested 1 week after the operation in an experimental chamber. Electrical stimulation of the lip was carried out with a series of three pulses, each 1 msec in duration, with an interval of 100 msec between them, and with an intensity of 2 to 100 V. To assess behavioral responses a modified scale of reaction levels, worked out for rats [1, 2], was used. The electromyogram (EMG) of the digastric muscle was recorded during stimulation of the lip (10 V, 0.3 msec). EMG responses were averaged on a specialized computer for 10 presentations, AES was carried out through electrodes applied to the base of the concha auriculae, with a frequency of 1 Hz and pulse duration of 1.2 msec, with intensity sufficient to cause twitching of the surrounding tissues [1-1.5 mA], for 15 min. Retesting began 15 min after the end of AES.

EXPERIMENTAL RESULTS

In response to stimulation of the lip of increasing intensity four levels of the animal's response could be distinguished. Level I was expressed as twitching of the whiskers, eyelids, and ears, characterizing the threshold of sensory perception of stimulation. The threshold of the JOR was the threshold of level II of response. Level III was thrusting forward the head, and movements of the limbs, which can be regarded as the nociceptive threshold of stimulation. Emotionally affective manifestations of pain constitute level IV of response.

As a result of AES facilitation of JOR was observed during stimulation of the lip of the unrestrained animals. This was clearly manifested as an increase in amplitude of the EMG of the digastric muscle (Fig. 1). Lowering of the threshold of JOR (level II) by 20% also was observed. An increase in the sensitivity to non-nociceptive stimuli took place against the background of depression of nociceptive sensitivity. Depression of the emotional-affective component of the nociceptive response was particularly marked; this constituted more than 30% of the initial level (Fig. 2). Defensive movements of the paws became more lethargic and of shorter duration, and vocalization was less frequently observed.

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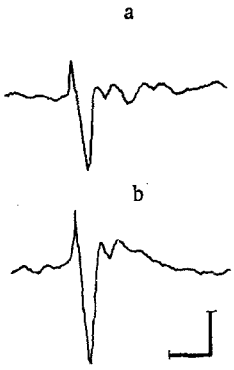

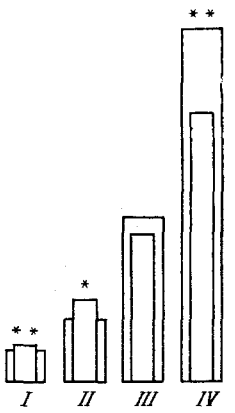

Fig. 1 Fig. 2

Fig. 1. Effect of AES on EMG of digastric muscle during stimulation of the lip. a) Before AES; b) 15 min after AES. Calibration: 100 μV. 100 msec.

Fig. 2. Effect of AES on profile of nociceptive sensitivity. Columns denote thresholds of levels I-IV of response during stimulation of the lip (narrow — before AES, wide — after AES). *$p < 0.05$; **$p < 0.01$.

JOR in response to low-intensity stimulation was unaccompanied by typical manifestation of pain. They appeared only when the threshold of this reflex was exceeded by 2-3 times. The possibility of evoking JOR by non-nociceptive stimulation also has been demonstrated by other workers in experiments on animals and in investigations on man [12, 14, 15]. This is evidence that JOR cannot serve as a criterion for the assessment of pain. It can be postulated that it performs the function of protection of structures of the oral cavity against possible damage. This is confirmed by data showing that the oral trigeminal nucles, which is closely connected with the superior colliculus [13], participates in the mechanism of JOR [6, 14]. The system of connections of the superior colliculus enables a combination of orienting reactions, performing the function of alertness, to take place. Meanwhile facilitation of JOR after AES in unrestrained animals is evidence of strengthening of the protective reflex response of the animal. An increase in amplitude of the EMG of the digastric muscle during AES was observed previously in acute experiments on unanesthetized animals during stimulation of the pulp [8]. Data on the inhibitory effect of AES on JOR [15] can be explained by the fact that the experiments were done on anesthetized animals. The use of anesthesia not only made AES less effective [8]. Depending on its level, the effect of conditioning stimulation on this reflex could be reversed [10]. The results confirm the view expressed by the writers previously that facilitation of JOR may be connected with intensification of the conduction of non-nociceptive impulses through the oral trigeminal nucleus after AES [3]. This is also in agreement with the lowering of the threshold of level I of response, which we observed. It must also be pointed out that facilitation of condition of impulses during stimulation of the lip has been observed in the superior colliculus [5]. Taken as a whole, these data may be evidence of facilitation of sensomotor integration during motor defensive responses after AES.

Depression of the emotional-affective component of pain observed in these experiments as a result of AES correlates closely with inhibition of evoked nociceptive activity in structures of the nonspecific system of the brain: the ventromedial region of the caudal trigeminal nucleus [3], the parafascicular complex of the thalamus [7], and the orbito-frontal cortex [9]. Activity of the nonspecific system is linked with conduction of the protopathic component of the nociceptive signal, responsible for emotional-affective behavior in animals [1, 4]. Our results are in agreement with data obtained by other workers, who have studied the effect of electroacupuncture on the emotional-behavioral component of the nociceptive response [2].

As a result of AES, activation of the various defensive systems of the body thus takes place. One of them is the antinociceptive system, causing depression of nociceptive sensi-

tivity. Another is the system of reflex, motor, orienting reactions, enabling the quickest avoidance of a dangerous stimulus. Interaction between these systems is very worthwhile in the combined response of the animal to pain.

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LOW BLOOD HEPARIN LEVEL REDUCES SENSITIVITY TO THE HYPOGLYCEMIC ACTION

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Heparin is known to play an important role in physiological responses aimed at maintaining glucose homeostasis. For instance, if all the circulating reactive heparin in healthy rats is bound with protamine sulfate, a state of total resistance to the hypoglycemic action of both exogenous and endogenous insulin arises [7, 8]. Meanwhile, administration of heparin protects against the diabetogenic action of alloxan and reduces hyperglycemia in animals in the early stages of alloxan diabetes, and promotes restoration of μ-cell function [4, 6]. Insulin, in the form of a complex with heparin, has a stronger hypoglycemic action [5], and the diabetogenic factor loses its ability to induce hyperglycemia [9].

In the light of these data it is interesting to study the hypoglycemic effect of exogenous insulin in animals with a low blood heparin level. In this investigation animals with alloxan diabetes, whose blood heparin concentration is significantly below normal [10], and aging animals with reduced anticoagulating potential of the blood due to depression of the function of the anticlotting system and to atherosclerosis, aggravated by keeping the animal for a long time on an atherogenic diet [3], were used as the model.

EXPERIMENTAL METHOD

In the experiments of series I noninbred male rats weighing 160-200 g, kept on the ordinary laboratory diet, were used. Diabetes was induced by intravenous injection of alloxan (Spofa, Czechoslovakia) in a dose of 40 mg/kg after starvation for 24 h. Animals

*Deceased.

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