By using the same techniques as in the present investigation, in a study of the parietal cortex [2] we obtained similar data on the arrival of specific heterogeneous volleys at different afferent inputs. It was shown under these circumstances that impulses of this kind determine monovalent responses of nerve cells in neuronal complexes, by contrast with heterogeneous nonspecific impulses that are addressed to neuron populations composed of both kinds of cells, but responding to these impulses like multivalent nerve cells. We also found projections of specific volleys of each modality to separate neuronal complexes in other polysensory formations both of the cortex and of the thalamus [1, 3]. Thus, for polysensory structures located at different levels of the CNS, including for NDB, arrival of specific heterogeneous afferentation at different neuron populations is a general rule and is an important condition for the reception and processing of information in this brain structures.

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ROLE OF THE SOMATOSENSORY CORTEX IN THE DEVELOPMENT OF REFLEX ANALGESIA

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KEY WORDS: electroacupuncture; somatosensory cortex; nociception.

Much attention is currently being paid to the study of the neurochemical and neurophysiological mechanisms of reflex analgesia [3-9]. The writers showed previously [7, 8] that electroacupuncture (EAP) leads to functional changes in the afferent systems of the brain and blocks conduction of nociceptive impulses. The important role of the second somatosensory area of the cortex in this process also has been demonstrated in acute experiments [4, 5, 9]. However, for further elucidation of the role of the somatosensory cortex and, in particular, of its second area, in the mechanisms of reflex analgesia, investigations on freely behaving animals are necessary.

In the investigation described below the characteristics of development of reflex analgesia in animals during free behavior after removal of the first (SI) and second (SII) somatosensory areas of the cortex were studied.

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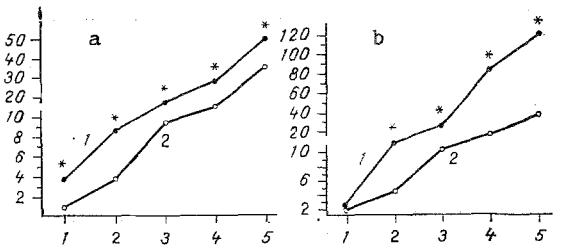

Fig. 1. Changes in profile of nociceptive response after EAP (a) and after injection of morphine (b). Abscissa, level of nocicpetive response; ordinate, strength of stimulation (in thresholds). 1) profile of nociceptive response in experiment, 2) profile of nociceptive response in control. *P < 0.05.

METHODS

Experiments were carried out on 19 unrestrained cats in an experimental chamber. Nociceptive electrical stimulation was applied through bipolar electrodes sutured to the dorsal part of the forearm of both forelimbs by bursts of pulses (following frequency of pulses in the burst 5/sec, pulse duration 1 msec, duration of burst 1 sec). The strength of stimulation was increased gradually from 100 mV to 30 V. EAP was applied in the region of cociceptive stimulation of the limbs through implanted needles. The frequency of EAP was 1-3 pulses/sec and the strength of stimulation under these circulstances was limited by the appearance of local muscular contractions; the duration of stimulation was 30 min. To compare analgesia observed after EAR, morphine was injected subcutaneously in a dose of 2.5-3 mg/kg. Somatosensory areas of one cerebral hemisphere were removed by electrical coagulation under hexobarbital anesthesia (30 mg/kg, intraperitoneally). To remove area SI the posterior sigmoid and coronal gyri were removed (6 animals), to remove SII the anterior ectosylvian gyrus was removed (6 animals), and both SI and SII were removed together in 4 animals. As the control, part of the lateral and suprasylvian gyri of the parietal association cortex, equal in area to SI, was destroyed in 3 cats. The animals began to be tested 8 days after the operation to remove the cortical areas. The results were assessed on a conventional scale, as used on experimental aniamsl in [2, 3] and adapted by ourselves for recording the response of unrestrained cats to pain: at level 1 of response, the threshold response, twitching of the eyelids, flexion of the stimulated limb; at level 2, flexion of the stimulated limb, shaking of the head, closing the eyes tightly, pressing the ears against the head; at level 3, shaking of the whole body, licking, changes in posture, single locomotions, vocalization; at level 4, intensive and frequent crying, constant moving about, restlessness; at level 5, generalized movements, running with a cry, aggressiveness. The significance of the results was estimated by Student's test. At the end of the investigation the locations of the lesions were verified morphologically.

RESULTS

During EAP with intensity sufficient to evoke local muscular contractions in the region of stimulation, no signs of unease were observed in the animals. Testing the animals after EAP revealed significant elevation of thresholds at all levels of response (Fig. 1a). The duration of the analgesic effect after EAP varied from 1.5 to 3 h. The analgesia observed after EAP developed only on the side of stimulation. This unilateral change in the profile of pain sensitivity after EAP is evidence of selective activation of the endogeneous antinociceptive system, exerting control over ascending nociceptive information, the somatotopic organization of which was demonstrated previously [11].

To compare the analgesic effect of EAP the classical narcotic analgesic, morphine, was used. Injection of morphine into the animals caused a change in the profile of pain sensitivity. Unlike EAP, however, elevation of the thresholds after morphine took place bilaterally. The analgestic effect appearing after morphine was most marked for stimuli evoking severe pain with clear emotional-affective manifestigations, and the primary sensory threshold of the response was virtually unchanged (Fig. 1b).

The results are in good agreement with data in the literature according to which analgesia after administration of narcotic analgesics is due largely to depression of the emotional-affective response to pain on account of inhibition predominantly of nociceptive impulses in the nonspecific ascending system responsible for formation of the emotional-affective component of the nociceptive response [1, 2].

Subsequent testing of the animals after cortical ablation revealed that unilateral removal of both SI and SII led to ineffectiveness of EAP on the contralateral side only and did not affect the development of analgesia following EAP given on the side of the cortical ablation.

Control destruction of part of the lateral and suprasylvian gyri of the parietal association cortex did not change the profile of nociceptive sensitivity in cats and did not change the effectiveness of subsequent EAP.

According to data obtained by other workers [12-14], in order to obtain the maximal analgesic effect after EAP, the greatest possible strength of current must be used without, however, causing unpleasant sensations. In order to rule out the absence of an analgesic effect of EAP after cortical ablation on account of an inadequate intensity of stimulation applied, the strength of the current was therefore increased during application of EAP.

After removal of area SII, incidentally, there was no analgesic effect of such EAP, whereas an increase in the intensity of EAP after removal of area SI led in some cases to a brief (up to 20 min) elevation of the thresholds of nociceptive sensitivity. A similar increase in the intensity of EAP after simultaneous destruction of SI and SII had no analgesic effect.

The absence of change in the profile of nociceptive sensitivity after EAP and removal of areas SI and SII is evidence of the important role of these structures in the mechanism of reflex analgesia. However, considering that analgesia can be obtained by increasing the intensity of EAP, applied after removal of area SI, and the ineffectiveness of EAP in the case of simultaneous ablation of areas SI and SII, or removal of SII only, it can be tentatively suggested that area SII plays a definite role in the development of analgesia of reflex nature. This conclusion is in good agreement with results obtained by the writers previously in acute experiments, which showed that area SII exerts marked corticofugal influences on the central gray matter, one of the principal antinociceptive structures [8]. The modulating influence of area SII on depression of nociceptive responses in the thalamic parafascicular complex [9] and the orbitofrontal cortex [5] during EAP also was demonstrated. The conclusion drawn from the present investigation, namely that area SII is one of the leading cortical structures determining the development of the analgesic effect during reflex stimulation, is also in good agreement with the results of investigations [10] which showed, in chronic experiments, that direct electrical stimulation of area SII potentiates the analgesic effect of EAP.

The results are thus evidence that the somatosensory cortex plays an important role in reflex analgesia. Area SII, which modulates the endogenous antinociceptive system, is particularly important in this respect.

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EFFECT OF PHOSPHATIDYLCHOLINE ON BODY TEMPERATURE AND POSTERIOR HYPOTHALAMIC UNIT ACTIVITY IN ANIMALS

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KEY WORDS: phosphatidylcholine; body temperature; hypothalamic neurons.

The effect of synthetic preparations and biologicals on body temperature and on its regulation in various animals and man has now been studied on a sufficiently wide scale [1, 3]. The participation of many neurotransmitters, neurohormones, and other substances in central mechanisms of temperature regulation has been established [1, 7]. Information has recently been obtained that certain exogenous phospholipids can induce changes in metabolism of certain neurotransmitters [5, 9]. However, the effect of phospholipids, as the most important structural components of biological membranes, on the temperature regulating apparatus has not been studied. In particular, we have no information on the effect of liposomes, composed of phospholipids, when introduced into the body on its temperature and on neuronal activity in temperature-regulating centers.

The aim of this investigation was to study the effect of phosphatidylcholine (PCh) liposomes on body temperature and on unit activity in the posterior hypothalamus, which performs integrative functions in central mechanisms of temperature regulation.

METHODS

The effect of PCh on body temperature was studied in 36 albino rats weighing 160-180 g and in 16 rabbits weighing 2.5-3.2 kg, under thermoneutral conditions (20-24°C). The animals' body temperature was measured in the rectum (at a depth of 3 cm in rats and 6 cm in rabbits) by means of a TPÉM-1 electrothermometer. The effect of PCh on units activity in the posterior hypothalamus (coordinates $P_1L_1H_{13-15}$ according to Sawyer's atlas) was investigated in 8 rabbits anesthetized with urethane (1.5 g/kg, intraperitoneally). The method of extracellular recording of spontaneous unit activity and of its analysis was described previously [2]. Unit activity was studied during heating of the body of an animal previously cooled (to 36°C) in air. Heating was carried out in a special thermally insulated chamber by means of a current of heated (40-42°C) atmospheric air from an EK-3 electrocalorifier. The brain temperature (mesencephalic region) was measured continuously by a miniature thermistor (diameter 0.8 mm) and readings were recorded on a V7-21A Universal Measuring Instrument. PCh liposomes were made as follows. An alcoholic solution of PCh was evaporated to dryness *in vacuo* in a current of nitrogen at 30°C. The cooled suspension of PCh in distilled water (0.5 g to 100 ml) was sonicated on a UZDN-1 apparatus for 10 min (frequency 22 kHz, current 0.2 A).

Standard egg PCh, carbachol, and L-noradrenalin bitartrate monohydrate (Calbiochem, USA) were used in the experiments. Aqueous solutions of PCh and also liposomes were injected into the lateral ventricles: in the experiments on rats in a volume of 20 µl under

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