

REPRESENTATION OF THE PHRENIC NERVE IN THE CEREBRAL CORTEX

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Stimulation of the phrenic nerve evokes early (latent period 8-12 msec) and late (latent period 30-40 msec) surface-positive potentials in the contralateral sensomotor cortex. The early potentials arise in two localized areas of the posterior sigmoid gyrus - near the medial edge of the posteruciate dimple and near the lateral edge of the cruciate sulcus. The late potentials, on the other hand, are recorded not only over the whole surface of the posterior sigmoid gyrus, but also in the anterior sigmoid gyrus. The early component of the response is evidently evoked by stimulation of the first group of muscle afferents. Impulses reaching the cortex from the phrenic nerve may perhaps participate in the mechanism of the sensation of breathlessness.

KEY WORDS: phrenic nerve; primary responses; cortical representation of first group of muscle afferents; dyspnea.

The representation of all the large somatic and visceral nerves in the cerebral cortex has been described [1, 2, 12, 16, 17]. The only exception is evidently the phrenic nerve, the cortical representation of which is not yet known. However, this is a matter of great interest in connection with analysis of the role of impulses spreading along the phrenic nerve in the mechanism of the sensation of breathlessness.

The investigation described below showed that the phrenic nerve is represented in the first somatosensory area of the cortex at two points, coinciding roughly with the representation of the first group of muscle afferents of the forelimb.

EXPERIMENTAL METHOD

Experiments were carried out on 21 cats weighing 3.2-3.9 kg anesthetized with α -chloralose (60-70 mg/kg, intraperitoneally). Cortical evoked potentials were recorded from the right hemisphere by a monopolar technique with silver ball electrodes, lightly pressed against the surface of the cortex by means of miniature spring clips. The reference electrode (a steel needle) was fixed in the nasal bones. Cortical responses were evoked by stimulation of the left phrenic nerve, the left greater splanchnic nerve, and the left forelimb. The forelimb was stimulated by bipolar needle electrodes inserted beneath the skin of the dorsal surface of the foot or into the central footpad. To stimulate the phrenic and greater splanchnic nerves the animals were artificially ventilated, the 5 or 6 last ribs were removed on the left side, and the central ends of the nerves divided near the diaphragm were placed on bipolar platinum electrodes. The thorax was then covered with vinyl chloride film to prevent the nerves from drying. Cortical responses were recorded in animals immobilized with tubocurarine.

EXPERIMENTAL RESULTS

Stimulation of the phrenic nerve evoked responses in the first somatosensory area of the cortex of the contralateral hemisphere which began with a surface-positive wave of potentials (Figs. 1A, B, E and 3A). The earliest component of the response had a latent period of 8-12 msec, a duration of 18-28 msec, and an amplitude of 30-90 μ V. This was followed as a rule by a second positive wave, which merged smoothly with a longer negative wave. The latent period of the second positive wave was 30-40 msec and its amplitude 30-120 μ V. The latent period, shape, and amplitude of the negative wave were much more variable than those of the positive components of the response.

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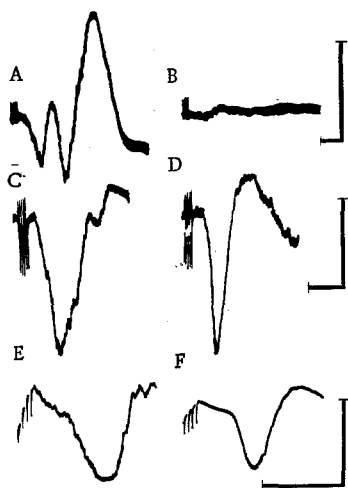


Fig. 1

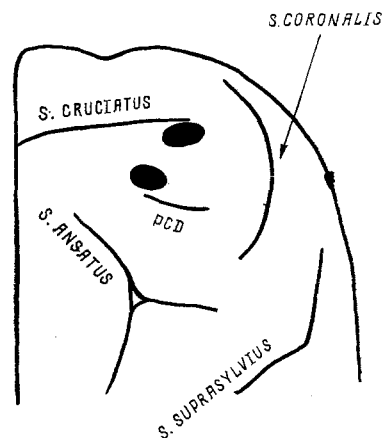


Fig. 2

Fig. 1. Responses of posterior sigmoid gyrus to stimulation of contralateral splenic nerve (A, C, E), contralateral greater splanchnic nerve (D), and contralateral forelimb (F). Potentials recorded near medial border of postcruciate dimple (A-D) and near lateral border of cruciate sulcus (E, F) in three cats: A-B, C-D, and E-F. After crushing of the phrenic nerve proximally to the point of stimulation responses to stimulation of this nerve disappeared (B). Voltage calibration: 50 μ V (for C, D), 100 μ V (for A, B, E), and 400 μ V (for F). Time calibration 20 msec.

Fig. 2. Scheme of dorsal surface of rostral part of cat cortex. Areas of cortex in which the early component of response to stimulation of contralateral phrenic nerve was recorded are shown in black. PCD) Postcruciate dimple.

Crushing the phrenic nerve above the point of stimulation completely abolished this response (Fig. 1B). Consequently, it was caused by afferent impulses spreading along the phrenic nerve.

The latent period of the early component of the response coincided with the latent periods of classical cortical primary responses to stimulation of the contralateral forelimb and the contralateral greater splanchnic nerve. This fact is illustrated in Fig. 1, C-F. Responses recorded at two points of the posterior sigmoid gyrus are illustrated here: Near the medial edge of the postcruciate dimple (C, D) and near the lateral edge of the cruciate sulcus (E, F). Responses near the postcruciate dimple were evoked by stimulation of the contralateral phrenic nerve (C) and the contralateral greater splanchnic nerve (D). Responses near the lateral edge of the cruciate sulcus appeared to stimulation of the contralateral greater splanchnic nerve (E) and the contralateral forelimb (F). The latent periods of all three responses evidently almost coincided. They were 9.7 msec (C), 9.3 msec (D), 12 msec (E), and 11.5 msec (F). The latent periods of the responses to stimulation of the splanchnic nerve (D) and forelimb (F) agreed with those described in the literature [3, 6, 11, 12].

Like known cortical primary responses, the early component of the response to phrenic nerve stimulation was recorded in very localized areas of the cortex. These areas are shown in Fig. 2 as black ovals. They are located in two regions of the posterior sigmoid gyrus - near the medial edge of the postcruciate dimple and near the lateral edge of the cruciate sulcus. Moving the recording electrode only 2-3 mm away from these areas led to disappearance of the early component of the response from the record. The late waves were recorded from wider areas of the cortex: They could be recorded not only in all parts of the posterior sigmoid gyrus, but even in the anterior sigmoid gyrus.

An important difference between the early component of the response described here and primary responses evoked by stimulation of the forelimb and greater splanchnic nerve is that the early component was strongly dependent on temporal summation. As Fig. 3 shows, a well-marked cortical response occurred only to application of a high-frequency volley of pulses to the phrenic nerve (A), and a single stimulus evoked no response (B). Conversely, temporal summation was not required for the primary responses evoked from the forelimb or the greater splanchnic nerve: If the stimulating current was strong enough maximal primary responses appeared not only to a high-frequency volley of stimuli (C), but also to a single stimulus (D).

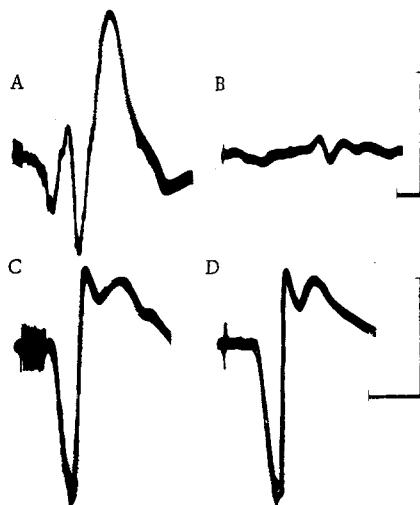


Fig. 3. Dependence of responses evoked in cortex by phrenic nerve stimulation on temporal summation. Responses near medial edge of postcruciate dimple (A, B) and near lateral edge of cruciate sulcus (C, D) evoked by stimulation of contralateral phrenic nerve (A, B) and contralateral forelimb (C, D). Response to stimulation of phrenic nerve arise to high-frequency series of stimuli (A) only and not to single stimulus (B). Conversely, during stimulation of forelimb responses are independent of whether they are evoked by high-frequency series of stimuli (C) or single stimulus (D). Voltage calibration: 100 μ V (A, B) and 500 μ V (C, D). Time calibration 20 msec.

The early positive waves of potentials described in this paper must evidently be a primary response. This is shown by the latent period of this wave, which was the same as that of the primary cortical response to stimulation of the forelimb and of the greater splanchnic nerve, and the very small size of the area of cortex in which this wave was recorded. This primary response was evoked by impulses which evidently spread along the first group of muscle afferents of the phrenic nerve. This is shown not only by the short latent period, but more especially by the localization of the response; it coincided almost exactly with the representation of the first group of muscle afferents of other nerves participating, like the phrenic nerve, in the brachial plexus [14-16]. Further support is given by two other features distinguishing this primary response: Its extremely low amplitude and its strong dependence on temporal summation. These features are known to be characteristic of primary responses evoked by stimulation of a very small number of primary afferents, literally only two or three fibers [4, 9, 10]. Histological investigations [8] have shown that the phrenic nerve in the cat contains just this small number of fibers belonging to the first group of muscle afferents.

The later positive-negative wave of potential with a latent period of 30-40 msec is a nonprimary response. The latent period, shape, and localization of this wave are characteristic of evoked potentials known as nonprimary responses of the second type [5].

The view was originally widely held that discomfort due to breathlessness is caused by impulses from the respiratory muscles of the chest [7]. However, it was later found that dyspnea can still develop when these impulses are blocked by spinal anesthesia and respiration is maintained by the activity of the diaphragm alone [13]. The data described in this paper suggests that in this case the dyspnea may be caused by impulses spreading along the phrenic nerve.

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AGE DIFFERENCES IN TEMPERATURE DEPENDENCE OF REPOLARIZATION OF THE ADRENOCORTICAL CELL MEMBRANE

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In experiments on isolated adrenals of male rats of two age groups (5 and 28-29 months) no age differences were found in the membrane potential of cells in the zona fasciculata of the cortex. The temperature dependence of repolarization of the cell membrane in the zona fasciculata of the adrenal cortex was investigated in rats of different ages within the temperature range from 7 to 17°C after preliminary cooling of the adrenals. The temperature coefficient of repolarization, calculated in old animals ($Q_{10} = 2.732$) was significantly higher than in the young animals ($Q_{10} = 1.481$). With age, the contribution of reactions with high activation energy increases in total balance of processes determining repolarization of the adrenocortical cell membrane.

KEY WORDS: Aging of the endocrine system; adrenal cortex; membrane potential; temperature dependence.

An important place in the study of the mechanisms of age changes in the functions of cells is occupied by the study of their biophysical properties, which are mainly determined by the state of the membrane: its polarization, excitability, and transport function. The level of polarization of the cell membranes plays an important role in the control of cell metabolism also [1, 5, 14].

Much factual evidence has now been gathered on age changes in the glands of internal secretion [2, 4, 6, 7], yet there is no information on the electrical properties of their cell membranes: the membrane potential (MP), details of active transport, and the supply of energy for it.

MP of the cells of certain organs and tissues (the liver, various structures of the nervous system, muscles, some epithelial cells) preserves its relative constancy during aging, despite significant changes in the concentrations of electrolytes in the tissues, and a decrease in the intensity of formation and in the concentration of high-energy phosphorus compounds, which provide for the work of the sodium pump [3, 8, 9, 15]. It can therefore be postulated that during aging changes arise in the mechanism of maintenance of MP of the cells.

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