TRANSMISSION OF NOCICEPTIVE INFORMATION ALONG THE SPINOCERVICOTHALAMIC COMPONENT OF THE LEMNISCAL SYSTEM

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All information from somatic receptors is transmitted to the cerebral hemispheres along two main afferent systems — lemniscal and extralemniscal. The lemniscal system of the cat is represented at the spinal cord level by the dorsal columns and spinocervical tracts (SCT). The remaining spinal pathways projecting to the cerebral hemispheres belong to the extralemniscal system [1, 2, 10]. In the generally accepted view, nociceptive information reaches the brain only via the extralemniscal pathways; the lemniscal system, on the other hand, serves for the transmission of signals from low-threshold mechanoreceptors [2, 10]. In accordance with this view only those neurons of SCT which are excited purely by tactile stimulation of the skin belong to the lemniscal system. The numerous neurons of this tract which are excited by nociceptive stimulation, however, are considered to belong to the extralemniscal system [2]. However, there is evidence that not only the purely "tactile" neurons, but those other neurons of SCT which are excited by nociceptive stimuli also should be included among the lemniscal system [1].

In this paper the writer gives new and more direct evidence in support of this suggestion, evidence of fundamental importance for the understanding of the mechanisms of pain localization.

EXPERIMENTAL METHOD

Twelve cats weighing 2.9-4.2 kg were anesthetized with α-chloralose (50-70 mg/kg, intraperitoneally) and immobilized with tubocurarine and artificially ventilated. Primary responses (PR) were recorded by monopolar silver ball electrodes, pressed lightly by miniature springs against the surface of the posterior sigmoid gyrus of the right hemisphere in the area of representation of the hind limb. The reference electrode (a steel needle) was secured in the nasal bones. Testing stimuli were applied to the superficial peroneal nerve of the left hind limb, to neurons of SCT in the left dorsal horn of the spinal cord at level L6-L7, and to the axons of these neurons in the left dorsolateral funiculus at the level L2-L3. Bipolar stimulation (with platinum electrodes) was applied to the nerve, monopolar stimulation (by a steel needle electrode) to the spinal cord. The needle electrodes were insulated with bakelite varnish or viniflex throughout their length except at the tip, the diameter of which was about 20 μ. The reference electrode, connected to the anode of the stimulator, was secured in the spinal muscles. To prevent exposed areas of the nerve, spinal cord, and cerebral cortex from drying, they were flooded with warmed mineral oil. Heating the whole of the dorsal surface of the left foot by means of a contact thermode was used as nociceptive stimulation. The temperature on the skin surface reached a maximum (44-60°C) in the course of 15-20 sec. Heating in this way, if continued as in these experiments for minutes, excites only polymodal nociceptors with unmyelinated afferent fibers [1, 7]. The remaining receptors were completely inactivated at this temperature after only a few seconds of heating.

EXPERIMENTAL RESULTS

The great majority of SCT neurons lie in the dorsal horn of the spinal cord [4, 6]. In response to stimulation of the ipsilateral superficial peroneal nerve these cells, together

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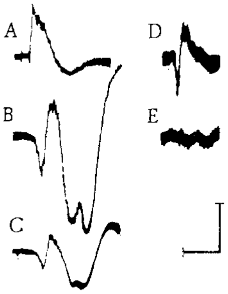

Fig. 1

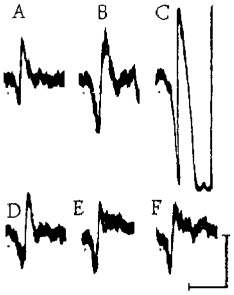

Fig. 2

Fig. 1. Electrophysiological identification of SCT neurons. A) N wave in dorsal horn L7 evoked by stimulation of ipsilateral superficial peroneal nerve (tip of recording electrode at depth of 1.7 mm from dorsal cord surface and 0.9 mm from midline); B, C) primary and secondary responses in medial part of posterior sigmoid gyrus evoked by strong (2 and 3 times threshold respectively) stimulation of contralateral dorsal horn (B) and contralateral superficial peroneal nerve (C). Dorsal horn stimulated at point at which N wave shown in trace A was recorded; D) primary response in medial part of posterior sigmoid gyrus to threshold stimulation of contralateral dorsal horn L7 (tip of stimulating electrode at depth of 1.2 mm from dorsal cord surface and 0.9 mm from midline); E) after moving stimulating electrode in dorsal direction through 300 μ PR disappeared. Calibration: A, B, C) 20 msec, D, E) 40 msec; A, C) 300 μV, B) 90 μV, D, E) 30 μV.

Fig. 2. Facilitatory action of nociceptive skin heating on PR evoked in cortex by direct stimulation of SCT neurons. PR in medial part of posterior sigmoid gyrus of right hemisphere evoked by threshold stimulation of SCT neurons (A-C) and of dorsolateral funiculus (D-F) on opposite side of body before (A, D), and 30-40 sec after (B, E) and 3-4 min after (C, F) beginning of continuous heating (to 49°C) of dorsal surface of left foot. Calibration: 40 msec and 30 μV.

with other neurons located in the same place, generate the negative component (N wave) of the dorsal surface potential of the spinal cord [9]. In order to locate the tip of the stimulating electrode as near as possible to these cells, the point on the surface of the spinal cord at which the N wave was particularly well marked was identified. Next, the needle electrode was inserted from this point parallel to the sagittal plane to the depth at which the N wave reached its maximal amplitude (Fig. 1A). In this position the tip of the microelectrode lay at the level of layers IV-V of the dorsal horn in the immediate neighborhood of SCT neurons [6, 9]. In the present experiments the microelectrode tip occupied this position when it was at level L6-L7, at a distance of 0.8-1.2 mm from the midline, and at a depth of 1-1.7 mm from the dorsal surface of the spinal cord, in good agreement with data in the literature on the location of SCT cells [5, 6, 8, 11].

Single stimulation of the dorsal horn at this point evoked a positive-negative response with a latent period of 8-9 msec on the surface of the posterior sigmoid gyrus of the contralateral hemisphere in the representation of the hind limb (Fig. 1B). This response was indistinguishable in every respect other than latent period from the classical PR to stimulation of the superficial peroneal nerve (Fig. 1C). The latent period of response of the cortex to stimulation of the nerve was 10-12 msec, i.e., it was longer by the time taken up in conduction from the nerve to the SCT neurons [9].

In the cat spinal cord there are two pathways stimulation of which can evoke cortical PR. One is SCT, the other consists of primary afferents ascending in the dorsal column to the nuclei of the dorsal columns in the medulla [1, 3]. If the PR recorded in the present experiments had been evoked by stimulation of the dorsal column, moving the stimulating electrode from the dorsal horn in the dorsal direction ought to have been accompanied by an increase in PR. In fact, movement of the electrode in that direction by only 200-300 μ was sufficient to cause a sharp reduction or even total disappearance of PR (Fig. 1D, E). Consequently, they were evoked by stimulation of SCT neurons and not of the dorsal column.

Nociceptive heating of the dorsal surface of the foot evoked very strong facilitation of PR to testing stimulation of SCT neurons receiving information from the heated area of the skin. During the first 30-40 sec after switching on the thermode the amplitude of PR evoked by threshold stimulation of the dorsal horn increased by about 15-30% (Fig. 2B). The amplitude of the PR increased by 50-80% 1-2 min after switching on the thermode. Facilitation reached a maximum after continuous nociceptive stimulation for 3-4 min, when the amplitude of PR was increased two-fourfold (Fig. 2C). After the thermode had cooled down the facilitation remained, but it gradually weakened during the next 5-8 min. Conversely, PR to threshold stimulation of the dorsolateral funiculus, with the fibers of SCT contained therein, remained unchanged during nociceptive stimulation (Fig. 2D-F).

The facilitatory action of nociceptive stimulation on PR evoked by direct stimulation of SCT neurons and the absence of such action on PR arising in response to stimulation of the axons of these neurons in the dorsolateral funiculus is evidence that this facilitation was due entirely to increased excitability of SCT neurons. Since nociceptive stimulation does not affect the purely "tactile" neurons of this tract [4], the facilitation observed must be the result of an increase in excitability of SCT neurons activated by nociceptive stimuli. This means that cortical PR are evoked by impulses generated not only by tactile neurons, but also by the nociceptive neurons of SCT. It is generally accepted that PR, at least their early components, are evoked by impulses reaching the cortex along the lemniscal system. That is why the participation of the nociceptive neurons of SCT in provocation of PR is direct evidence that the lemniscal system transmits information not only from low-threshold mechanoreceptors, as is generally considered, but also from nociceptors to the cerebral cortex. If it is recalled that the lemniscal system, by contrast with the extralemniscal, is somatotopically organized to an extremely fine degree [1, 2, 10], this fact will explain the animal's ability to determine the precise location of a painful stimulus.

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