FACILITATION OF THE SPINO-CERVICAL TRACT UNDER THE INFLUENCE OF IMPULSES FROM A FOCUS OF INFLAMMATION

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UDC 616-002-07:616.832.22-072.7

Earlier investigations in the authors' laboratory [1-4, 8] showed that impulses from a focus of inflammation in a limb facilitates the specific projection pathway along which information from the injured limb is transmitted to the cerebral cortex. The facilitation was shown by the fact that, after development of a focus of inflammation in the limb, the primary responses in somatosensory areas I and II in the cortex and in the ventral posterolateral nucleus (VPL) of the thalamus and the responses in the medial lemniscus, i.e., along the whole cerebral part of the specific projection pathway of the injured limb, appeared to less strong stimulation of the nerve of the affected limb than before the injury.

It was soon discovered [2] that this facilitation is observed only if the responses are evoked by stimulation of the nerve of the injured limb. If similar responses are evoked by direct stimulation of the medial lemniscus or VPL, when the animal is deeply anesthetized and the ascending activating system of the brain stem is blocked, no facilitation of the responses is observed under the influence of impulses from a focus of inflammation. Consequently, impulses from the focus of inflammation during deep anesthesia do not produce facilitation of neurons situated above the medial lemniscus, i.e., in the VPL and in somatosensory areas I and II of the cortex. It was therefore postulated that facilitation of the responses evoked by stimulation of the nerve of the injured limb is associated with an increase in the excitability of the neurons of the specific somatosensory projection pathway lying below the medial lemniscus.

In the present investigation the effect of impulses from a focus of inflammation in the limb was studied on the neurons of the first synaptic relay of the specific somatosensory projection pathway – the neurons of the spino-cervical tract [5, 6, 10-12].

EXPERIMENTAL METHOD

Experiments were carried out on 25 cats anesthetized with Nembutal (40 mg/kg, intraperitoneally). A focus of inflammation was produced by injection of 0.5-1 ml of turpentine beneath the skin of the dorsal surface of the foot of one of the hind limbs. The responses in the spino-cervical tract at the level of L1 were recorded by monopolar steel needle electrodes (diameter of point 10-50 μ), insulated except at the point by bakelite varnish. The responses were evoked by stimulation of the superficial peroneal nerves, supplying the part of the foot where the inflammatory process was situated.

In the dorso-medial part of the lateral lemniscus, the dorsal spino-cerebellar tract lies side by side with the spino-cervical tract. Both tracts respond to stimulation of afferents of the ipsilateral limb. They may therefore easily be confused. To identify the spino-cervical tract, the fact was used that this tract, unlike the dorsal spino-cerebellar tract, does not respond to stimulation of low-threshold muscle afferents of the same limb, for example, to stimulation of the gastrocnemius nerve [9].

EXPERIMENTAL RESULTS

Impulses from the focus of inflammation evoked primarily a decrease in the threshold of stimulation of the nerve of the injured limb to produce a response in the spino-cervical tract. It is clear from Fig. 1, A that before injury the weakest stimulation of the nerve to evoke responses in the spino-cervical tract was 350 mV. Two hours after injury to the right hind limb, the whole foot, as far as the ankle was highly edematous. At this time the threshold of stimulation of the nerve of the injured limb was appreciably

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Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 63, No. 3, pp. 38-41, March, 1967. Original article submitted July 21, 1965.

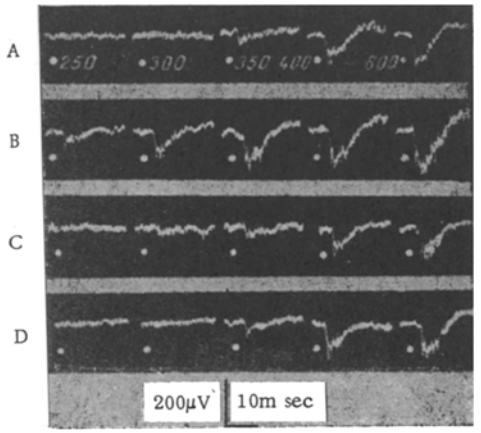

Fig. 1. Responses of the spino-cervical tract to stimulation of the ipsilateral superficial peroneal nerves before (A, C) and after (B, D) the development of a focus of inflammation in the right hind limb. A, B—responses to stimulation of the nerve of the right limb; C, D—responses to stimulation of the nerve of the left limb. The figures show that strength of stimulation of the nerve (in mV). The duration of the stimulus in all cases was 0.05 msec. Here and in Figs. 2 and 3 a positive deviation of the potential beneath the active electrode corresponds to a downward deflection of the beam.

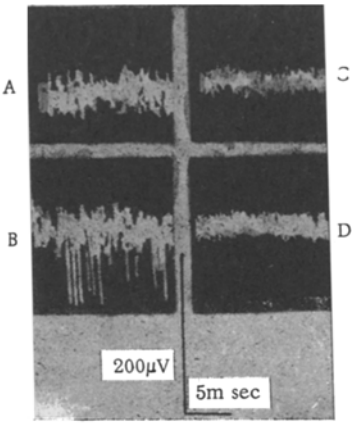

Fig. 2. Appearance of spontaneous discharges in single axons of the spino-cervical tract after injury to the ipsilateral limb (B); before injury (A), and also on the intact side before (C) and after (D) injury to the contralateral hind limb no spontaneous discharges are present.

lower—responses began to appear after stimulation of this nerve with a voltage of 250 mV (Fig. 1, B). A similar lowering of the threshold was observed in 22 of the 25 cats. A slight fall in the threshold (by as much as 20%) was sometimes noted during the first minutes after injury to the limb. From 2 to 5 h after injury, when an extensive focus of inflammation had developed in the limb, the threshold fell much more—usually by 30-40%. In four experiments it fell more still—by 50 or even by 60%.

At the same time the threshold of stimulation of the nerve of the intact limb usually remained unchanged (Fig. 1, C, D). In some experiments it actually was raised slightly.

Besides the lowering of the threshold, the impulses from the focus of inflammation in the limb also produced a marked increase in the amplitude of the response in the spino-cervical tract to maximal stimulation of the nerve of the injured limb for the low-threshold group of cutaneous afferents. It will be seen in Fig. 1 that after injury (B) the amplitude of the response evoked by such maximal stimulation of the nerve of the injured limb (600 mV) was greater than the amplitude of the response evoked by the same stimulus before injury (A). On the intact side no such changes in amplitude were ever observed (Fig. 1, C, D).

In a series of experiments comparatively thin microelectrodes were used (diameter of the points approximately 10 μ). By means of these microelectrodes discharges of individual axons of the spino-cervical tract could be recorded. These experiments showed that the neurons of the spino-cervical tract, which did not discharge spontaneously before injury (Fig. 2, A), began to discharge continuously after injury to the ipsilateral limb (Fig. 2, B).

Simultaneously with all these changes, the impulses from the focus of inflammation also led to an improvement in the reproduction of a high frequency of stimulation of the nerve of the injured limb by the spino-cervical tract. It can be seen in Fig. 3 that before injury (A) the spino-cervical tract reproduced a frequency of stimulation of the nerve of 50 cps badly, and hardly reproduced a frequency of 100 cps at all. After injury, when a focus of inflammation developed in the limb, this same tract began to reproduce clearly frequencies of stimulation of the nerve not only of 50 cps, but also of 100 cps (Fig. 3, B). In the spino-cervical tract on the uninjured side, no such improvement in reproduction of a high frequency of stimulation of the nerve took place.

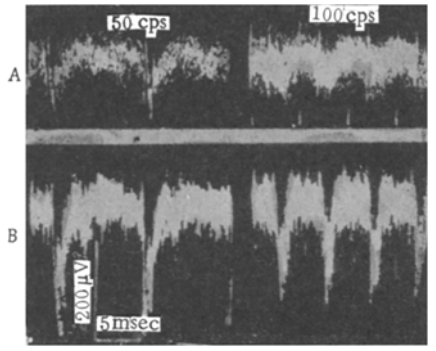

Fig. 3. Responses of the spino-cervical tract to maximal stimulation of the superficial peroneal nerve with frequencies of 50 and 100 cps before (A) and after (B) the development of a focus of excitation in the ipsilateral limb.

The facts described above show that impulses from a focus of inflammation cause depolarization of the postsynaptic membrane of the second-order neurons of the spino-cervical tract. As a result of this depolarization, the threshold of stimulation of these neurons is lowered. For this reason, they begin to respond to a weaker presynaptic volley of impulses, i.e., to weaker stimulation of the nerves of the injured limb. In addition, as a result of this depolarization, fresh neurons of the spino-cerival tract, not responding before injury even to maximal stimulation of the nerve, become involved in the zone of the discharge. The amplitude of the response is thus increased. The appearance of spontaneous discharges of individual neurons of the spino-cervical tract observed in these experiments after injury to the limb can be explained only by the depolarizing action of the impulses from the focus of inflammation.

Finally, this same depolarization may also account for the improvement in reproduction of a high frequency of stimulation of the nerve by the spino-cervical tract after injury to the limb. In fact, depolarization diminishes the after-hyperpolarization of the neurons of the spino-cervical tract described by Eccles and coworkers [7], i.e., it weakens the inhibitory mechanism lowering the frequency of discharges of the neurons. For this reason, after injury to the limb, the neurons of the spino-cervical tract become capable of reproducing a higher frequency of stimulation of the nerve of the injured limb.

All these facts provide an explanation of the facilitation of the specific somatosenory projection pathway of the injured limb, observed in earlier investigations [1-4, 8] by an increase in the excitability of the neurons of the first synaptic relay in the spino-cervical tract.

It is interesting that impulses from the focus of inflammation evoke appreciable changes in excitability of the neurons only of the first relay of the specific projection pathway of the injured limb. Meanwhile, despite the appearance of a continuous flow of impulses along the spino-cervical tract of the injured side, the level of excitability of the neurons of the higher structures in this same specific projection pathway (the VPL, somatosensory areas I and II of the cortex) shows no apparent change [2]. This fact is possibly associated with the bringing into play of inhibitory mechanisms, preventing an excessive increase in the excitability of the cortical and thalamic neurons [3, 4, 8]. As a result, these neurons remain capable of performing their higher integrative functions.

LITERATURE CITED

1. L. N. Smolin, In the book: Physiology and Pathology of the Nervous System [in Russian], Moscow (1964) p. 86.
2. L. N. Smolin, Pat. Fiziol., No. 5, 51 (1966).
3. L. N. Smolin, Abstracts of Proceedings of the 10th Congress of the I. P. Pavlov All-Union Physiological Society [in Russian], 2, No. 2, 279, Moscow–Leningrad (1964).
4. S. I. Frankshtein, Z. G. Biyasheva, and L. N. Smolin, Byull. éksp. Biol., No. 5, 27 (1965).
5. S. A. Andersson, Acta physiol. scand., 56, Suppl 194 (1962).
6. J. V. Catalano and G. Lamarche, Am. J. Physiol., 189, 141 (1957).
7. J. C. Eccles, R. M. Eccles, and A. Lundberg, J. Physiol. (London). 154, 89 (1960).
8. S. I. Frankstein, Z. G. Biyasheva, and L. M. Smolin, Nature, 205, 245 (1955).
9. A. Lundberg and O. Oscarsson, Acta physiol. scand., 51, 1 (1961).
10. R. F. Mark and J. Steiner, J. Physiol. (London), 142, 544 (1958).
11. F. Morin, Am. J. Physiol., 183, 294 (1965).
12. U. Norrsell and P. Voorhoeve., Acta physiol. scand., 54, 9 (1962).
13. B. Rexed and A. Brodal., J. Neurophysiol., 14, 399 (1951).