FUNCTIONAL ISOLATION AS A PRINCIPLE OF ACTIVITY OF THE NERVOUS SYSTEM (DESCENDING AND CONTRALATERAL INFLUENCES OF SPINAL MECHANISMS OF PROTECTIVE LIMB REFLEXES)

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In contrast to facilitation of flexor reflexes produced by impulses from an injured limb, the descending influence of the pyramidal tract on flexor reflexes of an injured limb is depressed. The influence of afferent components of the flexor reflex of the intact limb on flexor reflexes of the injured limb is likewise diminished. Impulses from pathologically changed tissues thus give rise to functional isolation of an injured organ.

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Previous investigations [1, 6-8] have shown that impulses from a focus of injury to a limb give rise to a focus of static excitation in the flexor center of the spinal cord. Mono- and polysynaptic flexor reflexes evoked by afferent stimuli from the injured limb are increased, to correspond to the protective, flexor attitude of the limb.

In this paper it is shown that the flexor center of the spinal cord, when in a state of excitation, is functionally isolated from other influences of the central nervous system: descending (from the pyramidal tract) and contralateral (from afferent components of the flexor reflex of the intact limb).

EXPERIMENTAL METHOD

Experiments were carried out on 25 adult cats anesthetized with Nembutal (20-30 mg/kg, intraperitoneally). Inflammation was produced in one hind limb by subcutaneous injection of 0.3-0.5 ml turpentine.

The pyramidal tract was approached by removal of the larynx, part of the pharynx and esophagus, and the long muscles of the neck and division of the basal bone. Stimulating electrodes were applied to the pyramidal tract and recording electrodes to roots L_7 and C_1. A monosynaptic reflex was evoked by stimulation of the deep peroneal nerve and the nerves to the medial and lateral heads of the gastrocnemius muscle.

Contralateral inhibition of the flexor reflexes of the hind limb was investigated as follows. The spinal cord was divided at the level T_6-T_8. Flexor polysynaptic reflexes were produced by stimulation of the divided ipsilateral superficial peroneal nerve and recorded in the nerves to the posterior head of biceps and the semitendinosus muscles. Conditioning stimuli were applied to the contralateral superficial peroneal nerve. The strengths of the testing and conditioning stimuli were 2-3 and 10-40 times above threshold for group 1a fibers, respectively. The interval between conditioning and test pulses was 50-80 msec. The magnitude of the polysynaptic responses was measured from their area.

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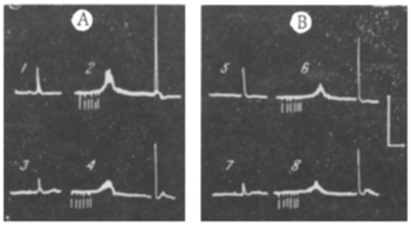

Fig. 1. Facilitatory action of pyramidal volleys on flexor monosynaptic reflex before (A) and after (B) injury to right limb in a cat (Nembutal anesthesia). 1, 3, 5, 7) monosynaptic reflex recorded in ipsilateral ventral root of segment L_7 to stimulation of deep peroneal nerve; 2, 4, 6, 8) same reflex facilitated by stimulation of pyramidal tract; 1, 2, 5, 6) responses in right ventral root; 3, 4, 7, 8) responses in left ventral root. After development of inflammation in right hind limb, facilitatory action of pyramidal tract on monosynaptic flexor reflex of injured limb becomes much less marked (B, 6). Each recording obtained by superposition of two sweeps of beam. Calibration 300 μV and 5 msec.

EXPERIMENTAL RESULTS

Influence of the Pyramidal Tract on Flexor Reflexes of Injured Limb. As shown originally by Sherrington [16], and subsequently confirmed [2, 3, 9, 10-14], stimulation of the pyramidal tract facilitates flexor reflexes of the contralateral limb. This observation was confirmed by the present experiments.

Facilitatory stimulation of the contralateral pyramidal tract increased the monosynaptic flexor reflex on the average by 350% (Fig. 1). The increase was greatest when the interval between facilitatory and test stimuli measured 20-25 msec. The limb swelled 1-2 h after subcutaneous injection of turpentine. At this time facilitatory stimulation of the pyramidal tract caused an increase of only 150-200% in the monosynaptic reflex. The facilitatory effects of the pyramidal tract on monosynaptic flexor reflexes of the healthy limb remained within normal limits.

Effect of Afferent Components of Flexor Reflex of Intact Hind Limb on Flexor Reflexes of Injured Limb. Sherrington [16] followed by other investigators [9, 14, 15], showed that stimulation of afferent components of the flexor reflex of one hind limb inhibits flexor reflexes of the contralateral limb. In the present experiments conditioning stimulation of afferent components of the contralateral limb likewise caused prolonged inhibition of the flexor reflex (230-250 msec). After injury, this inhibition diminished by 30-40%.

Hence, both facilitatory influences from the pyramidal tract and contralateral inhibitory influences on the flexor center are considerably reduced when this center is in a state of excitation. Comparatively recently one of the writers [5] found that Renshaw inhibition on an injured limb is also diminished.

Analogous functional isolation of autonomic centers of the nervous system have also been demonstrated in the past during localized injuries to the heart, lungs, stomach, and kidneys [6, 7].

The same mechanisms as were established in the body for responding to adequate stimuli are also used in reflexes to pathological stimulation. In the light of this, the discovery that functional isolation can also be found in physiological reactions is of undoubted interest. Two examples only will be given.

The orienting reflex is known to be accompanied by increased cortical electrical activity. Most workers regard activation as a manifestation of excitation. However, the following objection must be considered. The activation response is accompanied by depression of conditioned reflexes, or what Pavlov called "external inhibition." There is no real contradiction here. In a state of excitation evoked by the orienting reflex, the cortex ceases to respond to other additional stimuli.

It has recently been shown in Gurfinkel's laboratory [4] that Renshaw inhibition in an animal is depressed during locomotion. This is in complete agreement with the depression of Renshaw inhibition, mentioned above, during the flexor reflex evoked by injury to a limb. In both cases the center is in a state of excitation: in one case as the result of impulses from the pathological focus, in the other as a result of impulses evoked by locomotion.

It is interesting to compare the principle of functional isolation with the other general principle of activity of the nervous system — the mechanism of the dominant, also due to an increase in excitability of the center. In the dominant state the center easily responds to various additional stimuli, including those to which hitherto it did not respond. In a state of functional isolation, the reflex responses of the center are suppressed.

Functional isolation may be due to pre- or postsynaptic inhibition or to cathodic depression. The problem requires further investigation. One way or another, functional isolation evidently ensures relative rest for the pathological focus, and it is thus one of the mechanisms of healing.

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