EFFECT OF IMPULSATION FROM AN INFLAMMATORY FOCUS ON RECIPROCAL INHIBITION

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Impulsation from an inflammatory focus produces weakening of reciprocal inhibition of the extensors.

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Previous investigations in this laboratory showed that impulsation from an inflammatory focus produces both excitation and inhibition of spinal cord motoneurons. The object of the present investigation was to study the effect of impulsation from an inflammatory focus on reciprocal inhibition.

EXPERIMENTAL METHOD

The work of Renshaw [4] showed that reciprocal inhibition is most marked in the extensor muscles. Accordingly in our experiments we investigated the effect of antidromic stimulation on responses of extensor motoneurons. The degree of reciprocal inhibition was determined from the amplitude of monosynaptic reflexes (test responses) before and after antidromic stimulation (facilitatory stimulation).

Experiments were performed on cats after transection of the spinal cord at the level T_6-T_8 under nembutal- chloralose anesthesia (nembutal 6 mg/kg, chloralose 12 mg/kg). Monosynaptic reflexes were evoked by stimulation of the central ends of the divided dorsal roots of L_7 or S_1 and recorded in the nerve to the lateral head of the gastrocnemius muscle. Antidromic facilitatory stimulation of the motoneurons was produced by stimulating the nerves to the medial head of gastrocnemius and to the soleus muscle. Amplitudes of the monosynaptic responses were investigated at intervals from 0 to 50 msec between facilitatory and test stimulation. The amplitude of the monosynaptic response in each interval was taken as the arithmetical mean of five applications (one application every 5 msec). The strength of test and facilitatory stimulation was twice the threshold level for group 1a fibers. The nerves were stimulated with single square pulses, 0.1 msec in duration. Inflammation was produced by injection of 0.5-1 ml turpentine beneath the skin of the dorsum of the foot of one hind limb. Altogether 32 experiments were performed: 18 on animals with an inflammatory focus and 14 on healthy animals (control).

EXPERIMENTAL RESULTS

The curves in Fig. 1 show that impulsation from the inflammatory focus not only did not strengthen reciprocal inhibition but, on the contrary, weakened it. The thin line in Fig. 1 shows the curve of reciprocal inhibition of monosynaptic responses from the left hind limb, while the thick line shows the same from the right hind limb. The left limb was inflamed, the right intact. Inhibition clearly was less marked from the inflamed limb than from the intact. The results of investigation of inhibition in healthy animals are shown in Fig. 2. Inhibition was equal from both left and right limbs. Statistical analysis of the results confirmed their significance.

An inflammatory focus in the limb is known to evoke a protective flexor reflex. Activity of the flexor motoneurons is correspondingly increased and that of the extensor motoneurons reciprocally inhibited. A decrease in flow of impulses from motoneurons to the effector muscle is evidently accompanied by a simultaneous decrease in the flow of impulses spreading along reciprocal collaterals of the axon to the inhibitory Renshaw neuron, as a result of which reciprocal inhibition of the extensors is weakened in the affected limb.

Another mechanism is also possible. As a result of Wilson's investigations [5], the decrease in reciprocal inhibition can be attributed to "inhibition of inhibition." Wilson showed that discharge of the

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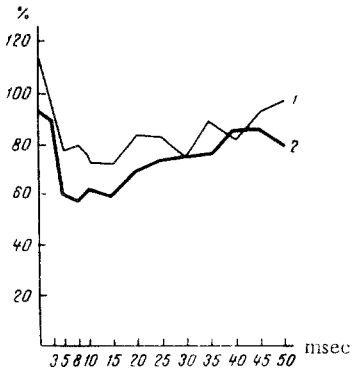

Fig. 1

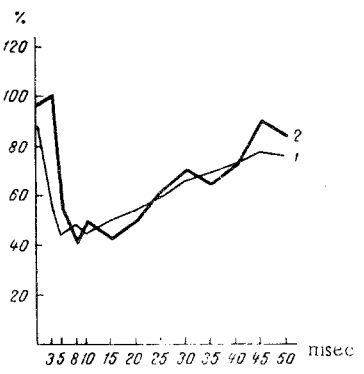

Fig. 2

Fig. 1. Aggregated curves of reciprocal inhibition from left (1) and right (2) limbs. Abscissa, intervals between facilitatory and test stimulation (in msec); ordinate, amplitudes of facilitated monosynaptic reflexes (in percent). Amplitude of unfacilitated monosynaptic response taken as 100. Left limb inflamed.

Fig. 2. Aggregated curves of reciprocal inhibition in healthy animals. Legend as in Fig. 1.

Renshaw cells is inhibited by impulses from the skin during nociceptive stimulation (pinching, etc.), just as during electrical stimulation of cutaneous fibers with a strength sufficient to cause excitation of Aσ fibers. Wilson suggests that inhibition of Renshaw cells is produced by other inhibitory interneurons which are excited by impulses arriving, in particular, from Aσ and C cutaneous fibers. Impulses from a pathological focus [2, 3, 6], as in the case of an inflammatory focus, developing at a site of injury [1], spread along Aσ and C fibers. Weakening of reciprocal inhibition in the affected limb may therefore arise because of "inhibition of inhibition."

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