

CHARACTERISTICS OF RECIPROCAL INHIBITION OF EXTENSOR MOTONEURONS AFTER EXTINCTION OF BACKGROUND SUPRASPINAL INFLUENCES

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Reciprocal inhibition of extensor motoneurons in the cat spinal cord is increased 1.5-2 h after chordotomy. An important role in the formation of spinal shock after chordotomy is assigned to the active decrease in excitability of the extensor motoneuron as the result of the strengthening of reciprocal inhibition under these conditions.

Previous investigations into the functional role of background activity of the brain centers by Dontsova [3] revealed its great importance in the formation of the functional systems of the brain and in their coordination. For this reason the question of the functional role of background supraspinal influences in the formation of spinal reflexes is of great interest because it can help to shed light on new mechanisms of integrative activity of the nervous system.

The object of the present investigation was to determine the concrete role of background supraspinal influences in the formation of reciprocal inhibition and in its sequelae.

EXPERIMENTAL METHOD

Experiments were carried out on cats 6-7 h after induction of anesthesia (100 mg/kg hexobarbital intraperitoneally). Laminectomy was performed in the regions of segments L5-S1 and T12-L1. Testing stimuli (0.1-0.2 msec, 1.5-2 times threshold strength) were applied through buried silver electrodes to the nerve to the gastrocnemius muscle or to the posterior tibial nerve. Conditioning antidromic stimulation was applied to a filament of the divided ventral root of L7 or S1 by the method described by Brooks and Wilson [5]. The strength of the conditioning stimulus was chosen so that the amplitude of the testing monosynaptic reflex was depressed by 10-20%, so that changes in this reflex could be observed whether in the form of an increase or a decrease. Single stimuli were applied from a three-channel electronic stimulator with radiofrequency output. After preliminary amplification the potentials were photographed from the screen of a type S1-19 cathode-ray oscilloscope. The background supraspinal flow was blocked by chordotomy at the level of the last rib. Further observations were made after 1.5-2 h when, according to data in the literature, degeneration of the structures of the spinal cord had not yet occurred but when the traumatic effects had passed off and absence of background supraspinal influences was the dominant factor.

EXPERIMENTAL RESULTS AND DISCUSSION

In all 7 experiments, 1.5-2 h after chordotomy a distinct change in amplitude of both the monosynaptic and the polysynaptic components of the ventral root potential was recorded, in agreement with previous observations [2, 4]. The amplitude of the monosynaptic reflex of extensor motoneurons was reduced after chordotomy on the average by 20% (within the range 5-30%). Meanwhile definite facilitation and synchronization of the polysynaptic reflexes, or their appearance if they had previously been absent when the chord was

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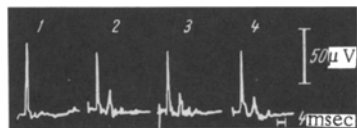


Fig. 1

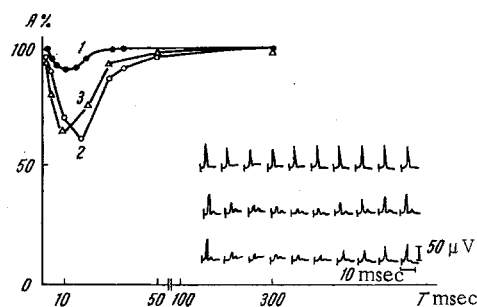


Fig. 2

Fig. 1. Effect of chordotomy on monosynaptic and polysynaptic components of the potential recorded from the ventral root of a segment of the cat's spinal cord: 1) spinal cord intact; 2, 3, 4) 1, 1.5, and 2 h respectively after chordotomy.

Fig. 2. Dynamics of reciprocal inhibition of extensor motoneurons with intact spinal chord and after chordotomy: 1) spinal cord intact; 2) 1.5 h after chordotomy; 3) 2 h after chordotomy. Top row of records with spinal cord intact; middle row 1.5 h after chordotomy; bottom row 2 h after chordotomy. In each row from left to right: 1st record is testing monosynaptic reflex, 2nd obtained for interval of 2 msec between conditioning and testing stimuli, 3rd - 6 msec, 4th - 8 msec, 5th - 10 msec, 6th - 30 msec, 7th - 50 msec, 8th - 100 msec, 9th - 200 msec, 10th - 300 msec.

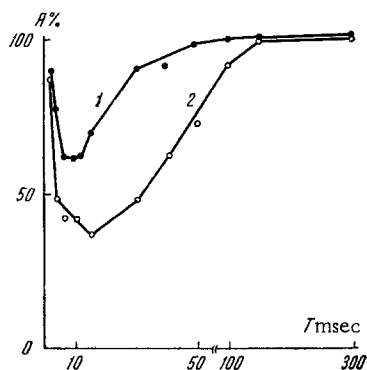


Fig. 3. Changes in reciprocal inhibition of extensor motoneurons after chordotomy (mean results of 7 experiments): 1) spinal cord intact; 2) 1.5 h after chordotomy.

intact, were recorded (Fig. 1). It is important to note that simultaneously with these effects, chordotomy also changed the character of appearance of the reciprocal inhibition (Fig. 2). When the spinal cord was intact, the monosynaptic extensor reflex began to be inhibited after 2 msec. The inhibition ended after 30 msec, with a maximum at 8-10 msec; the monosynaptic reflex was then inhibited by 10%. Inhibition of the monosynaptic reflex increased more rapidly 1.5-2 h after chordotomy. By 8 msec the monosynaptic reflex was inhibited by 35%, i.e., by 25% more than before chordotomy, and it was restored later - after 50 msec. The changes observed 30 min after chordotomy were unstable, evidently because of traumatic effects. Changes of the same character in reciprocal inhibition were also observed in the other experiments: in 6 of 7 experiments it was strengthened on the average by 22%, and only in 1 experiment was it very slightly reduced (by 8%). In 2 experiments reciprocal inhibition of the monosynaptic reflexes, although deeper in intensity, remained the same in duration as when the spinal cord was intact.

The mean results for all the experiments are summarized in Fig. 3. Statistical analysis of the results showed that differences were significant.

The decrease in amplitude of the monosynaptic reflexes of the extensor motoneurons with a simultaneous increase in their polysynaptic discharges indicated that 1.5-2 h after chordotomy the excitability of the monosynaptic reflex arcs was reduced and, at the same time, the activity in their polysynaptic pathways was increased. This was evidence that the conditions had arisen for equalization of the degree of excitability of the two pathways. Accordingly, responses not only of monosynaptic, but also of polysynaptic reflex arcs appeared to the stimulation of the same threshold strength as before. The question arises: by what mechanisms does the decrease in activity of the extensor motoneurons take place after chordotomy. Sherrington [7], who observed a decrease in the excitability of muscle extensor reflexes, postulated that in the intact spinal cord there could be initial stimulating supraspinal influences on the extensor motoneurons. Their cessation after chordotomy is the cause of the decrease in excitability of these motoneurons.

The increase in number of polysynaptic discharges recorded after chromotomy by the writers and by other investigators [1, 2, 4] must apparently have evoked a tendency in those animals toward prolonged extension similar to that observed in decerebrate rigidity. However, this was not observed. Accordingly, the fact should be noted that a decrease in amplitude of the monosynaptic reflexes took place not only in response to a simultaneous increase in activity of the polysynaptic arcs, but also during an increase in the degree and duration of reciprocal inhibition of the extensor motoneurons. The fact established by Maclean and Leffman [6], that evoked stimulation of various brain structures can inhibit the Renshaw cells and, consequently, weaken reciprocal inhibition, must also be mentioned. Comparison of this fact with the results of the present experiments suggests that in the intact spinal cord similar influences from certain supraspinal structures on the central mechanisms of reciprocal inhibition arise not only in response to special evoked stimulation, but also in its absence, i.e., that background supraspinal inhibition of Renshaw cells can take place, just as with other interneurons. One result of this state of affairs is that in the intact spinal cord there is a mechanism of background inhibition of segmental reciprocal inhibition of extensor motoneurons. Blocking the background supraspinal influences can thus be a cause of the strengthening of reciprocal inhibition of extensor motoneurons. Although strengthening of the polysynaptic actions on extensor motoneurons after chordotomy evokes discharges in them, at the same time, it can also lead to an increase in the number of impulses traveling along the recurrent axon collaterals of the motoneuron, which, in turn, can cause the strengthening of the reciprocal inhibition. As a result, no impulses leave the motoneurons for the muscles. All these factors evidently combine to cause the absence of long extension after chordotomy.

These results suggest that spinal shock after chordotomy may be the result, not only of causes identified by other workers, but also to a larger extent of an increase in the activity of the segmental mechanism of reciprocal inhibition of the extensor motoneurons, leading under these conditions to a decrease in their excitability.

They confirm the view expressed by Dontsova [3] that the mechanism of background influences and background activity of the brain and spinal cord is one of the important principles governing the formation of regulatory mechanisms of the nervous system.

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