RECIPROCAL INFLUENCES ON WORK OF SINGLE MOTONEURONS DURING CONTROLLED LOCOMOTION

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Susceptibility of motoneurons to antidromic inhibition was compared when activated under three different conditions: 1) during tonic stretching of the corresponding muscles; 2) during passive cyclic stretching of the muscles; 3) during locomotion of mesencephalic cats on a treadmill, evoked by stimulation of a particular region of the tectum mesencephali.

During evoked locomotion antidromic stimulation was found to have no noticeable effect on the frequency of motoneuron discharges and the duration of its discharge volley; during passive cyclic stretching of the muscles the discharge frequency showed little change but the duration of the volley was reduced.

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The effects of antidromic stimulation of motoneurons (reciprocal inhibition and facilitation) on immobilized preparations have been extensively studied [1, 4, 9, 10]. It has also been shown that supraspinal [8] and afferent [11] influences can modify the activity of the Renshaw cells giving rise to inhibitory interaction between motoneurons. However, technical difficulties have so far prevented the investigation of reciprocal influences on motoneurons during motor activity.

In the present investigation the action of antidromic stimulation of a ventral root on the work of motoneurons was compared during walking and during a stretch reflex, using a preparation with controlled locomotion [3].

EXPERIMENTAL METHOD

Tracheotomy and laminectomy at the level L_5-L_7 were performed on cats anesthetized with ether; the ventral root of L_7 was dissected and divided at its point of emergence from the dura. The carotid arteries were then ligated and precollicular decerebration performed. The anesthesia was ended, the animal's head was fixed in a stereotaxic holder, and the limbs were placed on the belt of a treadmill [3]. The spine was fixed in the lumbar region by two pairs of metal prongs. Under these conditions locomotion simulating natural can be evoked by electrical stimulation of a certain part of the mesencephalon at the border with the pons (Korsley-Clarke coordinates of the end of the bipolar electrode P2, U4, H0), and impulse activity of single motoneurons in a thin filament from the ventral root of L_7 can be recorded during walking [2]. The whole of the rest of the root was placed on another pair of electrodes for antidromic stimulation.

After isolation of a filament with a functionally isolated motoneuron generating impulses during locomotion, the possibility that it could be activated tonically by stretching a certain muscle group other than during locomotion was investigated. For this purpose passive movements were performed at different joints of the corresponding hind limb. The tonic impulse activity of the motoneuron thus obtained was inhibited by antidromic stimulation of the ventral root of L_7 (with the exception of the filament isolated from it). Antidromic stimulation was then carried out (with the same parameters—square pulses, 0.1 msec, 70-100/sec, 10-20 V) during evoked locomotion.

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TABLE 1. Frequency of Discharges (pulses/sec) of Motoneurons Activated Under Different Conditions

No. of motoneuron	Static passive stretching		Cyclic passive stretching of muscles		Controlled locomotion	
	without antidromic stimulation	with antidromic stimulation	without antidromic stimulation	with antidromic stimulation	without antidromic stimulation	with antidromic stimulation
1	35	15	41	26	Sup. 40 Tr. 16	35 16
2	22	12	33	25	43	42
3	34	24	30	30[1]	Sup. 34 Tr. 49	32 48
4	14	11	—	—	36	33
5	15	8	21	17	37	32
6	30	0	30	0[2]	55	60
7	15	0	—	—	Sup. 31 NS Tr. 46	30 43
8	31	0	30	25[1]	44	43
9	33 25	26 20	1Hc39 2 » 41	32[1] 34[1]	NS 34 S48	33 46
10	26 44	18 26	1 » 38 2 » 42	40[1] 50[1]	NS 43 S57	39 56
11	34	0	1 » 40 2 » 40	40[1] 41[1]	NS 47 S 70	42 68

1 Duration of volley of impulses reduced.
2 In most cycles inhibition was complete, but in some from 1 to 3 impulses remained from the whole volley.
Note. Each number in the table represents the mean or three adjacent cycles (in the 4 right columns) or for two or three adjacent tests not less than 1 sec in duration (in the 2 left columns). NS) walking without stimulation of mesencephalon; S) walking evoked by stimulation of brain stem (see also in text); Sup) phase of support; Tr.) phase of transfer.

EXPERIMENTAL RESULTS

Reciprocal influences during locomotion were investigated only on those motoneurons in which antidromic stimulation reduced the frequency of discharges evoked by passive movements at a joint. In 5 experiments 11 such motoneurons were recorded (Table 1). Three of these neurons (Nos. 1, 3, 7) could be activated by passive movements of the hip joint, the rest only by dorsiflexion of the ankle. Motoneurons of the muscles of the hip joint gave volleys of impulses during evoked locomotion both in the phase of support and in the phase of transfer of of the limb, but the ankle motoneurons gave volleys only in the phase of support.

In two experiments in which discharges were recorded from motoneurons (Nos. 7-11), the brain stem was divided so that the preparations were still able to walk in the absence of stimulation of the brain stem along a slowly (up to 0.5 m/sec) moving belt [3]. In another three experiments, the preparations were unable to make stepping movements without stimulation of the brain stem. During walking without stimulation activity was recorded in neurons Nos. 7 and 9-11. Discharges of the last three motoneurons were also recorded during locomotion evoked by stimulation of the brain stem. Motoneurons Nos. 1-6 and 8 were investigated only during walking evoked by mesencephalic stimulation.

The effect of antidromic stimulation on discharges of neurons Nos. 9 and 10 evoked by passive static stretching of the muscle was investigated both before and after locomotion. In the case of neurons Nos. 9-11, the effect of antidromic stimulation was investigated during activation by passive cyclic movement of the joint with a frequency of 1 and 2/sec (in the other cases a frequency of 1/sec only was used).

Traces obtained during an experiment with motoneuron No. 8 are given in Fig. 1. It did not generate impulses spontaneously. During maximal static flexion of the ankle it fired at the rate of 31 discharges/sec. Antidromic stimulation completely inhibited this activity (A). During evoked locomotion this motoneuron fired in the supporting phase at the rate of 44/sec. Antidromic stimulation lowered the frequency of the discharges in the volley only slightly (the last step in Fig. 1B)—to 42/sec. An intermediate result was observed during activation of the motoneuron by cyclic passive flexion of the ankle not during locomotion, in the absence of stimulation of the brain stem. This motoneuron under these circumstances fired at the rate of 30/sec and antidromic stimulation lowered the frequency of discharges in the volley to 25/sec, appreciably shortening the duration of the volley (Fig. 1B).

During antidromic stimulation of four motoneurons the impulse activity evoked by static stretching of the muscle ceased completely, and in another four its frequency fell by 8-20 (12) impulses/sec. In all these neurons the frequency of discharge during locomotion was reduced only slightly by the same antidromic stimulation. In the case of neurons Nos. 4, 5, and 9, antidromic stimulation lowered the frequency of discharges evoked by static stretching of the muscle by only 3-7/sec. In two of these cases an almost identical decrease in frequency of impulse activity took place during locomotion also, while in the third, inhibition was weakened during locomotion. The motoneurons whose frequency of tonic impulse activity was essentially reduced under the influence of antidromic stimulation thus hardly reacted to it at all during locomotion. Motoneurons showing weak inhibition during myostatic activation sometimes failed to show a significant decrease in susceptibility to antidromic inhibition during locomotion.

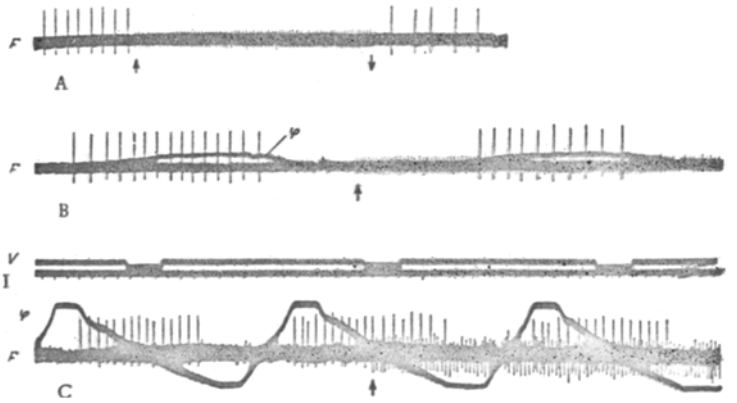

Fig. 1. Discharges of motoneuron during static stretching of ankle extensors (A), during rhythmic stretching of extensors (B), and during evoked locomotion (C). F) activity of filament; φ) longitudinal displacement of foot during walking (upward deflection of curve corresponds to phase of transfer of limb) or during passive flexion of joint (upward deflection of curve denotes decrease in angle of ankle joint); I) marker of stimulation of brain stem by square pulses, 1 msec, 30/sec; V) velocity of movement of treadmill belt (during interval between two marks belt advances 0.5 m). Arrow above denotes end of antidromic stimulation of ventral root of L_7 at 100/sec. Stimulation artifacts also visible.

In nearly all motoneurons investigated during cyclic passive stretching of the muscle the frequency of their discharges fell during antidromic stimulation to a lesser degree than during static stretching, but the duration of the volley was usually shortened. Because of the shortening of the low-frequency "tail" of the volley, the mean frequency of impulses in it was sometimes unchanged or even increased (neuron No. 10). During locomotion the effect of antidromic stimulation was weaker still than during cyclic passive stretching of the muscle: not only the frequency of the discharges but also the duration of the volley usually remained unchanged.

Granit and Renkin [7] found that during static activation of a motoneuron by stretching the corresponding muscle, antidromic stimulation lowers the frequency of impulse activity of the motoneuron by the same extent regardless of the initial frequency of its impulse activity over a wide range of frequencies. However, the effectiveness of antidromic inhibition is appreciably influenced by the magnitude of "excess excitation" not reflected in the frequency of impulse generation by the motoneuron [6]. This phenomenon of "excess excitation," whose mechanism has not yet been explained, may be the reason for the difference in effectiveness of antidromic stimulation during locomotion and during myotatic activation of motoneurons. During locomotion the motoneuron generates volleys of impulses lasting only a few tenths of a second, so that the "excess excitation," if present under these conditions, is unable to disappear, thus leading to weakening of antidromic inhibition.

However, the ineffectiveness of antidromic inhibition during evoked locomotion may be due also to inhibition of the Renshaw cells. An important factor here must clearly be the cyclic pattern of motoneuron activity. This is shown by the decrease in effectiveness of antidromic inhibition during activation of the motoneuron by cyclic passive muscle stretching. In this connection it is interesting to note the absence of reciprocal inhibition of the motoneurons of the diaphragm [5], which under natural conditions operate almost entirely on a cyclic pattern. However, the effectiveness of antidromic inhibition during evoked locomotion is appreciably reduced even when compared with what is observed during cyclic activation of motoneurons by passive muscle stretching.

Antidromic stimulation of none of the investigated motoneurons lowered the frequency of successive impulses in the volley during walking by more than 5/sec. Bearing in mind that under these circumstances nearly all axons of the ventral root were stimulated at very high frequency, the observed effects of antidro-

mic inhibition can be taken as maximal. During natural walking only some of the motoneurons are excited and the frequency of their impulse activity is much lower. During walking, reciprocal inhibition evidently does not play an essential role in determining the frequency of impulse activity of the motoneurons.

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