INFLUENCE OF DISCHARGE OF MOTONEURONS
UPON EXCITATION OF NEIGHBORING
MOTONEURONS

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INTRODUCTION

Some spinal motoneurons are equipped with recurrent collaterals which
arise from the axon near its origin and terminate in association with other
neurons of the ventral horn (cf. Cajal, 1909). An impulse that sweeps over
the motor axon must also invade its collaterals. Does it then affect the excita-
bility of the neurons in association with which the collaterals terminate?
Müller (1835) could produce no muscular contractions by stimulating the
central end of a cut motor root. Others have likewise failed to find evidence
that an antidromic volley produces either a centrifugal discharge in motor
axons or activity in other nerve tracts (Mislawski, 1895; Bernstein, 1898;
Eccles, 1931). In the absence of known excitatory effects it has been sug-
gested that impulses in recurrent collaterals might lead to inhibition of the
activity in the neurons to which they pass (Graham Brown, 1914; Gesell,
1940). The collaterals would then be an important part of the mechanism
for reciprocal innervation. Forbes and his collaborators (1933) put the sug-
gestion of Graham Brown to a careful experimental test. They found that
a contralaterally evoked reflex discharge into the tibial nerve is not condi-
tioned by antidromic volleys arriving at the cord in the motor axons of the
peroneal nerve.

The present experiments show that the antidromic activation of certain
groups of motoneurons does condition the reflex discharges of other moto-
neurons. The conditioning effect is often inhibitory. It is then neither pre-
ceded by facilitation nor delayed; inhibition is present when the antidromic
volley reaches the cord approximately simultaneously with an afferent volley
which fires the testing motoneurons directly after a single synaptic delay.
The inhibition must then be caused by events occurring during the synaptic
delay at the motoneurons—a period of only 0.9 msec. or less (Lorente de
Nó, 1938; Renshaw, 1940). The conditioning volley cannot have fired either
the tested motoneurons or premotor interneurons in time for the refractori-
ness (subnormality) which follows activity to mediate the response deficit
(cf. Gasser, 1937a, b; Lorente de Nó, 1936).

In a discussion of this phenomenon it would be misleading to focus atten-
tion only upon the possible role of recurrent collaterals. It is not necessary
to infer from the early onset of inhibition that a specific inhibitory action is
produced by the arrival of impulses at the synapses made by the recurrent
collaterals with other neurons. As Grundfest (1940) has pointed out, an al-
terative explanation for findings of this sort is suggested by the fact that
activity in axons can alter the excitability of adjacent, structurally independent axons (Adrian, 1930; Jasper and Monnier, 1938; Arvanitaki, 1940b; Katz and Schmitt, 1940). Evidence has also been supplied to show that activity in neurons in the central nervous system can affect the discharges of adjacent neurons in the absence of synaptic associations (Barron and Matthews, 1935; Gerard and Libet, 1940).

In the experiments about to be described it has been necessary to activate the conditioning motoneurons antidromically. The results have a more general significance, however, for it may be supposed that in any experiment synaptic excitation of the same motor cells would have produced similar effects (Sherrington, 1900).

METHODS

The preparations were cats, either decerebrated or under light barbiturate anesthesia (Dial; Nembutal). Reflex discharges of motoneurons were routinely induced by shocks applied to dorsal roots or to the dorsal columns. Particular use was made of the fact that the first portion of the reflex discharges to these stimuli represents the direct activation of motoneurons by the primary afferent fibers after a single synaptic delay; the size of the first wave of the reflex discharge to a constant afferent volley is then a measure of the synaptic excitability of the motoneurons (Renshaw, 1940). Stimulating and recording leads were also applied to various groups of ventral rootlets, to mixed nerves of the leg, and to the branches to individual muscles of the hind limb. Spinal transections and the intradural cutting of dorsal roots were performed according to the requirements of specific experiments. The usual differential amplifier and stimulating apparatus were used.

RESULTS

1. Centrifugal discharge in motor fibers following an antidromic volley. A volley of impulses backfired toward the spinal cord over a group of motor fibers sets up, after a short central delay, a centripetally directed discharge in some motoneurons. A typical experiment is illustrated in Fig. 1. The seventh lumbar ventral root was severed intradurally and four electrodes were placed on each of two portions of it, A and B. A shock, approximately maximal for alpha fibers, delivered to A through electrodes 3 and 4 set up a volley of impulses which travelled centrally in the motor fibers. This was recorded at low amplification through electrodes Al-2 (record c). Record d, at 25 times the amplification of c, clearly shows that the deflection due to the large centripetal volley was followed by a smaller diphasic response of reversed sign. Therefore the arrival of the antidromic volley at a central region evoked, after a short delay, a centrifugal discharge in some motor fibers. In records a and b the stimulating electrodes were placed central to the recording electrodes. The axons were killed under electrode 4. Both the direct response to the shock and the delayed volley of central origin then appeared as monophasic deflections. The delayed volley of central origin is seen to have occupied 2 to 3 per cent of the number of fibers involved in the centripetal discharge which evoked it.

The centrifugal discharge following an antidromic volley also occurs in the large limb nerves, such as the tibial and peroneal nerves, and in the nerves to individual muscles of the leg. It appears after the dorsal roots which contain the sensory fibers of the nerve under examination have been cut.
It is not possible to confuse the efferent discharge which follows an antidromic volley with spontaneous firing on the negative after-potential at the cut distal end of the prepared nerve. High partial pressure of carbon dioxide about the distal end greatly reduces the spontaneous firing without altering the centrifugal discharge.
Several additional facts point to a central origin for the discharge which follows an antidromic volley. Not only do the impulses of the discharge pass in a centrifugal direction (Fig. 1d), but their presence depends upon the integrity of the spinal cord. The efferent discharge is reversibly abolished during the subtotal asphyxia of the cord, which may be induced by temporary occlusion of the descending aorta at the level of the upper lumbar segments. Lastly, the centrifugal discharge is conditioned by a preceding dorsal root volley which fires few motoneurons (Fig. 2).

While these experiments demonstrate that the centrifugal impulses are set up at the spinal cord, they do not permit a more specific inference about its locus of origin. All that can be said is that the discharge arises at or central to the point of emergence of the motor axons from the cord.

The central latency for the centrifugal discharge evoked by an antidromic volley may be determined from the total latency by subtracting the conduction times in the motor axons for the centripetal and the centrifugal impulses. The total conduction time in the fastest motor axons may be approximated by the sum of the shock-response intervals for m waves (Lorente de Nó, 1939, page 409) set up by direct electrical stimulation of the motoneurons in the ventral horn and detected at the recording leads and at the stimulating cathode. These corrections for oscillogram b of Fig. 1 amount to ca. 0.3 msec. The minimal central latency, therefore, is ca. 0.9 msec., provided that some of both the centripetal and the centrifugal impulses pass in fibers of maximal or nearly maximal conduction velocity. This assumption is verified by the fact that apparent central latencies calculated in this way from records made on nerves in the thigh are equal to or only slightly greater than the central latencies calculated from the data of ventral root leads.

The central latencies for the centrifugal discharge generally lie between 0.8 and a little over 1.0 msec. The similarity of these values to the synaptic delays at motoneurons (Lorente de Nó, 1938; Renshaw, 1940) immediately suggests that the centrifugal impulses may arise from the synaptic excitation of motoneurons by impulses of the antidromic volley arriving over recurrent collaterals. Several lines of evidence render this possibility very unlikely. Instead, the centrifugal discharge seems to be due to repetitive activity in a fraction of the antidromically activated motoneurons. It is, therefore, in some ways formally comparable with the "effet pseudoréflexe" of Arvanitaki (1938, page 101; 1940a).

First, the centrifugal discharge never appears in a group of motor fibers other than that in which the antidromic volley passes. This is true when the fibers used for stimulating and recording are groups of ventral rootlets from the same or from adjacent levels of the cord (Fig. 1e and f). It is also true when mixed nerves of the leg are used (dorsal roots cut), and the same result obtains with motor branches to the same or different muscles.

Second, contributory evidence comes from the relationship between the size of an antidromic volley and the size of the efferent discharge which it
produces. If the efferent discharge were synaptically excited, it would be expected that as the size of the antidromic volley approached its maximal value, the number of centrifugal impulses set up by it would decrease, owing to the large number of refractory motoneurons. Actually a roughly linear relationship exists between the numbers of centripetal and centrifugal impulses. Figure 3 shows some of the responses obtained in one experiment in which the stimulating shock was progressively increased from submaximal strengths to values supermaximal for A fibers.

Additional proof is supplied by the effects which an antidromic volley exerts upon the reflex discharges of other motoneurons. As stated above, an antidromic volley in isolation never sets up centrifugal impulses in other motoneurons. It does, however, condition the reflex discharges into other motor axons. A detailed discussion of these effects follows. In brief, the facilitation which may occur is always delayed; inhibitory effects on the other hand appear immediately upon the arrival of the antidromic impulses into the cord. Therefore, at the time an antidromic volley, which arrives at the cord in some motoneurons, is setting up its centrifugal discharge, it produces only inhibitory effects on the synaptic excitation of other motoneurons. The efferent discharge cannot, therefore, be in motoneurons other than those invaded by the backfired impulses. Detonator facilitation produced by the arrival of impulses at the terminal knobs of the recurrent collaterals has not yet been discovered. One might suppose that such excitation exists; but as far as other motoneurons are concerned, any such action is masked by a more effective inhibitory process acting at the same time.

2. Effect of antidromic volley in some motor fibers on reflex discharges of other motoneurons. Antidromic volleys in certain deafferented motor nerves condition subsequent reflex discharges into other motor branches (Fig. 4–8; Table 1). For a given pair of nerves the effect is relatively constant in different preparations. The conditioning is frequently inhibitory; but facilitation, usually preceded by inhibition, also occurs.

An antidromic volley in one group of ventral rootlets often acts to inhibit subsequent testing discharges in the motor axons of adjacent rootlets. In contrast to the results ob-
tained when motor nerves are used for conditioning and testing, the effects are variable and sometimes absent. The reason for the relative constancy of the conditioning effect when motor nerves, rather than groups of ventral rootlets, are used is obvious in the light of cer-

Table 1. Effects of antidromic volleys in some motor nerves on reflex discharges into other motor nerves

<table>
<thead>
<tr>
<th>Motoneurons occupied by conditioning volley</th>
<th>Test motoneurons</th>
<th>Effect of conditioning volley on tested reflex*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Conditioning volley and tested reflex in motor nerves to the same muscle or muscle group</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>biceps (one part)</td>
<td>biceps (another part)</td>
<td>Inhibition (33, 65)</td>
</tr>
<tr>
<td>biceps (one part)</td>
<td>semitendinosus</td>
<td>Inhibition (50, 64, 75, 70, 75, 20)</td>
</tr>
<tr>
<td>biceps (one part)</td>
<td>biceps (one part)</td>
<td>Inhibition (80, 40, 85, 20)</td>
</tr>
<tr>
<td>semimembranosus (one part)</td>
<td>semimembranosus (another part)</td>
<td>Inhibition (92)</td>
</tr>
<tr>
<td>semimembranosus (one part)</td>
<td>semimembranosus (lateral head of gastrocnemius)</td>
<td>Inhibition (35)</td>
</tr>
<tr>
<td>medial head of gastrocnemius</td>
<td>biceps (other half)</td>
<td>Inhibition (35)</td>
</tr>
<tr>
<td>lateral head of gastrocnemius</td>
<td>sartorius (other half)</td>
<td>Inhibition (50, 33, 50, 75)</td>
</tr>
<tr>
<td>quadriceps (one half)</td>
<td>sartorius (remainder)</td>
<td>Inhibition (60, 60)</td>
</tr>
<tr>
<td>sartorius (one part)</td>
<td>Inhibition (10, 10, 15, 10, 10)</td>
<td>Inhibition (60, 70)</td>
</tr>
<tr>
<td><strong>Conditioning volley and tested reflex in nerves whose motoneuron pools lie in different portions of the ventral horn</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tibial</td>
<td>peroneal</td>
<td>Facilitation (to 140 percent), preceded by slight inhibition</td>
</tr>
<tr>
<td>peroneal</td>
<td>tibial</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>hamstring</td>
<td>hamstring</td>
<td>Inhibition (61, 85)</td>
</tr>
<tr>
<td>peroneal</td>
<td>tibial</td>
<td>Inhibition (80, 85), followed by facilitation (118, 120)</td>
</tr>
<tr>
<td>hamstring</td>
<td>hamstring</td>
<td>Inhibition (75, 82)</td>
</tr>
<tr>
<td>peroneal</td>
<td>tibial</td>
<td>Inhibition (85, 92)</td>
</tr>
<tr>
<td>hamstring</td>
<td>peroneal</td>
<td>Inhibition (54, 88, 95)</td>
</tr>
<tr>
<td>quadriceps (lateral head of gastrocnemius)</td>
<td>quadriceps</td>
<td>Facilitation (117)</td>
</tr>
<tr>
<td>quadriceps (quadriceps)</td>
<td>quadriceps</td>
<td>Inhibition (76)</td>
</tr>
<tr>
<td>tibial</td>
<td>tibial</td>
<td>Slight facilitation (?)</td>
</tr>
<tr>
<td>quadriceps (tibialis anticus)</td>
<td>quadriceps</td>
<td>Slight inhibition (?)</td>
</tr>
<tr>
<td>peroneal</td>
<td>peroneal</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>quadriceps (quadriceps)</td>
<td>quadriceps</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>sartorius (sartorius)</td>
<td>sartorius</td>
<td>Slight inhibition</td>
</tr>
<tr>
<td><strong>Conditioning volley and tested reflex in motor nerves to individual antagonistic muscles</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>gastrocnemius (tibialis anticus extensor longus)</td>
<td>tibialis anticus gastrocnemius</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>tibialis anticus extensor longus</td>
<td>flexor longus</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>digitorium</td>
<td>hallucis</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>flexor longus</td>
<td>extensor longus</td>
<td>No significant effect observed</td>
</tr>
<tr>
<td>hallucis</td>
<td>digitorium</td>
<td>No significant effect observed</td>
</tr>
</tbody>
</table>

* The figures in parentheses represent the maximal conditioning seen in specific experiments and expressed as:

\[
\text{size of conditioned reflex} \times 100 = \frac{\text{size of unconditioned reflex}}{100}.
\]
taining anatomical details (Sherrington, 1892; Marinesco, 1904; Cajal, 1909; Bok, 1928). The fibers of any small ventral rootlet are the axis cylinders of motoneurons lying in the cord at approximately the segmental level at which the rootlet makes its exit. They lie scattered throughout the cross-section of the ventral horn at this segmental level (Cajal). The motoneurons associated with any given muscle, on the other hand, extend continuously over two or more segments of the cord: but their cell bodies always occupy a specific, restricted locus in the cross-section of the ventral horn (Marinesco, Bok). The motor axons to each muscle thus arise from cells occupying a specific portion of the ventral horn. They become randomly mixed with other motor fibers in the ventral roots, and eventually segregate again into the motor nerve. The segmental variation in the innervation of any particular muscle is considerable, but the neurons representing the various muscles always preserve the same relative positions longitudinally in the cord (Sherrington). Thus in all preparations the motoneurons supplying any muscle stand in the same axial and cross-sectional relationship to those of every other muscle. No such fixed relationship exists between the motoneurons supplying any two groups of rootlets.

Control experiments demonstrate that antidromic conditioning as shown in Fig. 4 to 8 depends upon the arrival at the cord of impulses in motor A fibers. (i) The routine procedure has been to section the dorsal roots at, above, and below the segments which typically receive afferent fibers from the antidromically stimulated nerve. More extensive deafferentation in a few experiments has served to exclude the possibility that the conditioning is mediated by impulses in aberrant sensory fibers. In one such experiment the cord was transected at both caudal and upper lumbar levels. All dorsal roots on both sides of the isolated lumbosacral segments were cut intradurally. Impulses from the periphery could then arrive only via the intact motor roots. A shock applied to the dorsal columns at L4 served to set up a small discharge in a motor branch to the quadriceps. This discharge was inhibited by a preceding antidromic volley in the other branches of the crural nerve, just as when only the homolateral lumbar and sacral dorsal roots had been severed. (ii) The records of Fig. 4 demonstrate that impulses in the A fibers of the conditioning nerve are the ones which produce the inhibition. In the experiment from which the records of Fig. 4 are taken, stimulation of the homolateral dorsal columns at L4 produced a discharge in the nerve to the lateral head of the gastrocnemius (record a). In the following records (b–e) the column stimulus was preceded by a shock applied to the nerve of the medial head of the gastrocnemius. The inhibi-

![Image of Figure 4](http://jn.physiology.org/Downloadedfrom/10.2353/jn.1960.381.333)
tion of the reflex increased as the conditioning shock was strengthened from threshold to a value maximal for A fibers (record d). A 20-fold increase of the stimulus (record e) then produced no additional effect. (iii) The remote possibility that the antidromic volley produces conditioning peripherally rather than centrally has been excluded. The greatest conditioning effect is produced when the antidromic volley has entered the cord several milliseconds before the reflex impulses leave, a time at which the large peripheral changes due to the action currents of the antidromic impulses would have disappeared. Furthermore, when motor discharges are known to have been set up in motor fibers, as after electrical stimulation, they are never affected by antidromic volleys in other motor fibers.

The most conspicuous effect of an antidromic volley in some motoneurons on the discharges of others is found when the conditioning and tested nerves are branches to the same muscle or muscle group. The two pools of motoneurons then occupy the same portion of the ventral horn, axially as well as cross-sectionally. In all such experiments the effect of the antidromic volley has been inhibitory. The inhibition has a characteristic time course which is shown in Fig. 5. In the experiment reproduced in this figure the caudal cord was transected and all the dorsal roots on the right side were severed from L2 to the caudal transection. A stimulus applied to the right

![Diagram](image-url)
dorsal column at L4 produced a motoneuron discharge which was recorded in the nerve to the lateral head of the homolateral gastrocnemius. This discharge was inhibited by a preceding antidromic volley backfired into the motoneurons supplying the medial head of the gastrocnemius. As shown in Fig. 5, the inhibition reached a maximum when the testing impulses were set up shortly after the antidromic volley. The response deficit then gradually declined and disappeared as the shock interval approached 50 msec. Similar results were obtained from experiments with the various branches to the hamstring muscles, with branches to the quadriceps, and with branches to the sartorius (Table 1).

Of particular interest is the fact that the inhibition of a tested discharge by an antidromic volley in a related group of motoneurons appears when the antidromic volley reaches the cord as late as simultaneously with the testing impulses which fire the motoneurons after a single synaptic delay. The illustrative data of Fig. 6 are taken from an experiment in which the conditioning volley and the tested motor discharge occupied the two principal branches to the quadriceps. The caudal cord was transected and all homolateral dorsal roots as far cephalad as L1 were severed intradurally. The conditioning curve is shown in Fig. 6A. It is clear from Fig. 6B, which shows the data on which the point x of 6A is based, that the testing response was definitely inhibited when the conditioning shock preceded the testing stimulus by only 0.7 msec. Oscillograms taken with the stimuli in this temporal relation appear in Fig. 6C. The sequence of potential changes evoked by the conditioning antidromic volley and recorded with a needle electrode within the ventral horn is shown in the upper record. The middle record shows the unconditioned testing response, and the lower record the testing response inhibited by an antidromic volley set up 0.7 msec. before the testing impulses.

More significant than the shock interval is the relationship between the time of arrival of the antidromic volley at the cord and the period of the central latency for the tested motor discharge. Most of the motoneurons of the quadriceps group are located in L5 and L6 (Sherrington, 1892). The upper record of Fig. 6C shows that impulses of the antidromic volley reached the ventral horn of these segments 0.8–0.9 msec. after delivery of the conditioning shock. Direct electrical stimulation of the motoneurons at L5–L6 produced an m wave (Lorente de Nó, 1939, p. 409) at the recording electrodes after 0.8 msec. Therefore, 0.8 msec. was approximately the time taken for the tested motor impulses to travel from the cord to the recording electrodes. Thus the second arrows in the lower two records of Fig. 6C indicate the time at which these impulses left the cord. Records from the dorsum of the cord show that the testing impulses arrived at L5–L6, 0.2–0.3 msec. after the testing shock, as indicated by the first arrows on the lower records of Fig. 6C. The central latency of the testing reflex in these segments is given by the interval between the arrows—ca. 0.9 msec., or the duration of a single synaptic delay (Lorente de Nó, 1938; Renshaw, 1940). It is apparent from these conduction times that when the conditioning shock preceded the test-
Fig. 6. Conditioning of motor discharges into part of the crural nerve by antidromic volleys in other branches to the quadriceps. Cat under light anesthesia (Nembutal). The caudal cord had been transected, and all sacral and lumbar dorsal roots on the tested side were cut. The tested "reflexes" were initiated by stimulation of the dorsal columns at L4. 6A, the conditioning curve, ordinates and abscissae as in Fig. 5. The arrow indicates the time at which the conditioning and testing volleys reached the ventral horn simultaneously. 6B, the data on which point x of 6A is based. Note the definite inhibition superposed upon a slow rhythmic variation of the discharge. 6C, oscillograms identified on the figure. The abscissae are on the same scale as those of 6A. The conditioning and testing shocks have the temporal relations of point x of 6A. 6D, a diagram to show all the possible neurons and synaptic connections which might have been concerned in the production of the response deficit at point x of 6A.

ing stimulus by 0.6–0.7 msec., the fastest antidromic impulses and the testing impulses in the primary neurons reached the region of the quadriceps motoneuron pool at the same time. This time is indicated by the arrow in Fig.
6A. Inhibition (point x) was already pronounced when the testing impulses arrived very slightly after the conditioning volley. Thus inhibition was first induced when an antidromic volley arrived approximately simultaneously with the testing impulses which fired the tested motoneurons after a single synaptic delay. When the antidromic impulses arrived a little earlier, they produced a greater inhibition; but when they arrived later, no facilitation was apparent.

Conditioning of the discharges of some motoneurons by antidromic volleys in other motoneurons also occurs when the conditioning and tested motor nerves are not branches to the same muscle or muscle group (Table 1). Although it is apparently necessary that the two pools of motoneurons occupy the same segmental levels of the spinal cord, their cell bodies need not lie in the same portion of the ventral horn. The effects for most pairs of nerves are relatively small. Also, as never occurs when the two motoneuron groups occupy the same place in the ventral horn, facilitation is sometimes observed.

In Fig. 7 are shown the conditioning curves obtained in one illustrative experiment in which the antidromic volleys and tested motor discharges occupied the motoneurons of the tibial, peroneal and hamstring nerves. The segmental distribution of the motoneurons of each of these three nerves was determined at the end of the experiment by measuring the size of the motor volleys set up in each nerve by stimulation of the various lumbar and sacral ventral roots. The results are tabulated in Fig. 7D. The comparative similarity in the axial distribution of the motoneurons supplying the three nerves stands in contrast with the cross-sectional segregation of the three groups of cell bodies (Fig. 7E, after Marinesco). In confirmation of Forbes et al. (1933), an antidromic volley in the peroneal nerve did not significantly condition reflex discharges into the tibial nerve (Fig. 7A). However, an antidromic volley in the tibial did affect subsequent reflex discharges into the axons of the peroneal nerve. It produced a prolonged period of facilitation of the two-neuron arc discharge to an afferent volley (Fig. 7A, also 8). The facilitation appeared only when the tested impulses followed the conditioning volley by several milliseconds. Initially slight inhibition, which was more apparent in other experiments, occurred. An antidromic volley in the hamstring nerve inhibited the motor discharge into the peroneal, and vice versa (Fig. 7B). An antidromic volley in the tibial likewise inhibited motor discharges into the hamstring nerve, but in the reverse relation the initial inhibition gave way to prolonged facilitation (Fig. 7C).

The conditioning effect of an antidromic volley in one group of motoneurons is not dependent upon the origin of the impulses that serve to excite the tested motoneurons. A shock applied to the ventral columns excites fibers which produce, after a single synaptic delay, a discharge of motoneurons located 2 to 3 cm. caudal of the stimulating electrodes (Lloyd, 1941a). The collaterals which the ventral column fibers send to the ventral horn are oriented, on the average, in the direction opposite to the reflexo-
FIG. 7. The interaction between motoneuron discharges and antidromic volleys in the tibial, peroneal, and hamstring nerves of a cat under light anesthesia (Nembutal). The sacral cord had been transected, and all dorsal roots as far cephalad as L3 were severed on the tested side. The motoneuron discharges ("reflexes") were evoked by stimulation of the dorsal columns at L4 with one or two shocks. A, antidromic volley in the tibial nerve conditioning the motor discharge into the peroneal (○); vice versa (▲). B, antidromic volley in the hamstring nerve conditioning the discharge in the peroneal (○); vice versa (▲). C, antidromic volley in the hamstring nerve conditioning the discharge into the tibial (○); vice versa (▲). D, the segmental distribution of the motoneurons of the tibial, peroneal, and hamstring nerves of this preparation. E, the positions of the motoneuron pools of the tibial, peroneal, and hamstring nerves in the cross-section of the ventral horn (after Marinesco). The cell bodies of the peroneal motoneurons show retrograde degeneration.
motor collaterals of the primary afferent neurons. Yet the conditioning effects of an antidromic volley in one group of motoneurons upon the discharges of another group are the same whether the tested motoneurons are excited by ventral column volleys or by impulses in primary afferent neurons. The records of Fig. 8 demonstrate this fact for both inhibitory and facilitatory effects.

![Fig. 8. The conditioning by antidromic volleys of reflexes initiated by testing impulses in primary afferent fibers (a, b) and in ventral column fibers (c, d). Column I, same experiment as Fig. 6. Conditioning volleys and tested motor discharges in two nerves to the quadriceps. The tested discharges are inhibited. Column II, conditioning antidromic volleys in the tibial nerve; tested discharges in the peroneal. The tested discharges are facilitated. From an experiment on a cat under light anesthesia (Nembutal). The cord was transected at L1 and in the caudal region; all dorsal roots on the tested side of the isolated lumbar and sacral segments were severed. The conditioning produced by the antidromic volleys is similar, whether the tested discharges are set up by ventral column activity or impulses in primary afferent neurons.](http://jn.physiology.org/)

The magnitude of the conditioning effect, although not its qualitative nature, is dependent upon the size of the tested motor discharge. As Tables 2 and 3 show, the larger the tested discharge, the less is the fractional inhibition or facilitation produced by a fixed conditioning volley. This is because the actual number of motoneurons removed from or added to the motor discharge by the conditioning volley increases relatively little. The affected motoneurons may be assumed to be those stimulated approximately at threshold by the testing volley.
Table 2. Motor discharge in nerve to tibialis anticus, set up by stimulation of dorsal column at L5 and conditioned by a preceding antidromic volley in nerve to gastrocnemius. Dorsal roots cut. IV-13-40

<table>
<thead>
<tr>
<th>Relative size of tested 2-neuron arc discharge into nerve to tibialis anticus</th>
<th>Size of conditioned discharge</th>
<th>Size of unconditioned discharge</th>
<th>Per cent facilitation</th>
<th>Relative number of facilitated motoneurons</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>126</td>
<td>105</td>
<td>26</td>
<td>1.0</td>
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<tr>
<td>6</td>
<td></td>
<td></td>
<td>5</td>
<td>1.15</td>
</tr>
</tbody>
</table>

Table 3. Motor discharge in nerve to semitendinosus, set up by stimulation of the dorsal column at L6 and conditioned by an antidromic volley in a branch to the biceps; stimulus interval, 5.5 msec. Dorsal roots cut. IV-16-40

<table>
<thead>
<tr>
<th>Relative size of the tested 2 neuron arc discharge into nerve to semitendinosus</th>
<th>Size of conditioned discharge</th>
<th>Size of unconditioned discharge</th>
<th>Per cent inhibition</th>
<th>Relative number of inhibited motoneurons</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64</td>
<td>36</td>
<td>36</td>
<td>1.0</td>
</tr>
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<td>2</td>
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<td>20</td>
<td>20</td>
<td>1.1</td>
</tr>
<tr>
<td>4</td>
<td>88</td>
<td>12</td>
<td>12</td>
<td>1.3</td>
</tr>
<tr>
<td>20</td>
<td>96</td>
<td>4</td>
<td>4</td>
<td>2.2</td>
</tr>
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</table>

**DISCUSSION**

It is most unlikely that antidromic conditioning volleys in the present experiments significantly altered the testing volleys in fibers of either the primary afferent neurons or the ventral columns before the impulses reached the ventral horn. For a large safety factor is associated with the propagation of impulses in axons (Hodgkin, 1937; Tasaki, 1939); and, except in the ventral horn, activity of the conditioning motoneurons produces very little current flow which might polarize axons and block conduction. Similarly, there is no evidence that impulses once initiated in the tested motor axons are blocked by antidromic volleys in other axons (page 174). An antidromic volley produces its conditioning effect either by altering the excitability of neurons to synaptic stimulation or by altering the stimuli delivered to postsynaptic elements by the testing impulses in axonal terminations.

It has been shown that activity in one group of motoneurons can condition the activity of another group (Fig. 4 to 8). The initial effect is a response deficit, which is present when the conditioning volley reaches the ventral horn at the beginning of the synaptic delay at the tested motoneurons (point x of Fig. 6A). Available for the explanation of these facts are: (i) the arrival of conditioning impulses at the terminal knobs of recurrent collaterals, and (ii) other effects not primarily dependent upon synaptic associations. The present data do not permit a complete resolution of the mechanisms for the conditioning. Some delimitation, however, is possible for the mechanism of the initial inhibitory effect. Inhibition such as that at
point \( x \) of Fig. 6A cannot be explained by the sequelae of detonator excitation associated with the arrival of conditioning impulses at the terminals of recurrent collaterals, as shown in hypothetical form at 4 and 5 of Fig. 6D, because detonator effects at these terminals would be expected to sum with those of testing impulses arriving simultaneously at endings 2 and 3 to produce facilitation. It is, therefore, necessary to conclude that the well-known detonator (excitatory) process associated with the arrival of impulses at synapses is not the only mechanism by which an active neuron can affect other nerve cells. This conclusion is corroborated by recent observations on inhibition in which the conditioning volleys occupy primary afferent fibers (Lloyd, 1941b).

The role played by the recurrent collaterals of spinal motoneurons is not known. Hence any explanation for the antidromic conditioning (inhibition, or as the case may be, facilitation) which involves the collaterals is purely speculative. Of the other possible mechanisms there comes to mind most prominently the polarizing action of electrical currents. Currents are in fact set up in the ventral horn by the activity of the conditioning motoneurons (Fig. 6C), and it is known that flow of current through the cord alters the size of testing two-neuron arc discharges (Renshaw, 1940). The only question is whether the currents set up in the cord by the conditioning motoneurons are large enough to produce the effects observed.

For conditioning to occur, the conditioning and the tested motoneurons must lie at the same segmental levels of the cord. A variety of curves is found when the two groups of motoneurons occupy the same axial position but different parts of the cross-section of the ventral horn (cf. Fig. 7). It is obvious that the observed inhibition and facilitation, whatever their basis, must depend upon details of the anatomical substratum in the ventral horn which are as yet unknown. The conditioning effects are not obviously related to reciprocal innervation (cf. Table 1).

The reduction of the tested discharges of motoneurons by their active neighbors in the same pool (Fig. 5) has approximately the same time course as the subnormality of the activated motoneurons to synaptic stimulation (Eccles, 1931; Gasser, 1939; Lorente de Nó, 1939). Thus the firing of some motoneurons in any nucleus produces a decrease in the responsiveness of most or all the motoneurons in the pool. Thereby the susceptibilities of the motoneurons to firing by premotor neurons would be synchronized.

The effects produced by a centripetal volley in a mixed nerve upon reflex discharges into the motor axons of other nerves have commonly been assumed to be referable entirely to impulses in primary afferent axons. The present findings emphasize that effects produced by the antidromic impulses in the motor axons cannot be disregarded.

**Summary**

An antidromic volley in a group of motoneurons produces a small centrifugal discharge from the spinal cord into some of the motor axons which
carry the antidromic impulses. No centrifugal impulses appear in the axons of other motoneurons. The centrifugal impulses appear to be repetitive discharges set up at some central portion of the motoneurons, rather than reflex discharges synaptically excited through recurrent collaterals.

Antidromic volleys do, however, condition synaptically excited discharges of other motor cells. Inhibition typically occurs if the tested and the conditioning motor nerves are branches to the same muscle or muscle group. The response deficit then reaches its maximum when the conditioning antidromic volley arrives at the cord 2 to 4 msec. before the tested motor discharges are set up. The amount of inhibition then gradually declines. It disappears when the antidromic volley precedes the tested discharge by ca. 50 msec.

A particularly significant feature of the inhibition is its early onset. A response deficit is present if the antidromic volley reaches the ventral horn simultaneously with the testing impulses which fire the tested motoneurons after a single synaptic delay. This finding cannot be explained on the assumption that the only effect which an active neuron exerts upon other neurons is the detonator excitation produced by the arrival of impulses at synapses.

Conditioning also occurs when the antidromic volleys and the tested motor impulses occupy the nerves to different muscles or muscle groups. Both facilitation and inhibition have been observed. Facilitation usually follows a brief initial period of inhibition. Maximal facilitation is attained when the conditioning antidromic volley precedes the tested discharge by ca. 25–30 msec. It then declines and disappears only when the interval between conditioning and tested volleys exceeds 100 msec.

REFERENCES


