# EFFECT OF DORSAL ROOT REFLEXES ON IMPULSES ENTERING THE SPINAL CORD

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In response to repetitive stimulation of a dorsal root with relatively short intervals between stimuli (10-17 msec) a decrease in the amplitude of the second antidromic action potential of the series recorded from the divided dorsal root $L_7$ and peripheral nerves was observed. This decrease may be the result of refractoriness and after-changes in excitability in the fibers of the dorsal column after passage of the dorsal root reflex (DRR) discharges along them. The time interval for which a decrease in amplitude of the antidromic response was observed corresponds to the duration of the DRR.

Gotch and Horsley in 1891 first described an antidromic discharge of impulses in the dorsal roots connected with the entry of afferent impulses into the same or adjacent roots. This phenomenon, known as the dorsal root reflex (DRR), has been studied in detail by Matthews [7] and by other workers [1-6, 8-10]. In the modern view the cause of the DRR is depolarization of terminals of primary afferent fibers (DPAF) due to the action of special neuron systems [12]. During natural activity because of the asynchronous entry of afferent impulses into the spinal cord the DPAF hardly reaches the level required for action potential generation [1], but nevertheless the DRR must be taken into consideration in neurophysiological experiments and, in particular during the analysis of phenomena connected with DPAF and presynaptic inhibition.

This paper describes the results of experiments confirming this point of view.

## EXPERIMENTAL METHOD

Experiments were carried out on anesthetized cats (hexobarbital, 70 mg/kg intraperitoneally). Laminectomy was performed in the thoracic and lumbosacral regions of the spinal cord. The sural and popliteal nerves, the dorsal root (DR) of $L_7$ and a strip of the posterior column (PC) in the inferior thoracic region down to $L_1$ were dissected. Changes in excitability of the terminals of the primary afferent fibers were determined by Wall's method [11]. Thin bipolar electrodes were inserted into the spinal cord for a distance of 1.2 mm in the region $L_7$. Repetitive stimuli were applied to PC or to the terminals of the primary afferents. The frequency of stimulation varied from 10 to 100/sec, and in this case, the interval between stimuli at the maximal frequency was commensurate with the duration of the dorsal root reflex (DRR). The animal's body temperature was maintained at 37-38°C. Antidromic action potentials were recorded from the screen of a type S1-16 oscilloscope after preliminary amplification if recordings were taken from the divided DR of $L_7$ or from peripheral nerves. The strength of the stimulating current was such as to evoke submaximal antidromic responses (not exceeding 50-60% of maximal).

## EXPERIMENTAL RESULTS

During repetitive stimulation of an isolated strip of PC, series of antidromic action potentials were recorded in the DR of $L_7$ (Fig. 1A). These action potentials had the following characteristics: a) antidromic potentials were accompanied by dorsal root reflex discharge which were completely inhibited in response

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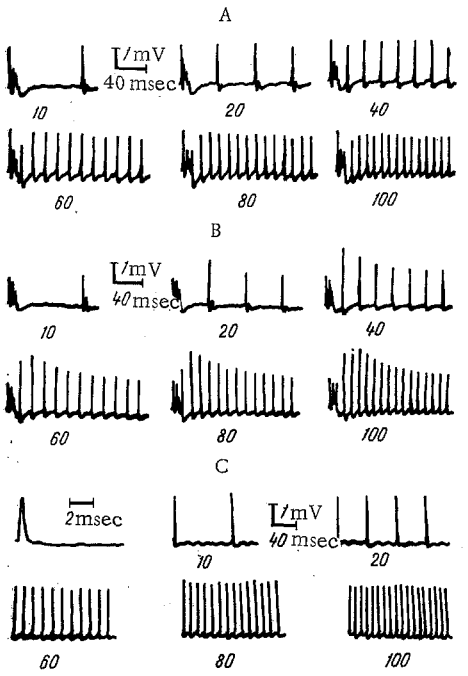


Fig. 1. Antidromic responses evoked by repetitive stimulation of isolated posterior column (A, C) and of terminals of primary afferents in region $L_7$ (B) and recorded from dorsal root of $L_7$. Numbers beneath records denote frequency of stimulation (in $sec^{-1}$).

II, starting from intervals of 50 msec between stimuli, i.e., at a frequency of 20/sec; b) with relatively short intervals between stimuli (from 10-17 msec) a marked decrease in amplitude of the second antidromic action potential was observed and the amplitudes of the subsequent potentials were equal in amplitude to the first potential.

A similar picture also was observed in recordings from the peripheral nerves (sural, popliteal) after division of the lumbar and sacral ventral roots.

Records illustrating changes in antidromic action potentials evoked by repetitive stimulation of terminals of the primary afferent fibers in region $L_7$ are shown for comparison in Fig. 1B. In this case, the amplitude of the antidromic potentials evoked by repetitive stimulation increased to reach a maximum at the 40th-50th millisecond, after which it decreased and became stationary at a level a little higher than that of the first potential of the repetitive series. The envelope of the antidromic potentials characterizes changes in excitability and, consequently, the degree of depolarization of the stimulated terminals of the primary afferent fibers. During stimulation of terminals of the primary afferent fibers the possibility of direct excitation of terminals of the axons of the depolarizing neurons forming axo-axonal synapses cannot be ruled out. However, judging by the character of the observed changes, the strength of the current applied, which was above threshold for terminals of the afferent fibers, was insufficient to evoke excitation of endings of axons of the depolarizing neurons and the observed DPAF took place through the participation of those same neuron systems which are activated by stimulation of the afferent nerves (Fig. 2).

The principal problem in the group of experiments being examined is that of the mechanisms of inhibition of the second antidromic response in the series of spikes evoked by stimulation of PC fibers. Under these conditions of stimulation the changes in the antidromic action potential were unconnected with polarization changes in the excitability of the afferent fibers under the stimulating electrodes, as is observed in the case of application of test stimuli to terminals of the primary afferents. In the experiments under review it was a question of changes in the amplitude of the "en passant" action potentials spreading along afferent fibers. Two hypotheses can be put forward regarding the mechanism of this inhibition.

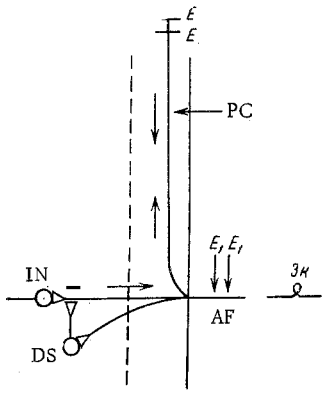


Fig. 2. Hypothetical scheme explaining decrease in amplitude of antidromic action potentials evoked by repetitive stimulation of fibers of the posterior column and recorded from dorsal root $L_7$. AF) Afferent fiber and its collaterals; PC) afferent fiber of posterior column; IN) interneuron; DS) depolarizing system; EE) stimulating electrodes; $E_1E_1$) recording electrodes. Arrows show spread of DRR impulses from presynaptic endings to fibers of posterior column and opposite flow of antidromic action potentials.

First, the decrease in amplitude of the "en passant" antidromic response may arise through the spread of depolarization from the presynaptic endings of collaterals to the afferent PC fibers forming these collaterals and, in particular, to the DR fibers from which the antidromic action potentials were recorded. In that case, the observed decrease in amplitude of the antidromic action potentials could be regarded as the result of a decrease in the membrane potential of the nerve fibers under the recording electrode. However, this hypothesis cannot explain the results of the present experiments because the temporal characteristics of inhibition of the antidromic responses evoked by stimulation of PC did not correspond to the temporal characteristics of primary afferent depolarization.

Second, inhibition of the "en passant" antidromic action potential may be the result of the multiple discharge (DRR) arising in the depolarized presynaptic endings and spreading along the PC fibers. The observations actually showed that inhibition of the second antidromic potential of the repetitive series evoked by stimulation of the PC fibers is observed only when the first stimulus evokes not only an antidromic response, but also a DRR (Fig. 1A). In some experiments in which DRR discharges were absent (evidently because of the irregular character of the DRR) no decrease in amplitude of the second antidromic action potential was observed (Fig. 1C). The results described explain the observed decrease in amplitude of the "en passant" antidromic action potential as the result of refractoriness and of after-changes in excitability in the PC fibers after passage of a series of impulses arising in the depolarized presynaptic endings (DRR) along them. The time interval at which the decrease in amplitude of the "en passant" antidromic response was observed corresponded to the duration of the DRR.

A scheme illustrating the mechanism of inhibition of the "en passant" antidromic responses is shown in Fig. 2.

It seems to the writer that the action of the antidromic volley of impulses arising as a result of depolarization of the presynaptic terminals described above, i.e., the action of the DRR on the volley of impulses evoked in the opposite direction in the same afferent fibers by repetitive stimulation is of not only theoretical, but also practical importance. It must be remembered that a phenomenon of this type may also arise when conditioning and testing stimuli are applied to different afferent nerves, i.e., whenever a conditioning volley evokes depolarization in the terminals of the primary afferent fibers sufficient to generate a DRR. The inhibition of the reflex responses observed at relatively short time intervals between conditioning and testing stimuli (commensurate with the duration of the DRR) may be the result not only of weakening of the transsynaptic transmission of excitation due to depolarization of the presynaptic terminals, but also to blocking of impulses in the afferent fibers by the opposite flow of antidromic action potentials.

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