# THE NATURE OF SPINAL SHOCK
# COMMUNICATION IV. ATONY OF THE MOTOR NEURON IN SPINAL SHOCK

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In research carried out in 1945 [1], we regarded spinal shock as a syndrome of atony of the motor neuron , although we had no direct proof of the localization of the developing atony.

In our third communication we showed that the sensory links of the reflex arcs of the spinal cord play no part in the development of spinal shock. The quantitative and qualitiative characteristics of the volleys of impulses produced by standard stimulation of the receptors of the skin were not altered by cordotomy over the whole extent of the afferent conducting pathways of the spinal cord.

In the present investigation the problem of the site of development of atony in the spinal reflex arc was investigated by the study of myotatic reflexes caused by stretching of a muscle or by electrical stimulation of the posterior root of the spinal cord.

## METHOD

In the first series of experiments we recorded the action potentials of the gastrocnemius muscle of the frog (Rana chensinensis) during reflex contractions of this muscle in response to its stretching. Ths hip and knee joints of the limb under study were firmly fixed, all the muscles with the exception of that being studied were denervated, and the skin of the limb was removed. Dorsiflexion of the foot was carried out by a standard method using pulleys, with a constant angle of stretching and with equal velocity. The electromyogram was recorded with the film moving at a speed of 25 mm/sec, with paired electrodes, 30 $\mu$ in diameter, inserted into the muscle and with no insulation in cross section.

In a second series of experiments, in order to eliminate the monosynaptic impulses, the tracing was made at a higher speed of movement of the photographic film (250 mm/sec). The potentials were recorded from the central end of the anterior root of the IXth nerve during stimulation of the posterior of the same nerve with closure of a constant current. All the anterior roots innervating the limb under study were divided.

The potentials were recorded on a two-channel cathod oscillograph.

## RESULTS

Conventionally, in the first series of experiments the electromyogram reproduced the myotatic reflex of its own particular level. During stretching of the gastrocnemius muscle (Fig. 1,A,B,C), in the initial state the volley of monosynaptic and polysynaptic impulses was interrupted by a period of silence. This reflex pause period [4], of differing duration and depth, is characterized by anelectrontonic hyperpolarizational inhibition, arising in the motor neuron in response to recurrent impulses [3,5,6].

In the experiments in which a state of increased excitation followed immediately after cordotomy (Fig. 1,D), the period of reflex silence disappeared, and in its place there was a continuous, intensive volley, with considerable after-action.

In the other experiments in which spinal shock was developing, we were able to discern three types of reaction: complete disappearance of monosynaptic and polysynaptic impulses (Fig. 1,A), disappearance of the initial impulses with the preservation of a short volley on the cessation of stretching (Fig. 1,B), and finally, the development of reflex anelectrotonic inhibition of the period of silence (Fig. 1,C), becoming more intensive later on (the third segment of the electromyogram) until the retention of one single initial monosynaptic potential.

After 4-6 minutes, when spinal shock had passed over, the initial electromyogram was restored (Fig. 1,B). During the change over from spinal shock to the state of increased excitation, the electromyogram showed greater activity than was observed before cordotomy (third cut on Fig. 1, A, and fourth cut on Fig. 1, C).

The disappearance of the potentials of the myotatic reflex showed convincingly that the genesis of spinal shock was predetermined by the motor neuron cell, and individual experiments testifying to the intensification of the reflex period of silence (Fig. 1,C) suggest the hyperpolarization (anelectrotonic state) of the motor neuron in the period of spinal shock.

In the second series of experiments we examined the fate of the initial monosynaptic potentials in the

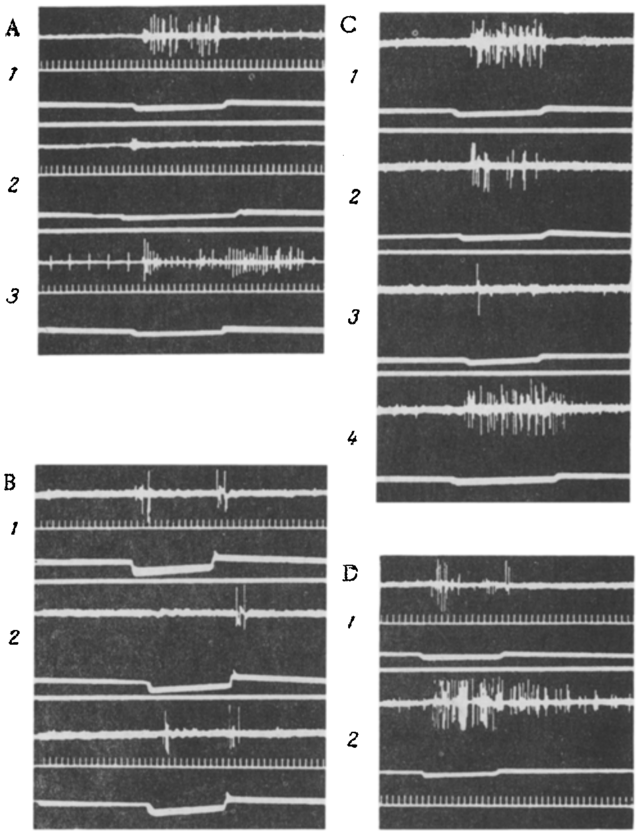

Fig. 1. Electromyogram of the gastrocnemius muscle. (First series of experiments: No. 30—A; 28—B, 16—C and 31—D). 1) Before cordotomy; 2) immediately after cordotomy; 3 and 4) a few minutes after cordotomy. In each cut, the top line represents the oscillogram and the bottom line the stimulation marker (stretching the muscle). Time marker—50 millisec.

anterior root in response to electrical stimulation posterior root of the same segment (the IXth) on the same side. In experiment No. 30(Fig. 2,A), after cordotomy, when spinal shock was developing, the monosynaptic potentials disappeared completely and were reestablished after 6 minutes, with a considerable shortening of the duration of central inhibition. In experiments Nos. 36(Fig. 2,B) and 2(Fig. 2,C) the spinal shock was characterized by a considerable decrease in the monosynaptic potential, which rose considerably in the period of increased excitation of the spinal reflexes and appeared sooner in time than in the original state before cordotomy.

Both series of experiments described in the present communication show that the link responsible for the development of spinal shock is the motor neuron cell, which as the final common spinal path, becomes passively atonic when deprived of supraspinal impulses. The state of atony of the motor neuron cell is shown by the fact that its polarization is increased – an anelectrotonic state develops, which diminishes it preparedness for non-incremental excitation.

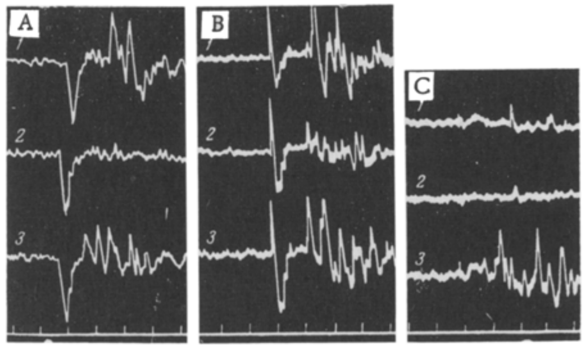

Fig. 2. Potentials of the anterior root. (Experiments of the second series: No. 30—A; 36—B and 2—C). 1) Before cordotomy; 2) immediately after cordotomy; 3) a few minutes after cordotomy. Stimulation of the posterior root is indicated by an artefact on the oscillogram. Time marker—10 millisec.


## SUMMARY

The condition of motorneurons was studied during spinal shock in experiments on frogs. The myotatic reflex of the gastrocnemius muscle was recorded by electromyograms in the first series of experiments. The disappearance of the initial impulses or an increase of the anelectrotonic "silence period" was noted in the monosynaptic and polysynaptic impulses in spinal shock. In the second series of experiments, the authors recorded the potentials of the IXth anterior root during electric stimulation of the corresponding posterior root. The disappearance or reduction of the intial monosynaptic potential following chordotomy, and its restoration after recovery from spinal shock, pointed to the fact that the motorneurons cells are the site of the spinal shock development. The anelectrotonic condition of the motorneuron during the shock period is an atonia provoked by the exclusion of the supraspinal impulses.


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