

Excitability of motor neurones in spinal shock in man¹

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The transient reflex depression caudal to a lesion of the spinal cord, 'spinal shock', can be due to depressed excitability of motor neurones and interneurons at the segmental level or to a decrease of fusimotor activity rendering the muscle spindles less sensitive to stretch or to both. In cats Hunt, Meltzer, and Landau (1963) reported a transient depression of fusimotor activity in that the afferent nerve volley to a brief stretch was diminished and associated with a depression of the reflex response. In the spinalized monkey depression of the reflex was not accompanied by a decrease in the afferent volley. In man early in spinal shock the ankle jerk was severely depressed or absent whereas the electrically evoked H-reflex was unaffected or slightly reduced, suggesting that fusimotor activity was depressed (Weaver, Landau, and Higgins, 1963). While the maximal H-reflex is a measure of the excitability of motor neurones to single afferent volleys, other abnormalities of excitability might be revealed by conditioning the H-reflex with a preceding stimulus. We have studied the recovery of the test H-reflex in patients one to four days after a lesion of the spinal cord when deep reflexes were absent or severely depressed. The examination was repeated both when the reflexes had returned and when there was spasticity.

MATERIAL AND METHODS

Ten patients (three males and seven females) aged 15 to 41 years with lesions of the cervical or upper thoracic spinal cord were examined. Eight suffered from a compression or dislocation fracture of the spine, one had haematomyelia, and one transverse myelitis. The examination was performed one to four days, one to four weeks, and, in five patients, two to three months after the lesion. Findings from 15 male subjects aged 19 to 48 years without signs or symptoms of neuromuscular disease are given for comparison.

ANKLE JERKS Maximal ankle jerks were elicited manually with strong taps of a reflex hammer. The closing

¹For preliminary reports see Diamantopoulos and Zander Olsen (1965 a and b).

of a contact in the head of the hammer released the sweep of the oscilloscope.

STIMULATION AND RECORDING The stimuli were rectangular pulses, 0.7 msec. in duration, applied to the posterior tibial nerve in the popliteal fossa with needle electrodes placed along the nerve. The action potentials were led off from the triceps surae muscle with subcutaneous needle electrodes, one placed over the belly and one over the distal tendon of the muscle.

PROCEDURE The patients were in a supine position with the thighs supported to flex the knees 20°. The ankle joints were kept flexed at 90° by sandbags. The potentials associated with maximal ankle jerks, the H-reflexes and the direct motor responses were recorded at intervals of 10 seconds (Fig. 1). The peak-to-peak amplitude given for each patient was the mean of at least five recordings. The electrical responses to maximal ankle jerks and the maximal H-reflexes were expressed as percentages of the direct motor responses to supra-maximal stimulation of the nerve (J/M and H/M ratio). The recovery of the H-reflex was studied by paired stimuli of equal strength, in one series evoking maximal H-reflexes, in another H-reflexes just above threshold. The interval between the conditioning and the test stimuli was increased in 24 steps from 1 msec. to 4 sec. A more detailed description of the procedure has already been given (Olsen and Diamantopoulos, 1967).

CLINICAL FINDINGS

One to four days after the lesion of the spinal cord the patients had flaccid paralysis of the lower extremities and absent or weak deep reflexes (Table I). The blood pressure was normal. After one to four weeks there were still no voluntary movements but in most patients the deep reflexes had returned and three had spasticity. After two to three months the patients were spastic with brisk reflexes and flexor spasms. Three were still paralytic.

ELECTROPHYSIOLOGICAL FINDINGS

ONE TO FOUR DAYS AFTER ONSET OF SPINAL SHOCK The maximal ankle jerks were absent or severely depressed and the J/M ratio was reduced (Table II).

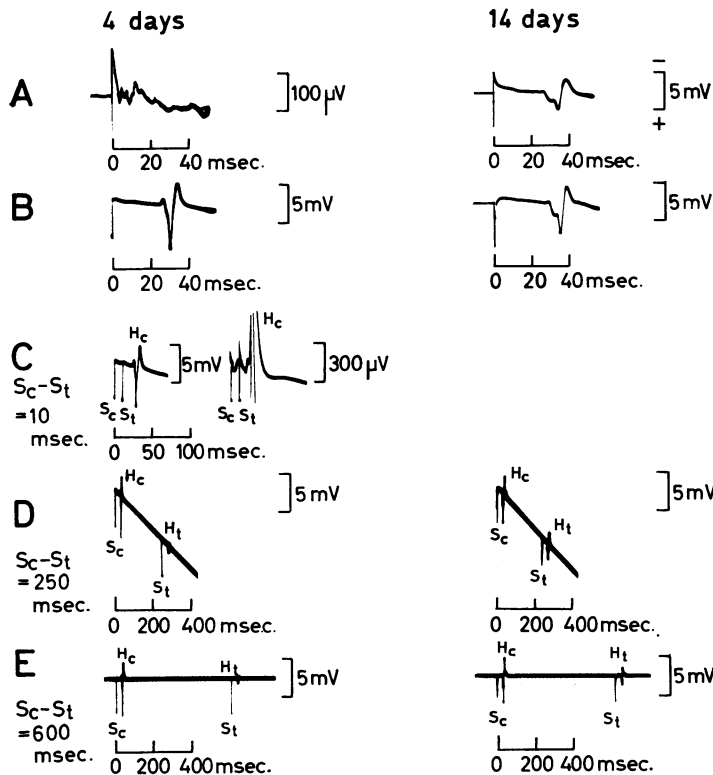


FIG. 1. Action potentials accompanying maximal ankle jerks and H-reflexes recorded from the calf muscles of a 15-year-old female (K.B.N.), four and 14 days after the onset of spinal shock. After four days the ankle jerk was absent (A) and the maximal H-reflex had normal amplitude (B). C-E: Recovery of the test H-reflex (H_t) 10, 250, and 600 msec. after a conditioning H-reflex (H_c). S_c : conditioning and S_t : test stimulus. H_t was more diminished after four days than after 14 days.

The single maximal H-reflexes and the H/M ratio were on the average below normal although in six patients still within the normal range. After conditioning with a preceding electrical stimulus the test H-reflexes were severely depressed both with maximal H-reflexes and with H-reflexes just above threshold (Figs. 2 and 3). The early facilitation (interval A) was absent or reduced. The test reflexes reappeared at about the same time interval after the conditioning stimulus as in normals and the rise in amplitude was slower (interval B). The

peak in amplitude of the test reflex which normally occurred at an interval of 200 to 300 msec. between the stimuli ('second facilitation') was absent except in three patients in whom there was a small second facilitation when reflexes were just above threshold. At greater time intervals the test reflexes were still depressed until 4 sec. after the conditioning stimulus (interval C).

ONE TO FOUR WEEKS AFTER ONSET OF SPINAL SHOCK
The dissociation between ankle jerks and H-reflexes

TABLE I
CLINICAL FINDINGS IN THE LOWER EXTREMITIES DURING AND AFTER SPINAL SHOCK

Time after Lesion	Total No. of Patients	No. of Patients with		Flaccidity	Spasticity		Knee Jerks			Ankle Jerks			Plantar Response			Sensibility ¹		
		Paralysis	Paresis		Severe	Slight	Absent	Weak	Normal	Brisk	Absent	Weak	Brisk	Absent	Flexion	Extension	Anaesthesia	Hypoaesthesia
		Severe	Slight															
One to 4 days	9	9	0	0	9	0	0	9	0	0	0	6	3	0	1	7	1	9
One to 4 weeks	10	10	0	0	7	0	3	3	6	1	0	2	4	4	1	1	8	7
Two to 3 months	5	3	1	1	0	4	1	0	0	1	4	0	0	5	0	0	5	4

¹Touch, pinprick, and temperature

TABLE II

AMPLITUDE OF MUSCLE ACTION POTENTIALS¹ ACCOMPANYING MAXIMAL ANKLE JERKS AND H-REFLEXES DURING AND AFTER SPINAL SHOCK

Time after Lesion	No. of Patients	Maximal Ankle Jerks (mV)		J/M Ratio ²		Maximal H-reflex (mV)		H/M Ratio ²	
		Mean	Range	Mean	Range	Mean	Range	Mean	Range
One to 4 days	9	1.0±0.2	0.5-0	4.1±2.9	0-18	9.4±1.8	2.7-17	38±9.1	13-62
One to 4 weeks	10	3.5±1.0	0.3-9.0	18±6.0	3.0-38	8.8±1.5	2.5-20	54±11.7	16-91
Two to 3 months	5	9.1±1.9	4.3-14	69 ³	53-84	8.8±2.1	4.8-15	69 ³	69-69
Normals	15	10.5±1.1	5.0-18	37±3.2	18-60	14.1	1.3-7.0	51±3.8	19-90

¹Mean values with standard errors.

²Amplitude of the action potentials recorded above the calf muscles accompanying the maximal ankle jerk or a maximal H-reflex as a percentage of the response to a supramaximal stimulation of the nerve.

³Two patients.

was less pronounced than shortly after the lesion, the maximal ankle jerks and the J/M ratios being less depressed. The maximal H-reflexes were on the average still below normal but the H/M ratio was normalized because the direct maximal motor response was diminished (Table II).

The reduction of the direct electrical responses was due to denervation from compression of nerve fibres in paralytic patients as evidenced by the occurrence of profuse fibrillations (Landau and Clare, 1959) and to the inactivity atrophy of the muscle fibres (Fudema, Fizzelle, and Nelson, 1961). The

recovery of the H-reflexes to conditioning stimuli was normal (Fig. 2 and 3) except in one patient in whom the second facilitation of the test reflex (interval B) was still absent with H-reflexes just above threshold.

TWO TO THREE MONTHS AFTER ONSET OF SPINAL SHOCK The action potentials accompanying maximal ankle jerks had increased further: the J/M and H/M ratios were determined in only two patients and were increased in both (Table II). The recovery of the H-reflexes to conditioning stimuli was faster

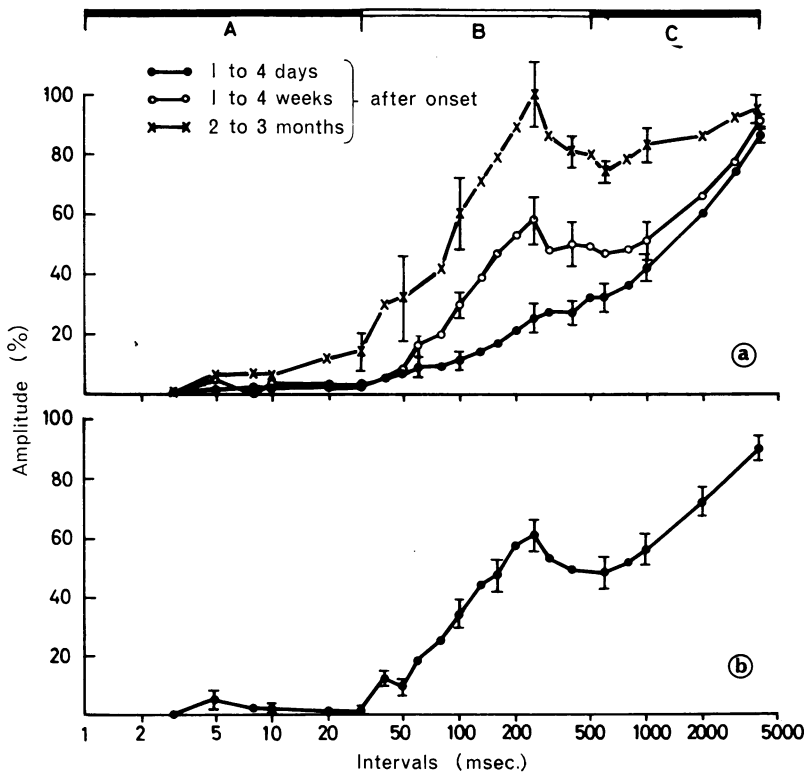


FIG. 2. Recovery of the maximal test H-reflex after a conditioning reflex. (a) Nine patients one to four days, 10, one to four weeks, and five, two to three months after the onset of spinal shock; (b) Fifteen normals. Abscissa: Time interval between conditioning and test stimuli, logarithmic scale. Ordinate: Amplitude of the test H-reflexes as percentages of their conditioning reflexes. The vertical bars denote the mean errors. Interval A: Phase of early facilitation in normals. Interval B: Phase of the second return of the test reflex in normals. Interval C: Phase of late depression of the test reflex.

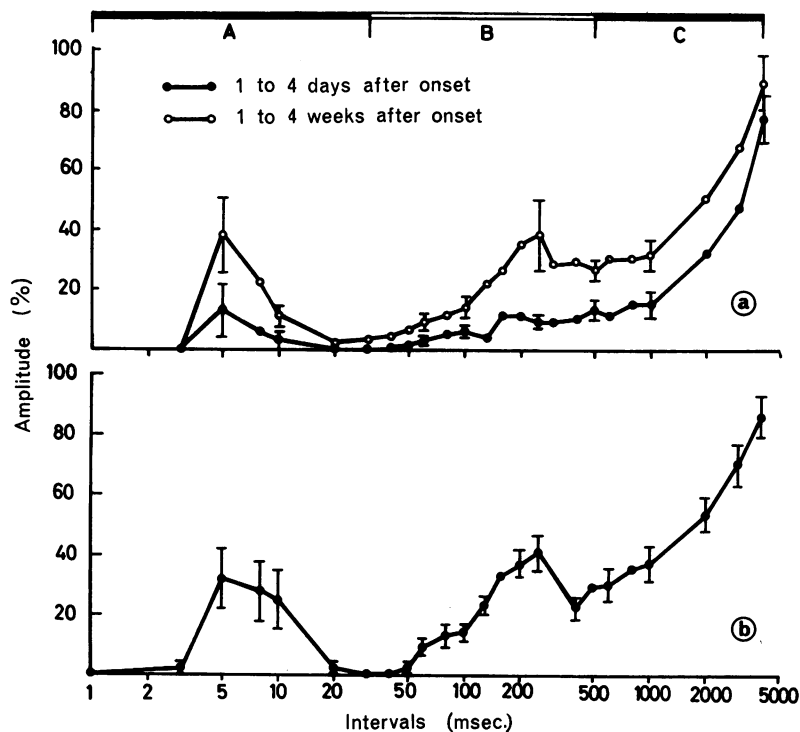


FIG. 3. Recovery of the test H-reflex after a conditioning reflex.

The reflexes were elicited by stimuli of equal strength just above threshold. (a) Nine patients; (b) thirteen normals. Coordinates as in Figure 2.

than in normals (Fig. 2) because the test reflexes occurred at a shorter interval between the stimuli and the amplitudes recovered faster. The second facilitation was more pronounced (interval B) and the following depression less pronounced (interval C).

DISCUSSION

In acute spinal shock in man maximal H-reflexes were normal or slightly reduced when tendon reflexes were absent or severely reduced (Weaver *et al.*, 1963); this finding was confirmed in our patients. It suggests that the reduction of the tendon jerk was mainly due to a depression of fusimotor activity rendering the muscle spindles less sensitive to stretch. The recovery of the H-reflex after a conditioning reflex revealed that the central excitability was also impaired. One to four days after the spinal lesion the recovery of the test H-reflexes was severely depressed. This depression indicates a diminished excitability of interneurons and of the fusimotor system rather than of motor neurones because the excitability to a single stimulus was almost normal. The second facilitation of the test response during the recovery after a conditioning stimulus was absent in most patients one to

four days after the spinal lesion. This is supposed to be caused by the depression of muscle spindle activity because the second facilitation has been considered to be due to activation of the muscle spindles stretched when the muscle lengthens again after the shortening during the conditioning reflex (Paillard, 1955).

One to four weeks after the onset of the spinal shock the maximal ankle jerks had somewhat increased but were still below normal. In view of the normal single and test H-reflexes this indicates that the impairment of fusimotor activity persists longer than the depression of central excitability.

Two to three months after the onset of the spinal shock when deep reflexes were hyperactive, the action potentials accompanying maximal ankle jerks had about the same amplitudes as the single maximal H-reflexes and there was no longer an indication of fusimotor depression. At this stage the recovery of the test H-reflex after a conditioning stimulus occurred faster than in normals, indicating increased central excitability.

SUMMARY

In 10 patients with spinal shock the action poten-

tials accompanying the maximal ankle jerks, the single maximal H-reflex, and the recovery of the test H-reflex after a conditioning stimulus were examined.

One to four days after the onset of spinal shock the maximal ankle jerks were absent or severely depressed whereas single maximal H-reflexes were normal or only slightly reduced. This suggests normal motor neurone excitability to a single afferent volley and the reduction of the ankle jerk is considered to be due to decreased fusimotor activity. On the other hand, the recovery of the test H-reflex after a conditioning stimulus was severely depressed. This indicates a depressed excitability of interneurons in addition to the reduced fusimotor activity.

One to four weeks after the onset of spinal shock maximal ankle jerks were still somewhat depressed, indicating a persisting impairment of fusimotor activity. The recovery of the test H-reflex to a conditioning stimulus was normal.

Two to three months after the onset of the spinal shock the ankle jerks were hyperactive and the action potentials accompanying them had increased to

the level of maximal H-reflexes. The recovery of the test H-reflex after a conditioning stimulus was faster than in normals indicating increased central excitability.

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