PROLONGATION OF DISTAL MOTOR LATENCIES — CHARACTERISTIC FINDING IN GUILLAIN-BARRE SYNDROME

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SYNOPSIS

In an electrophysiological study of 45 patients with Gullian-Barre Syndrome, seven showed mark prolongation of distal motor latencies of all nerves tested. While marked slowing of conduction velocities is seen in many demyelinating peripheral neuropathies, such findings of widespread prolongation of motor latencies with values exceeding twice normal range is, in our experience very suggestive of Guillain-Barre Syndrome. The changes are reversible and do not reflect rate of recovery. Segmental demyelination at sites prone to compression is the current accepted explanation.

INTRODUCTION

Electrophysiological abnormalities in Guillain-Barre Syndrome or acute polyradiculopathy (1) have been well discussed by Bannister et al (2), Walsh et al (3) and other workers (4, 5, 6, 7, 8). However, there has been little elaboration or emphasis on the diagnostic value of delayed distal motor latencies. Isch et al (6) had commented on the diagnostic usefulness of prolonged distal or terminal latencies. In our clinical and electrophysiological study of 45 consecutive cases of Guillain-Barre Syndrome seen in the Singapore Hospital, 7 cases showed this finding.

METHOD & RESULTS

Guillain-Barre Syndrome was diagnosed according to the criteria discussed by Osler and Sidell (1). All patients had the right median (motor, mixed, sensory), ulnar (motor, mixed, sensory), lateral popliteal, posterior tibial and sural nerves studied. The results of the terminal motor latencies of these 7 patients are given in Table 1 together with the mean normal values as determined in our laboratory by Tong et al (9). The median and ulnar nerves were stimulated at a fixed distance of 13 cm from the base of the respective digits. The lateral popliteal nerve was stimulated distally at the level of the lateral malleolus between the tibialis anterior and extensor hallucis longus. The posterior tibial nerve was stimulated at the level of the tip of the medial malleolus. Surface recording electrodes were used. Six patients had repeat nerve conduction studies after full clinical recovery and those latencies have improved to near normal.

TABLE 1
TERMINAL MOTOR LATENCIES IN SEVEN CASES OF ACUTE POLYRADICULOPATHY

No.	Age/Sex	Terminal Motor Latencies in msec				Comment on Repeat Studies & Time (Months later)
		Median	Ulnar	Lateral Popliteal	Posterior Tibial	
1	26/M	18.5	18.4	29.7	32.5	(–) Not done
2	64/M	20.8	15.8	Absent	Absent	(24) normal
3	29/F	20.5	12.6	21.1	14.2	(18) normal
4	35/M	7.3	8.4	8.7	15.7	(12) normal
5	52/M	7.2	6.0	8.0	11.5	(12) normal
6	55/M	6.2	Not done	12.2	11.2	(4) normal
7	62/M	24.7	7.8	18.2	17.3	(6) near normal
	Normal mean ± SD	3.4 ± 0.5	3.0 ± 0.4	4.3 ± 0.8	5.1 ± 0.8	

DISCUSSION

This finding is not common, occurring in about only 15% of our cases. However, when such marked delay of motor terminal latencies does occur in several nerves simultaneously in a patient who was previously well, it is very suggestive of Guillain-Barre Syndrome in our experience. Prolongation of distal motor latencies does occur in several types of neuropathies, e.g. diabetic, Charcot-Marie-Tooth disease, but these values do not revert to normal and progress with time. Marked slowing of nerve conduction velocities was also noted in 4 of these cases, but in itself is not a characteristic finding since it is also observed in chronic demyelinating neuropathies like Charcot-Marie-Tooth disease, Dejerine Sotta's and Refsum's disease. In compression syndromes e.g. the median nerve in carpal tunnel syndrome, terminal motor latency seldom exceeds twice the normal value.

Lambert and Mulder (5) have attributed this phenomenon to segmental demyelination at sites prone to mechanical compression resulting in conduction block (10). While this is a possibility, it does not satisfactorily explain delay of the distal latency of the lateral popliteal nerves which are not prone to compression at the ankles. We feel that joint mobility is the more important factor in triggering or worsening the demyelinating process.

Although no data is provided here, our experience is that such delay in distal latencies does not reflect on rate of recovery.

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