Original Article

Nerve Conduction study in Unilateral Limb Weakness Patients- A Retrospective Descriptive Analysis

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Abstract

Background: Limb weakness is most common complaint in neurology patients. Nerve conduction studies (NCS) help in differentiating between various classes of neuropathies, with the help of various NCS parameters. Hence, this research was planned to evaluate nerve conduction parameters in unilateral limb weakness patients. **Material and Methods:** This was a retrospective, descriptive research carried out in 78 unilateral limb weakness patients. We studied distal motor latency (DML), compound muscle action potential amplitude (CMAP), motor nerve conduction velocity (MNCV), F minimal latency, sensory nerve action potential amplitude (SNAP) and sensory nerve conduction velocity (SNCV) in unilateral median, ulnar, radial, suprascapular, axillary, musculocutaneous, tibial, peroneal and sural nerves using RMS-Aleron. **Results:** Neurophysiology data of total 78 cases was obtained. Out of those cases, 41 were upper limb and 30 were lower limb weakness cases. Among upper limb cases, motor axonal neuropathy was found in median nerve (22%), ulnar nerve (31.7%) and radial nerve (41.46%). Sensory axonal neuropathy was found in median (29.26%), ulnar and radial nerves (39% each). Motor demyelinating neuropathy was found in median nerve (9.75%), in ulnar nerve (12.19%) and radial nerve (24.39%). Sensory demyelinating neuropathy was found in median and ulnar (26.82% each), and radial nerves (39%).Among lower limb cases, motor axonal neuropathy was found in tibial in 20%, and peroneal in 60% cases. Sensory sural axonal neuropathy was found in 36.67%. Motor demyelinating neuropathy was found in tibial nerve in 10%, and peroneal involvement in 36.67% cases. Sensory sural demyelinating neuropathy was found in 20% cases. **Conclusion:** Axonal neuropathy was observed more frequently than demyelinating pattern in both motor and sensory nerve conduction parameters among patients with unilateral upper or lower limb weakness. Combined unilateral upper and lower limb weakness were assumed to be upper motor neuron lesion.

Keywords: Electroneurography, F waves recording, Peripheral Neuropathies, Mononeuropathy, Radial Palsy.

Received: 19 July 2026 **Revised: 01 August 2026** **Accepted: 13 August 2026** **Published: 16 September 2026**

Introduction

Limb weakness is most common complaint in neurology patients which may or may not be accompanied with sensory symptoms i.e. tingling and numbness. Lesions in upper motor neuron (UMN), lower motor neuron (LMN), the neuromuscular junction or the muscle itself are the aetiologies causing such weakness. Diseases affecting muscles are generally proximal and symmetrical.[1]

Patients are usually referred to neurophysiology laboratory to rule out neurological cause of weakness. Nerve conduction studies (NCS) and electromyography are essential to find out cause of limb weakness.[2]

NCS helps in differentiating between various classes of neuropathies, if its demyelinating, or axonal or mixed type.[1] These modalities are simple and reliable tests of peripheral nerve function, however are time consuming and demand a skillful, experienced elicitation.[2,3] Very few studies are published, that focus on use of electrodiagnostic methods in limb weakness patients. Hence, with this background, we decided to pool the NCS data of unilateral limb weakness patients from previously available records for analysis to identify type of neuropathy in these patients.

Materials and Methods

This was a retrospective, descriptive research carried out in 78 unilateral limb weakness patients, of any age and either sex, which were selected from previously available data at neurophysiology OPD of our tertiary care teaching hospital. The data belonged to patients reporting to neurophysiology OPD between Jan 2018-Dec 2020. Data of patients who reported to neurophysiology OPD with complaints of one sided limb weakness of either upper limb or lower limb or both limbs, was included in this study. Those cases, in which incomplete data was recorded, were excluded. All the available study parameters of affected (weak) limb of these patients were tabulated in Microsoft excel sheet.

As study data was pooled from neurophysiology OPD, and all

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DOI:
10.21276/amit.2026.v13.i3.1045

How to cite this article: Motewar SS, Asia AA, Atram PG. Nerve Conduction study in Unilateral Limb Weakness Patients- A Retrospective Descriptive Analysis. Acta Med Int. 2026;13(3):210-214.

the clinical assessment was done in Orthopaedics/Medicine departments, the clinical data like grading of weakness and sensory loss was not included in present study.

Our study parameters for motor nerve conduction were distal motor latency (DML), compound muscle action potential amplitude (CMAP), motor nerve conduction velocity (MNCV) and F-minimal latency in affected limb, focusing on median, ulnar, radial, tibial and peroneal nerves. For suprascapular, axillary and musculocutaneous nerves, only CMAP was considered. Sensory nerve action potential amplitude (SNAP) and sensory nerve conduction velocity (SNCV) were done to assess sensory conduction in median, ulnar, radial and sural nerves. All above parameters were recorded using RMS-Aleron machine for neurophysiology.

The study was started after approval letter (Out.No./SVNGMC/IEC Boi.Res./179/2025) from institutional ethics committee (for biomedical research) with due consideration to protect the confidentiality of personal, clinical and laboratory data.

Working definitions:

- Axonal neuropathy- Decreased CMAP (motor) or SNAP (sensory) accompanied with normal DML and NCV is considered as axonal neuropathy.
- Demyelinating neuropathy- Increased DML (or onset latency in case of sensory nerve) and Decreased NCV, accompanied with normal CMAP or SNAP is considered as demyelinating neuropathy.

Methodology: At our set up, for motor nerve conduction studies, a common setting was used with slight variation for each nerve study. The gain was set at 2–5 mV/division. The active electrode was placed over the centre of the respective muscle belly, and the reference electrode was positioned distally over/near the tendon. The stimulation pulse duration was fixed at 100 μs, and the current intensity ranging from 50-100 mA.

The gain was set at 10-20 μV/division for eliciting sensory component of median, ulnar, radial and sural nerves. Ring electrodes, placed over distribution area of particular sensory nerve were used to record antidromic sensory waveforms. The active electrode was placed within 8 cm of stimulator. The distance between active and reference electrode was kept at 3–4 cm. A current intensity of 5–30 mA with a pulse duration of 100μs was used, as sensory fibre have low threshold stimulation.[4]

Median motor nerve conduction parameters: Abductor pollicis brevis muscle was used as a location for keeping recording electrodes. Two waves for two sites of stimulation i.e. middle of the wrist and antecubital fossa were recorded. However, for our research, we considered only distal (wrist) stimulation wave parameters viz DML and CMAP. MNCV was calculated using distance between middle of the wrist and antecubital fossa. The distance measured was entered in the RMS-Aleron software to calculate MNCV. F minimal latency was recorded by stimulating at wrist and recording 8-10 waves on a rastered trace.[4]

Median sensory nerve conduction parameters: Two ring electrodes were placed over index finger at a distance of 3-4cm and stimulation was given at middle of wrist. The current was slowly increased from 0-50mA till, a clear waveform, to record onset latency and SNAP. The distance between active electrode and stimulator was measured. The number was entered in the software to get SNCV of the median sensory nerve.[4]

Ulnar motor nerve conduction parameters: Abductor digiti minimi muscle was used to place recording electrodes. Stimulation was given at two points i.e. medial wrist and below elbow. MNCV was calculated, using distance between elbow point and wrist point.[4,5]

Ulnar sensory conduction parameters: Ring electrodes were placed over little finger and stimulation performed at medial wrist, till we obtain a clear sensory waveform. We measured the distance between active electrode and stimulator. The number was entered in the software to get SNCV of the ulnar sensory nerve.[4]

Radial motor nerve conduction parameters: Active electrode was placed over extensor indicis proprius whereas reference electrode was placed over styloid process of wrist. Ground was placed in between the two electrodes. The stimulation was given at two sites, Viz. forearm and spiral groove. MNCV was calculated using two stimulation points.[4]

Radial sensory nerve conduction parameters: Disc active electrode was placed over superficial radial nerve and reference electrode was placed 3-4 cm distally over the thumb. Stimulation was given over the distal-mid radius until, a clear sensory waveform. The distance between active electrode and stimulator was measured. The number was entered in the software to get SNCV of the radial sensory nerve.[4]

Suprascapular, axillary, musculocutaneous motor nerve conduction parameters-Active electrodes were placed on supraspinatus, deltoid and centre of the biceps muscle belly, respectively for above nerves. Reference electrodes were placed 3-4 cm distally on these muscles respectively. Stimulation was given at Erb's point. Duration of current, for stimulation, was set at 500μSec. CMAPs were recorded for these nerves in affected limb as well as in normal limb for the purpose of comparison.[4]

Patients presenting with lower limb weakness underwent motor NCS of the tibial and peroneal nerves. Late responses, including F-wave minimal latencies, were also recorded. Patients with complaints of proximal lower limb weakness had additionally undergone motor nerve conduction studies of the femoral nerve. Sensory nerve conduction studies were performed on the sural nerve of the affected lower limb.

Tibial, Peroneal and Femoral motor nerve conduction: Active electrodes were placed on abductor hallucis brevis for tibial nerve, extensor digitorum brevis for peroneal nerve and rectus femoris for femoral nerve. Reference electrodes were placed at metatarso-phalangeal joint of the great toe, metatarso-phalangeal joint of the little toe, over a bony prominence at the knee, respectively. Stimulation was performed at above and posterior to the medial malleolus and in popliteal fossa for tibial nerve motor conduction. Stimulation was given at anterior ankle and below the fibular head for peroneal motor nerve conduction study. For femoral motor nerve conduction, stimulation was given at middle of the inguinal area, slightly lateral to the femoral pulse.[4]

Data Analysis: Previously available NCS data of 78 unilateral limb weakness patients were filled in pre-designed Case record forms (CRF) and Microsoft excel sheet. Using various filters, cases were separated in three groups viz. unilateral upper limb, unilateral lower limb and unilateral upper with lower limb weakness. Further motor and sensory nerve conduction parameters were tabulated separately. Percentages were calculated for each axonal and demyelinating neuropathy.

Results

This research was started with the aim to study nerve conduction parameters viz. CMAP, DML, MNCV, SNAP, SNCV, and F-minimal latency in unilateral limb weakness patients. Of the total 78 patients, 41 had upper limb weakness, 30 had lower limb weakness and seven had both upper with lower limb weakness.

[Table 1] shows that, out of 41 patients, four (9.75%) patients showed increased/absent DML with decreased/absent MNCV in median nerve, suggestive of motor demyelinating neuropathy. Nine (22%) patients showed decreased/absent CMAP with absent F minimal latency suggesting motor axonal neuropathy.

In ulnar nerve, five (12.19%) patients showed demyelinating and 13 (31.7%) patients showed axonal neuropathy. Radial nerve conduction study showed demyelinating neuropathy in 10 (24.39%) patients and axonal neuropathy in 17 (41.46%) patients. Only CMAP was assessed in patients of proximal nerve involvement and it showed axonal neuropathy in seven (17.07%) patients in axillary nerve, six (14.63%) patients in musculocutaneous nerve and four (9.75%) patients in suprascapular nerve.

Out of 41 patients, 12 (29.26%) patients showed decreased/absent SNAP suggesting sensory axonal neuropathy in median nerve and 11 (26.82%) patients showed decreased/ absent SNCV suggesting sensory demyelinating neuropathy. Sixteen (39%) patients demonstrated sensory axonal neuropathy in ulnar nerve and 11(26.82%) patients showed sensory demyelinating neuropathy. Radial nerve conduction study showed axonal and demyelinating neuropathy in 16 (39%) patients. [Table 2]

[Table 3] shows that, out of 30 patients of lower limb weakness, three (10%) patients showed motor demyelination in tibial nerve and six (20%) patients showed motor axonal neuropathy. Peroneal motor demyelinating neuropathy was observed in 11 (36.67%) patients while peroneal motor axonal neuropathy was found in 18 (60%) patients. As like upper limb cases, only CMAP was assessed in proximal nerve involvement patient. Three (10%) patients presented with femoral motor axonal neuropathy.

Out of 30 unilateral lower limb weakness patients, we found demyelinating sural neuropathy in six (20%) patients and sensory axonal sural neuropathy in 11 (36.67%) patients. [Table 4]

[Table 5] suggests that, out of seven patients of unilateral upper with lower limb weakness, only one patient each was found with median, tibial and peroneal nerve motor mixed (demyelinating along with axonal) neuropathy. This observation of only one patient with deranged NCS parameters can be a chance finding. Other reason of weakness in both upper and lower limbs on same side suggests hemiparesis or UMN (upper motor neuron) lesion. Hence NCS parameters are normal in such patients. One patient was found with axillary, musculocutaneous and one with femoral motor axonal neuropathy.

Out of seven patients, only one patient presented with median and ulnar sensory axonal neuropathy and three patients showed sural axonal neuropathy. Two patients showed demyelinating sural neuropathy. [Table 6]

Table 1: Unilateral upper limb weakness motor conduction (n= 41)

	DML				CMAP				MNCV				F minimal latency		
	Reference Values[4,5] (ms)	Normal	Absent	Increased	Reference Values[4,5] (mv)	Normal	Absent	Decreased	Reference Values[4,5] (m/s)	Normal	Absent	Decreased	Reference Values[4,5] (ms)	Absent	Increased
Median	3.77 ±0.40	37	4	0	8.10 ±2.62	28	4	9	58.52±3.76	32	4	5	<32	9	2
Ulnar	2.59±0.40	36	3	2	8.51± 2.03	28	3	10	61.45±5.73	32	3	6	<32	10	3
Radial	2.4± 0.4	16	7	3	13.0± 8.2	9	7	10	62.0± 5.1	9	7	10	NA	NA	NA
Axillary	NA	NA	NA	NA	7 to 13	6	1	6	NA	NA	NA	NA	NA	NA	NA
Musculocutaneous	NA														
Suprascapular															

Table 2: Unilateral upper limb weakness sensory conduction (n= 41)

	SNAP				SNCV			
	Reference Values4,5 (µV)	Normal	Absent	Decreased	Reference Values4,5 (m/s)	Normal	Absent	Decreased
Median	8.91± 4.48	29	4	8	45.45± 9.40	30	4	7
Ulnar	5.54± 2.37	25	5	11	54.17± 6.10	29	5	7
Radial (n=26)	4.0 to 13.0	10	6	10	58.0 to 71.0	10	6	10

Table 3: Unilateral lower limb weakness motor conduction (n=30)

	DML				CMAP				MNCV				F minimal latency			
	Reference Values[2,4,5] (ms)	Normal	Absent	Increased	Reference Values[2,4,5] (mv)	Normal	Absent	Decreased	Reference Values[2,4,5] (m/s)	Normal	Absent	Decreased	Reference Values[2,4,5] (ms)	Normal	Absent	Increased
Tibial	3.11±0.55	27	2	1	6.09±1.05	24	2	4	46.64±4.45	24	2	4	<56	23	4	3
Peroneal	3.38±0.41	19	10	1	6.18±0.64	12	10	8	47.61±3.98	17	10	3	<56	13	16	1
Femoral (n=11)	NA	NA	NA	NA	0 to 3	8	1	2	NA	NA	NA	NA	NA	NA	NA	NA

NA- Not available

Table 4: Unilateral lower limb weakness sensory conduction (n=30)

	SNAP (Sural midcalf amplitude)				SNCV			
	Reference Values[2,4,5] (µV)	Normal	Absent	Decreased	Reference Values[2,4,5] (m/s)	Normal	Absent	Decreased
Sural	16.58± 2.34	19	3	8	59.68± 2.42	24	3	3

Table 5: Unilateral upper and lower limb weakness motor conduction (n= 7)

	DML				CMAP				MNCV				F minimal latency			
	Reference Values [2,4,5] (ms)	Normal	Absent	Increased	Reference Values [2,4,5] (mv)	Normal	Absent	Decreased	Reference Values [2,4,5] (m/s)	Normal	Absent	Decreased	Reference Values [2,4,5] (ms)	Normal	Absent	Increased
Median	3.77±0.40	6	0	1	8.10 ±2.62	6	0	1	58.52± 3.76	6	0	1	<32	6	1	0
Ulnar	2.59± 0.40	7	0	0	8.51 ± 2.03	7	0	0	61.45± 5.73	7	0	0	<32	7	0	0
Tibial	3.11±0.55	7	0	0	6.09 ± 1.05	6	0	1	46.64± 4.45	6	0	1	<56	6	1	0
Peroneal	6.18± 0.64	6	0	1	6.18 ± 0.64	6	0	1	47.61± 3.98	6	0	1	<56	6	1	0
Axillary (n=2)	NA	NA	NA	NA	7 to 13	1	0	1	NA	NA	NA	NA	NA	NA	NA	NA
Musculocutaneous (n=2)	NA	NA	NA	NA	2.5 to 12.7	1	0	1	NA	NA	NA	NA	NA	NA	NA	NA
Femoral (n=2)	NA	NA	NA	NA	0 to 3	1	0	1	NA	NA	NA	NA	NA	NA	NA	NA

NA- Not available

Table 6: Unilateral upper and lower limb weakness sensory conduction (n= 7)

	SNAP				SNCV			
	Reference Values[4,5] (µV)	Normal	Absent	Decreased	Reference Values[4,5] (m/s)	Normal	Absent	Decreased
Median	8.91± 4.48	6	0	1	45.45± 9.40	6	1	0
Ulnar	5.54± 2.37	6	0	1	54.17± 6.10	6	1	0
Sural	16.58± 2.34	4	1	2	59.68± 2.42	5	1	1

Discussion

These findings suggest that majority of patients with limb weakness were having motor axonal neuropathy as compared to motor demyelinating neuropathy. This may be due to late presentation, secondary to delayed reporting. These patients report to neurophysiology OPD, with an intention to get handicap certificate, so that they can avail benefits of various

government schemes. In earlier stages, when the injury is purely demyelinating, they do not report to our OPD.

Jain S et al 2016, also found similar observations as like present research.[1] They performed NCS on 40 cases of bilateral lower limb weakness patients and found significant changes in study parameters in cases as compared to controls with inclination towards axonal neuropathy.[1] Primarily axonal polyneuropathies mainly affect nerve amplitudes i.e. SNAPs & CMAPs, whereas demyelination leads to prolonged latencies (DML) with reduced velocities (MNCV & SNCV).[1]

Nerve injury progresses in stagewise manner, depending upon the type and grade of injury. It advances as a part of orthograde & retrograde degeneration, which takes around 6 weeks.[6] At an early stage, there are chances of finding a nerve in demyelinating stages like neuropraxia, where we find decreased velocity (<75% of reference value) unequivocally with or without marked prolongation of distal latency.[4,5]

However, most of our cases present late in the stages of axonotmesis/complete nerve degeneration, where we get axonal injury in NCS parameters in the form of reduced amplitudes, slightly slowed velocity with mildly prolonged distal latency.[4,6] Amplitudes reflect the number of underlying live nerve axons, which is observed in the form of decreased CMAP & SNAP in a NCS.[4,5] Hence, irrespective of cause and type of injury, after a certain duration, we endup finding axonal loss injuries on NCS.

Limitations: As this was record-based retrospective research, we could not find the exact reasons of observations. Future studies of prospective design, involving detailed history taking, clinical examination with a larger sample size will help us to find the aetiology. Additions of EMG findings are also suggested for such future studies.

Conclusion

Among 78 patients presenting with unilateral limb weakness, NCS demonstrated diverse patterns of neuropathy. Motor axonal neuropathy was the most common finding among these patients, with abnormalities noted in CMAP and SNAP. In contrast, NCS were normal in patients with combined unilateral upper with lower limb weakness, suggesting an UMN lesion.

Conflict of interest- There is no conflict of interest to declare.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

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