

Nerve Conduction Studies In Lower Limb Weakness

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Abstract: **Introduction:** Nerve conduction studies (NCS) are an essential part of the work-up of peripheral neuropathies. NCS with EMG allows diagnostic classification, understanding and separation of different neuropathies. Symmetrical lower limb weakness of neurological origin often demands EDX study. Neuropathies may be mixed or motor or sensory. Further it may be either axonal or demyelinating. Aims and objectives: To assess nerve conduction studies in symmetrical lower limb weakness patients with peripheral neuropathies. To estimate prevalence of neuropathies in this cohort. Material methods: Forty cases and equal no of controls underwent NCV study. Tibial peroneal motor and sural sensory nerve conductions were done. Data was stored in excel sheet for analysis. Different NCV variables were compared between the groups. Unpaired t'test was used for comparison of variables. Level of significance was kept at p value <0.05. Results: It was observed that DML, CMAP/SNAP amplitude, and CV of study group were significantly different as compared to control group (p value <0.05). Neuropathy was present in 31 (77.5%) cases. 25 (80.65%) cases were axonal and 6 (19.35%) were demyelinating neuropathies. NCV was normal in 9 (22.5%) cases. Conclusion: NCV study proved an essential tool in diagnosis of neuropathies in lower limb weakness cases. Axonal neuropathies were more prevalent as compared to demyelinating one. Mixed neuropathies were more frequent than isolated motor or sensory neuropathies. Presence of conduction block suggests acquired demyelination. [Vijay v NJIRM 2016; 7(4): 56-60]

Key words: Nerve conduction study, conduction block, distal symmetrical neuropathy, limb weakness.

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Introduction: Weakness is a common presenting concern in neurology. It is often part of more systemic disorders, and is a common end result of many diseases, including rheumatologic disorders, endocrine, infectious and neoplastic disorders, not to mention psychiatric conditions. Occasionally, patients with other disorders of the motor system, such as rigidity or incoordination, may describe themselves as being weak because they have difficulties in performing motor acts. Patients are usually referred to neurophysiology laboratory to rule out neurological cause of weakness. Nerve conduction studies and electromyography successfully differentiates upper and lower motor neuron lesions. Further, it also delineates the level at which lesion is located in lower motor neuron.^{1,2}

True weakness can be caused by problems affecting upper motor neurons, lower motor neurons, the neuromuscular junction or the muscle itself. Muscle diseases are generally proximal and symmetrical. Bilaterally symmetrical paresis or paralysis of lower limbs is often referred to neurophysiology laboratory for evaluation. There are several etiologies like prolapsed inter-vertebral disc, cauda equine syndrome, amyotrophic lateral sclerosis, lumbosacral radiculoplexopathies, acute poliomyelitis, inflammatory demyelinating polyneuropathies, critical illness neuropathy and myopathy, neuromuscular

disorders, toxic neuropathies, etc that may cause bilaterally symmetrical weakness in lower limbs.^{3,4,5} Variety of EDX patterns are observed in these patients in nerve conduction studies.⁶

With this background nerve conduction studies were done in patients with bilaterally symmetrical weakness in lower limbs. Aim was to compare effect of a neurological cause for weakness on nerve conduction studies between cases and controls. Different EDX patterns were also observed in this category of patients.

Methods: The study was conducted during January 2013 to March 2016 in Clinical Neurophysiology Laboratory, Department of Physiology, GMERS medical college Gotri Vadodara. An approval from Institutional Human Ethics Committee was taken for conducting the research before start of this study. Forty cases were selected with supportive inclusion and exclusion criteria under supervision of consultant physician, orthopedic surgeon and pediatrician depending on outpatient or inpatient case of corresponding department. Inclusion criteria consisted of acute weakness of bilateral lower limbs with complete or partial sensory or motor loss. Irrespective of diagnosis like diabetes mellitus, chronic alcoholism, hypothyroidism, poliomyelitis, prolapsed inter-vertebral disc, and other causes of acute flaccid

paralysis/paresis; we selected all those cases of bilateral weakness in lower limbs with complete or partial motor/sensory loss. Cases with unilateral weakness were not included in study. Duration of weakness was from time of onset to three months. Weakness of more than 3 months was not included in the study. Patients with local injuries/lesions that may interfere with the electrophysiological study were excluded from the study. All cases were subjected to electrophysiological evaluation as described below. The control group included forty healthy subjects, mostly recruited from hospital staff and their relatives with no previous or current history of significant disease, in particular weakness in upper or lower extremity. They were selected on age and height basis so as to be matched with patient population after thorough physical and neurological examination by consultant physician. Electrophysiological studies similar to cases were performed and data was obtained for comparison. EMG was not done in any of the participants of control or study group. A written informed consent was taken from all the subjects screened under above inclusion and exclusion criteria who underwent the study. A detailed bilateral lower limb nerve conduction studies comprising of tibial and peroneal motor nerve conduction, Sural sensory nerve conduction were done.

Electrophysiological Study: Bilateral tibial and peroneal motor nerve conduction studies (MNCS) consisted of standard Belly-Tendon montage placement of active and reference electrodes. In tibial MNCs, active electrode was placed on Abductor Hallucis (AH) muscle and reference on its tendon at ball of great toe. In peroneal MNCS, active electrode was placed at Extensor Digitorum Brevis (EDB) muscle and reference on its tendon on dorsum of foot at fifth distal inter-phalangeal joint. Duration of stimulation strength was kept at 200ms, sweep speed at 5ms/D, sensitivity at 5mV/D and supramaximal strength was given with maximum strength not more than 100mA. Compound muscle action potentials (CMAP) were obtained and its features like distal motor latencies (DML), amplitude (Amp) and conduction velocity (CV) were recorded and stored in excel sheet for further analysis.

Bilateral Sural sensory conduction studies (SCS) consisted of antidromic recording of sensory nerve action potentials (SNAPs) by placing active and reference electrode 3 centimeters (cm) apart on skin

at ankle posteriorly to lateral malleolus. Sural nerve was stimulated at calf 15 cm away from active electrode. Settings for SCS were; duration-100ms, maximum strength 100uV, sweep speed- 2ms/D, sensitivity- 20uV/D and 20 consecutive stimuli were live averaged to obtain a Sural SNAP. Its features amplitude and CV were recorded and stored for further analysis. (Preston et al)

Statistical Analysis: We sought help of biostatistician for analysis of data. It was done using Graph Pad Prism online software designed by California based Graph Pad Prism Inc Ltd Company. Data was expressed as mean and standard deviation in all demographic and electrophysiological features. Comparison between cases and controls was done using unpaired t'test with level of statistical significance set at values less than 0.05.

Results: Forty cases (11 female, 29 male) with age range 32 to 60 years and equivalent controls (6 female, 34 male) with age range 15 to 70 years underwent electrophysiological evaluation for motor and sensory nerves in lower limbs. Mean and standard deviation values related with demographic profile are depicted in table no 1. Statistically significant difference was only observed in height parameter between the two groups (p value<0.05).

Table No. 1: Demographic profile of control and cases of population under study

Parameters	Control	Cases	P value
Age	44.35±8.95	45.875±15.4	0.5890
Height	166.25±3.90	160.22±6.22	1.547E-06*
Weight	59.6±8.4	59.125±14.20	0.855

(Note: * Suggests p value <0.05)

Usually, an increase in height is associated with decrease in the CV but comparison shows that trend is exactly opposite in control and cases as well. This nullifies possibility that differences in height feature had affected CV values in study population. Hence we can very well go ahead with nerve conduction studies (NCS) observations between control and cases in study.

Table No 2: Comparison of Motor conduction studies variables in control and cases of study population

Nerve	Side	Features	Control	Cases	P value
Tibial	Left	DML	3.54±0.74	4.44±1.2	0.000109*
		Amplitude	16.29±5.93	9.85±6.29	9.7285E-06*
		CV	47.54±3.68	40.17±6.34	1.15615E-08*
	Right	DML	3.517±0.61	4.32±1.16	0.000234*
		Amplitude	16.35±6.57	9.97±5.32	1.2081E-05*
		CV	48.602±4.08	41.0±6.49	2.69339E-08*
Peroneal	Left	DML	3.817±0.60	4.44±1.21	0.00533*
		Amplitude	8.257±2.46	5.72±3.86	0.00094*
		CV	51.35±3.64	43.23±9.83	6.4568E-06*
	Right	DML	3.81±0.38	4.43±1.19	0.00238*
		Amplitude	8.17±.72	4.47±3.69	3.36732E-06*
		CV	51.75±4.4	41.96±7.07	2.24911E-10*

(Note: * Suggests p value <0.05 hence statistically significant, DML= Distal motor latency, CV= Conduction velocity)

Table no 2 shows that comparison between almost all the features of motor conduction studies (DML, Amp and CV) among two groups is statistically significant i.e. p value <0.05. Thus in cases group latencies are prolonged, amplitude and conduction velocities are reduced significantly.

Table No 3: Comparison of Sural sensory conduction studies in control and cases of study population

Nerve	Side	Features	Control	Cases	P value
Sural	Left	Amplitude	21.89±14.4	14.17±5.86	0.00444*
		CV	51.16±5.11	49.09±7.5	0.2137
	Right	Amplitude	21.99±12.16	14.12±5.36	0.000896*
		CV	49.42±4.35	46.18±9.46	0.0718

(Note: * Suggests p value <0.05 hence statistically significant, CV= Conduction velocity)

Table no 3 shows that only sural nerve SNAP amplitude is grossly affected/ reduced whereas conduction velocity remains normal in cases as compared to

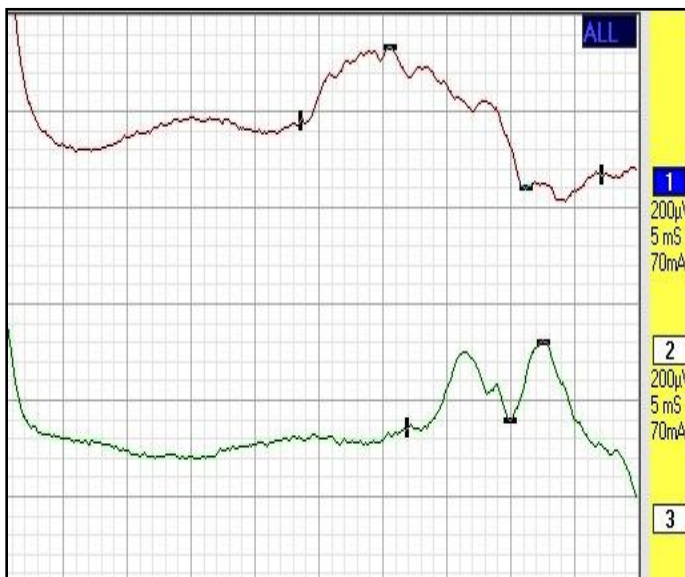
controls. This suggests that it's more often an axonal loss than demyelination in cases of bilateral lower limb weakness.

Discussion: In present study almost all the cases were reported to this laboratory within one week period and no final diagnosis were made at the time of referral except nine cases that were labeled with acute flaccid paralysis lower limbs with query cause Guillain-barre` syndrome (GBS). Therefore, in majority of cases electromyographer had no clue regarding etiology of presentation whether neurological or non neurological. Clinical examination however supported neurological involvement of lower limbs in the form of motor or sensory loss. Comparison of nerve conduction studies between cases and control as

depicted in table no 2 and 3 confirmed significant difference in the MNC (tibial and peroneal) parameters and SNAP amplitude of Sural SNC. Only parameter that did not show significant difference was SNC conduction velocity. Average values for peroneal CMAP amplitude were the only feature that showed marked reduction as compared to standard reference values in literature. Literature on bilateral symmetrical lower limb weakness shows that specific neurological issues were selected as cohort and then EDX study was done. We discuss few studies and its relevance to our findings.

Martel J et al studied Charcot-Marie-tooth disease patients with symmetrical weakness. Nerve conduction studies showed abnormal conduction velocities in these patients. In progressive lower limb weakness with sensory ataxia nerve conduction studies play useful role in diagnosis of this rare entity.⁷ Rubens O et al observed only prolongation of sensory and motor latencies with normal CV and amplitude in patients with peripheral neuropathy in chronic occupational lead exposure. There was no correlation observed between clinical biochemical and electrophysiological data.⁸ GBS also presents with lower limb weakness. It is more commonly proximal than distal muscle weakness. Al-Shekhlee A et al compared GBS and critical illness polyneuropathy (CIP) patients with new EDX criteria for AIDP, and found that amplitude reduction was more frequent phenomenon in CIP and conduction block (CB) in almost 10% nerves in AIDP cases.^{9, 10} CB were reported in present study too in 6 out of 40 cases (Figure1)

Figure No 1: Conduction block and temporal dispersion in a GBS patient.



Chen CC et al reported a case of hyperthyroidism that reported with lower limb weakness in proximal muscles. NCV study was normal and the case recovered after medications.¹¹ In sporadic adult-onset progressive muscular atrophy patients NCV abnormalities were seen in all variables.¹² Distal symmetrical neuropathy in lower limbs is more often seen in diabetes patients. Kakrani AL et al reported that tibial and Sural nerve involvement is more common in diabetic neuropathy patients.¹³ In present

study we observed involvement of peroneal nerve. Polyneuropathies associated with symmetrical weakness needs clinical and EDX evaluation. Routine nerve conduction studies like motor, sensory, late responses and EMG are very accurate to diagnose the lesion. Sometimes underlying subclinical lesion may also be detected. Conduction block (CB) is also a useful tool in EDX studies to diagnose focal neuropathies and demyelination.¹⁴

Primarily axonal polyneuropathies mainly affect sensory and Motor nerve amplitudes i.e. SNAPs and CMAPs whereas demyelination leads to prolonged sensory and motor latencies, reduced conduction velocities. Acquired demyelination can be differentiated from hereditary by presence of conduction blocks (CB) and temporal dispersion.¹⁵ In present study, all motor parameters and SNAP amplitude variable is affected. In general, it suggests presence of sensory motor axonal more than demyelinating polyneuropathies in symmetrical lower limb weakness patients. Case to case EDX evaluation reveals, mixed neuropathies in 27 cases and motor neuropathies in 4 cases. 9 cases showed normal or borderline NCV values hence labeled no neuropathy cases. Out of 31 cases with neuropathy, 25 cases were axonal and remaining 6 acquired demyelinating neuropathies. No case of hereditary demyelination or pure sensory neuropathy was reported from study group. Conclusion: Present study highlights the electrophysiological profile of 40 cases with lower limb weakness of neurological origin. Axonal neuropathies were most common in these cases as evaluated through EDX studies. Peroneal nerve CMAP was most frequent abnormality in lower limb weakness. NCV study proved an essential tool in diagnosis of neuropathies in lower limb weakness cases. Axonal neuropathies were more prevalent as compared to demyelinating one. Mixed neuropathies were more frequent than isolated motor or sensory neuropathies. Presence of conduction block suggests acquired demyelination.

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