Section: Physiology

Original Research Article

AN OBSERVATIONAL STUDY OF ALTERATIONS IN NERVE CONDUCTION STUDIES OF ACUTE SPINAL CORD INJURIES PATIENTS

Vijay Meena[1], Vishal Goel[2], Ajit Singh[3], Sushma Sood[4]

[1]*Orthopaedic consultant at District Hospital Hindaun City, District, Karauli, Rajasthan, India.*
[2]*Assistant Professor, Department of Physiology, Kalpana Chawla Government Medical college & Hospital, Karnal Haryana, India.*
[3]*Assistant Professor, Department of Pathology, Kalpana Chawla Government Medical College & Hospital, Karnal Haryana, India.*
[4]*Professor Emeritus, Department of physiology, NC Medical College & Hospital, Israna, Panipat, India.*

Received *: 20/10 /2023*
Received in revised form *: 22/11/2023*
Accepted *: 12/12/2023*

Corresponding Author:
Dr. Vishal Goel
Assistant Professor, Department of Physiology, Kalpana Chawla Government Medical College & Hospital, Karnal Haryana, India.
Email: drvishalgoel31@gmail.com.

DOI: 10.5530/ijmedph.2023.4.17

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2023; 13 (4); 77-81

ABSTRACT

Background: Spinal cord injury (SCI) is a devastating neurological state producing physical dependency, morbidity, psychological stress and financial burden. For the last 30 years, its global prevalence has increased from 236 to 1298 cases per million populations. **Aim and Objectives:** The objective of this study was to assess the electrodiagnostic changes in the lower limbs as measured by nerve conduction studies (NCSs) in lower limb in acute SCI patients.
Materials and Methods: An observational descriptive study was carried out on 35 patients of acute SCI patients who were between the age of 16 and 65 years by using RMS EMG EP Mark-II. Motor and sensory studies were performed on peroneal tibial, and sural nerve respectively. Parameters recorded were latency, amplitude and conduction velocity.
Results: Mean latency of right and left peroneal nerve increase in subsequent follow - up but this change is non- significant. Increment in mean conduction velocity and mean amplitude of peroneal nerve was statistically significant ($p=0.003$, $p=0.004$) for right and left respectively; $p<0.001$ for amplitude of peroneal nerve (right and left both). Mean latency and mean conduction velocity of tibial nerve (right and left) increase with subsequent follow up but this change were non-significant. Increment in mean amplitude of right and left tibial nerve was statistically highly significant ($p<0.001$, $p=0.03$ for right, left respectively). Mean latency of right and left sural nerve was increased in subsequent follow up visits but this change was not significant. Increment in mean conduction velocity and mean amplitude was statistically highly significant ($p<0.001$) for right and left both of sural nerve.
Conclusion: It is concluded from this study is that impact of SCI on latency, amplitude and conduction velocity of tibial, peroneal and sural nerve is very detrimental at onset of injury. But with subsequent follow up it has improved significantly, which can be related with recovery of lower limb.
Keywords: Nerve conduction studies, spinal cord injury, amplitude, latency, conduction velocity.

INTRODUCTION

Acute traumatic spinal cord injury (SCI) represents one of the most devastating injuries to afflict the human body. The injury has a high rate of prevalence in younger populations, creating physical, emotional, and economic burden on both the individual and society.[1]

A SCI refers to any injury to spinal cord that is caused by trauma instead of diseases. Depending on where the spinal cord and nerve roots are damaged, the symptoms can vary widely, from pain to paralysis to incontinence.[2]

These injuries are described as “incomplete” which can vary from having no effect on the patient to a “complete” injury which means a total loss of function. In a “complete” spinal injury, all function

below the injured area is lost. In an "incomplete" injury, some or all function below the injured area may be unaffected. If the patient has the ability to contract the anal sphincter voluntarily or feel a pin prick or touch around the anus, the injury is considered to be incomplete.[3]

In patients with spinal cord injury (SCI), clinical and electro physiologic parameters are useful in assessing the extent and level of SCI and also toward predicting functional recovery. Furthermore, they can be applied following SCI to assess the extent of spinal cord recovery and the relationship to the development of neurologic deficits. [4,5]

MATERIAL AND METHODS

This study was carried out on 35 patients of acute SCI presenting within 48 hours, 3 months and 6 months of injury to orthopaedics department of Pt. B. D. Sharma PGIMS, Rohtak during a period from June 2014 to November 2016. Detailed informative history of the patient was taken in a chronological order. General physical examination and neurological examination of the patient was performed. Prior written informed consent was taken from each patient explaining the procedure, risks and benefits.

Motor nerve conduction study was performed on the tibial and peroneal nerves, both proximally and distally along the leg below knee on both sides. Similarly, sensory nerve conduction was conducted on sural nerve. Parameter included motor distal latency, amplitude and conduction velocities of tibial, peroneal and sural nerves. This study was performed in accordance with ethical standards of the institute. Study was carried out at a controlled room temperature of 25˚C. RMS EMG EP Mark-II machine was used machine for determination of nerve conduction velocity. Two small button type silver electrodes were used as reference and recording electrode for nerve conduction studies. Ground electrode was used for earthing. The following electrophysiological tests were performed after explaining the procedure to patient in his/ her own language, to allay apprehension. For motor study÷ sensitivity: 2-5 Hz, low frequency filter: 2-5 Hz, high frequency filter: 10 kHz and sweep speed: 2-5ms/mm. For sensory studies÷ sensitivity: 10-20µv/mm, low frequency filter:5- 10Hz. High frequency filter: 2-3 kHz, sweep speed: 1-2ms/mm. Supramaximal strength of stimulus was used. Duration for motor and sensory study was at 100µs.

Exclusion Criteria

Patients with non-traumatic cause for spinal cord injury, patient with head injury/medically unstable condition, patient with previous implanted metallic devices, patient with claustrophobia, pacemakers and cochlear implants, patient presenting with previous neurological deficits, gunshot wounds were excluded from the study.

RESULTS

Table-1 shows the motor nerve conduction findings of tibial nerve initially and at follow ups i.e., 3 months, 6 months. The mean latency of right tibial nerve initially was 8.73 (± 4.34) and at 3 months and 6 months follow up was 9.02 ms (± 4.24) and 9.26 ms (±3.99) respectively. This difference in mean latency at various follow up visits was not significant. Similarly, the difference in mean latency of left tibial nerve at various follow up visits was not significant. [Table 1]

Table-2 shows the mean amplitude of right tibial nerve when measured initially was 5.50 mV (±6.20) which increase to 6.5 mV (± 6.65) after 3 months and 7.01 mV (±6.53) after 6 months. The difference in mean amplitude at various follow up visits was highly significant ($p<0.001$). Similarly, the mean amplitude of left tibial nerve when measured initially was 5.59mV (±6.11) which increased to 6.78mV (±6.54) after 3months and 7.55 mV (±6.32) after 6 months. This difference in mean amplitude at various follow up visits was statistically significant ($p=0.003$). [Table 2]

F Table-3 shows the mean conduction velocity of right tibial nerve when measured initially was 28.53m/s (±14.41) which increased to 29.83 m/s (±14.08) after 3 months and 31.14 m/s (±13.40) after 6 months. This difference in mean conduction velocity at various follow up visits was not significant ($p=0.079$). Similarly, the difference in mean conduction velocity of left tibial nerve at various follow up visits was statistically non-significant ($p=0.222$). [Table 3]

Table-4 shows the motor nerve conduction findings of peroneal nerve initially and at follow ups. The mean latency of right peroneal nerve initially was 4.55ms (±4.91) and at 3 months and 6 months follow up was 4.86ms (±4.86) and 5.36 (± 4.66) respectively. This difference in mean latency at various follow up visits was not significant ($p= 0.504$). Similarly, the difference in mean latency of left peroneal nerve at various follow up visits was not significant ($p=0.171$). [Table 4]

Table-5 shows the mean amplitude of right peroneal nerve when measured initially was 1.84mV (±2.94) which increased to 2.40mV (±3.23) after 3months and 3.09 mV (± 3.47) after 6 months. This difference in mean amplitude at various follow up visits was highly significant ($p<0.001$). Similarly, the mean amplitude of left peroneal nerve when measured initially was 1.74 mV (±2.87) which increased to 2.34 mV (±3.08) after 3 months and 2.90mV (±3.17) after 6 months. The difference in mean amplitude at various follow up visits was statistically significant ($p<0.001$). [Table 5]

Table-6 shows the mean conduction velocity of right peroneal nerve when measured initially was 21.43 m/s (±22.02) which increased to 23.80 m/s (± 22.46) after 3 months and 25.84 m/s (±21.80) after 6 months. This difference in mean conduction velocity at

various follow up visits was significant (p=0.003). Similarly, the difference in mean conduction velocity of left peroneal nerve at various follow up visits was also statistically significant (p=0.004). [Table 6]

Table-7 shows the sensory nerve conduction findings of Sural nerve initially and follow ups. The mean latency of right sural nerve initially was 4.30ms (±4.11) and at 3 months and 6 months follow up was 4.53ms (± 3.66) and 5.30ms (±3.74) respectively. This difference in mean latency at various follow up visits was not significant (p=0.274). [Table 7]

Table-8 shows the mean amplitude of right ulnar nerve when measured initially was 1.61mV (±4.08) which increased to 2.00mV (±3.58) after 3 months and 3.06 mV (±4.63) after 6 months. This difference in mean amplitude at various follow up visits was highly significant (p<0.001). Similarly, the difference in mean amplitude of left sural nerve at various follow up visits was also statistically significant (p< 0.001). [Table 8]

Table-9 shows the mean conduction velocity of right sural nerve when measured initially was 18.34 m/s (±16.98) which increased to 22.11 m/s (± 17.55) after 3 months and 24.23 m/s (±16.81) after 6 months. This difference in mean conduction velocity at various follow-up visits was highly significant (p< 0.001). Similarly, the difference in mean conduction velocity of left sural nerve at various follow up visits was also statistically highly significant (p<0.001). [Table 9]

Table 1: Showing latency of tibial nerve.

Tibial nerve	Latency (ms)			
	Initial	3 months	6 months	Significance*
Right	8.73 ± 4.34	9.02 ± 4.24	9.26 ± 3.99	p = 0.484
Left	8.86 ± 4.84	9.39 ± 4.40	9.43 ± 4.04	p = 0.480
Normative data	4.5 ± 0.8			

Mean ± S.D

Table 2: Showing Amplitude of tibial nerve

Tibial nerve	Amplitude (mV)			
	Initial	3 months	6 months	Significance*
Right	5.50 ± 6.20	6.50 ± 6.65	7.01 ± 6.53	p <0.001*
Left	5.59 ± 6.11	6.78 ± 6.54	7.55 ± 6.32	p = 0.003*
Normative data	12.9±4.8			

Mean ± S.D

Table 3: Showing conduction velocity of tibial nerve

Tibial nerve	Conduction Velocity (m/s)			
	Initial	3 months	6 months	Significance*
Right	28.53 ± 14.41	29.83 ± 14.08	31.14 ± 13,40	p = 0.079
Left	26.65 ± 14.78	28.38 ± 13.54	29.47 ± 14.17	p = 0.222
Normative data	47 ± 6			

Mean ± S.D

Table 4: Showing latency of paroneal nerve

Peroneal nerve	Latency (ms)			
	Initial	3 months	6 months	Significance*
Right	4.55±4.91	4.86±4.86	5.36±4.66	p =0.504
Left	4.50±4.67	5.01±4.74	5.72±4.99	p =0.171
Normative data	4.8± 0.8			

Mean ± S.D

Table 5: Showing amplitude of peroneal nerve

Peroneal nerve	Amplitude (mV)			
	Initial	3 months	6 months	Significance*
Right	1.84±2.94	2.40±3.23	3.09±3.47	p<0.001*
Left	1.74±2.87	2.34±3.08	2.90±3.17	p<0.001*
Normative data	5.9±2.6			

Mean ± S.D

Table 6: Showing conduction velocity of peroneal nerve

Peroneal nerve	Conduction Velocity (m/s)			
	Initial	3 months	6 months	Significance*
Right	21.43±22.02	23.08±22.46	25.84±21.80	p =0.003*
Left	20.09± 20.57	22.48±20.86	24.51±20.86	P= 0.004*
Normative data	47±4			

Table 7: Showing latency of Sural nerve

Sural nerve	Latency (ms)			
	Initial	3 months	6 months	Significance*

Right	4.30±4.11	4.53 ± 3.66	5.3 ± 3.74	p = 0.270
Left	4.58 ± 4.27	4.78 ± 3.74	5.61 ± 3.89	P = 0. 274
Normative data	2.9 ± 0.003			

Mean ± S.D

Table 8: Showing amplitude of sural nerve

Sural nerve	Amplitude (mV)			
	Initial	3 months	6 months	Significance*
Right	1.61 ± 4.08	2.00 ± 3.58	3.06 ±4.63	p < 0.001*
Left	1.54 ± 4.13	2.23 ± 2.34	3.00 ± 4.52	p <0.001*
Normative data	16.6 ± 7.5			

Mean ± S.D

Table 9: Showing conduction velocity of sural nerve

Sural nerve	Conduction velocity (m/s)			
	Initial	3 months	6 months	Significance*
Right	18.34 ± 16.98	22.11 ± 17.55	24.23± 16.81	p < 0.001*
Left	16.92 ± 15.61	20.38 ± 15.61	22.47 ± 15.17	p < 0.001*
Normative data	50.9 ± 5.4			

Mean ± S.D. **ms=milliseconds, mV=millivolts, m/s meters per second**

DISCUSSION

In this study, 35 patients with bilateral nerve conduction study (NCS) of two motor nerves (tibial and peroneal) and one sensory nerve of lower limb (sural) was done.

Mean latency and mean conduction velocity of tibial nerve (right and left) was increased with subsequent follow up but this change was non-significant. Increment in mean amplitude of right and left tibial nerve was statistically highly significant (p<0.001, p=0.03 for right, left respectively). Tables -1,2,3.

Mean latency of right and left peroneal nerve increased in subsequent follow up but this change is non-significant. Increment in mean conduction velocity and mean amplitude of peroneal nerve was statistically significant (p=0.003, p=0.004) for right and left respectively; p<0.001 for amplitude of peroneal nerve (right and left both). Tables-4,5,6.

Mean latency of right and left sural nerve was increased in subsequent follow up visits but this change was not significant. Increment in mean conduction velocity and mean amplitude was statistically highly significant (p<0.001) for right and left both. Tables-7,8,9.

Kirshblum et al was found that there were no significant differences between the groups with regard to sural nerve latency, peroneal nerve latency or tibial nerve latency. In contrast statistically significant difference was found between the groups on sural amplitude, peroneal CMAP, peroneal NCV, tibial CMAP and tibial NCV (p <0.0001).[6]

Iseli et al was found that the SSEP recording in parallel to clinical examinations. In both group, the mean amplitude of tibial SSEP (trauma n=39, amplitude0.42 µV(SD ± 0.56 µV); Scheffe' s test, (p<0.05) ANOVA testing of the tibial SSEP latency value of control group (latency 40.99ms (SD ±3.8) and two patients group : traumatic :50.83 (SD 10.27ms); ischemic : 48.80 (SD ±7.3) indicated a significant difference between the healthy subject and patients (p<0.05).[7]

Few studies in the literature have reported normal NCV after SCI.[8,9]

The tibial SSEP can be used to predict the recovery of lower limb function and is related to outcome of ambulatory capacity. It has been reported that a loss of tibial SSEP in patients with an acute spinal cord injury indicates poor recovery and patient with an initially elicitable tibial SSEP show some recovery.[10]

CONCLUSION

It is concluded from this study is that impact of Spinal Cord Injury on latency, amplitude and conduction velocity of tibial, peroneal and sural nerve is very detrimental at onset of injury. But with subsequent follow up it has improved significantly, which can be related to functional recovery of lower limb.

REFERENCES

1. Nobunaga AI, Go BK, Karunas RB. Recent demographic and injury trends in people served by the model spinal cord injury care systems. Archives of Physical Medicine Rehabilitation 1999; 80: 1372-82.
2. George LW, Wood II. Fractures, dislocations and fracture-dislocations of the spine. In: Canale ST, Beaty JH, editors. Cambell's operative orthopaedics. 11th ed. Philadelphia: Mosby Elsevier; 2008.pp.1761-850.
3. Sekhon LH, Fehlings MG. Epidemiology, demographics and pathophysiology of acute spinal cord injury. Spine 2001; 26:2-12.
4. Curt A, Dietz V. Neurographic assessment of intramedullar moto-neuron lesions in cervical spinal cervical spinal cord injury: consequences for hand function. Spinal Cord 1996; 34: 326-332
5. Curt A, Keck MF, Dietz V. Functional outcome following spinal cord injury: Significance of motor evoked potentials and ASIA Scores. Archives of Physical Medicine Rehabilitation 1998; 79:81-6.
6. Kirshblum S, Lim S, Garstang S, Millis S. Electrodiagnostic changes of the lower limbs in subjects with chronic complete cervical spinal cord injury. Archives of physical Medicine Rehabilitation 2001;82(5):604-7
7. Iseli E, Cavigelli A, Dietz V, Curt A. Prognosis and recovery in ischemic and traumatic spinal cord injury: Clinical and

electrophysiological evaluation. Journal of Neurology, Neurosurgery & Psychiatry 1999; 67(5) :567-71.
8. Nyboer VJ, Johnson HE. Electromyographic findings in lower extremities of patients with traumatic quadriplegia. Archives of Physical Medicine and Rehabilitation1971; 52(6) :256-9.
9. Cambell JW, Herbison GI, Chan YT, Jaweed MM, Gussner CG. Spontaneous electromyographic potentials in chronic spinal cord injured patients; relation to spasticity and length of nerve. Archives of Physical Medicine and Rehabilitation 1991; 72(1) :23-7.
10. Li C, Houlden DA, Rowed DW. Somatosensory evoked potentials and neurological grades as predictors of outcome in acute spinal cord injuries. Journal of Neurosurgery1990; 72(4) :600-9.