

## “Nerve Conduction Velocity of Common Peroneal Nerve in Post Stroke Patients”-An Observational Study

Dr. Zainab M M Chatriwala<sup>1</sup>, Dr. Mahendra Shende<sup>2</sup>

<sup>1</sup>Post Graduate Student, <sup>2</sup>Associate Professor, Department of Neurosciences Physiotherapy,  
Dr. A.P.J Abdul Kalam College of Physiotherapy, PIMS, Loni, Maharashtra, India.

---

### Abstract

**Introduction:** Stroke is the third leading cause of death and most common cause of disability among adults in US and a major health problem in India. Spasticity emerges in about 90 percent of cases. Stroke patients with persistent distal weakness of ankle dorsiflexors and spastic plantar flexors are often unable to actively dorsiflex the foot during swing phase which is referred to as foot drop. Common peroneal neuropathies are one of the causes of foot drop a condition which is evaluated using electrodiagnostic method.

**Objectives:** Primary objective of this study was to find out the Nerve Conduction Velocity of common peroneal nerve of the affected lower extremity in stroke participants. Secondary objective of this study was to compare the NCV values of common peroneal nerve with the unaffected lower extremity in the same participants.

**Methods:** Thirty participants between the age of 30- 60 years, with the clinical diagnosis of stroke with 3- 6 month duration. Nerve Conduction Velocity of the Common Peroneal Nerve was studied in participants affected and unaffected lower limbs. Outcome measures used in this study was Distal latency, Motor NCV and CMAP amplitude. After obtaining these values comparison was done between the affected and the unaffected lower limbs.

**Results:** The Study showed that there was statistically significant difference in all the outcome measures.

**Conclusion:** This study showed that the Distal latency were prolonged, Motor NCV and CMAP amplitude were reduced in the affected lower limbs compared to the unaffected lower limbs. Thus this study concluded that the spasticity of ankle plantar flexors and/or weakness of ankle dorsiflexors in stroke individuals could cause electrophysiological changes in the Common Peroneal Nerve.

**Keywords:** Stroke, Electrodiagnostic, Distal latency, CMAP amplitude, NCV.

---

### I. Introduction

In human body the most complex organ is the brain. To function effectively it requires constant supply of nutrients and oxygen from blood. Damage to the brain cells occur when there is interruption to this supply of nutrients and oxygen. There are many adverse effects occurring due to damage to the part of brain and can lead to death. One relatively common cause of death and brain damage is called stroke.<sup>1</sup> Over 2,400 years ago Hippocrates, the father of medicine, first recognized “Stroke”. During that time stroke was called apoplexy, which meant “Struck Down by Violence” in Greek.

The World Health Organization (WHO) defines stroke as “rapidly developing clinical signs of focal disturbance of cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than of vascular origin”.<sup>2</sup> It is the third leading cause of death.<sup>3,4</sup> In the young adults it is the most common cause of disability. It has been estimated that one third of all stroke survivors are functionally dependent after a year of stroke with motor function deficits. After 3 months around 20% of stroke survivors require institutional care while 15% to 30% are permanently disabled making it a foremost cause of functional impairment.<sup>5</sup> In India, stroke is a major health problem.<sup>6</sup> There is a limited access to reliable morbidity and mortality estimates for stroke in India attributable to certain factors like incomplete certification of death, incorrect classification of death and uncertainty of aetiology in cases of sudden death and miscellaneous co-morbidities.<sup>7</sup> The adjusted annual incidence per 100000 persons of stroke is 124 in rural area and 145 in urban area. In India incidence of stroke is high in young age group i.e. below 50 years of age and also in population with low socio-economic status. The probable risk factors are smoking and drinking which are extensively seen more in men.<sup>8</sup> Though many individuals survive with the stroke, they have problem with mobility due to physiological change in the muscle tone i.e. spasticity which emerges in about 90% of cases.<sup>9</sup> In lower limb spasticity is seen in the pelvic retractors, hip adductors, internal rotators, hip and knee extensors, ankle plantar flexors, invertors and toe flexors leading to the most common pattern, equinovarus foot<sup>10</sup>. Uncontrolled spasticity may lead to contracture of muscles, reduced range of motion, postural abnormalities and fitful motor performance which results in activity limitation such as dressing, bathing and walking.<sup>11</sup>

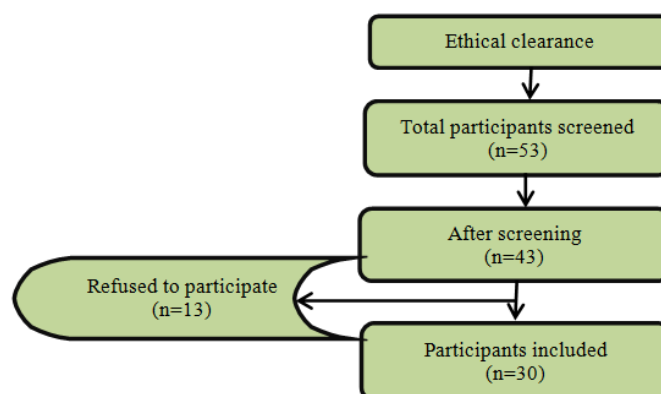
Walking is the most important physical activity which helps in maintaining healthy body structure and function. Patients with stroke may lack in the ability to adjust and stabilize proximal limbs and trunk

appropriately during movement, which results in postural abnormalities, balance impairments and increased risk of fall.<sup>9</sup>Stroke patients with persistent distal weakness of ankle dorsiflexors and spastic plantar flexors are often unable to actively dorsiflex the foot during swing phase which is referred to as foot drop.<sup>12</sup>Foot drop is usually caused by lower motor neuron (LMN) disease.<sup>13</sup>Some of the common cause of compression are wearing high heel boot/shoe, plaster casts, stocking or from sitting with the leg crossed for a prolonged period of time.<sup>14</sup> A study stated that people suffering from ankle sprain caused by inversion movement of the foot or ankle instability might have damage to the peroneal nerve.<sup>15</sup>Central nervous system pathology can also cause foot drop. There 52% to 67% of patients with spinal upper motor neuron (UMN) pathology who has suffered from foot drop. Central causes tends to occur where nerve fibers are highly condensed along the UMN tracts i.e. in the interhemispheric motor cortex homunculus (mass lesion, anterior cerebral artery stroke), corona radiata, internal capsule, cerebral peduncle, medulla, and spinal cord pyramidal tract (myelopathy).<sup>13</sup>Nerve and muscle generates electrical signals that deliver message to and from the brain. Injury or disease that affects nerves and muscles can slow down or halt the movement of these electrical signals. Measuring the speed and degree of electrical activity in muscles and nerves can help medical professionals make a proper diagnosis. This process is called as electrodiagnostic testing. The two tests commonly used are Electromyography (EMG) and Nerve Conduction Velocity (NCV).<sup>16</sup> This study aims to investigate whether spasticity of the ankle plantar flexors and/or weakness of ankle dorsiflexors in stroke patients cause electrophysiological changes in the common peroneal nerve, as reflected on Nerve Conduction Velocity (NCV) study.

## II. Materials And Methods

The research design used for the study was observational study. The source of data for this study was collected from Medicine &Neurophysiotherapy department, Pravara Rural Hospital. Participants included in the study were both male and female individuals with cerebro vascular accident who were referred to Neurophysiotherapy department. The sample included participants who fulfilled the inclusion and exclusion criteria and were willing to participate in the study. The inclusion criteria for the study were: Age 30 to 60 years, Participants with single episode of stroke with right or left side involvement, Participants with sub-acute stroke (3- 6 months), Modified Asworth Scale 1+ to 2, Brunnstrom recovery stage 2 to 3, Mini Mental State Examination score  $\geq 24$ .<sup>17,18,19</sup> The exclusion criteria for the study were: Participants with previous stroke presenting with bilateral hemiplegia, Neuromuscular disease, Radiculopathy or peripheral nerve injury in the lower limb, Diabetes mellitus, any other Neurological condition other than stroke.<sup>20,21</sup> Sampling design was convenient sampling. The equipment used for assessing the motor Nerve Conduction Velocity was RMSEMGEP MARK II was used.

### 2.1 Procedure:



**Figure 2.1: Procedure Flow chart**

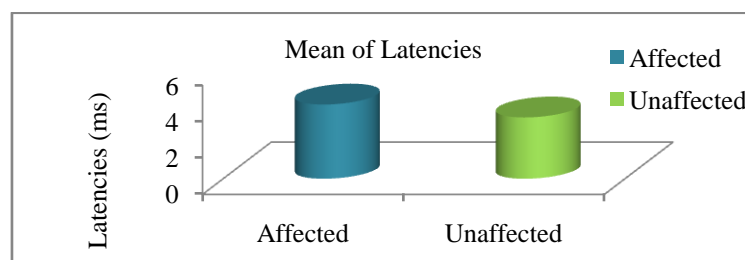
The study received ethical approval from the institutional ethical committee IEC, of PIMS, Loni (Ref. no. COPT/2015/1561/5). The participants were screened and after finding their suitability according to the inclusion and exclusion criteria, they were requested to participate in the study. The individuals who were willing to participate in the study were briefly explained about the nature of the study in the language best understood by them. They were encouraged to clarify queries regarding the study, if any. An informed written consent form, previously approved by the IEC was then obtained from the participants. The demographic data was obtained and detailed assessment was done. The study variables like Distal latency, CMAP amplitude, Motor Nerve Conduction Velocity of the Common Peroneal Nerve were assessed. All participants were allocated in a single group. The Distal latency, CMAP amplitude and Motor Nerve Conduction Velocity of the Common Peroneal Nerve of the affected and unaffected lower limb were obtained from the same participants.

After obtaining the Distal latency, CMAP amplitude and Motor Nerve Conduction Velocity, comparison was done between the affected and unaffected lower limb. The study was performed using NCV as a diagnostic tool. The participants were asked to lie in the supine position. Limb of the participants were placed in a relaxed position as any movement of the limb could hamper the results. Temperature significantly influences the Conduction Velocity and the amplitude of CMAP. Low temperature results in slowing of Nerve Conduction Velocity and increases the amplitude. Thus during the NCV study of the Common Peroneal Nerve the room temperature was maintained between 30°C and 36°C.<sup>22</sup>To study the Motor NCV of Common Peroneal Nerve three electrodes were used. The three recording electrodes were active electrode, G<sub>1</sub> (cathode), the inactive electrode, G<sub>2</sub> (anode) and the ground electrode. The recording site used to test the Common Peroneal Nerve was the Extensor Digitorum Brevis. On the dorsal lateral foot the G<sub>1</sub> electrode was placed over the muscle belly, G<sub>2</sub> electrode was placed distally over the metatarsophalangeal joint of the little toe. The ground electrode was placed between the recording electrodes and the stimulating electrode. The first stimulation site was the anterior ankle, slightly lateral to tibialis anterior tendon. The second stimulation site was below the fibular head i.e. lateral calf, one to two finger breadths inferior to fibular head. The distal distance was 9cm or 90 mm. At the stimulation site, to ensure that all axons have been depolarized, Supramaximal stimulation was performed. To achieve Supramaximal stimulation the current intensity was slowly increased until a point where the amplitude of the recorded potential no longer increased. After noting the latencies of the common peroneal nerve the distance between distal and proximal stimulation site was noted. The motor Nerve Conduction Velocity was calculated using the formula [Distance (mm)/proximal latency- distal latency (ms)].<sup>22</sup>After noting these values from the affected and unaffected lower extremities of the participants, comparison was done.

### III. Results

A total of fifty participants were screened of which 40 participants met the inclusion criteria. Out of these 30 participants agreed to participate in the study. Electrophysiological changes in the common peroneal nerve were observed in the affected and the unaffected lower limb of these participants. The confidence interval was set as at 95% and data was considered statistically significant with  $p < 0.05$  and highly significant with  $p < 0.01$ . The mean age of participants was  $46.067 \pm 7.70$  years. The average age of females was  $45 \pm 8.18$  years and for males was  $46.68 \pm 7.57$  years. The gender ratio was 19:11 (19 males and 11 females). The mean BMI of participants was  $22.828 \pm 3.63 \text{ kg/m}^2$ .

The Distal latency of the unaffected lower limbs were  $3.384 \pm 0.3911$  ms. The Distal latency of the affected lower limbs were  $4.096 \pm 0.2433$  ms. On comparing the Distal latency of the unaffected and the affected lower limbs of the stroke participants, it was observed that this difference was highly significant. ( $p < 0.001$ ,  $t = 8.463$  with  $df = 58$ ) (Figure: 3.1). The linear correlation between the two lower limbs was measured using Pearson coefficient. There was a positive correlation with regard to the difference between latencies of the two lower limb ( $r = 0.274$ ).



**Figure 3.1: Distal Latencies**

**Table 3.1: Distal Latencies (ms)**

Side	Mean	Standard Deviation
Unaffected	3.384	0.3911
Affected	4.096	0.2433

The MNCV of the Common Peroneal Nerve of the unaffected limb were  $47.220 \pm 2.788$  m/s. The MNCV of the Common Peroneal Nerve of the affected lower limb were  $44.605 \pm 3.580$  m/s. On comparing the Motor Nerve Conduction Velocity of the unaffected and the affected lower limbs, it was observed that this difference was highly significant. ( $p = 0.0025$ ,  $t = 3.156$  with  $df = 58$ ) (Figure: 3.2).

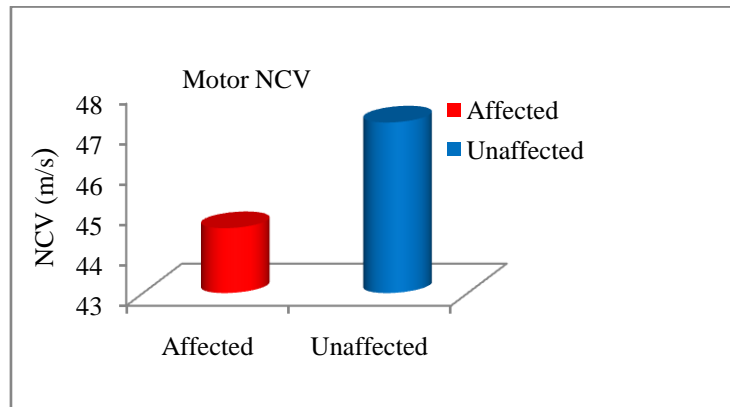


Figure 3.2: Motor NCV (m/s)

The CMAP amplitude of the Common Peroneal Nerve of the unaffected lower limb of the participants was  $4.963 \pm 1.235$  mv. The CMAP amplitude of the Common Peroneal Nerve of the affected lower limb of the participants was  $4.210 \pm 1.140$  mv. On comparing the CMAP amplitude of the affected and the unaffected lower limb, it was found that this difference was significant. ( $p = 0.0172$ ,  $t = 2.452$   $df = 58$ ) (Figure: 3.3).

The linear correlation with regards to the difference in CMAP amplitude between the two lower limbs was determined using Pearson coefficient and test based on it and it was found that there was a positive correlation ( $r = 0.921$ ).

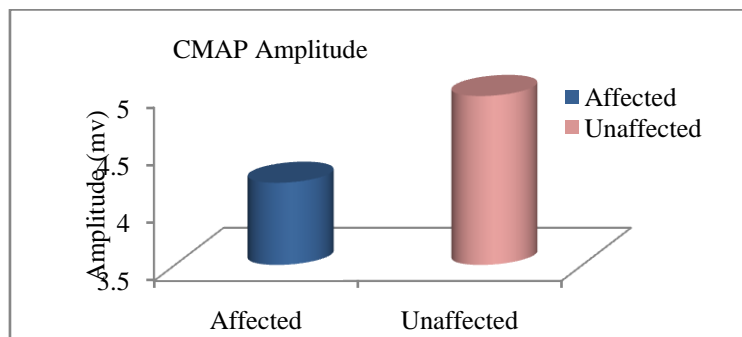


Figure 3.3: CMAP amplitude

#### IV. Discussion

The purpose of the study was to find whether spasticity of the ankle plantar flexors and/or weakness of ankle dorsiflexors in stroke patients cause electrophysiological changes in the common peroneal nerve, as reflected on Nerve Conduction Velocity (NCV) study. The result of the study showed that there were changes in the Distal latency, CMAP amplitude, Motor Nerve Conduction Velocity of the common peroneal nerve in the affected lower limb of stroke participants. It was observed that in the affected lower limb of the stroke patients the Distal latency was prolonged, CMAP amplitudes were reduced and Motor NCV was reduced. The result was statistically significant.

On comparing the Distal latency of the unaffected and the affected lower limbs of the stroke participants, it was observed that this difference was highly significant ( $p < 0.001$ ). The Distal latency was prolonged in the affected lower limb than in the unaffected lower limb. There was positive correlation ( $r = 0.274$ ) with regard to the difference between latencies of the two legs in the Extensor Digitorum Brevis muscle. Injury to the Common Peroneal Nerve around the head and neck of fibula is usually due to compression, traction or laceration and has been described mostly in sportsmen after severe ankle sprain.<sup>23,24</sup> The study on cadavers by Noble showed that the Common Peroneal Nerve and its branches of distribution are attached to the fibular neck along with peroneus longus muscle producing T- form osteo muscular tunnel. The extensibility of the Common Peroneal Nerve in the popliteal cave is limited to 10-25 mm. Severe inversion movement may lead to significant displacement of the osteo muscular tunnel causing nerve traction.<sup>25</sup> Walking is the most important physical activities which help in maintaining healthy body structure and function. A study by Wissel et al, showed that 25% of the patients with stroke suffer from spasticity within first 6 weeks of the event and is often seen in hip adductors, internal rotators, hip and knee extensors, ankle plantar flexors, invertors and toe flexors. Spasticity in ankle plantar flexors of the stroke patients was observed to be 66% and results in restriction of volitional movement. Thus the limb is positioned with equinovarus foot.<sup>10</sup> Baccari S et al. reported six cases of peroneal

nerve paralysis following ankle sprain, 0-3 days after the accident. These theories on peroneal nerve vulnerability were evidenced after ankle sprain during which the foot takes an equinovarus position.<sup>18</sup> Tsur A et al, conducted study on axillary nerve of stroke patients and found that continuous traction of the axillary nerve as in hypotonic shoulder may affect the electrophysiological properties of nerve. On studying the electrophysiological properties of the axillary nerve they found that the latency was prolonged.<sup>17</sup> Peroneal neuropathy can also be seen as a result of a variety of conditions like weakness of foot dorsiflexors, evertors etc.<sup>22</sup> Thus this result observed in stroke patients could be due to weakness of the ankle dorsiflexors or spasticity of ankle plantar flexors which leads to the equinovarus position of the foot and this could cause traction of the Common Peroneal Nerve and changes in the electrophysiological properties. On comparing the Motor Nerve Conduction Velocity of the affected and the unaffected lower limb, it was observed that the difference was highly significant. ( $p = 0.0025$ ). The results of this study is similar to the study conducted by Marco P et al., He conducted a study on post stroke hemiplegic patients to verify whether standard nerve conduction studies show significant difference in this group compared to the control group. The results showed that there was slower Nerve Conduction Velocity in the ulnar and Common Peroneal Nerve.<sup>26</sup> Takebe et al. conducted a study on the affected extremity of 27 hemiplegic patients and noted significant slowing in the Peroneal Nerve Conduction Velocities. They supposed that a decreased diameter of the nerve fiber as a result or cause of muscle atrophy could lead to decrease in Nerve Conduction Velocity.<sup>27</sup> Chokroverty et al. in their study measured the Motor Nerve Conduction Velocities of the Common Peroneal Nerve in 44 hemiplegic patients bilaterally and found a statistically significant difference between the two limbs.<sup>28</sup> A study was conducted to quantify Nerve Conduction Velocity differences in individuals with functional ankle instability compared to the healthy subjects and the results showed that participants with functional ankle instability might have damage to the peroneal nerve which resulted in slower Nerve Conduction Velocity.<sup>15</sup> On comparing the CMAP amplitudes of the affected and unaffected lower limb of the stroke participants, it was observed that this difference was significant ( $p = 0.0172$ ). Positive correlation was also found regarding the difference between the two lower limbs CMAP amplitude ( $r = 0.921$ ). CMAP amplitude reflects the number of muscle fibers that depolarize.<sup>22</sup> In this study the CMAP amplitude was reduced in the affected lower limb when compared to the unaffected lower limb. Emam F et al. conducted a study in 33 stroke patients to find out axillary nerve injury after shoulder subluxation. The electrophysiological properties like the CMAP amplitude, latency were assessed and the results showed that there were significant changes in the latency and amplitude in the paretic side compared to the non- paretic side.<sup>20</sup> The peroneal nerve is supplied by two to three vasa nervorum. Severe traction may either cause hematoma or ischemia inside the nerve. Nitz A et al, found that 17% of patients with ankle sprain had alteration in the electrophysiological properties of the peroneal innervated muscle, despite the lack of clinical signs and symptoms of nerve lesion suggesting a mild axonal injury.<sup>24</sup> Low CMAP amplitudes most often result from loss of axons.<sup>22</sup> Tsur A et al. in his study said that continuous inversion position, in foot drop may affect electrophysiological properties of the nerve. He said that when the compression is severe, ischemic changes occur and cause secondary axonal damage i.e expressed by reduction of the CMAP amplitude.<sup>18</sup> Thus the results obtained from this study could be attributed to the above explanations.

## V. Conclusion

The present study showed that there were changes in the electrophysiological properties of the Common Peroneal Nerve. The Distal latency were prolonged in the affected lower limb, the Motor NCV and the CMAP amplitude values were reduced on the affected lower limb compared to the unaffected lower limb.

**Limitation:** The limitation of this study was that it included a smaller sample size. The study did not have equal distribution of male and female participants.

## Recommendations

The future study can be conducted with a larger sample size and with equal distribution of male and female participants.

## Acknowledgement

I want to thank all the participants who co-operated in this study, Dr. S M Khatri for inspiring me, Dr. Mahendra Shende for his valuable contribution and encouragement which kept me moving.

## References

- [1]. Benjamin McDonald, Ann Witt, Natalie Reiss. MHMR services for the Concho Valley. STROKE. Introduction to stroke. Available at <file:///C:/Users/WELCOME/Desktop/full%20proj/references%20for%20intro/1/Stroke.html> accessed on 06-10-2015.
- [2]. Sacco R, et al. An Updated Definition of Stroke for the 21<sup>st</sup> Century. Stroke. 2013; 44: 2064-2089. Available at <http://stroke.ahajournals.org>. Accessed on 11-02-2015.
- [3]. Thomas T, Stephen B, Colin M. The global burden of cerebrovascular disease. Cerebrovascular disease.2006: 1-67. Available at

- [http://www.who.int/healthinfo/statistics/bod\\_cerebrovascularstroke.pdf](http://www.who.int/healthinfo/statistics/bod_cerebrovascularstroke.pdf) accessed on 03-02-2015.
- [4]. Bharucha N, Kuruvilla T. Epidemiology of Stroke in India. *Neurol J Southeast Asia*. 1998; 3:5-8.
- [5]. Steinwachs DM, Collins-Nakai RL, Cohn LH, Garson A Jr, Wolk MJ. The future of cardiology: utilization and costs of care. *Am Coll Cardiol*. April 2000; 35(5 Suppl B):91B-98B.
- [6]. Khadilkar S. Neurology: The Scenario in India. *JAPI*. 2012; 60:42-44.
- [7]. Pandian JD, Srikanth V, Read SJ, Thrift AG. Poverty and Stroke in India. *Stroke*, 2007; 38: 3063-3069.
- [8]. Taylor F C, Kumar S. Stroke in India factsheet. South Asia Network for Chronic Diseases, IIPH Hyderabad, Public Health Foundation of India. Available at <http://www.sanecd.org/Updated%20Stroke%20Fact%20sheet%202012.pdf>. Accessed on 06-10-2015.
- [9]. O’Sullivan S, Schmitz T. *Physical Rehabilitation: Stroke*. 5th Edition. Jaypee Brothers; 2007; Pg: 705-720.
- [10]. Thibaut A, Chatelle C, Ziegler E, Marie-Aure, Laureys S, Gosseries O. Spasticity after stroke: Physiology, assessment and treatment. *Brain Injury*. 2013; 1-13.
- [11]. Fayazi M, Dehkordi S N, Dadgou M, Salehi M. The Relationship between Spasticity and Lower Extremity Strength with Functional Mobility Following Chronic Stroke. *Int J Phys Med Rehabil*. 2014; 2(4): 1-5.
- [12]. Kluding P.M, Dunning K, O’Dell M.W, Wu S. S, Ginosian J, Feld J, Mc Bride K. Foot Drop Stimulation Versus Ankle Foot Orthosis After Stroke: 30-week Outcomes. *Stroke*. 2013; 44:1660-1669. Accessed on 24-05-2015.
- [13]. Westhout F D, Pare L S, Linskey M E. Central Causes of Foot Drop: Rare and Underappreciated Differential Diagnoses. *J Spinal Cord Med*. 2007; 30: 62–66.
- [14]. Chouhan S. Motor nerve conduction of common Peroneal nerve in young adult. *Current Neurobiology*. 2012; 3(1): 51-54.
- [15]. Simon J, Docherty C. Slower Nerve Conduction Velocity in Individuals with Functional Ankle Instability. *Int J Sports Med*. 2014; 35: 731-736.
- [16]. American Academy of Orthopaedic Surgeons. *Electrodiagnostic Testing*; 2007 (last viewed October 2007). Available at <file:///C:/Users/WELCOME/Desktop/full%20proj/references%20for%20intro/31/Electrodiagnostic%20Testing-OrthoInfo%20%20AAOS.html> accessed on 02-10-2015.
- [17]. Tsur A, Ring H. Axillary nerve conduction changes in hemiplegia. *Journal of Brachial Plexus and Peripheral Nerve Injury*. 2008; 26(3): 1-5.
- [18]. Tsur A. Common Peroneal Neuropathy in Patients after First-Time Stroke. *IMAJ*. 2007; 9: 866-869.
- [19]. Heart & Stroke Foundation Canadian Partnership for Stroke Recovery. Sub-acute Stage of recovery. Available at [www.Stroke.ca/glossary/sub-acute-stage-of-recovery/](http://www.Stroke.ca/glossary/sub-acute-stage-of-recovery/)
- [20]. Emam F E, Genedy A F, Mahmoud S A. Axillary Nerve Conduction Study in Paretic Limbs of Patients with Cerebrovascular Stroke. *The Egyptian Journal of Hospital Medicine*. 2014; 54: 94-100.
- [21]. Taksande B, Ambade M, Joshi R. Delayed Axillary Nerve Conduction Latency and Reduced CMAP Amplitudes in the Paretic Limbs of Patients With a Cerebrovascular Episode. *J Neurol Res*. 2011; 1(5): 210-214.
- [22]. Preston D C, Shapiro B E. *Electromyography and Neuromuscular Disorders- Clinical Electrophysiologic Correlations: Basic Nerve Conduction Studies*. Second Edition. Elsevier; 2005; Pg: 27-28.
- [23]. Baima J, Krivickas L. Evaluation and treatment of peroneal neuropathy. *Curr Rev Musculoskelet Med*. 2008; 1: 147-153.
- [24]. Nitz A J, Dobner J J, Kersey D. Nerve injury and grades II and III ankle sprains. *Am J Sports med*. 1985; 13(3):177-182.
- [25]. Noble W. Peroneal Palsy Due to Hematoma in the Common Peroneal Nerve Sheath after Distal Torsional Fractures and Inversion Ankle Sprains. *J Bone Joint Surg Am*. 1966; 48(8): 1484- 1495.
- [26]. Marco P, Benedetta V, Massimiliano M, Francesco I, Valter S. Peripheral Nerve Conduction Abnormalities in Nonparetic Side of Ischemic Stroke Patients. *Journal of Clinical Neurophysiology*. 2010; 27(1): 48-51.
- [27]. Takebe K, Narayan M G, Kukulka C, Basmajian J V. Slowing of nerve conduction velocity in hemiplegia: possible factors. *Arch Phys Med Rehabil*. 1975; 56(7): 285- 289.
- [28]. Chokroverty S, Medina J. Electrophysiological study of hemiplegia. Motor nerve conduction velocity, brachial plexus latency and electromyography. *Arch Neurol*. 1978; 35(6): 360- 363.