Axillary Nerve Conduction Study in Paretic Limbs of Patients with Cerebrovascular Stroke

Fahmy Emam Fahmy Emam*, Ahmed Fathy Mohammed Genedy**, Sobhia Ali Mahmoud*

* Rheumatology and Rehabilitation, Al-Azhar University, Faculty of Medicine for Girls
** Rheumatology and Rehabilitation, Director of Physical Medicine and Rehabilitation Centre for Armed Forces at El-Agouza

Abstract
To study the axillary nerve injury after shoulder subluxation in patients of stroke.

Methods:
All eligible and consenting patients of stroke (underwent) electrophysiology studies of both axillary nerve. The parameters include measurement of neuropathy motor latency, CMAP (compound muscle action potential) & SNAP (sensory nerve action potential) and the conduction velocity. This study included 33 patients with cerebrovascular stroke who were referred to the Physical medicine, Rheumatology and Rehabilitation department at Al-Zahraa University hospital between 1st April and 31st December 2012. They were 23 males and 10 females, their ages ranged from 27 to 72 years. The paretic limbs of the patients included were classified as case limbs, while the non-paretic limbs were classified as controls.

Results:
Axillary nerve latency and amplitude were high significant in shoulder subluxation in the hemiparaetic side in comparison with control significant.

Conclusion:
There is lower motor neuron axillary nerve in stroke patients, mixed type (demylinated and axonal), the incidence of axillary nerve lesion is mostly with stroke shoulder subluxation.

Keywords: Stroke; axillary nerve; nerve conduction studies.

Introduction
Cerebrovascular stroke is a sudden onset neurological deficit due to compromised blood supply to the brain. It is the third most common cause of death and disabling neurological damage. Worldwide about 15 million individuals have a stroke and about one third of them die\(^{(1)}\). There are two types of stroke; ischemic stroke, which occurs in approximately 85% of patients due to occlusion of the vascular supply to the brain by a thrombus or embolus. The other type is hemorrhagic stroke which occurs in about 15% of patients when impaired blood flow is due to hemorrhage in the brain\(^{(2)}\). A common sequela of stroke is hemiplegic shoulder pain that can hamper functional recovery and subsequently lead to disability. It can begin as early as 2 weeks poststroke but typically occurs within 2-3 months poststroke\(^{(3)}\). The most common manifestation of stroke is sudden weakness of the face, arm or leg, most often on one side of the body (Hemiparesis). Other warning symptoms can include: \(\star\)Sudden numbness of the face, arm or leg, especially on one side of the body. \(\star\) Sudden confusion, trouble speaking or understanding speech. \(\star\) Sudden trouble seeing in one or both eyes. \(\star\) Sudden trouble walking, dizziness, loss of balance or coordination. \(\star\) Sudden severe headache with unknown cause\(^{(4)}\).

Good shoulder function is a prerequisite for effective hand function, as well as for performing multiple tasks involving mobility, ambulation, and activities of daily living. A common sequela of stroke is hemiplegic shoulder pain that can hamper functional recovery and subsequently lead to disability. It can begin as early as 2 weeks poststroke but typically occurs within 2-3 months poststroke\(^{(5)}\).

Glenohumeral subluxation (GHS), a frequent complication for patients with a poststroke hemiplegia, is reported to be present in 17 to 81 percent of patients with hemiplegia following stroke\(^{(6)}\).

Patient and methods:
This study was conducted on 33 stroke patients with hemiparesis who were recruited from the Physical medicine, Rheumatology and Rehabilitation department at Al-Zahraa University hospital between 1\(^{st}\) April and 31\(^{st}\) December 2012. The paretic limbs of the patients included were classified as case limbs, while the non-paretic limbs were classified as controls.
**Inclusion criteria:**
1) History of stroke within the period of three months. 2) History of flaccid paralysis of the affected limb with motor power < grade 3 of Medical Research Council (MRC) at the onset of stroke\(^6\). 3) Sufficient cooperation for clinical and nerve conduction testing.

**Exclusion criteria:**
1) History of recurrent stroke. 2) Past history of trauma or nerve injury of either limb. 3) History of diabetes mellitus. 4) History of metallic implants as cardiac pacemaker and prosthetic valve. 5) Peripheral neuropathies.

All patients who met the inclusion criteria were subjected to the following:

A) **Written consents were taken.**
B) **Clinical assessment:**
Full medical history taking, - **Musculoskeletal examination:** was done with special emphasis on shoulder examination including inspection, palpation and range of motion of the shoulder for detection of any deformity, tenderness, limitation of range of motion and shoulder subluxation, presence of shoulder pain was assessed by Ritchie Articular Index (RAI) \(^7\). **Laboratory investigations:** fasting and 2 hrs postprandial blood glucose level, blood chemistry, coagulation and lipid profiles. **Radiological assessment:** Plain X rays of the shoulder antero-posterior view for detection of subluxation\(^8\)

**Electrophysiological studies:** Nerve conduction studies of axillary nerve of the included patients were done for the hemiplegic sides, and for the healthy sides as controls. There is no conduction technique to evaluate directly sensory component of axillary nerve. Therefore, the measurement of nerve conduction in the axillary nerve included the motor latency and amplitude of compound motor action potential (CMAP). Motor nerve conduction velocity is calculated by measuring the distance in millimeter between two points of stimulation, which is divided by the latency difference in millisecond. However, nerve conduction velocity is not calculated in the axillary nerve as it has only one point of stimulation. The test was performed using the EMG/NC unit. The apparatus used was (EMG/EP MEASURING SYSTEM MODEL MEB-9400K. NIHON KOHDEN CORPORATION, JAPAN).

![Fig. (1): Motor NCS of axillary nerve showing prolonged distal latency and reduced and amplitude of affected side as compared to control side.](image-url)
The electrodes used were:
1- Stimulator electrode. 2- Ground electrode. 3- Recording electrodes active and reference (Surface electrodes).

The technique used was as follows:
- The patients were asked to lay supine or sit with tested limb supported.
- Sterilization of the skin with alcohol.
- Temperature was adjusted.
- Stimulations were done at the erb's point (The cathode is placed in the supraclavicular fossa, just lateral to the sternocleidomastoid muscle and the anode is positioned proximal to the cathode). 3- Ground electrode was placed between the stimulator and recording electrodes. 4- Recording electrodes were placed so that active electrode on the muscle belly (over the most prominent portion of the middle deltoid muscle) and reference electrode on the tendon (over the insertion of the deltoid, approximately midway in the humerus).

Interpretation of the electrography:
CMAPs were assessed, latencies and amplitudes were determined and compared with the control side. The onset latency is the time in milliseconds from the stimulus artifact to the first negative deflection of CMAP. The amplitude of CMAP is measured in millivolts from the base line to the negative peak (base to peak).

- Delay in distal latency denotes demyelination.
- Reduction in amplitude denotes axonal lesion.

Statistical analysis:
All data were collected, tabulated and subjected to statistical analysis. Statistical analysis is performed by SPSS in general (version 17), also Microsoft office Excel is used for data handling and graphical presentation.

A. Descriptive statistics: Quantitative variables are described by Mean, Standard Deviation (SD), Range (Maximum – Minimum). Qualitative categorical variables are described by proportions and percentages.

B. Inferential statistics:
1. Cutoff values of parameters of NCS were determined (mean+2SD of the control value for the latencies and mean–2SD for the amplitudes).
2. Chi-square test and Fisher Exact test: are used for the analysis of categorical qualitative variables.
3. Student “t” test: is used for comparing the means of two groups. 4. ANOVA “F” test: is used for comparing the means of more than two groups of quantitative variables, followed by the application of Scheffe’s multiple comparison procedure to test the significance of difference of means between each two groups. 5. Pearson correlation coefficient “r” is used to assess the correlation between any two quantitative variables. Two Tailed tests are assumed throughout the analysis for all statistical tests.

The probability of error (P value): level of significance.
- If P>0.05: Non significant (NS).
- If P< 0.05: Significant (S).
- If P<0.01: Highly significant (HS).

Results:
The study included 33 patients with hemiparesis, they were 23 males (69.7%) and 10 females (30.3%), their ages ranged from 27 to 72 years with a mean of (53.5±10.8). The stroke duration ranged from 21 to 90 days with a mean of (51.58±22.44). 23 patients had ischemic stroke (69.7%) while 10 patients had hemorrhagic stroke (30.3%) as shown in Fig.(2). All patients were right handed. All male patients and three of female patients were working and stopped their jobs after the onset of stroke. The other female patients were housewives. All of male patients were smokers except three, while all of female patients were non smokers. 24 patients were hypertensive, 12 patients had a history of cardiac diseases (history of rheumatic or ischemic heart diseases) and 15 patients had dyslipidaemia (high cholesterol, TG or LDL levels). The demographic data of the included patients is shown in table (1).

Table (1): The demographic data of the included patients.

<table>
<thead>
<tr>
<th>Demographic variables</th>
<th>Patients Number (%)</th>
<th>Range</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>23 (69.7%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>10 (30.3%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>27-72</td>
<td>53.5 ± 10.8</td>
<td></td>
</tr>
<tr>
<td>Type of stroke:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic</td>
<td>23 (69.7%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hemorrhagic</td>
<td>10 (30.3%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroke duration (days)</td>
<td>21-90</td>
<td>51.58 ± 22.44</td>
<td></td>
</tr>
</tbody>
</table>
Radiological data:
Shoulder X ray findings were positive for subluxation in 23 patients (69.7%) and negative in 10 patients (30.3%) as shown in table (2) and Fig.(3).

Table (2): X-ray finding of shoulder subluxation.

<table>
<thead>
<tr>
<th>X ray: subluxation</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td>10</td>
<td>30.3%</td>
</tr>
<tr>
<td>Positive</td>
<td>23</td>
<td>69.7%</td>
</tr>
</tbody>
</table>

Comparison between patients with and patients without shoulder subluxation in X ray of the affected side as regard age, type of stroke.

Electrophysiological data
Motor nerve conduction studies of axillary nerve were done for the included patients for both the affected paretic sides and the other healthy sides (controls). Range, mean and SD of the motor latencies and amplitudes of axillary nerve of both affected and control sides are shown in table (3).

Table (3): The electrophysiological data of the included patients.

<table>
<thead>
<tr>
<th></th>
<th>Affected</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Motor Latency (ms)</td>
<td>3.10-5.40</td>
<td>4.21 ± 0.47</td>
</tr>
<tr>
<td>Amplitude (mV)</td>
<td>2.00-14.80</td>
<td>7.72 ± 2.72</td>
</tr>
</tbody>
</table>

ms = millisecond; mV= millivolt

1- Comparative studies:
Comparison between the affected hemiparetic sides and the healthy control sides regarding electrophysiological data was done and showed that: There was a highly significant statistical difference (p<0.01) between the affected and control sides regarding motor latencies of axillary nerve (with prolonged motor latencies in the affected sides than the control sides) as shown in table (4) and Fig.(4). 7 patients showed delay in axillary nerve motor latency more than the cutoff value (mean+2SD) of the control latency. There was a highly significant statistical difference (p<0.01) between the affected and control sides regarding motor amplitudes of axillary nerve (with lower motor amplitudes in the affected sides than the control sides) as shown in table (4) and Fig.(4). 6 patients showed reduction in axillary nerve motor amplitude less than the cutoff value (mean-2SD) of the control amplitude.

Table (4): Comparison between affected side and control side regarding motor latencies and amplitudes of axillary nerve.

<table>
<thead>
<tr>
<th></th>
<th>Affected Mean ± SD</th>
<th>Control Mean ± SD</th>
<th>Cut off value</th>
<th>t</th>
<th>P value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor Latency (mS)</td>
<td>4.21± 0.47</td>
<td>3.87± 0.36</td>
<td>4.59</td>
<td>3.295</td>
<td>0.002</td>
<td>HS</td>
</tr>
<tr>
<td>Amplitude (mV)</td>
<td>7.72 ± 2.72</td>
<td>9.64 ± 1.95</td>
<td>5.74</td>
<td>-3.302</td>
<td>0.002</td>
<td>HS</td>
</tr>
</tbody>
</table>
Fig. (2): Comparison between patients with and patients without shoulder subluxation in the hemiparetic side with each other and with control side regarding electrophysiological data was done and showed that:

➢ As regard motor latencies of axillary nerve, there was no statistical significant difference between patients with and patients without subluxation (p>0.05). Also, there was no statistical significant difference between patients without subluxation and control side (p>0.05) as shown in table (5) Fig.(7).

There was a highly significant statistical difference (p<0.01) between patients with subluxation and control side as shown in table (5) Fig.(7), with prolonged motor latencies in patients with subluxation than the control side, meaning a highly significant relation between shoulder subluxation and prolongation of motor latency of axillary nerve in the hemiparetic side.

**Table (6):** Comparison between patients with and patients without subluxation in the hemiparetic side and control side as regard motor latencies.

<table>
<thead>
<tr>
<th></th>
<th>Affected with positive subluxation</th>
<th>Affected with negative subluxation</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor Latency (mS)</td>
<td>4.28 ± 0.49</td>
<td>4.05 ± 0.38</td>
<td>3.87± 0.36</td>
</tr>
<tr>
<td>P value &amp; significance</td>
<td>0.34000 (NS)</td>
<td>0.49948 (NS)</td>
<td>0.00244 (HS)</td>
</tr>
</tbody>
</table>

Fig. (3): Comparison between patients with and patients without subluxation in the hemiparetic side and control side as regard motor latencies.

➢ As regard motor amplitudes of axillary nerve, there was a significant statistical difference (p<0.05) between patients with and patients without subluxation, while there was no statistical significant difference (p>0.05) between patients without subluxation and control side as shown in table (6) Fig.(9).

There was a highly significant statistical difference (p<0.01) between patients with subluxation and control side as shown in table (6) Fig.(9), with decreased motor amplitudes in patients with positive subluxation than the control side, meaning a highly significant relation between shoulder subluxation and reduction of motor amplitude of axillary nerve in the hemiparetic side.
Table (7): Comparison between patients with and patients without subluxation in the hemiparetic side and control side as regard motor amplitudes.

<table>
<thead>
<tr>
<th>Motor Amplitude (mV)</th>
<th>Affected with positive subluxation</th>
<th>Affected with negative subluxation</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean ± SD</td>
<td>7.01 ± 2.74</td>
<td>9.34 ± 1.91</td>
<td>9.64 ± 1.94</td>
</tr>
<tr>
<td>P value &amp; significance</td>
<td>0.02976 (S)</td>
<td>0.93454 (NS)</td>
<td>0.00031 (HS)</td>
</tr>
</tbody>
</table>

Fig.(4): Comparison between patients with negative and patients with positive subluxation in the hemiparetic side and control side as regard motor amplitudes.

Discussion:
In our study, a highly significant relationship was found between shoulder subluxation and shoulder pain. Several authors reported a high correlation between subluxation and shoulder pain \(^{(10, 11, 12)}\). In contrary \textit{Ikai et al.} \(^{(8)}\) reported no correlation between shoulder subluxation and shoulder pain. These inconsistent findings are due to the different assessment methods and times after the stroke occurred. Studies that report a correlation between shoulder subluxation and shoulder pain were performed in an acute stage of hemiplegia while the other studies frequently reported a large range of time since stroke \(^{(13,14,8)}\). Two mechanisms appear to be plausible explanations for why shoulder subluxation may be considered a source of shoulder pain. First, periarticular tissue may become overstretched and causing pain, since the capsule and ligaments contain high concentrations of pain receptors. Second, overstretching may be the origin of painful ischemia in the tendons of the supraspinatus and the long head of biceps muscles \(^{(15)}\). Shoulder subluxation was found to be significantly related to hemorrhagic type of stroke which agreed with \(^{(12)}\). Concerning motor nerve conduction studies of the axillary nerve, comparison between affected and control sides showed a highly significant statistical difference between affected and control sides as regard:

Axillary nerve motor latency, with prolonged motor latencies were found in the affected sides than the control sides (7 cases showed prolongation in axillary nerve latency more than the cut off value), Axillary nerve motor amplitude, with lower motor amplitudes were detected in the affected sides than the control sides (6 cases showed reduction in axillary nerve amplitude below the cut off value), These results are in agreement with \textit{Tsur and Ring} \(^{(16)}\) studies in which bilateral axillary nerve conduction studies were done for 21 hemiplegic patients between 44 to 67 days from the onset of stroke, they reported delayed axillary nerve latencies of the affected paretic limbs (3.54 ms±0.38) with a significant statistical difference when compared to the non-paretic limb \(p<0.001\), and reduced axillary nerve amplitude of CMAPs (10.51 mV±0.90) with a significant statistical difference when compared to the non-paretic limb \(p<0.001\). Our results also agreed with \textit{Kingery et al.} \(^{(17)}\) study in which nerve conduction studies were done for axillary nerve in hemiplegic patients between 2 to 20 weeks from the onset of the lesion, they reported reduction in axillary nerve amplitudes in the hemiparetic side compared to the normal side with mean of \((7.7 mV±2.7)\) in the affected side. The delay in motor latencies indicates
neuropathic demyelinating affection of axillary nerves, while the reduction in amplitudes indicates axonal affection. The factors contributing to this affection are subluxation, decreased temperature, uncontrolled range of motion during passive exercise or trauma during transfer. Also, the weight of the unsupported arm may cause traction damage to the nerve. These results suggested that axillary nerve demyelinating and axonal neuropathy were mostly associated with shoulder subluxation. These results also were in agreement with the results of Tsur and Ring, both studies reported axillary nerve neuropathic affection in hemiparetic patients with shoulder subluxation. They reported abnormal values of axillary nerve in all the assessed cases. Several authors reported dysfunction of the brachial plexus and other peripheral nerves in patients with shoulder subluxation. Studied the motor conduction of suprascapular, axillary, musculocutaneous, and radial nerves of 21 hemiplegic patients with shoulder subluxation. He found that distal latencies of all tested nerves were significantly delayed stroke presents a serious threat to adults as it is a leading cause of death as well as long-term disability, according to U.S. Centers for Disease Control and Prevention.

References