

## Ultrasonographic and Electrophysiologic Assessment of Ulnar and Median Nerves in Spastic Upper Limbs in Chronic Stroke Patients

Mohamed Salem Mosalam Ali\*, Ahmad Osama Hosny, Mohamed Ebrahim Negm, Mohamed Abdelfattah Elsamahy, Iman Ahmed Yassine

Department of Neurology, Faculty of Medicine, Suez Canal University

\*Corresponding author: Mohamed Salem Mosalam Ali, Mobile: 01091031863, Email: Mohmmmed.salem.mosalem@gmail.com

### ABSTRACT

**Background:** Stroke is the primary cause of long-term disability and mortality among adults and is classified as a chronic illness. After suffering a stroke, spasticity manifests in around thirty percent of cases. **Aim:** To evaluate the effect of spasticity in chronic hemiplegic upper extremities on electrophysiological and morphological characteristics of the median and ulnar nerves.

**Subjects and methods:** This was cross-sectional research that was performed on 50 patients with chronic stroke at the Clinical Neurophysiology Unit, Neurology Department of Suez Canal University Hospitals, Suez Canal University and Ultrasonography Unit, Diagnostic Radiology Department, Ismailia, Egypt, from May 2021 to January 2023. All the patients were subjected to clinical evaluations: (Spasticity evaluation: including Brunnstrom Recovery Stages (BRS) and ultrasonographic measurements.

**Results:** There was a statistically significant positive moderate association among ultrasonographic measures of median nerve (distal) and median nerve (proximal). On the paretic side, motor conduction velocities of the median nerve were significantly delayed (mean±SD) (48.3± 9.5) when compared with the non-paretic side (mean±SD) (54.4± 8.3) and p-value (0.020) demonstrated that there were statistically significant variances. The F-wave latencies of the median nerve were significantly higher on the paretic sides (mean ± SD) (32.2± 5.1) when compared with the non-paretic (mean ± SD) (27.5± 3.9) side, and the p- value (0.011) showed that there were statistically significant differences.

**Conclusion:** We concluded that stroke peripheral nerve cases seem to be influenced electrophysiologically on the paretic side. But according to US measurements, they weren't affected.

**Keywords:** Spasticity; Ulnar nerves; Median nerves; Chronic Stroke.

### INTRODUCTION

Stroke is classified as a chronic condition. A prevalent manifestation following a stroke; spasticity manifests in around thirty percent of cases and usually happens within the initial few days to weeks. However, spasticity can manifest in the short, medium, or long-term following a stroke, its onset is extremely variable (1).

A patient recovering from a stroke can have the development of numerous complications that impair their motor, sensory, and cognitive functions (2). Muscle spasticity and peripheral neuropathies are two of the most significant complications to consider. Peripheral neuropathies may be caused by excessive usage of non-paretic hand and wrist, the use of ambulatory assistive devices, edema, or nerve compression or traction. There have been reports of lower motor neuron alterations resulting from lesions in the upper motor neurons (3,4). According to the findings of nerve conduction studies (NCS), the limb of the hemiplegic upper extremity that is affected by paretic activity has a longer motor distal latency and a lower compound muscle action potential (CMAP) amplitude than the limb that is not affected by paretic activity (4).

Over the past several years, there has been an increase in interest in the influence that spasticity has on peripheral nerves in the event of a stroke. Alterations in posture caused by flexor spasticity, causing pronation of the forearm to occur, flexion of the elbow, and flexion of the wrist, have the potential to have an effect on the

median and ulnar nerves in the upper extremities. When the wrist and finger are in the flexed position, the median nerve is subject to compression by the flexor tendons that are located within the carpal tunnel. This has been established by previous studies (4).

The utilization of ultrasound (US) to visualize peripheral nerves and soft tissues has grown in popularity over the past decade. It gives several benefits in comparison to alternative imaging modalities, including its rapidity, low cost, dynamic nature, and non-invasive nature. As there aren't recognized adverse effects associated with the utilization of ultrasound diagnosis, no specific restrictions apply. Previous research has established that peripheral nerves are susceptible to morphological alterations in a variety of clinical conditions, including polyneuropathies and carpal tunnel syndrome (5).

The purposes of this study were primary objectives: To evaluate the impact of spasticity in chronic hemiplegic upper extremities and electrophysiological characteristics of median and ulnar nerves in order to evaluate the impact of spasticity in the chronic hemiplegic upper extremities on morphological features of the median and ulnar nerves. And secondary objective: To determine the possibility of use of neuromuscular US and nerve conduction study as a marker of severity of spasticity.

### SUBJECTS AND METHODS

This was cross-sectional research that was performed on 50 patients at Clinical Neurophysiology Unit,

Neurology Department and Ultrasonography Unit, Diagnostic Radiology Department, Suez Canal University Hospitals, Ismailia, Egypt from May 2021 to January 2023.

### Inclusion criteria

Ischemic or hemorrhagic stroke, stroke onset up to six months, adults (those over eighteen years old) with spasticity in the upper extremity (proximal, distal, or hand) with a minimum score of one on the Modified Ashworth Scale (MAS).

### Exclusion criteria

Peripheral neuropathies or comorbidities that impact the peripheral nervous system (e.g., diabetes mellitus, chronic alcoholism, chronic renal failure), cognitive, psychiatric illness, or significant medical, bony deformities, or fixed contractures that may impede NCS processes, pregnancy, or concomitant neurodegenerative disorders.

### Sample size:

$$n = 2 \left[ \frac{(Z_{\alpha/2} + Z_{\beta}) * \sigma}{\mu_1 - \mu_2} \right]^2$$

n= sample size

$Z_{\alpha/2} = 1.96$  (The critical value that divides the central ninety five percent of the Z distribution from the five percent in the tail)

$Z_{\beta} = 0.84$  (The critical value that separates the lesser twenty percent of the Z distribution from the upper eighty percent)

$\sigma$  = the calculate of the standard deviation = 0.21 mm<sup>(6)</sup>

So, by calculation, the subjects required for the study, were 50 patients approximately.

## METHODS

Each of the patients performed the subsequent: Clinical assessments: (Spasticity Assessment: involving Brunnstrom Recovery Stages (BRS): The motor recovery sequences subsequent to a stroke are identified by this assessment in accordance with the degree of spasticity and the emergence of voluntary movement<sup>(7)</sup>. MAS: The Modified Ashworth Scale; a six-point scale was utilized. The possible scores are between zero and four, with lesser scores indicating normal muscle tone and greater scores signifying spasticity<sup>(8)</sup>. The Motricity Index (MI) gives an overview of the motor capabilities of the arm, while the overall arm score is calculated through the assessment of shoulder abduction, elbow flexion, and pinch grip. The maximum possible score for the upper extremity is ninety-nine<sup>(9)</sup>.

Time since stroke: six to twelve months, twelve to thirty-six months, and >thirty-six months).

### Ultrasonographic imaging

Utilizing an electronic real time linear five-to-twelve-megahertz probe (Philips HD15 Pure Wave

Linear probe L12-5), (Royal Philips, Amsterdam, The Netherlands) ultrasonographic measurements were conducted. Bilateral scans of the ulnar and median nerves were performed on the unaffected and affected extremities of the cases. Supine with the distal forearm positioned to the wrist line and five centimeters proximal of the medial epicondyle to the brachial artery, the cross-sectional area (CSA) and short and long axes of the median nerve were calculated.

Additionally, measurements were taken of the ulnar nerve five centimeters distal to the terminus of the ulnar groove.

### Electrophysiological examination

Nerve conduction studies were conducted utilizing a Nihon Kohden two-channel electromyography instrument. Bilateral measurements were taken of the motor and sensory nerve latency, sensory nerve action potentials (SNAP) and CMAP amplitudes, of ulnar nerves and latency of the F-wave of the median, and motor and sensory nerve velocity.

Patients stayed supine with their skin temperature maintained between thirty-one and thirty-four degrees Celsius. Utilizing skin surface electrodes with the tendon and belly method, CMAPs were obtained. The median nerve received stimulation at the wrist and antecubital fossa, after which the abductor pollicis brevis (APB) muscle provided CMAP and F-wave latencies. At the distal elbow and wrist, the ulnar nerve was stimulated, and compound muscle action potential and F-wave latencies were acquired from the abductor digiti minimi muscle. The antidromic procedure was implemented in order to obtain the sensory NCS.

**Ethical Considerations:** The patient provided written informed permission to participate in the trial. Permission to perform the study was received from the Neurology Department at Suez Canal University (Approval No. 4225 in April 2021), in addition to clearance from the Research Ethics Committee of the Faculty of Medicine in Suez Canal. For the purpose of conducting research involving human subjects, this study has been carried out in conformity with the Declaration of Helsinki, which is the Code of Ethics of the World Medical Association.

### Statistical analysis

Statistical analysis was conducted on the gathered data using version 26 of the SPSS (Statistical Package for the Social Sciences) program. The normal distribution of the data was assessed by employing the Shapiro Wilk test. In order to represent qualitative data, relative percentages and frequencies were utilized. For quantitative data, the mean, standard deviation (SD), median, and range were utilized. The Pearson correlation, Student t, and Mann Whitney U tests were all utilized. A significance level of P-value less than 0.05 signified a significant distinction.

**RESULTS**

**Table 1** demonstrates that a total sample of 50 cases with chronic stroke with the mean age  $57.7 \pm 18.5$  years were studied. Among them 68% were males, 76% were married and 58% had secondary education. The mean body mass index (BMI) was  $26.1 \pm 5.5$  Kg/m<sup>2</sup>.

**Table 1: Sociodemographic data among the participants.**

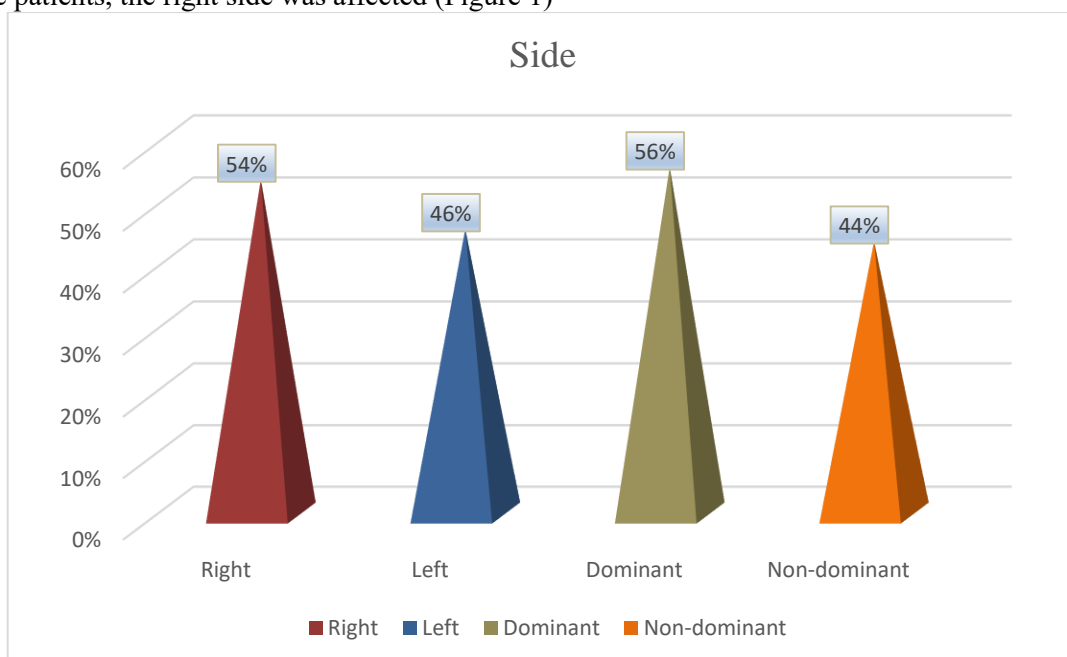
Variable		n= 50
Age (years)	Mean ± SD	57.7± 18.5
Gender	Male, n (%)	34 (68)
	Female, n (%)	16 (32)
Body mass index (Kg/m <sup>2</sup> )	Mean ± SD	26.1± 5.5
	Median (range)	25 (19, 28)
Marital status	Single, n (%)	3 (6)
	Married, n (%)	38 (76)
Education	Widow/er, n (%)	9 (18)
	Primary, n (%)	8 (16)
	Secondary, n (%)	29 (58)
	University, n (%)	13 (26)
Smoking	Smoker, n (%)	18 (36)
	Ex-smoker, n (%)	9 (18)
	Non-smoker, n (%)	23 (46)

**Table 3** shows that all participants had chronic stroke. Among them 72% had ischemic stroke and 28% had hemorrhagic stroke. The mean time since stroke was  $48.5 \pm 20.8$  months.

**Table 3: Stroke characteristics among the participants.**

Variable		n= 50
Type of stroke	Ischemic, n (%)	36 (72)
	Hemorrhagic, n (%)	14 (28)
Time since stroke (months)	6- 12	12 (24)
	12- 36	15 (30)
	> 36	23 (46)

In 54% of the patients, the right side was affected (Figure 1)



**Figure 1: Affected side among the participants.**

**Table 4** demonstrates that motor conduction velocities of the median nerve were considerably slowed on the paretic side when compared with non-paretic side. The F-wave latencies of the median nerve were significantly prolonged on the paretic sides when compared with non-paretic side.

**Table 4: Comparison between paretic and non-paretic sides regarding electrodiagnostic findings of the median nerve.**

Variable		Paretic side	Non-paretic side	P value
		Mean ± SD	Mean ± SD	
<b>N=50</b>				
<b>Median nerve</b>	CMAP amplitudes (mV)	<b>3.7± 0.9</b>	4.5± 0.6	<b>&lt;0.001*</b>
	SNAP amplitudes (mV)	28.7± 10.1	30.2± 9.9	0.220
	Motor nerve latency (msec)	4.7± 0.2	4.2± 0.6	<b>&lt;0.001*</b>
	Sensory nerve latency (msec)	3.1± 0.4	2.9± 0.5	0.03*
	Motor nerve velocity (m/sec)	48.3± 9.5	54.4± 8.3	<b>&lt;0.001*</b>
	Sensory nerve velocity (m/sec)	48.7± 6.6	50.1± 9.4	0.391
	F-wave latency (msec)	32.2± 5.1	27.5± 3.9	<b>&lt;0.001*</b>

\*: p is significant

**Table 5** shows that there was statistically significant variance among paretic and non-paretic sides regarding CMAP amplitudes (mV) and Sensory nerve latency (msec), while there wasn't statistically significant regarding other electrodiagnostic findings of the ulnar nerve.

**Table 5: Comparison between paretic and non-paretic sides regarding electrodiagnostic findings of the ulnar nerve.**

Variable		Paretic side	Non-paretic side	P-value
		Mean ± SD	Mean ± SD	
<b>N=50</b>				
<b>Ulnar nerve</b>	CMAP amplitudes (mV)	5.7± 1.4	6.6± 1.6	<b>&lt;0.001*</b>
	SNAP amplitudes (mV)	17.5± 11.1	19.3± 8.9	0.166
	Motor nerve latency (msec)	3.2± 0.6	3.2± 0.3	1
	Sensory nerve latency (msec)	3.6± 0.7	3.1± 0.5	<b>&lt;0.001*</b>
	Motor nerve velocity (m/sec)	50.9± 10.5	51.4± 9.3	0.801
	Sensory nerve velocity (m/sec)	50.6± 9.6	50.1± 9.5	0.794
	F-wave latency (msec)	28.3± 6.2	28.5± 4.4	0.853

\*: p is significant

**Table 6** shows that there was significant positive moderate association between weight and both of distal and proximal ultrasonographic measures of median nerve. There was significant positive weak association between BMI and ultrasonographic measures of median nerve (proximal). There was significant positive moderate association between ultrasonographic measures of median nerve; distal and proximal.

**Table 6: Correlation between patients' clinical data and ultrasonographic measurements.**

Variable	Ultrasonographic measures of median nerve (distal)		Ultrasonographic measures of median nerve (proximal)		Ultrasonographic measures of ulnar nerve-elbow	
	r	p	r	p	r	p
Age	-412.0	0.162	-0.602	0.162	-0.770	0.522
Weight (Kg)	0.632	<b>0.010*</b>	0.412	<b>0.022*</b>	0.435	0.100
Height (cm)	0.311	0.255	0.319	0.119	-0.511	0.877
Body mass index(kg/m2)	0.320	0.151	0.326	<b>0.009*</b>	0.328	0.261
Brunnstrom recovery stages	-0.234	0.610	0.144	0.400	0.188	0.155
Modified Ashworth Scale (MAS)	-0.818	0.129	-0.619	0.615	-0.429	0.091
Motricity Index(MI)	-0.651	0.303	0.523	0.212	0.722	0.772
Ultrasonographic measures of median nerve (distal)	-	-	0.512	<b>0.042*</b>	0.409	0.302
Ultrasonographic measures of median nerve (proximal)	-	-	-	-	0.823	0.411
Ultrasonographic measures of ulnar nerve-elbow	-	-	-	-	0.517	0.088

\*p is significant at <0.05.

## DISCUSSION

In this research, the average age of stroke cases was  $57.7 \pm 18.5$  years. A study goes in line with our finding, **Uğurlu et al.**, showed that among 33 cases with stroke the mean age was  $55.6 \pm 11.3$  and ranging from 26 to 72 years <sup>(10)</sup>.

In this study, there were (34) 68% males and (16) 32% females. So, males were more among the stroke patients. In accordance with our results, **Naseri et al.** found a higher prevalence of males among stroke patients (57.1%) than females (42.9%) <sup>(11)</sup>.

In this study, there were 54% had affected right side while there 46% had affected left side. Among the participants, only one was left-handed. So, there were 56% had affected dominant side. This goes in line with **İsnaç et al.** who discovered that fifty percent of the sample (n=30) of cases had a left hemisphere stroke that affected their dominant (right) side. <sup>(12)</sup>.

In this study, among the cases 72% had ischemic stroke and 28% had hemorrhagic stroke. Consistent with our findings, **Uğurlu et al.** found that among stroke patients, four (12.1%) had hemorrhagic and the others (87.9%) had ischemic stroke <sup>(10)</sup>. Similar to our results, When the stroke types were examined by **İsnaç et al.** it was discovered that 63.7 percent (n = 19) were of the ischemic cases, and 36.7 percent (n= 11) of them were hemorrhagic <sup>(12)</sup>.

according to our research, on the paretic side, the median nerve motor conduction velocities were much slower than on the normal side, (Mean  $\pm$  SD =  $48.3 \pm 9.5$ ) when compared with non-paretic side, (Mean  $\pm$  SD =  $54.4 \pm 8.3$ ) and the p value showed that there were statistically significant differences. **Uğurlu et al.** reported findings consistent with our own study, indicating that the paretic sides exhibited slowed motor conduction velocity of the median nerve, prolonged distal latency, and prolonged median nerve F-wave latency in comparison to the non-paretic sides (all P-value  $< 0.05$ ) <sup>(10)</sup>.

The median nerve F-wave latencies on the paretic side ( $32.2 \pm 5.1$ ) were significantly more than those on the non-paretic side ( $27.5 \pm 3.9$ ). An additional study conducted by **Picelli et al.** investigated nerve conduction studies and US parameters of the median and ulnar nerves in cases with spastic upper limbs who had suffered chronic stroke. This study provided further support for the accuracy of our results. The F-wave minimal latency in the affected and unaffected upper limbs was compared, and the results presented that there were significant variances in the median nerve (the affected were  $22.7 \pm 2.2$  milliseconds, while the unaffected were  $25.3 \pm 3.2$  milliseconds;  $p=0.045$ ) <sup>(4)</sup>.

Our study revealed that there wasn't statistically significant variation among paretic and non-paretic sides regarding ulnar nerve measures.

**Lukács** evaluated the electrophysiological changes in the early and late phases following ischemic stroke, which agreed with our findings. Regarding the measurement of the median and ulnar nerves, distal

motor and sensory latencies, as well as motor and sensory action potential amplitudes, there wasn't statistically significant distinction observed among the hemiparetic and unaffected sides <sup>(13)</sup>.

Unlike our results and according to **Uğurlu et al.**, a study was conducted on hemiplegic patients, where median, ulnar, and tibial nerve NCS were performed. The study found that the amplitudes of the CMAP of the median and ulnar nerves were significantly lower on the plegic side of the affected extremities <sup>(10)</sup>.

There was significant positive moderate association among weight and ultrasonographic measures of median nerve (distal). There was significant positive moderate correlation among weight and ultrasonographic measures of median nerve (proximal). There was significant positive weak association among BMI and ultrasonographic measures of median nerve (proximal). There was significant positive moderate association among ultrasonographic measures of median nerve (distal) and median nerve (proximal).

Multiple significant associations were identified among ulnar compound potential of muscular action, distal ulnar motor latency, median nerve cross-sectional area, and Modified Ashworth Scale in other investigations <sup>(14,15)</sup>.

## STRENGTH AND LIMITATIONS

This study possesses a number of benefits and disadvantages. Individuals who suffer from systemic disabilities that can cause polyneuropathy or those with electrophysiologically confirmed polyneuropathy were excluded, which constituted the study's greatest strength .

Concerning initial limitations, the duration since the stroke showed a broad range distribution. Determining the effect of the duration of time since the stroke on the present findings is thus challenging. Furthermore, the present study fails to go further into each of the present results for daily living activities.

## CONCLUSION

We concluded that stroke peripheral nerve cases seem to be influenced electrophysiologically on the paretic side. But according to US measurements, they were not affected.

## DECLARATIONS

- **Consent for publication:** I confirm that all authors have given their consent to submit the work.
- **Availability of data and material:** Available
- **Competing interests:** None
- **Funding:** No fund
- **Conflicts of interest:** Absence of any conflicts of interest.

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