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# Neurophysiological Changes in Post Stroke Spasticity Patients

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## Abstract

**Background** A stroke is a serious life-threatening medical condition that occurs when the blood supply to part of the brain is disrupted. Spasticity is a state, in which, there is an increase in muscle tone or stiffness, which might be associated with movement and speech disorders. Spasticity is usually caused by damage to nerve pathways within the brain or spinal cord that control muscle movement. The H-reflex is a compound electromyographic (EMG) response composed of group Ia (and large group II) afferents from muscle spindles (and Golgi tendon organs) that project monosynaptically (and to a lesser extent di- and tri-synaptically) to spinal efferent α-motoneurons (αMN), which in turn activate the muscle after an electrical current is applied to the nerve.

**Objective** To assess the Medical Research Council (MRC) scale in post stroke spasticity patients and to estimate the neurophysiological changes in those patients regarding H-reflex and Hmax/Mmax ratio.

**Methods** A randomized clinical trial involves 50 patients with stroke of both sexes. Their age ranged from (30-60 years old) suffering from upper and lower limb spasticity. They were assessed by the MRC scale to determine the grade of power of muscle. The neurophysiological test is done by using the H-reflex for both upper and lower limbs (flexor carpi radialis and soleus muscles, respectively). The maximum amplitude was measured from the peak of the first positive deflection to the peak of the negative one. The latency and the Hmax/Mmax ratio were also measured.

**Results** The result of this study showed a significant decrease in MRC in spastic patients (P value ≤0.001). H-reflex latency in lower limbs was significantly decreased from (30.3±2.4 ms) in the normal population to (18.5±1.3) in the spastic patient. The H reflex amplitude in the lower limb was significantly increased in post stroke spasticity patients (P value ≤0.05). In the upper limbs H-reflex latency in flexor carpi radialis was also significantly decreased. Regarding H reflex amplitude of the upper limb in the spastic patient was significantly increased. There is a noticeable increase in the H/M ratio in post stroke spasticity patient which was (0.51±0.92) with a significant P value of 0.005.

**Conclusion** Patient with post stroke spasticity had decreased in MRC scale, while H-reflex has decreased in latency and increased in amplitude, as well as decreases in Hmax/Mmax Ratio in both upper and lower limbs.

**Keywords** Post stroke spasticity, H-reflex latency, H-reflex amplitude, H/M ratio, MRC scale.

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**List of abbreviations:** FCR = Flexor carpi radialis, H/M = H-reflex/Mmax, MRC = Medical Research Council scale, PSS = Post stroke spasticity, UMN = Upper motor neuron

## Introduction

Spasticity was first described by Lance in 1980 as a motor disorder characterized by a velocity-dependent increase in tonic

stretch reflexes (muscle tone), with exaggerated tendon jerks, resulting from hyper-excitability of the neurons involved in stretch reflex, as a component of the upper motor neuron (UMN) syndrome [1]. Spasticity is common after stroke, which is ranging from 30% to 80% of stroke survivors [2,3].

The post stroke spasticity (PSS) is more often found in the flexor muscles of the upper limb (fingers, wrist, and elbow flexors) and extensor muscles of the lower limb (knee and ankle extensors). Wissel et al., observed that PSS developed most often in the elbow (79%), wrist (66%), ankle (66%), and shoulder (58%) [4].

It is assumed that all positive features of the UMN syndromes are related to changes in the balance between excitatory and inhibitory signals to the spinal motor neuron pool leading additionally to changes in soft tissue and muscle fiber density [5]. The disruption of spinal interneuron-mediated influences might reduce the inhibition of the antagonist muscle and increase the action potentials in the sensory neurons, thus leading to excessive muscle activation and hence spasticity develops [6].

Impaired movement is usually presented in stroke patients, which may be due to a combination of UMN syndromes, including spasticity, weakness, loss of coordination and dexterity, and sustained muscle contraction. Patients with spasticity exhibit impaired functions and have a poor quality of life [7].

The assessment of muscle power of upper and lower limbs by Medical Research Council (MRC) scale. The MRC scale of muscle strength uses a score of 0 to 5 to grade the power of a particular muscle group in relation to the movement of a single joint [8].

Paul Hoffmann described the H-reflex more than a century ago [9], and it was later named after him [10]. The H-reflex has long been and continues to be a significant tool for studying neuromotor control processes and clarifying neuromotor deficits [5,11].

The H-reflex is a compound electromyographic (EMG) response composed of group Ia (and large group II) afferents from muscle spindles (and Golgi tendon organs) that project monosynaptically (and to a lesser extent di- and tri-synaptically) to spinal efferent α-motoneurons (αMN), which then activate the muscle when an electrical current is applied to the nerve [12,13].

This study aimed to assess the motor state of PSS patients by using the MRC scale. Also, to explain the neurophysiological changes by H-reflex in upper and lower limbs in patients with PSS. In addition to measure the Hmax/Mmax in patients with post stroke spasticity.

## Methods

This is a randomized clinical trial conducted at the Clinical Neurophysiology Unit in Baghdad Teaching Hospital, Medical City from January to July 2022.

Fifty patients with the chronic attack of stroke of both sexes and aged 30 to 60 years complaining of upper and lower limb PSS were included in this study.

### Inclusion criteria

1. Patients with chronic stroke (more than 3 months after the onset of stroke) either ischemic or hemorrhagic stroke diagnosed by CT scan with focal spasticity.
2. The age ranged from 30-60 years old.

### Exclusion criteria

1. Patient with another neurologic disease, diabetes, orthopedic or rheumatological diseases.
2. Patient who takes muscle relaxant drugs.

For each patient neurological examination was done, scoring the muscle power of the upper and lower limbs by using the MRC scale. And grading of muscle power from (0-5) by MRC scale.

H-reflex for each patient was measured in the morning by EMG/EP machine (MEDTRONIC Keypoint, Denmark). The temperature of the neurophysiological unit was between 25-28°C during the examination, the patient temperature was between (32-34) °C which is measured by using a thermometer.

H-reflex was measured for both upper and lower limbs (flexor carpi radialis (FCR) and soleus muscles respectively). Using EMG/EP machine (MEDTRONIC Keypoint, Denmark). The maneuver was as follows:

A patient lying in the supine position and the paretic upper limb suspended over the bed. The patient head was resting on a pillow [14]. Before attaching the electrodes, the skin was cleaned with alcohol, ether, and acetone to reduce the impedance at the skin-electrode interface.

A pulse width of 0.5-1 ms was delivered at a frequency of one pulse per 2-3 s to the median nerve. The optimal site of stimulation was determined by moving the cathode until the site to elicit an H-reflex in FCR with the largest amplitude at a given intensity was identified. The input-output relations for the H-reflex and M-wave were determined, by progressively increasing the current in steps of 0.5-1 mA (five stimulations/step) until the M-wave amplitude reached a plateau (Mmax).

Electrical stimuli (1 ms duration) applied to the tibial nerve were delivered via a constant current stimulator. The optimal site of stimulation was determined by moving the cathode until the site to elicit an H-reflex in the soleus with the largest amplitude at a given intensity was identified. The input-output relations for the H-reflex and M-wave were determined, by progressively increasing the current in steps of 0.5-1 mA (five stimulations/step) until the M-wave amplitude reached a plateau (Mmax).

The H reflex latency was estimated by the first artifact of the stimulation till reached the base line first deflection.

The onset latency of the H-reflex was measured from the stimulus artifact to the first deflection from the baseline. The maximum amplitude measured from the peak of the positive to the peak of the negative deflections, and the Hmax/Mmax amplitude ratio was also measured.

**Statistical analysis**

Microsoft Excel 2016 (Microsoft Corporation, USA) and IBM SPSS (Statistical Package for Social Sciences) version 26 (IBM Corporation, USA) were used. A statistically significant p value was ≤0.05.

## Results

Data of the study group showed the mean age of the study group was 47.68±7.99 years with a range of 30-60. Twenty-four of them (48%) and females were 26 (52%). MRC scale was (2.38±0.73) in a spastic patient, this value was lower than normal subjects (4.5±0.5) As the results shown by Paternostro et al., [15] with significant p value <0.001 (Table 1)

**Table 1. Data of the study group (n=50)**

| Variable | | Value | P value |
|---|---|---|---|
| Age (years) | Mean±SD | 47.68±7.99 | |
| | Range | 30-60 | |
| Gender | Male, N (%) | 24 (48%) | |
| | Female N (%) | 26 (52%) | |
| Medical Research Counsel scale | Spastic patients | 2.38±0.73 | <0.001 |
| | Normal [15] | 4.5±0.5 | |

The normal value of H-reflex latency was (30.3± 2.4 ms) in lower limbs, as Buschbacher [16] mentioned in his study, while in spastic patient in PSS was (18.5±1.3 ms) with a significant difference of 0.001. The normal value of H-reflex amplitude in lower limb was (2.4±1.4), while in the spastic patient was (3.56±0.06) with significant value of 0.05 [17]. In the upper

limbs, the normal value of H-reflex latency in FCR was (15.88 ±1.27 ms) [18], but the results of H-reflex latency in the current study was (8.68±1.05) with a significant value of 0.001. The upper limb H-reflex amplitude in spastic patient was (1.23±0.25), which was significantly different compared to normal value (0.5±0.1) of Bodofsky [19]. There is a noticeable decrease in Hmax/Mmax ratio in PSS patient, which was (0.51±0.92) with significant P value of 0.005, while Hmax/Mmax ratio should not exceed 0.75 in normal individuals [20] (Table 2).

**Table 2. Data of the neurophysiological tests (n=50)**

| Variable | | Mean±SD | P value |
|---|---|---|---|
| H-reflex latency lower limbs (ms) | Spastic patients<br>Normal [16] | 30.3±2.4<br>18.5±1.3 | 0.001 |
| H-reflex amplitude lower limbs (mv) | Spastic patients<br>Normal [17] | 2.4±1.4<br>3.56±0.06 | 0.05 |
| H-reflex latency in upper limbs (ms) | Spastic patients<br>Normal [18] | 15.88±1.27<br>8.68±1.05 | 0.001 |
| H-reflex amplitude upper limbs (mv) | Spastic patients<br>Normal [19] | 0.5±0.1<br>1.23±0.25 | 0.05 |
| Hmax/Mmax ratio | Spastic patients<br>Normal [20] | 0.51±0.92<br>0.75 | 0.005 |

## Discussion

Post stroke spasticity is assumed that all positive features of the UMN syndromes are related to changes in the balance between excitatory and inhibitory signals to the spinal motor neuron pool leading additionally to changes in soft tissue and muscle fiber density [21]. Thus, spasticity can be divided into two components: spasticity mediated by the neural reflex and spasticity due to muscle contracture, which is often referred to as non-reflex spasticity. Damage to the UMNs disrupts communication between the brain and the spinal cord, resulting in a state of net disinhibition of the spinal reflexes [22].

The disruption of spinal interneuron-mediated influences might reduce the inhibition of the antagonist muscle and increase the action potentials in the sensory neurons, thus leading to excessive muscle activation, hence PSS results [6]. However, spasticity may also be explained by changes in the mechanical properties of muscles and not only by neural-mediated hyperreflexia. Several studies support the involvement of peripheral tissues, such as muscle fibers and connective tissue, in spasticity [23,24]. Currently, the commonly used spasticity assessment methods are the clinical scale methods such as the MRC scale, which could provide some useful information on whether spasticity exists or not and what the severity of spasticity is with several levels (such as 0-4) as what we found in results, the MRC scale was decreased significantly in PSS patients [8].

The result of this study showed that H-reflex latency decreased in post stroke spasticity patients in the upper and lower extremities this can be explained by the damage of interneuron inhibitory motoneuron or disruption of stimulant sensory neurons in spinal cord, so, the reflex will be delayed and shown as decreased latency in recording of H-Reflex.

The H-reflex amplitude was increased due to increased spasticity of muscle due to increased α motor neuron activation, which is shown in the neurophysiological record of the reflex as increase in the peak of the waves.

In conclusion, patient with PSS had decreased muscle power elicited by a decrease in MRC

scale while H-reflex had decreased in latency and increased in amplitude, as well as Hmax/Mmax ratio was also in both upper limbs and the lower limbs.

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## Author contribution

Dr. Fawzi: had collected the patients and made all the neurophysiological tests. Dr. Hamdan: put the idea of the research. Dr. Al-Gawwam: had referred the patients to the neurophysiological unit. Dr. Jaafar: had written the manuscript and made the statistical analysis.

## Conflict of interest

The authors declare there is no conflicts of interest.

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## References

1. Lance JW. What is spasticity? Lancet. 1990; 335(8689): 606. doi: 10.1016/0140-6736(90)90389-m.
2. Wissel J, Manack A, Brainin M. Toward an epidemiology of poststroke spasticity. Neurology. 2013; 80(3 Suppl 2): S13-9. doi: 10.1212/WNL.0b013e3182762448.
3. Opheim A, Danielsson A, Alt Murphy M, et al. Upper-limb spasticity during the first year after stroke: stroke arm longitudinal study at the University of Gothenburg. Am J Phys Med Rehabil. 2014; 93(10): 884-96. doi: 10.1097/PHM.0000000000000157.
4. Wissel J, Schelosky LD, Scott J, et al. Early development of spasticity following stroke: a prospective, observational trial. J Neurol. 2010; 257(7): 1067-72. doi: 10.1007/s00415-010-5463-1.
5. Burke D. Clinical uses of H reflexes of upper and lower limb muscles. Clin Neurophysiol Pract. 2016; 1: 9-17. doi: 10.1016/j.cnp.2016.02.003.
6. Mukherjee A, Chakravarty A. Spasticity mechanisms - for the clinician. Front Neurol. 2010; 1: 149. doi: 10.3389/fneur.2010.00149.
7. Sheffler LR, Chae J. Hemiparetic Gait. Phys Med Rehabil Clin N Am. 2015; 26(4): 611-23. doi: 10.1016/j.pmr.2015.06.006.
8. Bickerstaff ER. Muscle power examination. In: Prasad K, Spillane J, Yadav R (eds). Bickerstaff Neurological examination in clinical practice. Oxford London-Edinburgh: Blackwell Scientific Publications; 1998. p. 117-37.
9. Hoffmann P. Über die Beziehungen der SehnenreflexezurwillkürliehenBewegung und zum Tonus. Z Biol. 1918; 68: 351-70;
10. Magladery JW, McDougal DB Jr. Electrophysiological studies of nerve and reflex activity in normal man. I. Identification of certain reflexes in the electromyogram and the conduction velocity of peripheral nerve fibers. Bull Johns Hopkins Hosp. 1950; 86(5): 265-90.
11. Knikou M. The H-reflex as a probe: pathways and pitfalls. J Neurosci Methods. 2008; 171(1): 1-12. doi: 10.1016/j.jneumeth.2008.02.012.
12. Thompson AK, Wolpaw JR. Operant conditioning of spinal reflexes: from basic science to clinical therapy. Front Integr Neurosci. 2014; 8: 25. doi: 10.3389/fnint.2014.00025.
13. Thompson AK, Wolpaw JR. Targeted neuroplasticity for rehabilitation. Prog Brain Res. 2015; 218: 157-72. doi: 10.1016/bs.pbr.2015.02.002.
14. Dumitru D, Amato AA, Zwartz MJ. Nerve conduction studies. In: Dumitru D, Amato AA, Zwarts MJ (eds). Electrodiagnostic Medicine. 2nd ed. Philadelphia, PA: Hanley & Belfus; 2002.
15. Paternostro-Sluga T, Grim-Stieger M, Posch M, et al. Reliability and validity of the Medical Research Council (MRC) scale and a modified scale for testing muscle strength in patients with radial palsy. J Rehabil Med. 2008; 40(8): 665-71. doi: 10.2340/16501977-0235.
16. Buschbacher RM. Normal range for H-reflex recording from the calf muscles. Am J Phys Med Rehabil. 1999; 78(6 Suppl): S75-9. doi: 10.1097/00002060-199911001-00014.
17. Jerath N, Kimura J. Chapter 15 - F wave, A wave, H reflex, and blink reflex. Clinical neurophysiology: Basis and technical aspects. Handbook of clinical neurology,2019; 160: 225-39. doi: https://doi.org/10.1016/B978-0-444-64032-1.00015-1.
18. Miller TA, Newall AR, Jackson DA. H-reflexes in the upper extremity and the effects of voluntary contraction. Electromyogr Clin Neurophysiol. 1995; 35(2): 121-8.
19. Bodofsky EB. Contraction-induced upper extremity H reflexes: normative values. Arch Phys Med Rehabil. 1999; 80(5): 562-5. doi: 10.1016/s0003-9993(99)90200-9.
20. Matthews WB. Ratio of maximum H reflex to maximum M response as a measure of spasticity. J Neurol Neurosurg Psychiatry. 1966; 29(3): 201-4. doi: 10.1136/jnnp.29.3.201.
21. Burke D, Wissel J, Donnan GA. Pathophysiology of spasticity in stroke. Neurology. 2013; 80(3 Suppl 2): S20-6. doi: 10.1212/WNL.0b013e31827624a7.

**22.** Bhimani R, Anderson L. Clinical understanding of spasticity: implications for practice. Rehabil Res Pract. 2014; 2014: 279175. doi: 10.1155/2014/279175.

**23.** Tabary JC, Tabary C, Tardieu C, et al. Physiological and structural changes in the cat's soleus muscle due to immobilization at different lengths by plaster casts. J Physiol. 1972; 224(1): 231-44. doi: 10.1113/jphysiol.1972.sp009891.

**24.** Sinkjaer T, Toft E, Larsen K, et al. Non-reflex and reflex mediated ankle joint stiffness in multiple sclerosis patients with spasticity. Muscle Nerve. 1993; 16(1): 69-76. doi: 10.1002/mus.880160112.

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