ORIGINAL ARTICLE

IJPHY

Comparing The Effects Of Electrical Stimulation Of Antagonist At Motor Point And The Agonist At Musculotendinous Junction On Spasticity Of Biceps Muscle In Chronic MCA Stroke Patients. A Pilot Study

[*1]Rahul Shaik
[2]Kannuru Ramya Jyothi
[3]P. Sasikala

ABSTRACT

Background: Neuro-physiological conflict between reciprocal inhibition and nonreciprocal inhibition phenomena during electrical stimulation of spastic muscles may give scope for new technological advances in electrical stimulation techniques for spasticity reduction. This study compares the effects of antagonistic motor point stimulation and agonist stimulation at muscle–tendon junctions on biceps spasticity.

Methods: 11 post-stroke patients between 40 and 60 years of age were randomly assigned to two groups. In the antagonist-stimulation group, electrical stimulation is given to the triceps muscles, up to 30 contractions in a single session with the frequency of 40 Hz-100Hz and 0.7ms pulse duration. In the agonist-stimulation group, the patient's musculotendinous junction of spastic biceps is marked with the help of diagnostic ultrasound before starting the treatment. Spastic biceps was stimulated at the musculotendinous junction up to 30 contractions in a single session with a frequency of 40Hz to 70Hz and 0.7ms pulse duration. Both groups received single-treatment sessions per day for five days a week for four weeks. Stretch pain on VAS and ENMG (H reflex and F/M ratio) were recorded on the first day and after four weeks of treatment.

Results: Extremely significant reduction in spasticity (p=<0.0001) and stretch pain (p=<0.0001) were noted after both types of stimulations. Subjects in the agonistic stimulation group showed significant improvement in both outcome measures compared to the antagonistic stimulation group, with a mean difference of 2.965ms in H reflex and 1.235 in F/M ratio. More significant improvement in pain was noted in the agonistic stimulation group (p=0.0002). These results demonstrated a significant spasticity reduction in agonistic stimulation compared to agonistic stimulation.

Conclusion: Agonist stimulation at a musculotendinous junction is more effective than antagonistic stimulation at a motor point in reducing spasticity in MCA stroke subjects.

Keywords: Agonist-stimulation, Golgi Tendon Organ, Nonreciprocal inhibition, spasticity.

Received 18th May 2023, accepted 27th August 2023, published 09th September 2023

www.ijphy.org

10.15621/ijphy/2023/v10i3/1417

[2]GEMS College of Physiotherapy, Ragolu, Srikakulam, A.P, India.
[3]Professor, Department of Physiology, Narayana Medical College, Nellore, A.P, India.

CORRESPONDING AUTHOR

[*1]Rahul Shaik

GEMS College of Physiotherapy, Ragolu, Srikakulam, A.P, India.
Email: rahulbhagath09@gmail.com

INTRODUCTION

Stroke is the second leading cause of death worldwide [1] and accounts for almost 5% of all disabilities [2]. According to the global burden of diseases in 2016, the global lifetime risk of stroke increased from 22.6% in 1990 to 24.9% in 2016, and it's prevalent from the age of 25 years. Spasticity is a significant contributing factor to disability after stroke, with the prevalence ranging from 30% to 80% of stroke survivors. In upper limbs, spasticity will develop most commonly in the elbow (79%), wrist (66%), ankle (60%) and shoulder (5%). Spastic muscle may develop physical changes such as shortening and contracture that results in muscle stiffness [3]. Leading to reduced ROM. Usually, spasticity may reduce at least after one year without electrical stimulation, but with electrical stimulation, it recovers within three months [4]. Generally, the muscle spindle responds to increased stretch and Golgi [5]. Tendon organs respond to increased contraction and cause relaxation of stimulated muscle [6]. There is evidence for the electrical stimulation of antagonistic muscles at motor points and its role in reducing spasticity [7-11]. The recent focus on stimulation of the Golgi Tendon Organ in inhibiting the same muscle gave an insight into spasticity reduction through a nonreciprocal inhibitory mechanism [12,13]. So, there is a need to study the difference between physiology and its implications of two mechanisms, reciprocal and nonreciprocal inhibition, in the management of spasticity with electrical stimulation.

METHODOLOGY

All the ethical issues were noted, and ethical clearance was obtained from the research institution. This study is a part of the original trial, registered in the Clinical Trials Registry of India and Dr. NTR University of Health Sciences, Vijayawada, Andhra Pradesh, as a Ph. D research work.

All the safety precautions during electrotherapy were carried out according to the standard protocols. The current intensities used were too low to cause electric shock. Subjects were explained their right to discontinue the study during the research if they found any discomfort. MCA stroke patients with a first-time history between the ages of 40 and 60 years who agreed to participate in the study were recruited for this study. Biceps muscle spasticity between 1+ and 2 was set as a minimum criterion to include the subjects. This study included patients of any gender not less than six months after the occurrence of hemiplegia. Subjects with any hand dominancy were included. All the subjects have normal tactile and pain sensation.

Patients who have a pacemaker, a history of heart failure, epilepsy, broken skin at the stimulation site, and impaired mental status were excluded from this study. Patients with metal implants in affected upper limbs, chronic wounds, and other musculoskeletal and vascular-related conditions were excluded from this study.

Recovery in the spasticity was measured by ENMG changes in spastic biceps muscles in terms of changes in H- reflex and mF/Mratio. A visual Analog Scale was used to evaluate the level of pain perception while stretching the spastic biceps muscles.

PROCEDURE

Informed consent was obtained from patients and their attendants before enrolling the subjects. Eleven participants were selected for the study and divided into two groups using simple random techniques. Subjects in both groups were given the same rehabilitation protocols other than electrotherapy. Group A (5 subjects): Patients received antagonistic muscle stimulation (triceps stimulation) at the motor point in this group. Group B (6 subjects): Patients received agonistic muscle stimulation at the musculotendinous junction in this group. Data on all outcome measures was collected before starting treatment and after the end of 4th week.

Intervention for subjects in group A:

The patients were asked to sit with a semi-flexed elbow and arm supported on a high table, and electrical stimulation was given at the motor point of the Triceps muscle of the spastic upper limb. Thirty contractions in a single session were given five days a week and continued up to 4 weeks. The stimulation frequency is 40 Hz-100Hz, and the pulse duration is 0.7ms, using a square wave electrical pulse. The intensity was adjusted according to the patient's tolerance, and a visible contraction of the stimulated muscle was achieved in each stimulus.

Intervention for subjects in group B:

The musculotendinous junction of the biceps muscle was marked with the help of Diagnostic Ultrasound. During stimulation, patients were asked to sit with extended spastic upper limb supported on a high table, and electrical stimulation was given to the spastic biceps muscle at the musculotendinous junction up to 30 contractions in a single session for five days a week and continued up to 4 weeks.

The stimulation was given by placing the active electrode over the musculotendinous junction of the spastic biceps muscle. The stimulation Frequency is 40Hz to 70Hz, with an intensity of 50 mA or more, a pulse duration of 0.7 ms, and the square wave was used. The intensity will be set according to the patient's tolerance.

Data Analysis and Results:

Highly significant reductions in spasticity (neuro-physiological measures) and stretch pain were noted after both stimulations. Neurophysiological measures like H reflex and mF/M ratio of spastic biceps were significantly reduced in subjects treated with agonistic stimulation when compared to that of antagonistic stimulation technique. Considerable improvement in stretch pain was also noted in the subjects of the agonistic stimulation group compared to the antagonistic stimulation group.

Table 1: Pre and post-test comparison of outcome measures.

		Antagonist stimulation	Agonist stimulation	Between group comparison
H Reflex	Pre	24.947 ± 2.339	24.593 ± 2.125	0.5519
	post	29.085 ± 1.305	32.046 ± 1.5344	**<0.0001**
Within group comparison		**<0.0001**	**<0.0001**	
F/M Ratio	Pre	8.3984 ± 1.77011	8.4566 ± 1.07709	0.8376
	Post	6.229 ± 0.809127	4.9875 ±0.7798	**<0.0001**
Within group comparison		**<0.0001**	**<0.0001**	
VAS	Pre	8.7 ± 0.9944	8.767 ± 0.7279	0.7359
	post	6.00 ± 0.6948	5.767 ± 0.9643	**0.0002**
Within group comparison		**<0.0001**	**<0.0001**	

DISCUSSION

Electrical stimulation has been widely studied for spasticity suppression. However, the results have been contradictory. The different outcomes may be related to the wide variety of stimulation parameters, application methods, and quantification measurements used. Alfieri reported that spasticity was dramatically reduced during electrical stimulation by stimulating the antagonistic muscles of the wrist and finger flexors of hemiplegic patients [14,15]. However, some studies indicated that either increased or unchanged spasticity was found after ES [16,17].

These findings are in line with the previous study of S C Chen, Y S Chen (2004), in which stimulation of the musculotendinous junction of Gastrocnemius muscle in spastic stroke patients with twenty-four neurologically stable stroke patients revealed that surface electrical stimulation on the muscle-tendon junctions of spastic Gastrocnemius muscles is an effective way to suppress spasticity at the metameric site[13]. Delwaide and colleagues found that the Ib inhibitory effect was depressed or absent in the spastic limbs of stroke patients [18]. Reduction of the Ib inhibitory effect is one of the path mechanisms responsible for spasticity in stroke patients. So, stimulation of the Ib fibers may lead to a reduction of muscle tone. Alon and De Dominico (1987) explained that with stimulation of the agonist, the spastic muscle may inhibit its excitation due to muscle fatigue or autogenic inhibition through the increased response of the Golgi tendon organ. The researchers have hypothesized the reduction in spasticity as a result of the effects of antidromically propagated action potentials evoked in the motor neuron axons to spastic muscle. The inhibition of the Renshaw cells, the inhibitory interneurons inhibit the activity of the agonist (spastic muscle) itself [19].

CONCLUSION

Agonist stimulation at musculotendinous junction is more effective than antagonistic-stimulation at motor point in reducing spasticity (neuro-muscular physiological measures) in MCA stroke subjects. Clinical tests for spasticity were not considered in this study due to the possibility of less correlation between neuro-muscular physiological measures and clinical tests of spasticity due to less intervention duration.

REFERENCES

[1] Murray, Christopher J. L, Lopez, Alan D, World Health Organization, World Bank & Harvard School of Public Health. (996) Global health statistics: a compendium of incidence, prevalence and mortality estimates for over 200 conditions /Christophe J.L. Murray, Alan D. Lopez. Harvard School of Public Health.

[2] Kamalakannan, S., Gudlavalleti, A., Gudlavalleti, V., Goenka, S., & Kuper, H. (2017). Incidence & prevalence of stroke in India: A systematic review. The Indian journal of medical research, 146(2),175–185.

[3] Wissel, J., Schelosky, L. D., Scott, J., Christe, W., Faiss, J. H., & Mueller, J. (2010). Early development of spasticity following stroke: a prospective, observational trial. Journal of neurology, 257(7), 1067–1072. https://doi.org/10.1007/s00415-010-5463-1

[4] Crago, P. E., & Makowski, N. S. (2012). Muscle response to simultaneous stimulated and physiological action potential trains--a simulation study. Annual International Conference of the IEEE Engineering in Medicine and Biology Society. IEEE Engineering in Medicine and Biology Society. Annual International Conference, 2012, 1839– 1842.

[5] Edin, B. B., & Vallbo, A. B. (1988). Stretch sensitization of human muscle spindles. The Journal of physiology, 400, 101–111.

[6] Gregory, J. E., & Proske, U. (1979). The responses of Golgi tendon organs to stimulation of different combinations of motor units. The Journal of physiology, 295, 251–262.

[7] Merilynmackenzie-knapp, (1999). Electrical stimulation in the early stroke rehabilitation of upper limb with in attention, Australian Journal of Physiotherapy. Volume 45, Issue 3, 223-227.https://doi.org/10.1016/S0004-9514(14)60353-6

[8] Yang, Y. R., Mi, P. L., Huang, S. F., Chiu, S. L., Liu, Y. C., & Wang, R. Y. (2018). Effects of neuromuscular electrical stimulation on gait performance in chronic stroke with inadequate ankle control - A randomized controlled trial. PloS one, 13(12), e0208609.

[9] Vafadar, A. K., Côté, J. N., & Archambault, P. S. (2015). Effectiveness of functional electrical stimulation in improving clinical outcomes in the upper arm following stroke: a systematic review and meta-analysis. BioMed research international, 2015, 729768.

[10] Li-Ling Hope Pan, Wen-Wen Yang (2018). Effects of 8-week sensory electrical stimulation combined with motor training on EEG-EMG coherence and

motor function in individuals with stroke , Scientific Reports, volume 8, 9217.
[11] Cuesta-Gómez, A., Molina-Rueda, F., Carratala-Tejada, M., Imatz-Ojanguren, E., Torricelli, D., & Miangolarra-Page, J. C. (2017). The Use of Functional Electrical Stimulation on the Upper Limb and Interscapular Muscles of Patients with Stroke for the Improvement of Reaching Movements: A Feasibility Study. Frontiers in neurology, 8, 186.
[12] Mukherjee, A., & Chakravarty, A. (2010). Spasticity mechanisms - for the clinician. Frontiers in neurology, 1, 149.
[13] Chen, S. C., Chen, Y. L., Chen, C. J., Lai, C. H., Chiang, W. H., & Chen, W. L. (2005). Effects of surface electrical stimulation on the muscle-tendon junction of spastic gastrocnemius in stroke patients. Disability and rehabilitation, 27(3), 105–110.
[14] Alfieri V. (1982). Electrical treatment of spasticity. Reflex tonic activity in hemiplegic patients and selected specific electrostimulation. Scandinavian journal of rehabilitation medicine, 14(4), 177– 182
[15] Bakhtiary, A. H., & Fatemy, E. (2008). Does electrical stimulation reduce spasticity after stroke? A randomized controlled study. Clinical rehabilitation, 22(5), 418–425.
[16] Kanaka, T. S., & Kumar, M. M. (1990). Neural stimulation for spinal spasticity. Paraplegia, 28(6), 399– 405.
[17] Robinson, C. J., Kett, N. A., & Bolam, J. M. (1988). Spasticity in spinal cord injured patients: 2. Initial measures and long-term effects of surface electrical stimulation. Archives of physical medicine and rehabilitation, 69(10), 862–868.
[18] Delwaide, P. J., & Oliver, E. (1988). Short-latency autogenic inhibition (IB inhibition) in human spasticity. Journal of neurology, neurosurgery, and psychiatry, 51(12), 1546–1550.
[19] Roger Nelson M. Roger, Currier P. Dean & Karen W. (1999). Clinical electrotherapy. 3rd edition.