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ANTAGONIST VERSUS AGONIST MUSCLE NEUROMUSCULAR ELECTRICAL STIMULATION ON SPASTICITY IN STROKE PATIENTS

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ABSTRACT

Background: Spasticity is the common problem encountered in the treatment of hemiplegic patients. Various treatment techniques have been developed to reduce spasticity, neuromuscular electrical stimulation is one of them. Several studies have proved that stimulation of either spastic muscle or stimulation of antagonist muscle to spastic muscle results in a reduction of spasticity. However, there is no literature available on a comparative study to suggest which method is more effective in spasticity reduction. Hence this study was undertaken to find out the efficacy of each technique and to compare the two techniques of Neuromuscular electrical stimulation to determine the most effective technique.

Methods: In this study with pre and post-experimental design 30 post-stroke patients were selected and they were randomly assigned into two groups. Group A received antagonist (triceps) muscle Neuromuscular electrical stimulation and Group B received agonist (biceps brachii) muscle Neuromuscular electrical stimulation for 2 weeks, one session per day for a duration of 30 minutes. Outcome measures were recorded using modified Ashworth scale and deep tendon reflex grading scale.

Results: Statistical analysis was carried out by using Wilcoxon signed rank sum test and Mann-Whitney U test at 0.05 level of significance. There was a significant recovery after the treatment based on the Modified Ashworth Scale and deep tendon reflex grading scale scores before and after the intervention within the groups and between the groups with p-value < 0.001. The group receiving the antagonist muscle neuromuscular electrical stimulation showed better recovery with a mean difference of 1.8 and 1.2 on Modified Ashworth Scale and reflex grading scale respectively.

Conclusion: The study concluded that both the techniques resulted in reduction of spasticity and on comparison it was found that antagonist muscle (triceps) Neuromuscular electrical stimulation reduced spasticity more effectively than the agonist muscle Neuromuscular electrical stimulation

Keywords: Stroke; spasticity; NMES; antagonist muscle; agonist muscle; MAS.

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INTRODUCTION

Stroke is the largest single cause of neurological disability [1]. Spasticity is one of the long-term disabilities caused by stroke. Spasticity is the abnormal muscle tone following a stroke, which interferes with the normal postural control and makes active movement difficult, often requiring rehabilitation. Spasticity may be defined as a motor disorder characterized by velocity-dependent increased resistance to passive movement and hyperactivity of stretch reflexes [2]. About 20% to 30% of all stroke victims suffer from spasticity [3-5].

Spasticity limits the voluntary motor capabilities of the patient if left untreated complications such as joint dysfunction, contracture pain and undesirable compensatory movements develop leading to disability [1]. The marked functional impairments associated with spasticity have led healthcare providers from many areas, to search for clinical procedures to combat this problem.

The pathophysiologic basis of spasticity is due to abnormalities at different levels, including stretch reflex arc, spinal segmental influence, supraspinal mechanisms and mechanical factors [6,7].

There are various traditional treatment techniques available to treat spasticity which includes, prolonged stretching, proprioceptive neuromuscular techniques, cryotherapy etc. One of the recent treatment techniques includes neuromuscular electrical stimulation [8]. Neuromuscular electrical stimulation is a promising adjunct to physical therapy intervention and recent literature has created new interest in its potential [9].

Levine MG (1952), is one of the earliest researchers to report the benefits of antagonist muscle neuromuscular electrical stimulation on spasticity who reported a relaxation of hypertonicity within several seconds of initiating stimulation, as evidenced by a reduction in opposition to passive stretch of spastic muscle [10]. Robinson CJ (1998) documented long-term effects of surface electrical stimulation of spastic quadriceps muscle itself in 31 spinal cord injured patients and reported most of the participants had a decrease in spasticity after 8 weeks of electrical stimulation [11].

Thus, inhibition of spasticity can be accomplished through stimulation of either the agonist (spastic muscle) or its antagonist muscle [12,13]. However, there are no comparative studies done to determine which method of stimulation is more effective in spasticity reduction. So this study is aimed at determining the effect of Neuromuscular electrical stimulation of antagonist muscles over the effect of Neuromuscular electrical stimulation of agonist muscles in spasticity reduction in stroke patients. Hence the purpose of the study is to determine the most effective method of Neuromuscular electrical stimulation for spasticity reduction, which can be used as an adjunct to conventional treatment to improve the efficacy of rehabilitation.

MATERIALS AND METHODS

A total of 30 post-stroke patients were recruited from the

K.S. Hegde charitable hospital, Deralakatte, Mangalore, for this study, with the objective to compare the efficacy of neuromuscular electrical stimulation of antagonists (triceps) with neuromuscular electrical stimulation of agonists (biceps brachii) in spasticity reduction

Age of the patients participated ranged from 45 to 75 years. Patients having Biceps brachii muscle spasticity of grade 2 as per the modified Ashworth scale with duration of stroke within 6 months were included.

Subjects were randomly assigned into 2 groups using computer-generated table of random numbers. There were 10 male and 5 female patients in Group-A. In Group-B there were 8 males and 7 female patients. Group A received antagonist muscle (triceps) NMES and Group B received agonist muscle (Biceps brachii) NMES for 30 minutes each session. The electrodes were placed along the longitudinal axis of the muscle bulk, one on the origin and the other electrode placed on the motor point of the muscle. Intensity used was sufficient to evoke minimum visible muscle contraction. Patients in both the groups received single-treatment session per day for 5 days per week for 2 weeks.

Group A received antagonist muscle (triceps) NMES. Position of the patient: side lying on the normal side, head was slightly flexed and positioned over a thin pillow, trunk was straight with a pillow to support from the back, the affected side shoulder was protracted, flexed with the arm forward over a pillow in a relaxed supported position, elbow was slightly flexed, forearm pronated and wrist and fingers in neutral position. The affected lower limb was positioned on a pillow with hip and knee in slight flexion. Electrode placement: one electrode was placed on the origin of triceps i.e. upper part of the posterior humerus and the other electrode was kept on the motor point of the triceps muscle.

Group B received agonist muscle (Biceps brachii) NMES. Patient was positioned in supine lying position with the neck in a neutral position on a thin pillow, the trunk was positioned straight in line with the neck. The affected side shoulder was protracted on a pillow with the arm and forearm placed straight by the side with the elbow and wrist in neutral position, properly supported and relaxed. A small pillow was placed under the hip to prevent retraction of the pelvis and lateral rotation of lower limb. Electrode placement: one on the origin of the biceps brachii muscle and the other electrode was placed on the motor point of the biceps brachii muscle. The part to be treated was exposed and placed in the above-mentioned position, so that the therapist could observe the muscle contraction.

After the administration of neuromuscular electrical stimulation, patients of both the groups received conventional treatment for the duration of 30 minutes which included- Slow sustained passive stretching of spastic biceps muscle for 30 seconds with 3 repetitions and PNF techniques Hold relax and slow reversal techniques for upper limb were given.

Outcome measures were recorded on the first day before the treatment and repeated on the 15th day after the treat-

ment using Modified Ashworth Scale¹⁴ and deep tendon Reflex Grading Scale¹⁵. Both the outcome measures were assessed by a physical therapist, who was blinded for the study and has experience in using both the scales.

Statistical analysis

The data obtained were statistically analyzed using SPSS software version 17.0. the descriptive statistics were used to describe the mean and SD of demographic data. The differences in the mean of the outcome measures within the groups and between the groups were detected by

Using Wilcoxon signed rank sum test and Mann- Whitney U test at 0.05 level of significance.

To perform the computation of data, categorical variables of the modified Ashworth scale were assigned numerical values, designated as “Computed modified Ashworth scale scores” in this study i.e. modified Ashworth scale value 0=0, MAS value 1=1, modified Ashworth scale value 1+=2, modified Ashworth scale value 2=3, modified Ashworth scale Value 3=4, modified Ashworth scale Value 4=5.

RESULTS

Table 1: Descriptive data of the subjects participated in the study

	Group: A (Antagonist NMES)	Group: B (Agonist NMES)
Total Number of Subjects	15	15
Male	10	8
Female	5	7
Mean Age (years)	60	57.8
Mean duration from the time of onset of stroke (in months)	5.8	4.9
Side affected: Left Hemiplegia	9	8
Right Hemiplegia	6	7

The results revealed the reduction in spasticity on modified Ashworth scale found to be very highly significant in the antagonist NMES group with a mean difference of 1.80 (SD = 1.01) at $p < 0.001$ level.

There was a highly significant reduction of spasticity on MAS in agonist group with a mean difference of 1.00 (SD = 1.07) at $p < 0.007$.

There was a very highly significant reduction in spasticity on reflex grading scale in antagonist group with a mean difference of 1.20 (SD = 0.56) at $p < 0.001$.

There was a highly significant reduction in spasticity on reflex grading scale in the agonist group with a mean difference of 0.60 (SD = 0.63) at $p < 0.007$.

Table 2: Comparison of the effect of NMES on spasticity between pre & post-NMES using mean and standard deviation of MAS and reflex grading scale within the group- A and within the group B.

GRADING SCALE	GROUP		N	Mean	Std. Deviation
MAS	Antagonist (Group A)	PRE	15	3.0000	.0000
		POST	15	1.2000	1.0142
	Agonist (Group B)	PRE	15	3.0000	.0000
		POST	15	2.0000	1.0690
Reflex Grading scale	Antagonist (Group A)	PRE	15	3.8667	.3519
		POST	15	2.6667	.7237
	Agonist (Group B)	PRE	15	3.9333	.2582
		POST	15	3.3333	.6172

Graph 1: Comparison of effect of NMES on spasticity between pre & post NMES within group A (antagonist) & Group B (agonist) in terms of MAS & Reflex grading scale

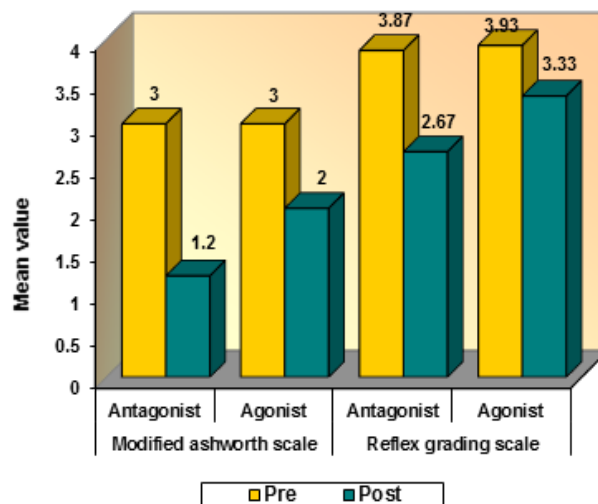
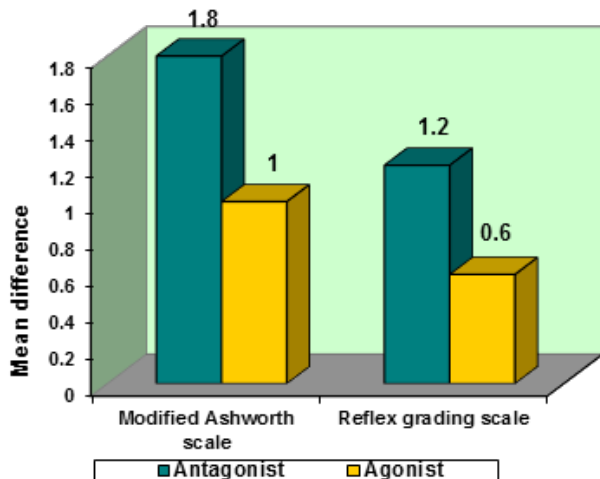


Table 3: Pre and Post NMES values of mean difference of MAS and reflex grading scale in Group:A and Group: B and its level of significance

GRADING SCALE	GROUP		Paired Differences		Z	p-value
			Mean difference	Std. Deviation		
MAS	Antagonist (Group A)	PRE -POST	1.8000	1.0142	3.228	.001 vhs
	Agonist (Group B)	PRE -POST	1.0000	1.0690	2.719	0.007 hs
Reflex Grading scale	Antagonist (Group A)	PRE-POST	1.2000	.5606	3.448	.001 vhs
	Agonist (Group B)	PRE-POST	.6000	.6325	2.714	0.007 hs

Graph 2: Comparison of mean difference of reduction of spasticity between antagonist NMES and agonist NMES group



DISCUSSION

In this study, statistical analysis revealed a significant reduction in spasticity after the completion of neuromuscular electrical stimulation for 15 days in both the groups on modified Ashworth scale and reflex grading scale scores.

On comparison of the pre-stimulation and post-stimulation scores of group A, it was found that there was a significant difference in modified Ashworth score ($z=3.228$, $p<0.001$) and reflex grading score ($z=3.448$, $p<0.001$).

These findings are in line with the previous study of Sahin N et al. (2012) who have conducted a randomized controlled study on the efficacy of electrical stimulation in reducing the post-stroke spasticity and concluded that NMES given together with stretching of the wrist extensor muscles was more effective than stretching of the wrist extensor muscles alone in reducing spasticity [16].

Also, these results are inconsistent with the finding of Bakhtiary AH et al, (2008) who conducted a randomized controlled clinical trial on 40 stroke patients aged between 42 – 65 years with ankle plantar flexor spasticity and demonstrated that electrical stimulation of antagonist muscles may help to reduce spasticity effectively in stroke patients [13].

The significant reduction in spasticity in antagonist muscle neuromuscular stimulation can also be attributed to neural plasticity caused by it as reported by Motta-Oishi AA et al (2013) [17].

On comparison of the pre-stimulation and post-stimulation scores of group B, it was found that there was a significant difference in modified Ashworth score ($z=2.719$, $p<0.007$) and reflex grading score ($z=2.714$, $p<0.007$).

These findings are in accordance with Theodore I. (1996), who conducted a study on “the effect of Neuromuscular Electrical Stimulation in Reducing Tone” by applying NMES for 10 minutes on spastic (agonist) wrist flexor muscle group. This study demonstrated the reduced resistance to passive movement of the spastic wrist flexor muscles after neuromuscular electrical stimulation¹⁸. According to

Alon and De Dominico (1987) 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.

When the mean values were compared, antagonist group (Group:A) showed a greater reduction in spasticity when compared to the agonist group (Group-B).

The neurophysiologic rationale for the effectiveness of neuromuscular electrical stimulation to the antagonist muscle seems to rest on the principle of “reciprocal inhibition” [19,20]. As neuromuscular electrical stimulation is applied to the antagonist muscle, the large diameter Ia muscle spindle afferent fibers originating in the muscle are excited. The action potentials generated in these fibers are transmitted to the spinal cord and excite the spinal interneurons, which in turn inhibit the activity in the motor neurons to the spastic muscles.

The exact mechanism by which neuromuscular electrical stimulation of spastic muscles may reduce spasticity is unknown. 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 agonist (spastic muscle) itself [20].

This study gives a new dimension in the treatment of stroke patients for normalizing tone, that is by using neuromuscular electrical stimulation to the antagonist of the spastic muscle, spasticity can be reduced more effectively.

CONCLUSION

Based on the statistical outcome in this study, it can be concluded that there is a significant difference in the effects of neuromuscular electrical stimulation on spasticity between the group receiving antagonist neuromuscular electrical stimulation and the group receiving agonist neuromuscular electrical stimulation. Also, from the results obtained in this study, we conclude that antagonist muscle neuromuscular electrical stimulation reduces spasticity more effectively when compared to agonist neuromuscular electrical stimulation.

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