



Effect Of Neuro- Muscular Electrical Stimulation (NMES) In The Management Of Spasticity In Post- Stroke Patients A Review Of Literature

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Abstract: Background and purpose – The effectiveness of neuromuscular electrical stimulation (NMES) has been investigated in post-stroke patients. The present study review examines whether NMES, gives during acute stroke, was more effective in reducing spasticity without any other physical therapy modality and interventions in post-stroke patients. **Methods-** 26 articles were searched for this review of literature after editing and filtering of data a total number of 10 articles were included in the study which were both experimental and review of literature. Articles were included that have sufficient information related to effect of electrical stimulation (NMES) in management of spasticity in post-stroke patients. **Results-** There was a total of ten trails regarding the application of NMES in post-stroke patient's spasticity. All of them were non-pharmacological and interventional. All of them focused on the effectiveness of NMES on the spasticity during post-stroke. The majority of the included studies utilized the MAS and / or electromyographic recordings in order to quantify the effect of NMES. In most cases, spasticity was decreased for at least two weeks post intervention. **Conclusions- NMES** can be used solo without combination with different physical therapy modalities in order to produce optimal result. Based on the existing literature, the authors believe that future studies on the subject of NMES in the management of post- stroke spasticity should focus on carefully examining each electrical parameter.

Index Terms- Stroke, spasticity, electrical stimulation.

I. INTRODUCTION

A cerebrovascular accident is a sudden, focal neurological deficit that lasts more than 24 hours. A stroke is an immediate or chronic manifestation of symptoms induced by a localised impairment of arterial brain circulation. (Tanovic, et al)

Strokes are the primary cause of serious long term disability in the United State. The American heart association predicts and overall stroke prevalence of 6.8 million Americans over the age of 20 years, accounting for 2.8% of population, based on the National health and nutrition examination survey data from 2017 to 2010. (Go AS, et al ;)

One of the main symptoms in the majority of post-stroke patients is spasticity, a motor disorder characterized by a velocity – dependent increase in tonic stretch reflexes with overstated ligament jerks, which could be a ordinary sign of upper motor neurone disorder. Spasticity is considered a “positive “include of UMNS due to the loss of hindrance of lower motor neurone pathways coming about from a sensory – motion control disorder in the muscle regulation system. Post-stroke patients more often than not show with an mixed, lateralized sensory and motor disorder with their influenced upper limb exhibiting a hypertonic flexion pattern whole the equilateral lower limb exhibits a hypertonic extension pattern.(Chasiotis, A., Giannopapas, et, al)

Spasticity is very variable during the post-stroke phase, and studies have shown that it develops and reaches its peak at 1 to 3 months post-stroke. Although the neural components of spasticity reaches its peak at 3 months post- stroke, the neural component may increase over time resulting in an increase spasticity incidence at 6 months after stroke. Spasticity is more commonly observed in the lower limb’s extensor muscles (knee and ankle extensor) and upper limb flexor muscles (finger, wrist and elbow flexors). (Marcolino, M.A.Z., et al.)

A cerebrovascular Affecting the motor cortex or internal capsule, it commonly causes initial hypotonia and absent tendon jerk, followed several days and weeks later by spastic hypertonia in the antigravity muscles .The upper limb adopts an adducted posture at the shoulder and a flexed posture at the elbow and wrist with the finger flexed into the palm. In the lower limb, there is hip and knee extension, with plantar-flexion at the ankle.

Spasticity, while a separate issue, is also involved in muscle contraction. Muscle contraction is the loss of movement over time caused by abnormally shortening of soft tissue structures. This restricts joint mobility and causes pain and stiffness. (Bavikatte G, et; al)

Spasticity and muscular contraction are separate issues, but spasticity also plays a role in the development of contractures. In some patients, contractures may even increase the severity of spasticity. (Bavikatte G, et; al)

Ability to move joints may be impaired in individuals following a stroke. This can happen within 2-6 weeks and get worse after 1-3 months. It may continue to get worse over 3-6 months. (Kuo C-L, et; al)

Electrical stimulation is a supplementary modality of several forms that is used to increase muscle strength, relief discomfort and diminished hypertonia in the affected limbs Neuro- muscular nerve stimulation (NMES) is a type of electrical stimulation that causes muscle contraction by applying an electrical stimulus to the distal section of a specific nerve. Because the electrical excitability of lower motor unit is usually impact, NMES can be used to stimulate the neuromuscular activity of the effected limbs with either direct stimulation of the affected muscle or stimulation of their antagonist alone or in parallel with robotic assistive devices. (Chasiotis, A., Giannopapas, et, al)

The purpose of this review is to investigate the effectiveness of NMES (electrical stimulation) in the management of spasticity in post stroke patients.

II. METHODOLOGY

A review of literature regarding the use and effectiveness of NMES in the management of spasticity in post-stroke patients.

Studies reporting interventions with NMES on spasticity were included while protocols presenting NMES treatments combined with physiotherapeutic and / or pharmacological techniques were excluded from the literature search. Most included studies based their design and the sample stratification using the modified Ashworth scale (MAS), hence the database search was adjusted accordingly. A review search was performed using the PubMed, Google Scholar, and sci-hub with the terms: electrical stimulation (NMES), stroke, spasticity, from 2010 to 2023. The corresponding flowchart is presented in *figure 1* and the included studies are presented in *Table 1*.

2.1 INCLUSION CRITERIA: The inclusion criteria will be the article on electrical stimulation effect on spasticity in stroke patients, NMES effect on spasticity in stroke patients, TENS affect on spasticity in stroke patients.

Most included studies were based their design and the sample stratification using the modified Ashworth Scale (MAS).

Articles are include those are published after 2010-2023 in English.

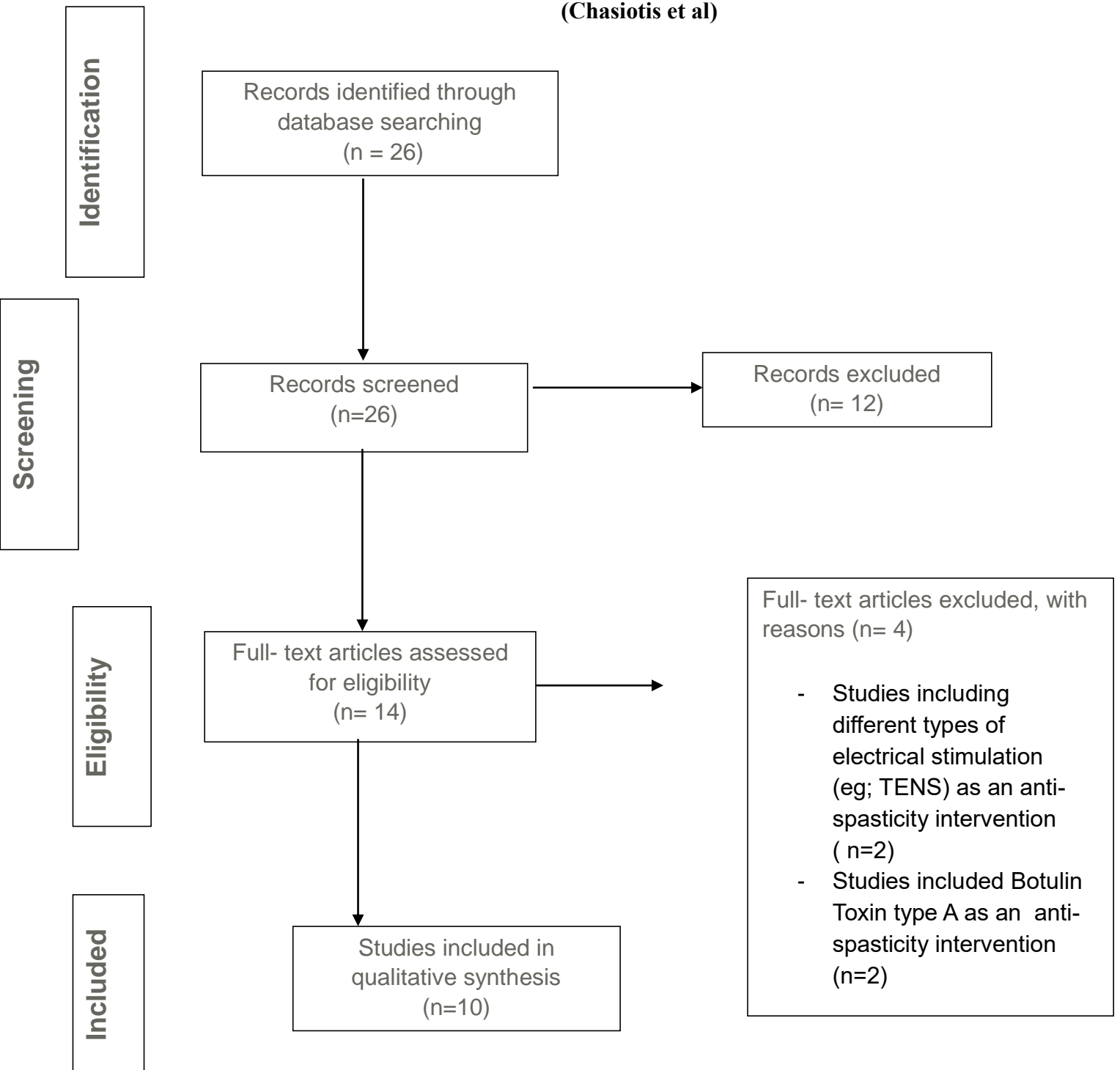
2.2 EXCLUSION CRITERIA: exclusion criteria will be the articles those are published in other languages and data related to study characteristics (i.e. author and year of publication) and copy that is also excluded.

NMES treatment combine with physiotherapeutic and / or pharmacological techniques were excluded from the literature search.

2.3

DATA COLLECTION PROCEDURE

(Chasiotis et al)



study	of study	patients	Electrical parameters	Results
Page MJ et al, 2021	Single-blinded RCT	37 stroke patients	WF: Symmetrical, biphasic. F: 30 Hz. PD: 300µsec. Ramp up/down: 1sec/1sec. ON/OFF time: 5sec/5sec. I: 0-90mA. AS: supraspinatus, deltoid. EC TD: 30min/session. 5 days/week, 3 weeks	Decreased MAS for 3 months p<.05 increased fma after weeks
Lin Z et al, 2011	RCT	50 stroke patients	WF: Pulsed, F: 100 Hz, PD: 0.1 msec (100µsec), Width between 2 pulses: 0.9msec (900µsec), Width between 2 cyclic pulses: 3 msec. Resting time: 9sec, AS: ECR, ECU. TD: 15min/session, 5 days/week, 1 month	Decreased MAS (p=.001). *Increased FIM (p=.028)
Boyaci et al, 2013	RCT	31 stroke patients	GROUP A: EMG-triggered NMES (active NMES).WF: Symmetrical, biphasic. F: 50Hz. PD: 200µsec. I: 20-47mA. Ramp up/down: 2sec/2sec. Sensitivity EMG biofeedback = 0-100µV. AS: ECC + ECU TD: 45 min/session, 5 days/week, 3 weeks. GROUP B: (passive NMES).WF: Symmetrical, Biphasic. F: 50Hz. PD: 200µsec. I: 20-47mA Ramp up/down: 2sec/2sec. Sensitivity EMG biofeedback = 0-100µV. AS: ECR + ECU. TD: 45min/session, 5 days/week, 3 weeks.	Improved ROM & FMA in passive NMES (P<.05 significant changes on the other measurements)
Malhotra et al, 2013	RCT	90 stroke patients	WF:300 µsec. ON/OFF time: 15sec/15sec. Ramp Up/Down: 6sec/6sec. F: 40Hz I: maximum muscle contraction. AS: wrist+ finger extensors. TD: 30min, 2 times/day, 5 days/week, 6 weeks	Decreased VRS (p=.02), *No changes in spasticity & stiffness (p=.02).
Lee et al, 2015	Double-blind sham RCT	39 stroke patients	WF: Symmetrical, rectangular, biphasic F: 30Hz PD: 200µsec AS: FD-ED, Pronator-Supinator TD: 20-30min	Decreased MAS (p=.017). *No improvement in FMA, MAS, SIS and ADL (p=.017).
Wang et al, 2016	RCT	72 stroke patients	WF: symmetrical, biphasic, square. PD: 200µsec, F: 20Hz, ON/OFF time: 5sec/5sec, I: sensory threshold, motor threshold & full-movement, NMES TD: 30min, 2 times/day, 5 days/week, 4 weeks AS: EC + EDL	Decreased CSS in full-movement NMES (p<.05 aads in full-movement nmes differences tugt>.05)

Kotaro et al, 2017	Review	67 stroke patients	WF: monophasic, biphasic, and burst (polyphasic) waves. PD: 150–300 μ s, on/ off time: 15 sec, for 30 min, 6 weeks	Decrease in spasticity
Yang et al, 2018	RCT	25 stroke patients	WF: biphasic, square. F: 50Hz. PD: 200 μ sec. ON/OFF time: 5/15sec. I: 50-0mV (full ROM contraction). AS: TA, MG TD:20min, 3 times/week, 7 weeks	Decreased MAS in NMES-TA group in static (p=0.028) and dynamic spasticity (p=.025). *Increased muscle strength in NMES-TA group (p=0.009). *Increased ankle plantar flexion during push off in NMES-TA group (p=.015).
Mano et al, 2021	RCT	61 stroke patients	WF: Symmetrical, rectangular. Biphasic F: 35Hz (1st group) & 50Hz (2nd group). PD:300 μ sec. I: maximum muscle contraction. Ramp Up/down: 2sec/2sec (the 1st week) & 1sec/1sec (rest of the study). Contraction-relaxation time: 5-25sec (2 weeks), 5-20sec (3rd week), 5-15sec (4thweek), 5-10sec (5th, 6thweek) & 5-5sec (final weeks) TD: 20min (first 2 sessions) & 30min (rest sessions), 3days/week, 8weeks & 2-months follow-up. AS:ECR (L+B) + EDC	Improved ROM in 35Hz and in 50Hz with greater results in 35Hz (p<0.01). *Improved grip strength in 35Hz and 50Hz with greater results in 35Hz (p=0.016). *Decreased MAS in 35Hz than 50Hz (p=0.002) *Larger EMG amplitude extensors in 35Hz (p>0.05) and ACR extensors in 50Hz (p>0.05). *Improved BI in 35Hz (p<0.01).
Sattam et al, 2023	A pilot RCT	19 stroke patients	WF: Sinusoidal, burst, constant current. F: 80 Hz ON/ OFF TIME: 5s/15s Treatment time: 30 min, 4 weeks	*Decrease in MAS (p= 0.008) *Improves 10 MWT (p= 0.028) *NMES reduce spastic and dynamic spasticity.

III. RESULT

There were a total of ten trails regarding the application of NMES in post-stroke patient's spasticity. All of them were non-pharmacological and interventional. All of them focused on the effectiveness of NMES on the spasticity during post-stroke. The majority of the included studies utilized the MAS and / or electromyographic recordings in order to quantify the effect of NMES.

A study by Lin and colleagues examined the effectiveness of NMES on spastic upper limb. All patients received neuromuscular electrical stimulation for 30 min, 5 days a week for 3 weeks. Measurements were re- corded before treatment, at the 2nd and 3rd week of treatment and 1, 3 and 6 months after treatment ended. Significant improvements were found in Modified Ashworth Scale scores after the 3rd week of treatment. (Page MJ et al.)

similar result regarding the management of post-stroke spasticity were presented by sahin and colleagues using higher frequency NMES (100Hz, 100 μ sec PD) in 50 post-stroke patients, which led to decreased spasticity for approximately one month post- intervention($p<.001$)(Lin Z et al.)

A study by kotaro and colleagues concluded that NMES is used not only for muscle strengthening and motor recovery of paralysed limbs as introduced in this review but also for reducing spasticity.(kotaro et al)

A study by sattam and colleagues concluded that NMES, as applied in the active NMES group, resulted in significant improvements in plantarflexor spasticity, walking ability, and functional ambulation. However, only plantarflexor spasticity showed a significant improvement during the comparison between groups.(sattam et al)

A study by Mano and colleagues concluded that improved ROM in 35Hz and in 50Hz with greater results in 35Hz ($p<0.01$). *Improved grip strength in 35Hz and 50Hz with greater results in 35Hz ($p=0.016$). *Decreased MAS in 35Hz than 50Hz

($p=0.002$) *Larger EMG amplitude extensors in 35Hz ($p>0.05$) and ACR extensors in 50Hz ($p>0.05$). *Improved BI in 35Hz ($p<0.01$). (Mano et al.)

In a mixed population RCT, Wang and colleagues examined the effects of 50 Hz, 200 μ sec, symmetrical biphasic square NMES combined with full active ROM movement in the reduction of spasticity focusing on the plantar flexors. Patients received 30-minute sessions of NMES on the motor points of the extensor hallucis and digitorum longus twice a day, five days per week for four weeks. The results were evaluated using the composite spasticity scale (CSS), ankle active dorsiflexion score (AADS), and walking time in the timed up and go test (TUGT). The authors reported significant reduction in the CSS scores ($p<.05$) and improvement in AADS scores ($p<.05$), which maintained at the two-week follow-up. (wang et al)

Yang and colleagues assessed the efficacy of 50 Hz, 200 μ sec PD, biphasic square waveform NMES on either the dorsi flexors or plantar flexors muscles during walking and gait performance in 25 stroke patients with inadequate ankle control. The participants were divided into three equal groups. The experimental group received 20 minutes of NMES either on the tibialis anterior muscle (NMES-TA) or the medial gastrocnemius muscle (NMES-MG) while the control group received 20 minutes of ROM and stretching exercises. The static

and dynamic spasticity of ankle plantar flexors during gait was assessed using the MAS and EMG activity of the muscles and their lengthening velocities. The authors reported a statistically significant reduction in static ($p=.028$) and dynamic spasticity ($p=.025$) of the ankle plantar flexors of the NMES-TA group as well as improvements in muscle strength ($p=.009$) and active ankle plantar flexion during push off ($p=.015$). (Yang et al)

IV. DISCUSSION

This review of literature showed that NMES can be used solo without combination of other physiotherapy modalities in order to produce optimal results, that provides improvements in spasticity and increase in range of motion in post-stroke patients.

The application of NMES in the rehabilitation of individuals with neurological illnesses has grown in recent years. Its spasticity-reducing benefits may be explained by its impacts on raising Ib fiber activity through processes that promote Renshaw cell recurrent inhibition, antagonist reciprocal inhibition, and enhancing cutaneous sensory impulses. (Stein et al.)

NMES can be used to improve spasticity in such patients. This would result in a greater benefit from the motor control programmes as well as a better improvement in functional activity. NMES cannot prevent the use of the affected hand, but it can improve and facilitate the patient in using the affected hand for everyday activities, resulting in improved range of motion. (Chan MK et al.)

V. Conclusion

NMES without combine with other interventions and physiotherapy modalities, solo provides the improvement in spasticity and improves the muscle strength. This data provides support for further use of NMES as an additional therapy technique to reduce spasticity in post-stroke patients. But the conduction the large scale and high quality RCTs are needed to establish its true efficiency

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