International Journal of Environmental Sciences
ISSN: 2229-7359
Vol. 11 No. 20s, 2025
https://theaspd.com/index.php

Effectiveness Of Surged Faradic Stimulation Combined With Static Cock-Up Splinting In Reducing Wrist Flexor Spasticity Among Chronic Stroke Patients

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Abstract
Background: *Wrist flexion spasticity is a common motor impairment seen in patients following a stroke, often affecting upper limb functionality and restricting voluntary wrist extension. Management strategies like stretching and splinting are widely used but may not be adequate when applied in isolation. Surged faradic stimulation has been proposed as an effective adjunct to enhance motor recovery through muscle re-education.*
Objective: *To compare the effectiveness of combining surged faradic muscle stimulation on wrist extensors with wrist flexor stretching and static cock-up splinting, versus using splinting and stretching alone in reducing wrist flexion spasticity among chronic stroke patients.*
Methods: *A total of 40 patients with chronic stroke and wrist flexor spasticity (MAS grades 2–3) were randomly assigned into two equal groups. Group A received wrist flexor stretching followed by the application of a static cock-up splint, while Group B received the same protocol along with additional surged faradic muscle stimulation on wrist extensors. The intervention period lasted for three weeks. Outcome measures included the Modified Ashworth Scale (MAS) and Active Range of Motion (AROM) of wrist extension.*
Results: *Both groups demonstrated statistically significant improvements in MAS scores and wrist AROM after the intervention period ($p < 0.05$). However, Group B showed more pronounced improvements in both outcome measures compared to Group A.*
Conclusion: *The combination of surged faradic muscle stimulation with static cock-up splinting and stretching proves to be more effective than splinting and stretching alone in reducing wrist flexion spasticity and enhancing wrist extension in chronic stroke patients.*
Keywords: *Stroke, spasticity, wrist extension, faradic stimulation, static cock-up splint, rehabilitation.*

INTRODUCTION

Stroke is a major global health burden and a leading cause of long-term disability. Among the motor impairments that follow a cerebrovascular accident, spasticity of the upper extremity—particularly involving the wrist flexors—poses significant challenges to functional independence and quality of life[1]. Wrist flexion spasticity limits voluntary movement and hinders participation in activities of daily living, making its management a critical component of stroke rehabilitation[2].
The upper limb, especially the wrist and hand, plays a central role in purposeful movement. After a stroke, the imbalance between muscle groups often leads to hyperactivity of wrist flexors, resulting in a flexed resting posture and reduced range of motion[3]. Chronic spasticity, if left unaddressed, contributes to soft tissue contractures, joint deformities, and further functional decline[4]. Managing this condition is thus not only about improving mobility but also preventing long-term structural changes that can exacerbate disability.

Traditional physiotherapeutic interventions for spasticity include stretching, positioning, and splinting[5]. Static cock-up splints are commonly used to maintain the wrist in a functional position, reduce abnormal tone, and prevent contracture formation[6]. These splints assist in redistributing muscle forces and facilitate better alignment during movement[7]. Moreover, when combined with consistent stretching of the wrist flexor muscles, splinting can contribute to lengthening of shortened soft tissues and improved passive range of motion[8].

However, passive modalities alone may not be sufficient to reverse or significantly reduce neural spasticity in all cases. Over the years, adjunctive therapies aimed at neuromuscular facilitation have gained prominence. Among these, electrical stimulation has shown considerable promise in activating weakened or inhibited muscles and promoting motor relearning[9]. Faradic stimulation, specifically in a surged mode, is a low-frequency current that produces visible muscle contractions and is often used for re-education of paretic muscles[10].

The application of surged faradic stimulation to the antagonist muscles (i.e., wrist extensors) may provide additional benefits by enhancing reciprocal inhibition of the hyperactive wrist flexors[11]. This reciprocal mechanism, combined with the direct activation of extensors, contributes to improved voluntary motor control and reduction in spastic tone[12]. Several studies have supported the idea that incorporating functional electrical stimulation (FES) or neuromuscular electrical stimulation (NMES) into rehabilitation enhances both cortical plasticity and functional outcomes in stroke survivors[13].

In clinical settings, the integration of electrical stimulation with conventional physiotherapy techniques such as stretching and splinting has gained attention. This multimodal approach addresses both neural and biomechanical components of spasticity. Moreover, it can be tailored to the needs of individual patients, thereby optimizing the therapeutic potential[14]. Despite the growing body of evidence, there remains a need to further explore and validate combinations of such interventions in a structured manner.

Existing literature has compared individual modalities like splinting and electrical stimulation, but relatively fewer studies have examined their combined effect in a controlled, comparative design[15]. Particularly, there is limited data on the effectiveness of surged faradic muscle stimulation in conjunction with stretching and splinting for reducing wrist flexor spasticity in the chronic stroke population. This gap in literature underlines the importance of studying whether adding faradic stimulation to a stretching-and-splinting protocol provides superior outcomes compared to stretching and splinting alone.

The current study attempts to address this gap by comparing the effect of two treatment protocols on wrist flexion spasticity in chronic stroke patients. Group A received conventional treatment comprising wrist flexor stretching followed by application of a static cock-up splint. Group B received the same intervention with the addition of surged faradic stimulation applied to the wrist extensors. The efficacy of these interventions was measured using two widely accepted clinical tools: the Modified Ashworth Scale (MAS) for spasticity grading and the Active Range of Motion (AROM) measurement for wrist extension.

The rationale behind this study lies in the hypothesis that active facilitation of antagonist muscles through faradic stimulation may yield better spasticity reduction and motor recovery than passive methods alone. Furthermore, the study aims to contribute practical insights to clinical decision-making in neurorehabilitation by identifying synergistic treatment combinations that are both effective and easily implementable in standard therapy settings.

MATERIALS AND METHODS

Study Design

This comparative interventional study aimed to evaluate the impact of surged faradic stimulation combined with static cock-up splinting versus splinting alone in mitigating wrist flexor spasticity in individuals with chronic stroke[16]. The study spanned three weeks, during which participants received structured, supervised physiotherapy sessions on a daily basis. Ethical clearance for the research was obtained from the Institutional Ethics Committee prior to participant recruitment.

Study Setting

The study was conducted within the Department of Physiotherapy at Government Netaji Subhash Chandra Bose Medical College, Jabalpur, Madhya Pradesh, India. All components of the research—including recruitment, assessment, intervention, and follow-up—were executed within the same institutional framework, ensuring standardization of the treatment environment and procedural consistency.

Sample Size and Sampling Technique

A total of 40 participants meeting the eligibility criteria were recruited through purposive sampling. Subjects were allocated into two groups of 20 each:

- **Group A (Control Group):** Participants received passive wrist flexor stretching followed by application of a static cock-up splint.
- **Group B (Experimental Group):** Participants received the same stretching and splinting protocol as Group A, along with surged faradic stimulation to the wrist extensor muscle group.

Inclusion Criteria

Participants were selected based on the following criteria:

- Diagnosed case of chronic stroke with duration exceeding six months.
- Age ranging between 45 and 70 years.
- Demonstrable wrist flexor spasticity classified as Grade 2 or 3 on the Modified Ashworth Scale (MAS).
- Ability to understand instructions and provide informed consent for participation.

Exclusion Criteria

Exclusion criteria comprised:

- Presence of cognitive or perceptual deficits interfering with command following.
- Skin infections, allergies, or open wounds at potential electrode or splint contact sites.
- Presence of implantable electrical devices such as pacemakers.
- Fixed deformities or contractures at the wrist that limit range of motion assessment or intervention.

Intervention Protocol

Group A – Control Group

Participants assigned to Group A underwent two therapeutic modalities:

1. **Passive Wrist Flexor Stretching:**
Stretching was conducted slowly within a pain-free range, targeting the wrist flexor muscles. Each session lasted approximately 10 minutes and aimed to elongate spastic musculature and reduce tone.
2. **Static Cock-Up Splinting:**
A custom-made thermoplastic cock-up splint was applied to maintain the wrist in a functional position of 20–30 degrees extension. Splints were worn for 6 hours daily, primarily during periods of functional activity. Participants were monitored weekly for signs of skin irritation or pressure points.

Group B – Experimental Group

Group B participants received the same protocol as Group A (passive stretching and splinting) along with the addition of electrical stimulation:

3. **Surged Faradic Stimulation to Wrist Extensors:**
Electrical stimulation was delivered using surged faradic current, targeting the extensor carpi radialis longus and brevis muscles. Treatment parameters were standardized as follows:
   - **Current Type:** Surged faradic
   - **Frequency:** 30–40 Hz
   - **Pulse Duration:** 0.1 milliseconds
   - **Surge On/Off Ratio:** 1:3
   - **Session Duration:** 15 minutes
   - **Electrode Placement:** Active electrode over the motor point of wrist extensors; passive electrode on the dorsal forearm
   - **Intensity:** Adjusted to elicit a visible contraction without causing discomfort
   - **Patient Positioning:** Seated with forearm pronated and supported on a table to ensure stability during stimulation

Duration and Frequency of Treatment

Each participant received a total of 18 treatment sessions over a period of three weeks, with sessions conducted six days per week. Each session lasted between 25 to 30 minutes, encompassing stretching, stimulation (in Group B), and splint application procedures.

Outcome Measures

1. Modified Ashworth Scale (MAS)

Wrist flexor spasticity was evaluated using the MAS, which grades resistance to passive muscle stretch on a scale from 0 (no increase in tone) to 4 (rigid in flexion or extension). Assessments were performed at baseline and after three weeks by a blinded assessor. All evaluations were carried out with the participant seated, elbow flexed at 90°, and forearm resting on a stable surface.

2. Active Range of Motion (AROM) of Wrist Extension

AROM was measured using a standard universal goniometer. The axis of the goniometer was aligned with the lateral aspect of the wrist joint; the stationary arm was aligned with the forearm, while the moving arm followed the fifth metacarpal. Wrist extension was actively performed by the participant, and the angle was recorded in degrees. Measurements were conducted three times per session, and the mean value was used for statistical analysis. Baseline and post-intervention assessments were performed under identical conditions.

Statistical Analysis

Data were analyzed using IBM SPSS Statistics Version 21. Descriptive statistics including mean and standard deviation were calculated. Within-group comparisons of pre- and post-intervention values were assessed using the paired t-test. Between-group comparisons were performed using the independent samples t-test. A p-value of less than 0.05 was considered statistically significant. Results were presented in both tabular and graphical formats to enhance interpretability.

Ethical Considerations

The study was approved by the Institutional Ethics Committee of Government NSCB Medical College, Jabalpur. Written informed consent was obtained from all participants after explaining the objectives, procedures, and their right to withdraw at any stage without penalty. Confidentiality of data was maintained throughout the study, and no identifying information was disclosed in any reporting.

RESULTS

A total of 40 participants completed the study, with 20 in each group. The baseline demographics were comparable between the two groups. Data analysis focused on changes in Modified Ashworth Scale (MAS) and Active Range of Motion (AROM) pre- and post-intervention within and between the two groups.

Baseline Characteristics

Table 1. Characteristics of participants

Variable	Group A (n = 20)	Group B (n = 20)
Age (mean ± SD)	58.2 ± 6.5 years	57.6 ± 5.8 years
Gender (M/F)	13 / 7	14 / 6
Stroke duration	11.4 ± 3.1 months	11.8 ± 2.9 months
Dominant hand	17 right / 3 left	18 right / 2 left

There was no statistically significant difference in baseline demographic parameters between the two groups ($p > 0.05$).

Within-Group Comparison (Pre vs Post)

Both groups showed statistically significant improvements in MAS scores and wrist AROM after 3 weeks of intervention.

Within-group comparison of MAS and AROM

Parameter	Group	Pre-Treatment	Post-Treatment	p-value
Modified Ashworth Scale	Group A	3.00 ± 0.00	2.00 ± 0.32	< 0.001
	Group B	3.00 ± 0.00	1.00 ± 0.24	< 0.001
AROM (degrees)	Group A	4.95 ± 1.55	12.65 ± 1.68	< 0.001
	Group B	5.30 ± 1.62	17.30 ± 1.83	< 0.001

Between-Group Comparison (Post-Treatment)

Group B (surged faradic + splint + stretch) showed significantly greater improvement in both MAS and AROM than Group A (splint + stretch only).

Between-group comparison of post-treatment outcomes

Outcome Measure	Group A (Post)	Group B (Post)	p-value
MAS (mean ± SD)	2.00 ± 0.32	1.00 ± 0.24	< 0.001
AROM (degrees)	12.65 ± 1.68	17.30 ± 1.83	< 0.001

The results suggest that the addition of surged faradic stimulation contributed to a significantly greater reduction in spasticity and improvement in voluntary movement of the wrist.

Figure 1.Comparison of AROM Before and After Intervention in Both Groups

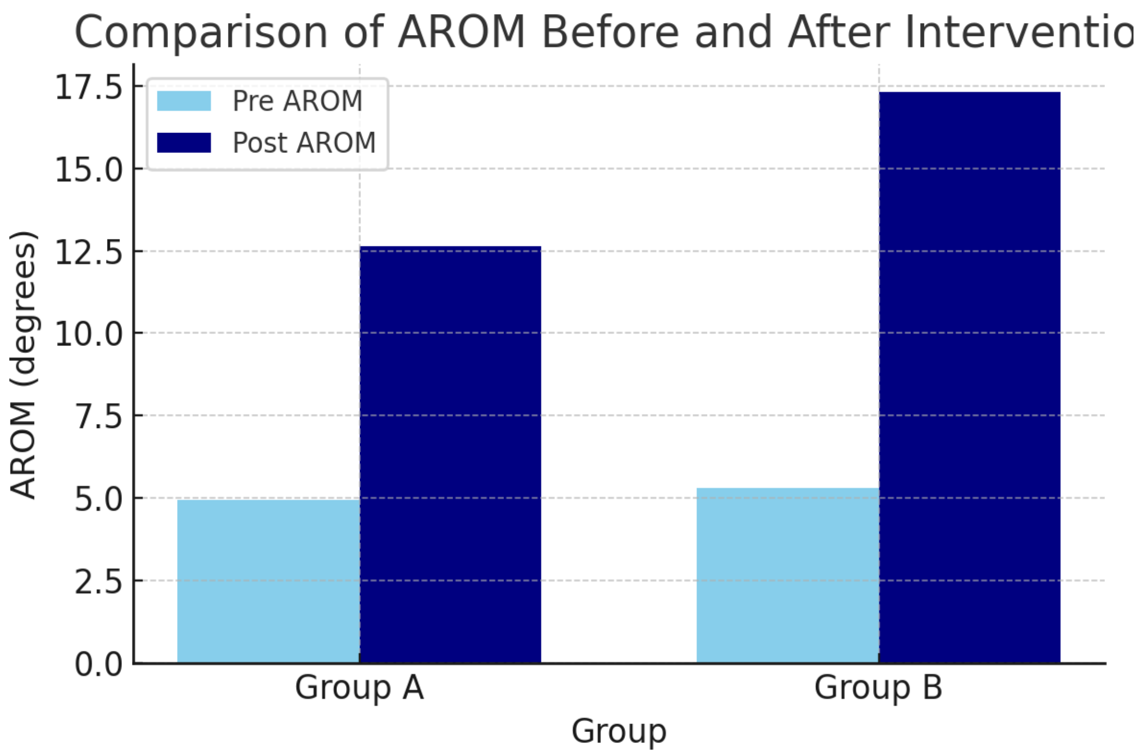
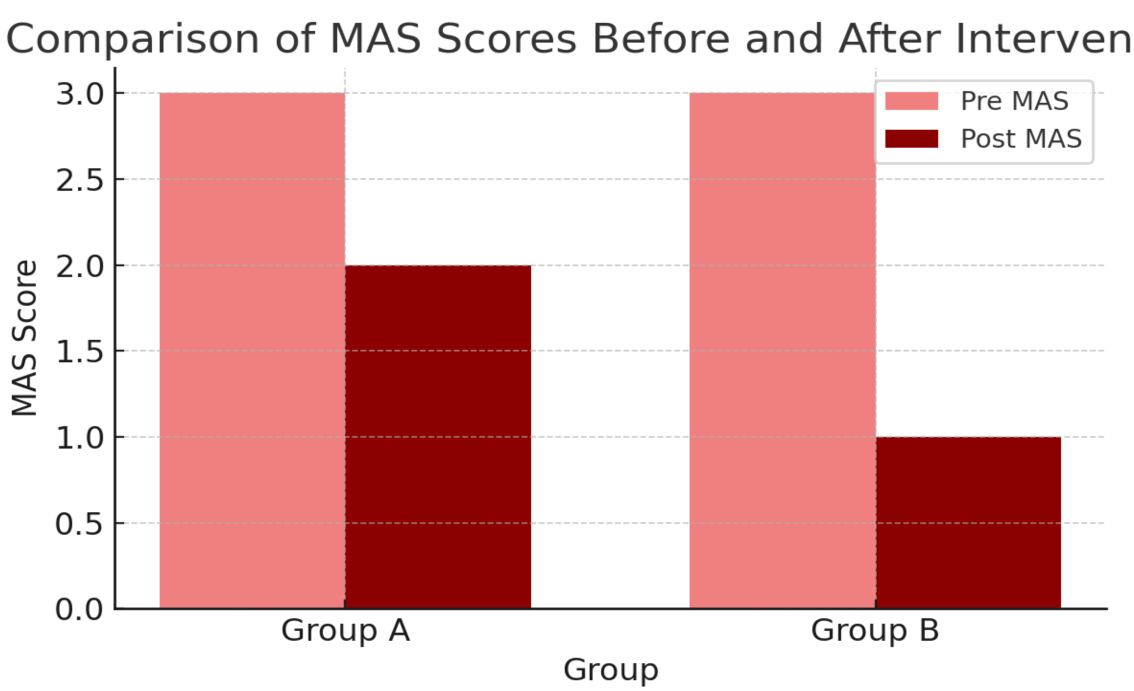


Figure 2.Comparison of MAS Scores Before and After Intervention in Both Groups



Summary of Results

- Both interventions led to clinically meaningful improvements.
- Group B (surged faradic + splinting + stretching) showed superior improvements in both spasticity (MAS) and range of motion (AROM).
- The findings support the beneficial role of faradic stimulation as an adjunct in chronic stroke rehabilitation.

DISCUSSION

The present study aimed to compare the effects of two rehabilitation protocols for managing wrist flexor spasticity in chronic stroke patients: (1) stretching with static cock-up splinting alone, and (2) the same protocol with the addition of surged faradic muscle stimulation applied to the wrist extensors. The results indicate that both groups showed significant improvement in spasticity and wrist range of motion; however, Group B demonstrated superior outcomes in both MAS and AROM measures. These findings suggest that the addition of faradic stimulation may enhance neuromuscular control and facilitate better motor recovery.
Spasticity in chronic stroke is primarily attributed to hyperexcitability of the stretch reflex arc and disruption of supraspinal inhibitory control[17]. In the upper extremity, the wrist flexors commonly present with high tone, leading to functional impairment. Static splinting and passive stretching aim to counteract this tone by maintaining muscle length, preventing contractures, and reducing soft tissue stiffness[18]. While these methods offer biomechanical benefits, their impact on neural control mechanisms is limited when applied in isolation[19].
The results of Group A (stretching and splint only) are consistent with earlier studies which demonstrated moderate reduction in spasticity through static positioning and elongation techniques[20]. Application of a cock-up splint helps maintain the wrist in extension, thereby inhibiting excessive flexor tone and improving alignment for functional movement[21]. Moreover, prolonged positioning in an anti-spastic pattern may reduce reflex activity over time and improve resting muscle tone[22].
However, Group B's significantly greater improvements in MAS and AROM reinforce the hypothesis that active facilitation of antagonist muscles contributes to better spasticity management. Surged faradic stimulation applied to the wrist extensors likely induced reciprocal inhibition of the wrist flexors, leading to reduced hypertonia and increased voluntary control[23]. This principle is well-supported in neurophysiological models, where activation of one muscle group suppresses activity in its antagonist through spinal inhibitory circuits[24].
Several studies support the clinical utility of electrical stimulation in stroke rehabilitation. Research has shown that neuromuscular electrical stimulation (NMES) can enhance sensorimotor integration, promote cortical plasticity, and improve volitional control of paretic muscles[25]. In particular, low-frequency stimulation in a surged pattern, as used in the present study, facilitates gradual recruitment of motor units, leading to better muscle endurance and coordination[26].
Kimberley et al. reported improved wrist and finger control following electrical stimulation therapy in chronic stroke patients[27]. Similarly, Chae et al. demonstrated that NMES applied to wrist extensors significantly reduced flexor tone and improved hand function compared to standard therapy[28]. The findings of the present study align with such literature and further emphasize the value of combining active modalities with passive techniques in neurorehabilitation.
The greater increase in AROM observed in Group B reflects not just reduced tone, but also improved voluntary recruitment of the wrist extensors. This could be attributed to better muscle re-education through repetitive contraction induced by the faradic stimulation. Over time, this may enhance neuromuscular coordination and task-specific movement patterns[29]. Moreover, electrical stimulation may serve as a biofeedback tool, allowing patients to relearn movements by experiencing muscle activation even in the absence of strong voluntary drive[30].
Another possible contributing factor is improved sensorimotor integration. The sensory input provided by electrical stimulation may help restore proprioceptive feedback pathways, which are often impaired post-stroke[31]. Enhanced afferent feedback may improve movement planning and execution, ultimately leading to better joint mobility and functional range[32].

It is also worth noting that the intervention in Group B was simple, safe, and well-tolerated by patients. Compliance was high, with no adverse effects reported. This highlights the feasibility of integrating such stimulation protocols in routine outpatient or home-based stroke therapy. Given that the intervention duration was only three weeks, the magnitude of improvement observed in Group B is clinically encouraging and suggests potential for even greater gains with longer therapy periods.

Despite the promising results, certain limitations must be acknowledged. The sample size was modest, with only 20 participants per group, which may limit generalizability. The study duration of 3 weeks may not reflect long-term outcomes or sustainability of benefits. Also, the study did not assess functional outcomes such as hand dexterity, grip strength, or ability to perform activities of daily living, which are crucial to evaluating real-life improvements[33]. Future research with larger sample sizes and longer follow-up periods should aim to explore these aspects.

Additionally, the study did not evaluate neural markers or imaging data (e.g., EMG, fMRI) to confirm changes in neural pathways, which could further substantiate the observed improvements. Including neurophysiological assessments in future trials would help clarify the underlying mechanisms driving recovery.

In terms of clinical implications, the findings support the integration of surged faradic stimulation into routine physiotherapy for upper limb spasticity post-stroke. When combined with stretching and splinting, it offers a multi-pronged approach that addresses both mechanical and neural contributors to spasticity. Moreover, the use of low-cost, portable electrical stimulators makes this modality accessible and practical in low-resource settings.

The study reinforces the importance of treating spasticity not only through passive correction but also by activating and retraining the neuromuscular system. This aligns with modern neurorehabilitation principles that emphasize task-specific training, sensory feedback, and plasticity-based interventions. Therapists should be encouraged to adopt multi-modal approaches and tailor interventions based on patient-specific needs and responsiveness.

In summary, the findings of this study suggest that the addition of surged faradic stimulation to a conventional splinting and stretching program provides superior outcomes in terms of spasticity reduction and voluntary wrist extension in chronic stroke survivors. The protocol is safe, simple to administer, and can be incorporated into standard rehabilitation practice with minimal cost or training.

CONCLUSION

The findings of this study demonstrate that both conventional stretching with static cock-up splinting and the combined approach involving surged faradic muscle stimulation are effective in reducing wrist flexor spasticity among chronic stroke patients. However, the group that received surged faradic stimulation in addition to stretching and splinting exhibited significantly greater improvements in both spasticity and active range of motion of the wrist.

The superior outcomes observed in the experimental group can be attributed to enhanced reciprocal inhibition, improved neuromuscular control, and better sensorimotor integration facilitated by the electrical stimulation. These results reinforce the potential of multimodal interventions in optimizing stroke rehabilitation outcomes, especially in addressing persistent upper limb spasticity.

Clinically, the protocol used in this study is practical, cost-effective, and easily applicable in routine physiotherapy settings. Incorporating faradic stimulation into rehabilitation programs may enhance patient outcomes and functional recovery, particularly in cases where passive modalities alone yield limited progress. Further studies with larger sample sizes, extended follow-up periods, and inclusion of functional performance measures are warranted to validate and expand upon these findings. Nevertheless, the present study offers valuable insights into the synergistic effect of combining active and passive physiotherapeutic approaches in the management of post-stroke spasticity.

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