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Comparative study between the effectiveness of myofascial release with a tennis ball Verses proprioceptive neuromuscular facilitation on spasticity and motor function of the upper limb in patients with stroke

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Abstract:
Background: *Spasticity of the upper limb is a common complication following stroke and significantly limits functional recovery. Physiotherapy interventions such as Myofascial Release (MFR) and Proprioceptive Neuromuscular Facilitation (PNF) are widely used, yet direct comparative evidence of their effectiveness in reducing spasticity is limited.*

Objective: *To compare the effectiveness of myofascial release using a tennis ball and proprioceptive neuromuscular facilitation on upper-limb spasticity in individuals with stroke.*

Methods: *A comparative interventional study was conducted on stroke patients meeting predefined inclusion criteria. Participants were randomly allocated to Group A (MFR with tennis ball + conventional physiotherapy) or Group B (PNF + conventional physiotherapy). Both groups received 30–40 minutes of therapy, three days per week for four weeks. Spasticity was assessed using the Modified Tardieu Scale (MTS) before and after the intervention. Pre–post changes within groups were analyzed, and mean differences were compared between groups.*
Results: *Both groups demonstrated statistically significant reductions in spasticity following treatment ($p < 0.0001$). Group A showed a mean MTS improvement of 1.20, while Group B showed a larger mean improvement of 2.37. Although both interventions effectively reduced spasticity, the magnitude of improvement was greater in the PNF group, indicating superior neuromuscular facilitation effects on modulating abnormal tone.*

Key Words: *MTS (modified tardue scale) , MFR (myofascil realease) , PNF (proprioceptive neuromuscular facilitation) ,WMFT (wolf motor function test) .*

RESEARCH QUESTION

Is proprioceptive neuromuscular facilitation technique more effective than myofascial release in reducing spasticity and improving motor function in 32 post-stroke patients?

1. INTRODUCTION:

STROKE is a clinically defined syndrome of acute, focal neurological deficit attributed to vascular injury (infarction, hemorrhage) of the central nervous system [1]. Stroke is a leading cause of disability in the United States and the fifth leading cause of death.

The socio-economic impact of stroke is considerable worldwide. Stroke is assuming an increasing impact in terms of media attention, patient and carer knowledge, service developments and research. It is estimated that there are 4.5 million deaths a year from stroke in the world and over 9 million stroke survivors. Almost one in four men and nearly one in five women aged 45 years can expect to have a stroke if they live to their 85th year. The overall incidence rate of stroke is around 2–25 per thousand population. The risk of recurrence over 5 years is 15–40%. It is estimated that by 2023 there will be an absolute increase in the number of patients experiencing a first-ever stroke of about 30% compared with 1983. There is a total prevalence rate of around 5 per thousand population. One year after a stroke, 65% of survivors are functionally independent, with stroke being the major cause of adult disability.
An acute ischemic stroke (AIS) occurs when an artery supplying the brain becomes occluded, leading to the death of brain tissue and focal neurological deficits.[2] Stroke causes motor disorders such as spasticity, which is caused by an imbalance of brain activity, spasticity is a high resistance to passive muscle stretching influence by velocity[3]sensorimotor function and more pain, reduced joint range of motion, and reduced sensibility. Spasticity prevalence after stroke varies from 18% to 60%. Spasticity affects the upper extremities more frequently than the lower extremities. This regional inclination contributes to the slower recovery of upper-limb function, with a near-permanent disability in hand and wrist function. Spasticity delays stroke recovery because it interferes with the ability to move normally and perform everyday tasks, including grooming, dressing, and bathing.
According to several researchers, dysfunctional reflexes linked to spasticity are the primary determinants of motor dysfunction. Spasticity is considered one of the factors that contribute to motor and functional impairment after stroke. However, it is not an autonomous agent because other primary factors, such as muscle weakness, may also be present. Spasticity in the upper limb is a strong component of scapular retractors, shoulder adductors, depressors, internal rotators, elbow flexors, forearm pronators, and wrist and finger flexors [4]. Grasp & release and manipulation are strategies of movement precision; muscle fatigue impairs the ability to grasp objects, having a great impact on daily living. Gross motor activities are affected, which leads to difficulty in performing daily activities like eating, combing hair, buttoning and unbuttoning, dressing and undressing, etc. Upper limb impairment compromises reaching, grasping, and other skills, partially or completely interfering with activities of daily living and reducing individual quality of life[5].

2. LITERATURE REVIEW:

1 **Dr. Maheshwari et al (2024)** "Immediate effect on wrist flexor spasticity in patients with stroke – a pre- and post-study was conducted on 36 stroke patients for 6 months. The results show that MFR showed a significant clinical difference, and spasticity decreased. The results showed that myofascial release showed a significant clinical difference in pre- and post-assessment values.R2-R1 values of post-treatment assessment compared to pre-treatment assessment decreased; this shows that myofascial release helps to decrease or treat spasticity.
2 **. Rutu J Parikh et .al (2024)** aimed to evaluate " myofascial release with tennis ball on spasticity and motor function in upper limb in patients with chronic stroke '' twenty two chronic patients were selected to conduct this study. A control group with conventional physiotherapy and another trial group with tennis ball MFR. Myofascial release performed with a tennis ball in conjunction with conventional physiotherapy has more beneficial effects on spasticity and motor functions of the upper extremity in patients with chronic stroke compared to conventional therapy alone.
3 **. Ujwal et al. aimed** to evaluate "effectiveness of proprioceptive neuromuscular facilitation on spasticity in hemiplegia ". Study was conducted on randomized controlled trial was conducted on thirty stroke patients with minimum grade 1 spasticity.PNF was significantly effective over conventional physiotherapy for reduce spasticity .in PNF group showed significant improvement as compared to conventional. The ROM also showed significant improvement in the PNF group. Conclusions: PNF technique is significantly effective over conventional physiotherapy for reducing spasticity and improving functional activities in hemiplegia.
4 **Anastasija et al (2024)** aimed to evaluate "the effectiveness of proprioceptive neuromuscular facilitation (PNF) in improving upper limb motor function in patients with stroke" and, as a result, a significant improvement in active range of motion using a manual goniometer in shoulder and elbow joint and wrist joint flexion, radial deviation for PNF appliedparticipant. The review examined five studies The review examined five studies on PNF effects in 210 stroke patients, all scoring six or more on the high-quality PEDro scale. Patients were, on average, 52 to 63 years old. Four of the five studies, using the Arm Motor Ability Test and Fugl-Meyer assessment, showed statistically significant improvements in upper limb motor ability and function post-PNF intervention.
5 **. Murphy SJ, Werring DJ. et al. (2020)** aimed to evaluate the prevalence of stroke .15% of strokes worldwide are the result of intracerebral haemorrhage, which can be deep (basal ganglia, brainstem), cerebellar, or lobar. Deep haemorrhages usually result from deep perforator (hypertensive) arteriopathy (arteriolosclerosis), while lobar haemorrhages are mainly caused by cerebral amyloid angiopathy or arteriolosclerosis. A minority (about 20%) of

intracerebral haemorrhages are caused by macrovascular lesions (vascular malformations, aneurysms, cavernomas), venous sinus thrombosis or rarer causes; these are particularly important in young patients (<50 years).

3. AIM

To find the effectiveness of myofascial release with a tennis ball and proprioceptive neuromuscular facilitation on reducing spasticity and improving motor function in the upper limb in stroke patients

OBJECTIVES

- To find the effectiveness of myofascial release with a tennis ball on spasticity and to reduce spasticity
- To find the effectiveness of proprioceptive neuromuscular facilitation to reduce spasticity.
- To find the effectiveness of myofascial release with a tennis ball and proprioceptive neuromuscular facilitation studies. Assessment of spasticity in upper limb pre and post-training.

4. METHODOLOGY :

- Study design - comparative study
- Study setting - study should be conducted in a tertiary care hospital at - Chh. Sambhajinagar
- Type of study - Interventional study
- Duration of study- 6 months
- Duration of intervention- 4 weeks
- Sample size - 16 in each group (total 32)
- Sampling technique - convenience sampling technique

Sample size calculation

$N = \frac{2[Z_\alpha + Z_\beta]^2}{\left(\frac{\mu1-\mu2}{\sigma}\right)^2}$ $\left(\frac{\mu1-\mu2}{\sigma}\right)^2$ is defined as effect size (ES) while μ1 and μ2 are means of two groups and σ is the standard deviation of the population being studied.
The probability of falsely rejecting a true null hypothesis (Type II error) is $\beta = 0.2$
Power = 1- β = 0.80, i.e., the power of the test is 80%.
for which $Z_\beta = 0.84$ and $Z_\alpha = 1.64$

$$\therefore N = \frac{2[1.64 + 0.84]^2}{(ES)^2}$$

For an effect size of 0.75.

$$N = \frac{2[1.28 + 0.84]^2}{(0.75)^2}$$

N = 15.98
A sample size of 16 per group, i.e., a total sample of 32, is considered for two groups.

INCLUSION

- Males & female both
- Age group – 20 to 70 years
- Type - sub-acute and chronic stroke
- Tone in flexor arm from 1 to 1+ spasticity on MAS

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- Patient with normal cognitive function

EXCLUSION: -

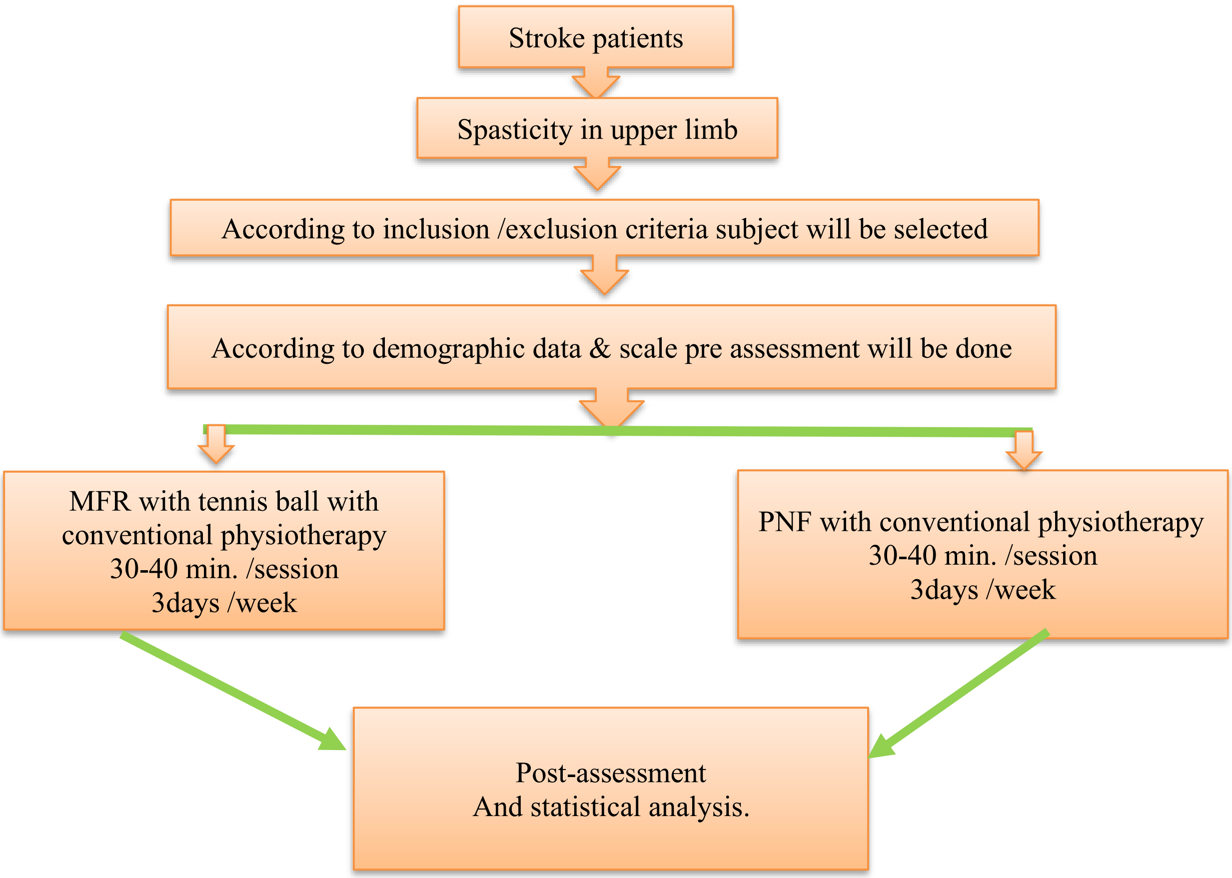
- Patient with complete flaccid stage
- Musculoskeletal disorder (eg.:fractures ,recent surgery of hand)
- Superficial and deep sensory loss of skin
- Patient on oral medication to release spasticity.

MATERIALS USED

- Pen
- Data collection sheet
- Tennis ball
- Chair (beside plinth)
- Consent form
- Scales - (a) modified tardive scale

  (b) wolf motor function test

Fig 1



5. PROCEDURE

Participants were divided into two groups. Experimental and control groups.

The experimental group included MFR with a tennis ball along with a conventional physiotherapy program, and the control group included PNF patterns in the upper limb.

GROUP A:-

- Myofascial release with tennis ball – A chair should be placed beside the plinth, and the participant should be made to sit on the chair so that the affected arm faces the plinth side.
- The affected upper limb should be placed on the plinth. Myofascial release will be applied to the patient, keeping the tennis ball below the patient's limb
- The therapist rolls the patient's limb over the tennis ball passively
- Tolerable pressure will be applied to the limb so that it presses the tennis ball
- Once the session of this therapy is given to the patient for about 2 min to the bicep brachii, pronator teres, and long finger flexor muscle for a total 6min / day
- MFR 6 min. And conventional physiotherapy 25 to 30 min. Total 30-40 min/ session

GROUP B

- This includes pattern of facilitation that is – D1 & D2 flexion, extension for upper limb with stretch, stimulus, and stretch reflex.
- D1 flexion - for upper limb included flexion, abduction with lateral rotation for 30 sec hold and relax
- D1 extension - included extension, adduction with medial rotation for 30 sec hold and relax
- D2 flexion - included flexion, adduction with lateral rotation for 30 sec hold and relax.
- D2 extension – extension, abduction with medial rotation for 30 sec hold and relax 2min for all movements = 5sets per session /10 min.
- The duration of the treatment session should be 20-30 min with conventional physiotherapy, including a rest period
- Session should be thrice in a week

CONVENTIONAL PHYSIOTHERAPY

The conventional physiotherapy intervention for both groups aims to reduce spasticity and improve motor function of the affected upper limb. Therapy sessions should be tailored according to patient needs, approx 20 to 30 min for 4 weeks.To improve range of motion, flexibility and manage spasticity.

(a) Active-assisted ROM movement should be performed with terminal stretch to maintain joint integrity and prevent contracture = 5min.
(b) Bilateral training involves activities that use both arms simultaneously, such as wall pushing- 6 sets 10 sec each
Ball catching - 15-20 catches = 15mins for bilateral hand training.
Wand exercises – 10 reps for 3 sets
(c) Static stretching- 10 min.

OUTCOME MEASURE

- Modified tardieu scale
- Wolf motor function test

6. OBSERVATION AND RESULT:

Table 1: Distribution of cases in the study groups according to age-group

Table 1 shows that the majority of participants in both Group A (43.7%) and Group B (43.7%) belonged to the 51–60 years age group. The mean age was 49.19 ± 7.75 years in Group A and 52.69 ± 10.59 years in Group B. The difference between groups was not statistically significant (t = 1.07, p = 0.294).

Age-Group [In years]	Group A		Group B	
	No	Percentage	No	Percentage
≤40	**04**	**25.0**	**02**	**12.5**
41-50	05	31.2	05	**31.2**
51-60	07	43.7	07	**43.7**
>60	00	00	02	**12.5**
Total	**16**	**100%**	**21**	**100%**
Mean±SD	**49.19±7.75**		**52.69±10.59**	
t-value	**1.07**			
P-value	**P=0.294 NS**			

NS = Not Significant

Fig 2

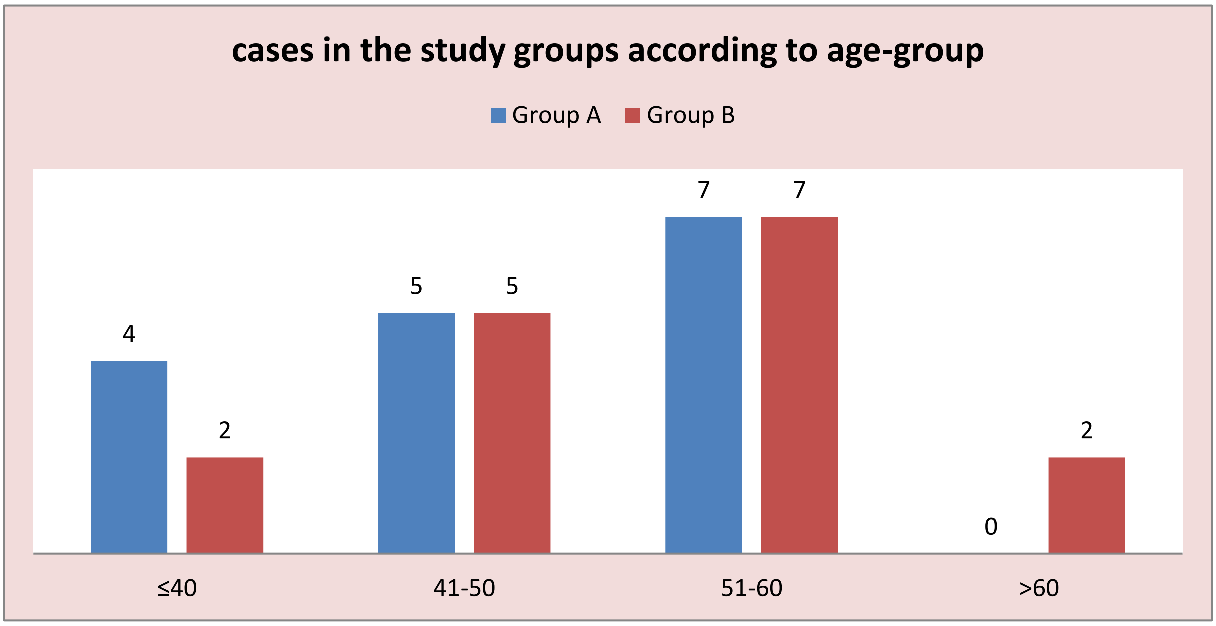


Table 2: Distribution of cases in the study groups according to Gender

Table 2 indicates male predominance in both groups, with 81.2% males in Group A and 68.7% males in Group B. Females constituted 18.8% in Group A and 31.3% in Group B. The gender distribution between the groups was not statistically significant ($\chi^2 = 0.667$, $p = 0.414$).

Gender	Group A		Group B	
	No	Percentage	No	Percentage
Male	13	81.2	11	68.7
Female	03	18.8	05	31.3
Total	16	100%	16	100%
Chi-square value	0.667			
P-value	P=0.414 NS			

NS = Not Significant

Fig 3

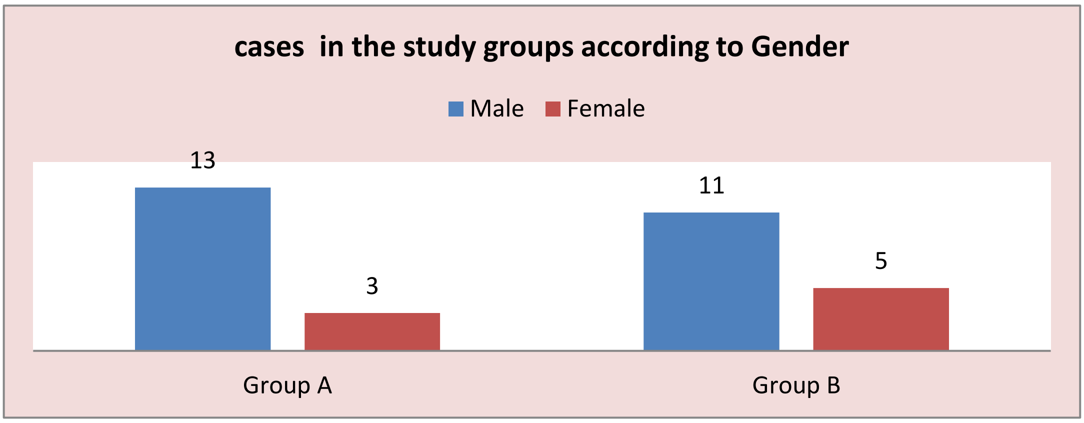


Table 3: Comparison of mean WMFT **pre and post treatment in study Groups**

Table 3 demonstrates no significant difference in pre-treatment WMFT scores between Group A (55.44 ± 11.96) and Group B (50.94 ± 15.06) (p = 0.352). Post-treatment WMFT scores significantly improved in both groups, with Group B (90.63 ± 7.16) showing significantly higher improvement than Group A (78.63 ± 8.39) (p = 0.003).

WMFT	Group A	Group B	t-value	P-value
Pre-treatment	55.44±11.96	50.94±15.06	0.936	P=0.352 NS
Post-treatment	78.63±8.39	90.63±7.16	4.35	P=0.003 S

NS = Not Significant, S = Significant

Fig 4

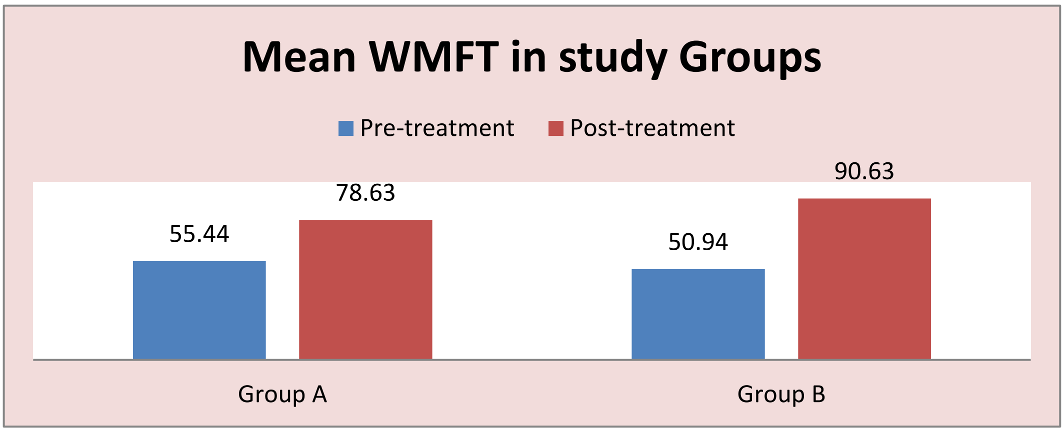


Table 4: Comparison of mean difference WMFT in pre and post treatment study Groups

Table 4 shows a significant improvement in WMFT scores within both groups following treatment. Group A showed a mean difference of 23.19, while Group B demonstrated a greater mean difference of 39.68. The improvement in both groups was statistically highly significant ($p < 0.0001$).

WMFT	Mean Difference	t-value	P-value
Pre Vs Post Treatment in Group A	23.19	11.55	P<0.0001 S
Pre Vs Post Treatment in Group B	39.68	19.42	P<0.0001 S

NS = Not Significant, S = Significant

Fig 5

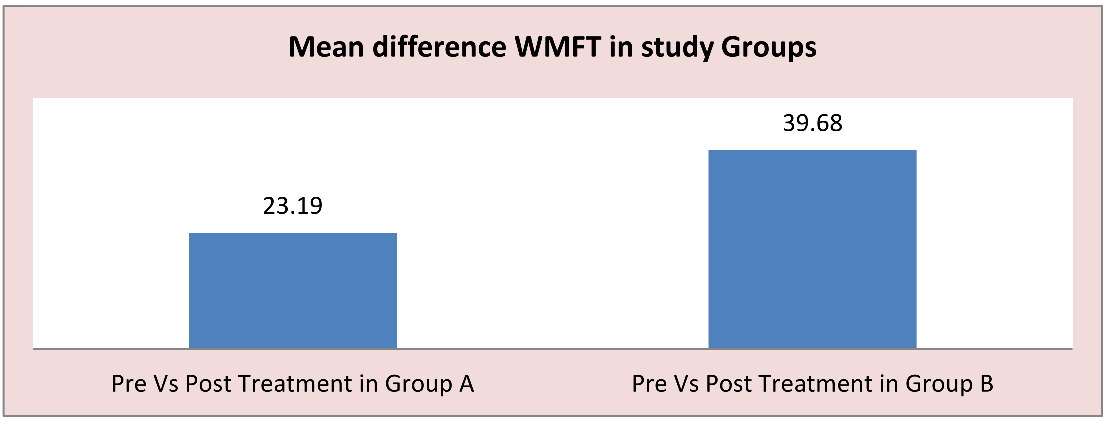


Table 5: Comparison of mean MTS in study Groups at pre- and post-treatment

Table 5 reveals comparable pre-treatment MTS scores between Group A (2.63 ± 0.88) and Group B (2.81 ± 0.65) with no significant difference ($p = 0.501$). Post-treatment, both groups showed significant reduction in spasticity, with Group B (0.44 ± 0.51) showing greater improvement than Group A (1.41 ± 0.81) ($p < 0.0001$).

MTS	Group A	Group B	t-value	P-value
Pre-treatment	2.63±0.88	2.81±0.65	0.681	P=0.501 S
Post-treatment	1.41±0.81	0.44±0.51	4.15	P<0.0001 S

NS = Not Significant, S = Significant

Fig 6

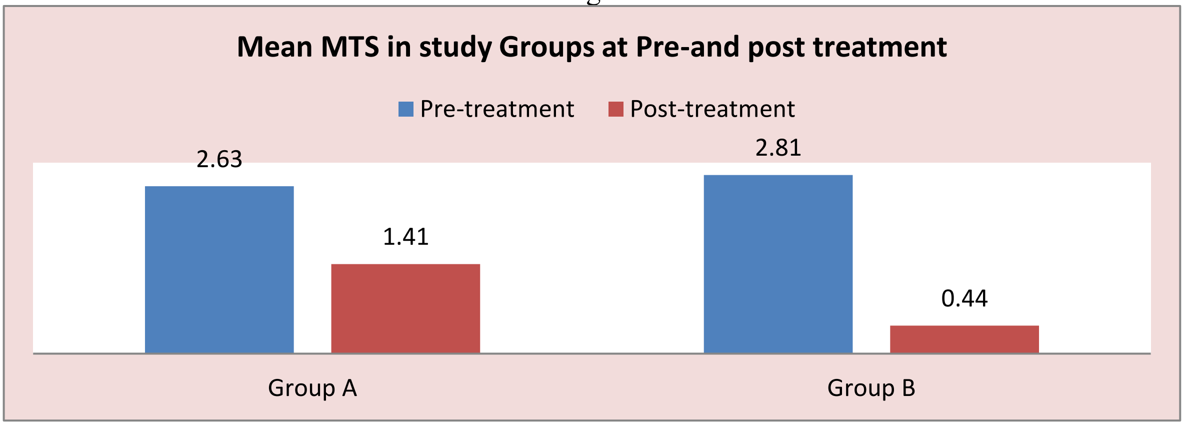


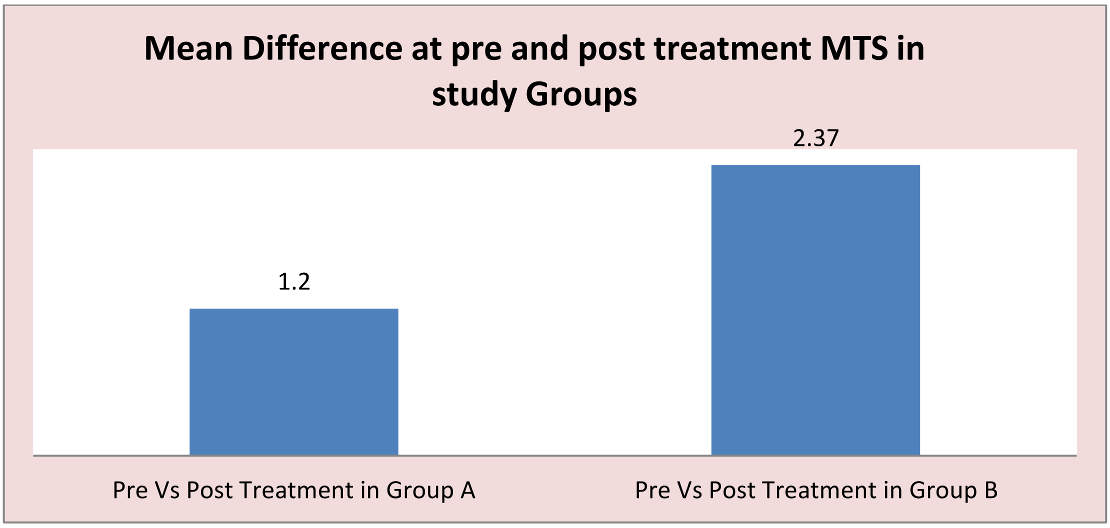
Table 6: Comparison of mean difference at pre and post treatment MTS in study Groups

Table 6 indicates a statistically significant reduction in spasticity within both groups after treatment. Group A showed a mean difference of 1.20, whereas Group B demonstrated a greater reduction with a mean difference of 2.37. The improvement in both groups was highly significant (p < 0.0001).

MTS	Mean Difference	t-value	P-value
Pre Vs Post Treatment in Group A	1.20	8.73	P<0.0001 S
Pre Vs Post Treatment in Group B	2.37	11.66	P<0.0001 S

NS = Not Significant, S = Significant

Fig 7



7. DISCUSSION

The present study aimed to compare the effects of Myofascial Release (MFR) using a tennis ball and Proprioceptive Neuromuscular Facilitation (PNF) exercises on spasticity in individuals following stroke. A total of 32 participants were included and randomly allocated into two groups: Group A (n = 16) received MFR with a tennis ball, and Group B (n =

16) received PNF exercises. Both interventions were administered over four weeks at a frequency of three sessions per week. The experimental design and statistical analysis confirmed normal data distribution in both groups.
Baseline demographic and clinical characteristics demonstrated that the two groups were comparable before intervention. There were no statistically significant differences between Group A and Group B with respect to age or gender distribution. Additionally, pre-treatment outcome measures revealed no significant differences in Wolf Motor Function Test (WMFT) scores or Modified Tardieu Scale (MTS) scores, indicating baseline homogeneity and allowing for valid comparison of post-intervention outcomes.
Post-stroke spasticity is a consequence of an upper motor neuron lesion, which disrupts supraspinal inhibitory control over spinal motor pathways. The loss of descending inhibition leads to increased excitability of spinal reflex circuits, hyperactivity of alpha motor neurons, and altered gamma motor neuron function, resulting in heightened muscle spindle sensitivity. Clinically, these neurophysiological alterations manifest as velocity-dependent resistance to passive stretch, abnormal synergistic movement patterns, increased muscle tone and stiffness, and impaired voluntary motor control. Prolonged spasticity further contributes to secondary musculoskeletal impairments, including muscle shortening, fascial tightness, reduced joint mobility, and compromised motor relearning, particularly affecting the upper limb.
The findings of the present study suggest that Myofascial Release exerts its therapeutic effects predominantly through peripheral and spinal-level mechanisms. Sustained pressure applied using a tennis ball likely resulted in mechanical deformation of fascial collagen fibers, reduction of fibrotic adhesions, enhanced interfascial glide, and increased tissue extensibility and muscle length. These mechanical effects are consistent with previous findings reported by Meltzer et al[7]. and Grieve et al[9]., who demonstrated reduced tissue stiffness and improved flexibility following myofascial interventions.
In addition to mechanical adaptations, MFR appears to produce neurophysiological effects by stimulating low-threshold mechanoreceptors, such as Ruffini endings and interstitial receptors. Activation of these receptors is associated with decreased sympathetic nervous system activity, reduced muscle spindle sensitivity, suppression of gamma motor neuron activity, and subsequent reduction in alpha motor neuron firing, leading to diminished stretch reflex excitability. These mechanisms provide a plausible explanation for the reduction in spasticity observed in Group A and are supported by the findings of Maheshwari et al. (2024),[8]who reported significant reductions in wrist flexor spasticity following MFR. In the present study, Group A demonstrated a mean reduction of 1.20 on the Modified Tardieu Scale along with statistically significant improvement in WMFT scores, indicating clinically meaningful but moderate effects. The relatively smaller magnitude of improvement suggests that MFR primarily influences peripheral soft-tissue components and reflex-mediated tone, with limited impact on central motor control mechanisms.
In contrast, Proprioceptive Neuromuscular Facilitation appears to act predominantly through central and spinal neurophysiological pathways, which may explain its greater effectiveness in modulating spasticity and enhancing functional outcomes. PNF techniques provide intense proprioceptive stimulation to muscle spindles, Golgi tendon organs, joint receptors, and cutaneous receptors, thereby enhancing afferent input and improving sensorimotor integration. During the execution of PNF diagonal patterns, activation of antagonist muscles facilitates reciprocal inhibition of spastic agonists, while sustained or resisted contractions activate Golgi tendon organs, resulting in autogenic inhibition. These inhibitory mechanisms collectively reduce alpha motor neuron excitability and muscle tone. Moreover, PNF movement patterns are functional, diagonal, spiral, multi-joint, and task-oriented, closely resembling activities of daily living. Repeated practice of these patterns promotes cortical reorganization, use-dependent neuroplasticity, and improved voluntary motor control by enhancing descending inhibitory pathways commonly impaired after stroke. PNF also assists in normalizing abnormal flexor synergy patterns in the spastic upper limb by promoting selective muscle activation and coordinated movement. In the present study, Group B demonstrated a greater reduction in spasticity, with a mean MTS reduction of 2.37, along with significantly greater improvement in WMFT scores. These findings are consistent with previous studies by Ujwal et al[13]. and Anastasija et al. (2024)[5], which reported significant improvements in spasticity, range of motion, and upper-limb functional performance following PNF interventions.
Although both interventions were effective in reducing post-stroke spasticity, the superior outcomes observed with PNF can be attributed to its broader influence on central nervous system function. PNF directly addresses central motor dysfunction by enhancing cortical and spinal inhibitory control, promoting long-term motor relearning, and improving functional use of the affected limb through task-oriented movement patterns. In contrast, Myofascial Release primarily targets peripheral tissue stiffness and muscle tightness, providing short-term modulation of tone with limited capacity for central neuromotor retraining. Consequently, while Group A demonstrated clinically significant improvements, Group B exhibited statistically significant and functionally superior outcomes. Collectively, these findings contribute to the growing body of evidence supporting the use of centrally driven, function-oriented rehabilitation strategies to enhance functional independence and quality of life in individuals living with post-stroke disability.

8. CONCLUSION

Both interventions—MFR using a tennis ball and PNF—were effective in significantly reducing upper-limb spasticity in stroke survivors. However, PNF demonstrated a larger reduction in MTS scores, indicating it may be more effective in modulating neuromuscular tone. These findings support the use of both techniques in clinical practice, but highlight PNF as the preferred intervention when the primary goal is rapid and pronounced reduction in spasticity. MFR remains valuable as an accessible, low-cost approach and may serve as a complementary method alongside PNF.

9. LIMITATIONS:

The study duration was limited to 4 weeks.
MFR dosage may influence results; different intensities or durations could produce larger effects.
The sample size, though adequate, may limit generalization to all stroke populations.
Only MTS was used to assess spasticity. Inclusion of EMG or neurophysiological testing would provide deeper insight.

10. FUTURE RECOMMENDATION

Incorporate combination protocols (MFR + PNF) to evaluate potential additive benefits.
Explore long-term retention of gains beyond 4 weeks.
Use instrumented assessments of muscle stiffness or neural excitability.
Expand sample size and include functional outcomes such as Fugl-Meyer or Box & Block Test to evaluate how spasticity reduction translates to motor performance.

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