Research Article

Effects of Reactive and Proactive Exercises on Ankle Spasticity in Patients with Chronic Stroke: A Randomized Clinical Trial

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Running title: Reactive and Proactive Exercises in Stroke

Abstract

Background: Stroke is a leading cause of adult disability. Spasticity is a neurological condition that occurs secondary to stroke and affecting the patient's quality of life. This study designed to compare the effect of reactive and proactive exercises on spasticity in people with stroke.

Materials and methods: In this single-blind clinical trial study, 30 people with stroke were randomly divided into three groups: control, reactive, and proactive, of which 27 patients completed the study. These people were treated for 12 sessions for 4 weeks (three times a week). The control group received conventional exercises, the reactive group received conventional exercises plus reactive exercises, and the proactive group received conventional exercises plus proactive exercises. The outcomes were clinical outcome of spasticity in baseline and neural properties that respectively evaluated using the Modified Modified Ashworth Scale and H-reflex latency and Hmax/Mmax ratio. All statistical analyzes were performed using SPSS version 20.

Results: In H-reflex latency ($F = 0.404$, $P = 0.672$, $\eta p^2 = 0.033$) and Hmax/Mmax ratio ($F = 0.878$, $P = 0.429$, $\eta p^2 = 0.071$) there were no significant differences between groups.
Conclusions: Intergroup comparisons did not show a significant advantage in favor of the use of proactive and reactive exercises on neural properties of spasticity compared to conventional exercises alone.
Keywords: Stroke; Spasticity; Exercise therapy

Introduction:

Stroke remains the principal cause of long-term disability in adults and the second most common cause of death worldwide (1). Spasticity, a frequent consequence of upper motor neuron lesions, exerts marked effects on skeletal muscle function (2). Lance originally defined spasticity as a velocity-dependent enhancement of stretch reflex activity in the absence of voluntary contraction (3). More recently, Dressler has described it as involuntary muscle overactivity in central paresis, triggered by either slow or rapid passive joint motion or by sensory input (4). Reported prevalence after stroke varies widely, ranging from 4–46% within the first month, 4.16–48% at 1–3 months, 6.9–63% at 3–6 months, and 7.6–49% beyond 6 months (5).

Clinically, spasticity is associated with increased muscle tone and stiffness, exaggerated tendon reflexes, and contracture formation. These abnormalities contribute to reduced voluntary strength, impaired balance, and deficits in motor control, ultimately limiting functional independence (6–8). Spastic overactivity of ankle plantar flexors is an important predictor of post-stroke falls (9, 10) and is closely linked to reduced quality of life (11).

Current management strategies include removal of nociceptive stimuli, use of anti-spastic positioning, active and passive range-of-motion and stretching exercises, night and standing ankle splints, chemo-denervation with botulinum toxin, and oral antispastic agents such as tizanidine, baclofen, and benzodiazepines (12, 13). More recent rehabilitation concepts emphasize task-specific, goal-directed, engaging, progressive, and adaptive training approaches to enhance motor recovery after stroke (14).

During the last decades, evidence-based frameworks have been proposed to optimize posture and movement in this population. These frameworks rely on principles of motor learning and exploit both feedback and feedforward mechanisms to improve motor control (15). Neurophysiological studies have shown that proactive motor control relies on feedforward mechanisms while reactive motor control relies on feedback mechanisms (16).

Anticipatory postural adjustments, often referred to as proactive exercises, are mediated through feedforward mechanisms and represent self-initiated, predictable reactions (17). They stabilize the trunk and stabilizing musculature before voluntary limb motion or predictable perturbations (18). In contrast, compensatory postural responses, or reactive exercises, are driven by feedback mechanisms and are elicited in response to unexpected perturbation (17). Reactive postural control therefore reflects the ability to respond rapidly after a perturbation (19). While anticipatory and compensatory strategies are related, they play distinct roles; efficient anticipatory control reduces reliance on compensatory reactions (18). Neurophysiological studies have shown that these responses engage spinal and subcortical pathways that enable precise action at the shortest possible latencies (20). In older adults and in individuals with neurological conditions such as stroke, anticipatory mechanisms are often delayed or impaired, resulting in a greater dependence on compensatory responses to maintain stability (18).

One commonly used method for quantifying spasticity is the Hoffmann (H) reflex. This electrophysiological measure reflects the excitability of α-motor neurons and correlates with

spastic hypertonia (21). Enhancement of the H reflex may arise from diminished inhibitory modulation of lower motor neurons or increased stretch reflex excitability due to reduced presynaptic inhibition (22). A number of studies have shown that H-reflex and M-wave parameters provide relatively objective indices of spasticity and can complement clinical assessment (23, 24). Although several investigations have explored the influence of exercise programs on spasticity in stroke (2, 14), there is limited evidence regarding training paradigms that explicitly engage feedback- and feedforward-based postural control. The present clinical trial therefore aimed to compare the effects of reactive and proactive exercises, designed to activate feedback and feedforward mechanisms, on ankle spasticity in patients with stroke.

Materials and methods:

Study setting:

The trial was carried out between 2022 and 2024 in the physiotherapy department of Ayatollah Kashani Hospital, Isfahan, Iran.

Participants and experimental design:

This study was a type of parallel single blind randomized clinical trial. Individuals experiencing a first unilateral stroke were identified through referrals from neurology outpatient services. Patients who expressed willingness to participate provided written informed consent. Eligible participants were then randomly allocated to one of three arms: a reactive exercise group, a proactive exercise group, or a control group receiving conventional therapy. In this study, block randomization method was used for random allocation. Using a statistical software, the statistician designed 10 blocks of 3, considering that we had 3 groups. Randomization was performed by the clinic secretary, who was unaware of the study.

The study protocol received ethical approval from the Ethics Committee of Isfahan University of Medical Sciences (IR.MUI.RESEARCH.REC.1399.302) and was prospectively registered in the Iranian Registry of Clinical Trials (IRCT20200101045970N4). All participants were fully informed about the study objectives and procedures prior to enrolment.

Inclusion and exclusion criteria:

Inclusion criteria were:

- first-ever unilateral stroke confirmed by CT scan or MRI and neurologist report (2);
- time since stroke ≥ 6 months (19);
- sufficient cognitive ability to comprehend and execute basic verbal instructions, as determined by a Persian version of the Mini-Mental State Examination (score ≥ 24) (2);
- ability to stand and walk independently without assistive devices (25);
- presence of gastrocnemius spasticity graded ≥ 1 on the Modified Modified Ashworth Scale (MMAS ≥ 1) (2).

Exclusion criteria comprised:

- additional neurological conditions such as Parkinsonism or diabetic neuropathy (2);
- severe visual, depth perception, or vestibular disorders (25);
- serious musculoskeletal problems in the lower limbs such as fixed contractures (2);
- intense lower-limb pain rated 75–100 mm on the VAS (26);
- participation in other concurrent physiotherapy interventions (2);
- use of medications affecting muscle tone, sensation, or strength (27);
- body weight >150 kg or height >2.1 m (19).

Sample size:
Based on a previous study (19) and using a significance level of α = 0.05 (Z = 1.96), power of 80% (Z1−β = 0.84), correlation ρ = 0.8, and one pre-test (υ = 1) and two post-intervention measurements (ω = 2), yielding R = 0.8, the required sample size was calculated as 10 participants per group.

$$n_{repeated} = R\left[\left[\frac{2\left(Z_{1\text{-}\alpha/2} + Z_{1\text{-}\beta}\right)}{\Delta^2} + \frac{Z^2_{1-\frac{\alpha}{2}}}{4}\right]\right]$$

$$R = \left[\frac{1 + (\omega - 1)\rho}{\omega}\right] - \frac{\upsilon\rho^2}{1 + (\upsilon - 1)\rho}$$

Patient recruitment:
A total of 68 individuals with stroke were screened. Thirty-eight did not meet the inclusion criteria, met at least one exclusion criterion, or declined participation. The remaining 30 participants were randomly assigned to the control, reactive, or proactive groups (10 per group) (Figure 1).

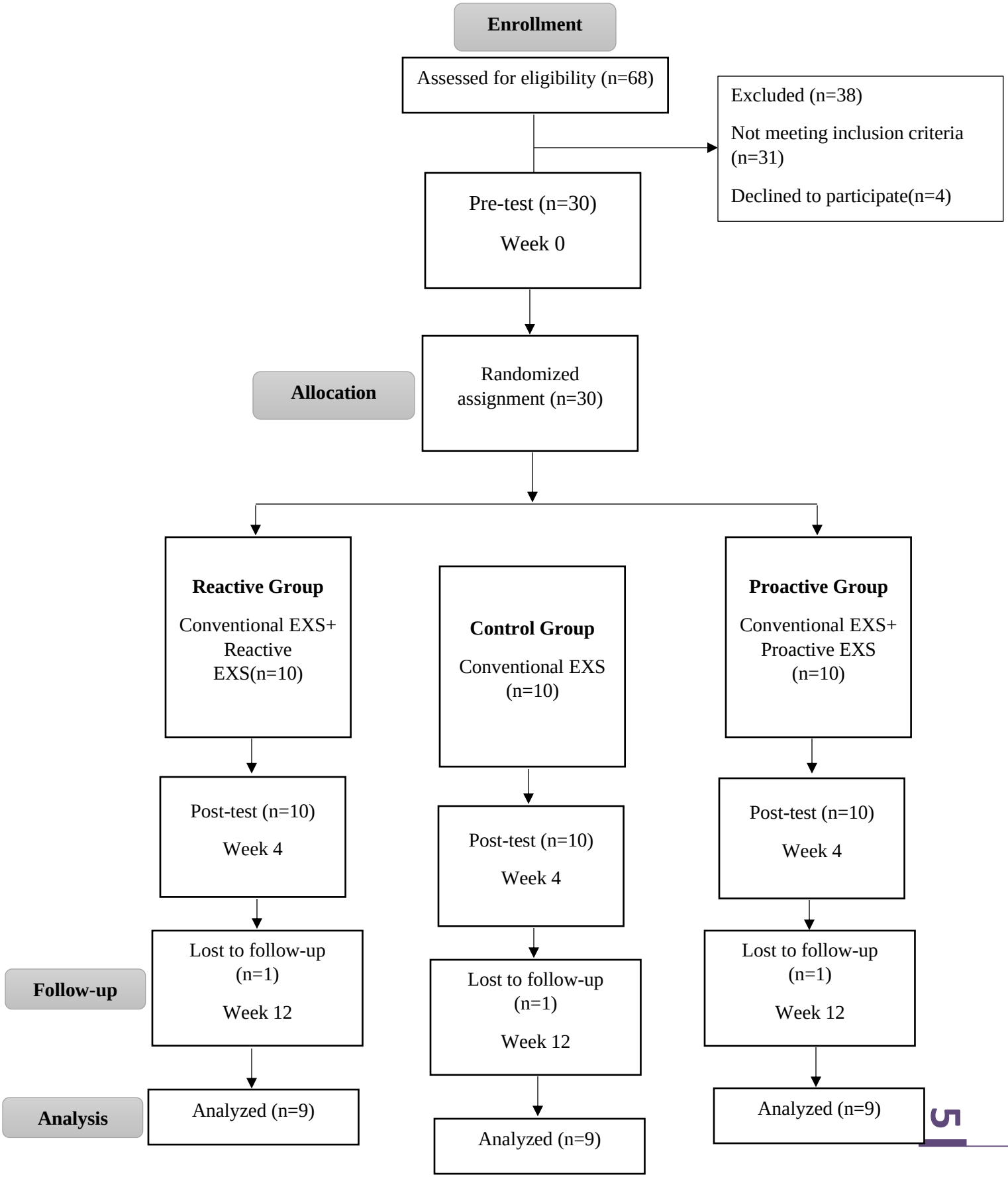
Enrollment
Assessed for eligibility (n=68)
Excluded (n=38)
Not meeting inclusion criteria (n=31)
Declined to participate(n=4)
Pre-test (n=30)
Week 0
Allocation
Randomized assignment (n=30)
Reactive Group
Conventional EXS+ Reactive EXS(n=10)
Control Group
Conventional EXS (n=10)
Proactive Group
Conventional EXS+ Proactive EXS (n=10)
Post-test (n=10)
Week 4
Post-test (n=10)
Week 4
Post-test (n=10)
Week 4
Follow-up
Lost to follow-up (n=1)
Week 12
Lost to follow-up (n=1)
Week 12
Lost to follow-up (n=1)
Week 12
Analysis
Analyzed (n=9)
Analyzed (n=9)
Analyzed (n=9)

Figure 1. The CONSORT flowchart

Treatment protocol:

Following baseline assessment, all groups received 12 treatment sessions over four weeks (three sessions per week on alternate days) (2).

- **Control group (conventional therapy):** Each session lasted 35 minutes: A 5-minute warm-up period, followed by 25 minutes of conventional balance-focused physiotherapy, and concluding with a 5-minute cool-down phase. Conventional therapy consisted of lower-limb range-of-motion activities, weight-bearing tasks on the affected side, gait training in parallel bars with forward, backward, and sideways steps, along with bridging exercises and muscle-stretching routines (28).
- **Reactive and proactive groups:** Each session was 60 minutes in duration, comprising a 5-minute warm-up, 25 minutes of the general balance exercises performed by the control group, an additional 25 minutes of group-specific balance training (reactive or proactive), and a final 5-minute cool-down.

For each group, five specific exercises were used, with 5 minutes allocated per exercise. Within each 5-minute block, the exercise was repeated two to five times according to patient capacity. All training sessions were performed under the direct oversight of a physiotherapist.

- **Reactive exercise examples:** examples include seated and standing perturbation exercises, therapist-applied push-and-release challenges, balance tasks on unstable surfaces, and "cowboy-style" movements carried out on a therapy ball.
- **Proactive exercise examples:** catching and throwing a ball, rotational movements, reaching tasks, single-leg stance, and tandem walking.

Outcome measurements:

All measurements were performed by an assessor who was not involved in treatment delivery and remained blinded to group allocation. Spasticity of the ankle plantar flexors was evaluated clinically in baseline and electrophysiologically at three time points: pre-intervention, immediately after the 4-week intervention, and two months after treatment completion.

Clinical assessment of spasticity:

Resistance of the hemiplegic ankle plantar flexors to passive stretch was graded using the Modified Modified Ashworth Scale (MMAS), a widely used and reliable instrument for quantifying spasticity (29). Using the validated Persian version of the MMAS, the blinded examiner rated resistance to passive movement on a 0–4 scale (30).

Electrophysiological assessment of spasticity:

H-reflex and M-wave responses of the soleus muscle were recorded using an electrodiagnostic device. Participants lay prone on the treatment bed with their feet extending beyond the edge. After shaving and cleansing the skin with soap-water–soaked cotton, a line was drawn from the midpoint of the popliteal fossa to the Achilles tendon and divided into eight equal segments to optimize electrode placement. The active electrode was positioned over the soleus muscle at the fifth or sixth distal segment, while the reference electrode was placed on the Achilles tendon. A ground electrode was located between the active electrode and the stimulation anode. The tibial nerve was stimulated in the popliteal fossa with the cathode proximal to the anode. Recording settings were:

gain 500 μV, sweep speed 10 ms/cm, stimulus duration 1 ms, and band-pass filter 2–10,000 Hz (31). Supramaximal stimuli were used to determine the maximal M-wave amplitude. The recorded variables included M- and H-wave amplitudes, H-reflex latency, and the Hmax/Mmax ratio. All electrophysiological measurements were obtained by an experienced neurologist blinded to group allocation.

Data analysis:
Data were analyzed using IBM SPSS Statistics version 20. Normality of distributions was checked using the Shapiro–Wilk test. For normally distributed variables, one-way ANOVA and repeated-measures ANOVA were applied. Non-normally distributed variables were evaluated with the Kruskal–Wallis test. A P-value < 0.05 was considered statistically significant.

Results:
Baseline characteristics:
The Mean ± SD values of age, body mass index (BMI), and post-stroke duration for the three groups are summarized in Table 1. One-way ANOVA revealed no significant between-group differences in age or BMI (p = 0.09), and the Kruskal–Wallis test showed no significant group differences in stroke duration (p = 0.29).

Table1. Demographic features of participants in the proactive, reactive, and control groups

Variable	Proactive group (n=10) Mean±SD	Reactive group (n=10) Mean±SD	Control group (n=10) Mean±SD	Confidence٪٩٥ Interval for mean		P value
				Lower bound	Upper bound	
Age (Year)	57.90±8.47	63.20 ± 10.64	52.90±10.33	٥۴٫١٠	۶١٫٩٠	**P=0.083**
BMI (Kg/m^2)	25.54±3.37	22. 73±2.40	25.54 ± 3.57	٢٣,٣٦	٢٥,٨٥	**P=0.090**
Post stroke duration (Month)	٢٦,٥٠±١٧٫٠۴	14.70 ±14.33	25.90 ±22.79	١٥,٤٣	٢٩,٣١	**P=0.288**

Body Mass Index.

Table1. Demographic features of participants in the proactive, reactive, and control groups
Table 2 presents the distribution of qualitative variables: sex, affected side, stroke type, use of assistive devices, and baseline spasticity scores. According to the Kruskal–Wallis test, none of these variables differed significantly among the groups (p > 0.05).

Table2. The frequency of qualitative background variables in proactive, reactive and control groups

Variable	Proactive group (n=10)	Reactive group (n=10)	Control group (n=10)	P value
Gender	F:2, M:8	F:4, M:6	F:2, M:8	**P=0.517**
Affected side	L:6, R:4	L:8, R:2	L:7, R:3	**P=0.631**
Stroke type	I:5, H:5	I:7, H:3	I:4, H:6	**P=0.404**
Assistive device	Yes:5, No:5	Yes:5, No:5	Yes:7, No:3	**P=0.592**
Spasticity Score	1:0, 2:7, 3:3, 4:0	1:2, 2:3, 3:5, 4:0	1:2, 2:5, 3:3, 4:0	**P=0.857**

F: Female, M: Male, L: Left, R: Right, I: Ischemic, H: Hemorrhagic

Electrophysiological outcomes:
Detailed electrophysiological data are provided in Table 3.

Table 3. Electrophysiological measurements of the study population

	Baseline			**Post training**			**Follow-up**		
Variable	Proactive group (n=10) SD± Mean	Reactive group (n=10) SD± Mean	Control group (n=10) SD± Mean	Proactive group (n=10) SD± Mean	Reactive group (n=10) SD± Mean	Control group (n=10) SD± Mean	Proactive group (n=9) SD± Mean	Reactive group (n=9) SD± Mean	Control group (n=9) SD± Mean
H-RL (ms)	33.15± 4.88	32.08± 5.42	32.9± 3.61	32.74± 4.15	31.45± 5.12	32.5± 3.53	33.40± 4.50	31.62± 5.42	32.96 ± 3.17
H/M R (mv)	.369± .056	.325± .035	.367± .054	.376± .064	.340± .039	.374± .058	.366± .062	.334± .035	.364± .059

H/M R, H max /M max Ratio; H-RL, H-reflex latency.

- **H-reflex latency:** Repeated-measures analysis showed no significant main effect of time across the three groups ($F = 1.467$, $P = 0.241$, $\eta p^2 = 0.058$). The time × group interaction was also non-significant ($F = 0.589$, $P = 0.673$, $\eta p^2 = 0.047$), and there were no significant differences between groups overall ($F = 0.404$, $P = 0.672$, $\eta p^2 = 0.033$). Based on the effect size index, none of the two treatment methods had a significant advantage over the change in the delay time of the H reflex, and the magnitude of the difference between groups for this outcome was not significant.

- **Hmax/Mmax ratio:** For the ratio of maximum H-reflex amplitude to M-wave amplitude, there was no significant main effect of time ($F = 1.491$, $P = 0.236$, $\eta p^2 = 0.061$). The interaction between time and group was non-significant ($F = 0.267$, $P = 0.898$, $\eta p^2 = 0.023$), and no significant group differences were observed ($F = 0.878$, $P = 0.429$, $\eta p^2 = 0.071$). According to effect size index, the magnitude of the difference between groups for this outcome was not significant.

Discussion:

This randomized trial did not demonstrate significant changes in H-reflex latency or in the Hmax/Mmax ratio within or between the reactive, proactive, and conventional therapy groups.

Post-stroke spasticity can cause considerable complications, including pain, impaired upper- and lower-limb function, reduced joint range, gait disturbances, limitations in daily activities, and substantial deterioration in quality of life (32–34). Both pharmacological and non-pharmacological modalities are used to address spasticity and improve functional outcomes, including conservative interventions, acupuncture, and dry needling (35). Exercise-based rehabilitation remains a central, low-cost strategy for reducing abnormal muscle tone and enhancing function in individuals after stroke. Zhang et al. reported that aquatic exercise resulted in better functional outcomes compared with land-based therapy, although the degree of spasticity was similar in both groups (36). Among conservative interventions, stretching is one of the most frequently prescribed components for increasing joint range of motion and managing spasticity in rehabilitation programs (37).

Previous work has shown that proactive and reactive training approaches can improve voluntary movement control, enhance rapid postural responses, reduce fall incidence, and augment reactive balance in people with stroke (15, 38, 39). Specific reactive exercises such as perturbation-based balance training (PBT) target these mechanisms and may therefore be particularly useful for fall prevention (39). Mansfield et al. evaluated body-weight–supported perturbation-based training (PBT) in comparison with traditional balance exercises in individuals with chronic stroke and reported that PBT resulted in superior gains in reactive balance control (19). There is increasing evidence that the cortex becomes engaged during reactive balance control as balance task difficulty increases (20). Recent work has demonstrated cortical involvement in reactive balance responses (40). It seems that the same situation occurred in our study, meaning that these exercises affected more areas of the cortex, and therefore our measurement methods that measured subcortical parameters were unable to track these changes.

The timing of intervention appears critical. PBT applied during the subacute phase of stroke may yield larger benefits than in chronic stages, because substantial spontaneous recovery and neuroplastic changes occur in the early weeks after stroke, plateauing around three months and declining thereafter; beyond six months, neurological recovery is typically limited (39). In the present study, all participants had chronic stroke with a duration > 6 months, which may have constrained the potential for neurophysiological change.

In addition to clinical outcomes, the current trial incorporated electrophysiological indices to evaluate potential neural adaptations induced by reactive and proactive training. Among reflex responses evoked by electrical stimulation, the H-reflex is widely used. H-reflex latency and the Hmax/Mmax ratio are considered reliable markers of α-motor neuron excitability (41). Prior research has documented increased H/M ratios in stroke survivors compared with healthy controls (42, 43). This ratio reflects the excitability level of motor neuron pools, which tends to rise in the presence of spasticity; typically, the H-reflex amplitude is elevated, whereas the M-wave amplitude may be reduced (44). Such changes may result from augmented loop gain within the

monosynaptic reflex arc, enhanced postsynaptic excitation from descending pathways, and/or diminished presynaptic inhibition (42, 45).

In contrast to some earlier reports, we did not observe significant modifications in H-reflex latency or the H/M ratio following either conventional therapy or the reactive and proactive protocols. Nor were there significant differences among groups. Another randomized trial has shown that functional stretching exercises can alter neural properties in chronic stroke patients (2), whereas some studies, such as that by Bakheit et al., have similarly reported no change in Hmax/Mmax ratio or H-reflex latency after a single session of isotonic or isokinetic stretching in spastic stroke (31).

A likely explanation for the absent neurophysiological changes in our study is the chronic stage of the participants. When a long time has elapsed since stroke, neural recovery is limited and plasticity is reduced. Furthermore, evidence suggests that stroke survivors can be categorized into two groups with low or high reflex torque based on neurological characteristics (46). In spastic hemiplegic patients, facilitation of Ia afferents during soleus H-reflex testing may be comparable to that seen in healthy individuals, suggesting preserved presynaptic inhibition. Consequently, reflex stiffness may be similar to that of controls, and mechanical alterations in muscle rather than changes in reflex pathways may account for much of the spasticity observed in chronic stroke.

This study has several limitations. The sample size was relatively small, which may have limited statistical power to detect subtle effects and there is a possibility of a type II error. The exercise time was not the same among the three groups. We also did not systematically measure adherence to the exercise protocols or participants' perceptions of acceptability and tolerability. Future research should consider larger cohorts and incorporate additional neurophysiological markers. As suggested by Heng-Yi Shen et al., the Hslp/Mslp ratio may provide a more sensitive indicator of spasticity in both upper and lower limbs in hemiplegic patients (19).

Conclusions

In this randomized controlled trial, reactive and proactive exercises did not show statistically significant differences over conventional exercises alone in terms of electrophysiological measures of ankle spasticity in patients with chronic stroke. It seems that the sensitivity of the measurements in this study was low and the use of more advanced methods to monitor the effects of these exercises on cortical and subcortical levels is needed in future studies.

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Conflict of interest

The authors declared no conflict of interest.

Authors' contributions

Conception and design: Ehsan Ghasemi; Funding acquisitions: Ehsan Ghasemi; Administrative, technical, or logistic support: Ehsan Ghasemi, Faezeh Kouhestani Dehaghi and Majid Ghasemi; Provision of study materials or patients: Majid Ghasemi; Data collection: Ehsan Ghasemi and Majid Ghasemi; Data analysis and interpretation: Ehsan Ghasemi and Faezeh Kouhestani Dehaghi ; Critical revising of the article for important intellectual content: Ehsan Gasemi, Faezeh Kouhestani Dehaghi, Tayebeh Roghani, and Majid Ghasemi; Final approval of the article: Ehsan Ghasemi and Tayebeh Roghani.

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