

Effects of dorsal foot sensory electrical stimulation on gait phase control in stroke survivors with cognitive impairment: a randomized controlled trial

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Abstract

Background

Post-stroke gait asymmetry is associated with impaired paretic weight bearing, reduced walking stability, and increased fall risk. Stroke survivors with cognitive impairment may have greater difficulty with gait phase control because of reduced attentional allocation and sensorimotor integration. This assessor-blinded randomized controlled trial examined whether dorsal foot sensory electrical stimulation improves paretic stance-phase control in stroke survivors with cognitive impairment.

Methods

In this assessor-blinded, parallel-group randomized controlled trial, 50 hospitalized stroke survivors with cognitive impairment, defined as an education-adjusted Montreal Cognitive Assessment score < 26, and National Institutes of Health Stroke Scale scores < 5 were randomized 1:1 to dorsal foot sensory electrical stimulation plus conventional rehabilitation or sham-device control plus conventional rehabilitation. Both groups received conventional rehabilitation for 4 weeks, with stimulation administered 5 times weekly for 30 minutes. The primary outcome was paretic stance-phase percentage during normal walking. Linear mixed-effects models were used for longitudinal analyses. A complete-case sensitivity analysis adjusted for concurrent gait speed.

Results

Seventy-eight participants were screened and 50 were randomized, with 25 in each group. The paretic stance-phase percentage decreased from 68.85% to 60.59% in the stimulation group and from 69.46% to 64.78% in the control group. The group-by-time interaction was significant in the intention-to-treat model after multiple imputation (chi-square = 38.84, $P < 0.001$), and the between-group difference in change at week 4 was - 3.40 percentage points (95% CI, -4.49 to -2.31). After adjustment for concurrent gait speed in complete cases, the group-by-time interaction remained significant (chi-square = 15.79, $P = 0.003$). Single-support phase percentage and knee flexion peaks also improved more in the stimulation group.

Conclusions

Four weeks of dorsal foot sensory electrical stimulation combined with conventional rehabilitation improved abnormal paretic stance-phase distribution in stroke survivors with cognitive impairment, and this effect was not fully explained by increased gait speed. These findings support sensory-driven gait rehabilitation as a potential strategy for improving paretic weight-bearing participation and gait phase control.

Trial registration

Chinese Clinical Trial Registry, ChiCTR2400081558, registered 5 March 2024, before participant enrollment.

Introduction

Even after regaining independent walking, patients with stroke often continue to show unstable gait, characterized by insufficient paretic weight bearing, delayed phase transition, and imbalanced bilateral gait distribution [1–3]. Abnormal paretic stance phase not only reflects changes in the temporal structure of gait but is also closely associated with insufficient paretic limb loading, reduced efficiency of weight transfer, impaired walking stability, and increased fall risk [2–6]. Therefore, paretic stance-phase percentage may serve as an important indicator of post-stroke deficits in gait stability-related control.

Abnormal paretic stance phase after stroke is not merely a consequence of insufficient motor output; it may also be related to cognitive decline. Gait control requires continuous attentional allocation, sensory integration, and postural adjustment. In stroke survivors with cognitive impairment, reduced gait automaticity, poorer active engagement of the paretic limb, and less efficient use of sensory information may make paretic limb underuse, abnormal stance-time distribution, and stability-related compensation more prominent [7–12]. These observations suggest that abnormal paretic stance phase in stroke survivors with cognitive impairment may partly arise from impaired cognitive-sensorimotor control, rather than solely from local muscle weakness or restricted joint motion.

If abnormal paretic stance phase partly reflects cognition-related sensorimotor control deficits, enhancement of peripheral sensory input may be a reasonable intervention strategy. Sensory electrical stimulation can increase paretic sensory input without directly inducing obvious muscle contraction, and may strengthen peripheral sensory cues during walking [13, 14]. The dorsum of the foot provides important sensory information for gait-cycle transition, preparation for foot contact, and regulation of weight bearing. Therefore, dorsal foot sensory electrical stimulation may improve paretic limb participation and gait phase control by enhancing peripheral cueing input [13–19]. However, previous studies have mainly focused on overall walking performance, such as gait speed, step length, or improvement in foot drop, whereas stance-phase percentage, which more directly reflects phase control and paretic weight-bearing participation, has received less attention.

On this basis, the present assessor-blinded randomized controlled trial focused on stroke survivors with cognitive impairment and examined the effect of 4 weeks of dorsal foot sensory electrical stimulation on paretic stance-phase percentage during normal walking. We further assessed whether this change persisted after adjustment for concurrent gait speed. Paretic single-support phase percentage, knee flexion peaks, Timed Up and Go (TUG), and MoCA scores were also examined to help interpret the kinematic and clinical significance of changes in gait phase.

Methods

Study design and participants

This was an assessor-blinded, parallel-group randomized controlled trial. The study protocol was approved by the Ethics Committee of University-Town Hospital of Chongqing Medical University and registered with the Chinese Clinical Trial Registry on March 5, 2024, before participant enrollment (ChiCTR2400081558). Written informed consent was obtained from all participants.

Inpatients with stroke were consecutively recruited from the Department of Rehabilitation Medicine, University-Town Hospital of Chongqing Medical University, between March 2024 and January 2025. The inclusion criteria were as follows: (1) diagnosis of ischemic or hemorrhagic stroke within the previous 6 months; (2) age 18–85 years; (3) cognitive impairment after stroke, defined as an education-adjusted Montreal Cognitive Assessment (MoCA) score < 26 , with 1 point added for participants with ≤ 12 years of education; (4) NIHSS score < 5 at enrollment; (5) ability to walk at least 10 m without an assistive device; (6) normal superficial sensation in the paretic lower limb; (7) at least 1 week after the most recent stroke onset, clinical stability, and no expected change in medication over the following 4 weeks; (8) ability to understand training instructions, cooperate with device operation, and provide adequate feedback; and (9) provision of written informed consent.

The exclusion criteria were as follows: (1) lower motor neuron injury; (2) lower-limb sensory loss or severe sensory abnormality preventing perception of the stimulation; (3) severe cardiac disease, such as myocardial infarction, congestive heart failure, or the need for a pacemaker; (4) current use of another electrical stimulation device; (5) metal hip or knee prosthesis in the lower limb; (6) epilepsy, autoimmune disease, or malignant tumor; and (7) pre-stroke cognitive or motor dysfunction.

Sample size estimation

The sample size was estimated on the basis of the change in the primary outcome, paretic stance-phase percentage, from W0 to W4. According to preliminary data, the expected between-group difference in change in the primary outcome was 2.5 percentage points, with a pooled standard deviation of 2.42 percentage points. With a two-sided alpha of 0.05, 90% power, and a 1:1 allocation ratio, at least 20 participants were required per group. Allowing for approximately 20% incomplete follow-up or attrition, the final planned sample size was 25 participants per group, for a total of 50 participants.

No important changes to the methods or trial outcomes were made after trial commencement. No interim analyses or stopping guidelines were planned. The trial ended after completion of the planned recruitment and follow-up.

Randomization and allocation concealment

Randomization was performed using a computer-generated random sequence. The random allocation sequence was generated by an independent researcher who was not involved in enrollment, intervention delivery, or outcome assessment. Eligible participants were enrolled by study therapists, and the independent researcher assigned participants to interventions according to the envelope sequence after baseline assessment. Participants were allocated in a 1:1 ratio, with no blocking, stratification, or other restriction. Allocation concealment was implemented using sequentially numbered, opaque, sealed envelopes to ensure the independence and unpredictability of the allocation process.

Blinding

The study was designed as an assessor-blinded trial. All gait data collection and outcome assessments were performed by independent assessors who were unaware of group allocation. Because the intervention required therapists to set and supervise device operation, therapists were not blinded. Participants wore devices with identical appearance; however, because dorsal foot sensory stimulation was perceptible, participant blinding may have been compromised. The success of blinding was not formally assessed. Statistical analyses were conducted according to the prespecified plan.

Interventions

The stimulation group received dorsal foot sensory electrical stimulation in addition to conventional rehabilitation. The control group wore an identical device, but the stimulation output was not activated and no therapeutically meaningful dorsal foot sensory electrical stimulation was delivered. The stimulation site was set on the dorsal skin over the distal first metatarsal. Stimulation intensity was set at 3 times the perceptual threshold (PT) and was kept within a sensory stimulation range that did not elicit muscle contraction, ensuring that the stimulation remained below the motor threshold. Each participant's PT was measured before the intervention and recalibrated weekly to maintain consistent stimulation intensity.

The stimulation was set and supervised by an independent therapist. The stimulation timing was synchronized with the gait cycle and was mainly delivered during the swing phase. The intervention was administered 5 times per week, 30 min per session, for 4 consecutive weeks. Both groups received conventional rehabilitation training at the same frequency. The intervention lasted 4 weeks, with 5 assessment time points: baseline (W0) and post-intervention weeks 1–4 (W1–W4).

Gait measurement

Kinematic data were collected using an optical motion capture system (Motion Analysis Corporation, USA) and the accompanying Cortex software. Reflective markers with a diameter of 14 mm were attached to the bony landmarks required for lower-limb gait analysis. Marker trajectories were recorded using Kestrel infrared cameras with a sensor resolution of 2048 x 1088 pixels. The system tracked

marker displacement through the camera array and reconstructed three-dimensional coordinates by triangulation. Bilateral lower-limb ground reaction forces were simultaneously collected using AMTI three-dimensional force plates (USA). The sampling frequency was 60 Hz for kinematic data and 1000 Hz for ground reaction forces. Stance-phase percentage, first peak knee flexion, second peak knee flexion, and single-support phase percentage were obtained from the raw output of the three-dimensional gait analysis for the paretic side and were repeatedly recorded at the prespecified time points [20–22]. Gait speed (m/s) was also extracted from the same three-dimensional gait analysis output.

Outcome measures

The primary outcome was stance-phase percentage during normal walking. First and second peak knee flexion were secondary kinematic outcomes, and single-support phase percentage was a secondary temporal outcome. Gait speed was analyzed as a complete-case supplementary gait outcome. TUG and MoCA were recorded only at W0 and W4 and were used as auxiliary observational indicators of overall functional mobility and global cognitive status, respectively. The total MoCA score is 30, with higher scores indicating better global cognitive function; 1 point was added for participants with ≤ 12 years of education.

Statistical analysis

Because gait speed was available only in the 42 participants who completed all observations from W0 to W4, it was analyzed as a complete-case supplementary outcome. Concurrent gait speed was further included in the stance-phase percentage model as an exploratory sensitivity analysis to evaluate whether the primary temporal outcome might be influenced by changes in overall walking speed.

The primary analysis followed the intention-to-treat (ITT) principle and included all 50 randomized participants. For participants who did not complete all follow-up assessments from W0 to W4, missing longitudinal outcome data were handled using multiple imputation. In addition, a complete-case supplementary analysis was performed in the 42 participants who completed all observations from W0 to W4 to assess the robustness of the findings. Because TUG and MoCA were measured only at W0 and W4, they were not imputed and were analyzed only as auxiliary outcomes.

Continuous variables were summarized according to their distribution: approximately normally distributed variables are presented as mean $\pm$ standard deviation, and skewed variables are presented as median (interquartile range). Categorical variables are presented as frequency and percentage. For stance-phase percentage, first peak knee flexion, second peak knee flexion, and single-support phase percentage, the primary analysis included all 50 randomized participants and used linear mixed-effects models after multiple imputation to compare whether the patterns of change over the 4-week intervention differed between groups. Multiple imputation was performed under the missing-at-random assumption. The imputation model included group, time, baseline demographic and clinical variables,

and observed outcome values at each time point; model estimates were pooled according to Rubin's rules. Fixed effects in the linear mixed-effects models included group, time, and the group-by-time interaction; participant was included as a random effect, and time was treated as a categorical variable. The overall interaction was tested using likelihood ratio tests, with chi-square values and P values reported. The estimated between-group difference in change from baseline to W4 (beta) and 95% confidence interval (CI) were also reported. The same model was repeated using non-imputed data in the 42 participants who completed all observations from W0 to W4 as a complete-case supplementary analysis. Because gait speed was analyzed only in these 42 complete cases, the same linear mixed-effects model was used as a supplementary analysis. Concurrent gait speed was further added to the stance-phase percentage model in the complete-case set as an exploratory sensitivity analysis. TUG and MoCA were measured only at W0 and W4, were not included in longitudinal mixed-effects models, were not imputed, and were compared only for between-group differences in change from W0 to W4 as auxiliary analyses.

All tests were two-sided, and $P < 0.05$ was considered statistically significant.

Results

Participant characteristics and analysis sets

A total of 78 participants were consecutively screened, of whom 50 met the eligibility criteria and were randomized, with 25 participants in each group (Fig. 1). All randomized participants received the allocated intervention and were included in the ITT analysis. Forty-two participants completed all observations from W0 to W4 and were included in the complete-case supplementary analysis; 4 participants in each group were lost to follow-up because they were discharged before the 4-week assessment. In the randomized population, 28 participants were men (56.0%), the mean age was 61.55 $\pm$ 15.06 years, the baseline NIHSS score was 2.84 $\pm$ 1.16, and the baseline MoCA score was 20.04 $\pm$ 3.30. Except for first peak knee flexion, baseline demographic, clinical, and main gait variables were generally comparable between groups. No serious or important adverse events related to the intervention were observed in either group during the study.

Table 1
Baseline characteristics.

Variable	Stimulation group, n = 25	Control group, n = 25	P value
Age, years, mean (SD)	60.85 ± 14.86	62.25 ± 15.53	0.746
Sex, male, n (%)	15 (60.00%)	13 (52.00%)	0.569
Height, cm, mean (SD)	163.07 ± 8.70	162.33 ± 8.46	0.762
Body weight, kg, mean (SD)	61.94 ± 9.27	61.73 ± 12.63	0.947
Education, years, mean (SD)	7.64 ± 1.89	6.88 ± 1.86	0.158
Smoking history, n (%)	18 (72.00%)	16 (64.00%)	0.544
Alcohol use history, n (%)	20 (80.00%)	17 (68.00%)	0.333
Hypertension, n (%)	23 (92.00%)	23 (92.00%)	1.000
Stroke type, n (%)			0.390
Ischemic	12 (48.00%)	9 (36.00%)	
Hemorrhagic	13 (52.00%)	16 (64.00%)	
Hemiparetic side, n (%)			0.258
Right	10 (40.00%)	14 (56.00%)	
Left	15 (60.00%)	11 (44.00%)	
Time from stroke onset to enrollment, months, mean (SD)	3.68 ± 0.90	3.72 ± 1.24	0.897
NIHSS score, points, mean (SD)	3.00 ± 1.15	2.68 ± 1.18	0.336
Married, n (%)	20 (80.00%)	21 (84.00%)	1.000
Stance-phase percentage, %, mean (SD)	68.85 ± 4.09	69.46 ± 4.37	0.612
First peak knee flexion, degrees, median (IQR)	10.39(8.98, 13.81)	12.49(11.07, 16.07)	0.028

Note: This table includes all 50 randomized participants, with 25 in the stimulation group and 25 in the control group. Normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed variables are presented as mean +/- standard deviation, non-normally distributed variables (first peak knee flexion and single-support phase percentage) are presented as median (interquartile range), and categorical variables are presented as n (%). MoCA indicates the education-adjusted score. P values are reported for descriptive baseline comparisons: independent-samples t tests or Mann-Whitney U tests were used for continuous variables, and chi-square tests or Fisher exact tests were used for categorical variables. Baseline gait speed was available only in the 42 participants who completed all observations and is therefore not shown in this table.

Variable	Stimulation group, n = 25	Control group, n = 25	P value
Second peak knee flexion, degrees, mean (SD)	40.79 ± 9.57	38.57 ± 9.79	0.359
Berg Balance Scale (BBS), points, mean (SD)	36.84 ± 5.23	35.32 ± 5.21	0.308
Montreal Cognitive Assessment (MoCA), points, mean (SD)	20.60 ± 3.61	19.48 ± 2.93	0.234
Single-support phase percentage, %, median (IQR)	25.66(22.04, 27.66)	24.03(19.76, 25.61)	0.207
Note: This table includes all 50 randomized participants, with 25 in the stimulation group and 25 in the control group. Normality of continuous variables was assessed using the Shapiro-Wilk test. Normally distributed variables are presented as mean +/- standard deviation, non-normally distributed variables (first peak knee flexion and single-support phase percentage) are presented as median (interquartile range), and categorical variables are presented as n (%). MoCA indicates the education-adjusted score. P values are reported for descriptive baseline comparisons: independent-samples t tests or Mann-Whitney U tests were used for continuous variables, and chi-square tests or Fisher exact tests were used for categorical variables. Baseline gait speed was available only in the 42 participants who completed all observations and is therefore not shown in this table.			

Dorsal foot sensory electrical stimulation improved abnormal paretic stance phase

As shown in Table 2, both the primary and secondary outcomes showed trends of change favoring the stimulation group during the 4-week intervention. Figures 2–4 further show group mean changes and individual trajectories for stance-phase percentage, gait speed, and knee flexion peaks. Results of the complete-case supplementary analysis in the 42 participants are shown in Table 3.

In the primary ITT longitudinal linear mixed-effects model after multiple imputation, the group-by-time interaction for stance-phase percentage was significant, indicating different patterns of change between the two groups during the 4-week intervention (chi-square = 38.84, $P < 0.001$) (Table 2 and Fig. 2). The paretic stance-phase percentage decreased from 68.85% to 60.59% in the stimulation group and from 69.46% to 64.78% in the control group. The between-group difference in change from baseline to W4 was – 3.40 percentage points (95% CI, -4.49 to -2.31; $P < 0.001$).

Because prolonged paretic stance time after stroke is generally considered to be related to reduced weight-bearing efficiency and stability-related gait compensation, these findings suggest that dorsal foot sensory electrical stimulation combined with conventional rehabilitation may further improve abnormal paretic gait phase in stroke survivors with cognitive impairment.

Improvement in stance phase was not fully explained by increased gait speed

Among the 42 participants who completed all observations from W0 to W4, gait speed increased from 0.25 +/- 0.09 m/s to 0.41 +/- 0.10 m/s in the stimulation group and from 0.25 +/- 0.06 m/s to 0.34 +/- 0.06 m/s in the control group. The group-by-time interaction was significant (chi-square = 74.52, $P < 0.001$), and the between-group difference in change from baseline to W4 was 0.07 m/s (95% CI, 0.05 to 0.08; $P < 0.001$).

Because gait temporal variables may be influenced by changes in gait speed, concurrent gait speed was further included in the stance-phase percentage model in the complete-case set as an exploratory sensitivity analysis. After adjustment for concurrent gait speed, the group-by-time interaction for stance-phase percentage remained significant (chi-square = 15.79, $P = 0.003$), and the between-group difference in change from baseline to W4 was -2.67 percentage points (95% CI, -3.96 to -1.37), indicating that changes in stance-phase percentage were not fully explained by increased gait speed (Table 4).

This finding suggests that the effect of dorsal foot sensory electrical stimulation on stance phase cannot be fully explained by improvement in overall walking speed, supporting a possible additional effect on gait phase control and paretic limb participation.

Improved gait phase was accompanied by changes in paretic lower-limb kinematics

As a secondary temporal outcome, paretic single-support phase percentage increased from 23.74% to 31.06% in the stimulation group and from 22.46% to 25.54% in the control group (Supplementary Figure S1). The group-by-time interaction was significant (chi-square = 43.39, $P < 0.001$), and the between-group difference in change from baseline to W4 was 4.21 percentage points (95% CI, 2.86 to 5.57; $P < 0.001$). The combination of decreased stance-phase percentage and increased single-support phase percentage suggests that the internal temporal structure of the paretic stance phase may have been redistributed, manifested as increased paretic single-limb weight-bearing time rather than a simple shortening of overall stance time.

To further examine whether these temporal changes were accompanied by changes in paretic lower-limb kinematics, we simultaneously analyzed first and second peak knee flexion (Fig. 4).

The group-by-time interaction for first peak knee flexion was also significant (chi-square = 41.94, $P < 0.001$), indicating different patterns of change between the two groups over 4 weeks. First peak knee flexion increased from 12.14 degrees to 20.61 degrees in the stimulation group and from 14.34 degrees to 19.52 degrees in the control group. The between-group difference in change from baseline to W4 was 3.40 degrees (95% CI, 2.24 to 4.56; $P < 0.001$). Because first peak knee flexion differed between groups at baseline, a sensitivity analysis was further conducted in the 42 complete cases by including baseline first peak knee flexion as a covariate when modeling changes from W1 to W4. After adjustment for baseline first peak knee flexion, the group-by-time interaction remained significant (chi-square = 28.86, P

< 0.001), and the between-group difference in change from baseline to W4 was 3.18 degrees (95% CI, 1.97 to 4.38).

The group-by-time interaction for second peak knee flexion was also significant (chi-square = 50.88, $P < 0.001$). After 4 weeks, second peak knee flexion increased from 40.79 degrees to 50.89 degrees in the stimulation group and from 38.57 degrees to 44.12 degrees in the control group. The between-group difference in change from baseline to W4 was 4.60 degrees (95% CI, 3.17 to 6.04; $P < 0.001$).

The increase in first peak knee flexion suggests improved shock-absorption control during loading response, whereas the increase in second peak knee flexion suggests enhanced lower-limb propulsion and flexion preparation during the transition from terminal stance to swing. These findings support the interpretation that improvement in gait phase was accompanied by improved dynamic kinematic performance of the paretic lower limb.

Sensitivity analysis

To assess the robustness of the findings, the same models were further repeated using non-imputed data in the 42 complete cases who completed all observations from W0 to W4. The group-by-time interactions for stance-phase percentage, single-support phase percentage, and knee flexion peaks remained significant, and the directions of effect were consistent with those in the primary analysis, supporting the robustness of the findings. Supplementary gait speed analysis and the stance-phase percentage model adjusted for concurrent gait speed are shown in Table 4.

Table 2
ITT longitudinal model results.

Outcome	chi-square	Interaction P value	Between-group difference in change, beta (95% CI)	Control, W0 to W4	Stimulation, W0 to W4
Stance-phase percentage (primary outcome)	38.84	$P < 0.001$	-3.40 percentage points (-4.49, -2.31)	69.46%→64.78%	68.85%→60.59%
Single-support phase percentage (secondary temporal outcome)	43.39	$P < 0.001$	4.21 percentage points (2.86, 5.57)	22.46%→25.54%	23.74%→31.06%
First peak knee flexion (secondary kinematic outcome)	41.94	$P < 0.001$	3.40 degrees (2.24, 4.56)	14.34°→19.52°	12.14°→20.61°
Second peak knee flexion (secondary kinematic outcome)	50.88	$P < 0.001$	4.60 degrees (3.17, 6.04)	38.57°→44.12°	40.79°→50.89°
Note: Table 2 presents the intention-to-treat analysis after multiple imputation and includes all 50 randomized participants, with 25 in the stimulation group and 25 in the control group. After multiple imputation of missing longitudinal outcome data from W0 to W4, random-intercept linear mixed-effects models were used for analysis. Fixed effects included group, time, and the group-by-time interaction, with time treated as a categorical variable. The interaction P value indicates the overall test of the group-by-time interaction. The beta value indicates the between-group difference in change at W4 relative to baseline. Values for W0 to W4 are descriptive means after imputation, whereas model inference is based on beta values and 95% CIs. CI indicates confidence interval.					

Table 3
Complete-case supplementary analysis.

Outcome	chi-square	Interaction P value	Between-group difference in change, beta (95% CI)	Control, W0 to W4	Stimulation, W0 to W4
Stance-phase percentage	35.23	$P < 0.001$	-3.34 percentage points (-4.45, -2.24)	69.77%→65.00%	68.43%→60.32%
Single-support phase percentage	39.37	$P < 0.001$	4.22 percentage points (2.85, 5.58)	22.59%→25.69%	23.86%→31.18%
First peak knee flexion	40.14	$P < 0.001$	3.43 degrees (2.24, 4.61)	12.97°→18.13°	11.45°→20.04°
Second peak knee flexion	46.74	$P < 0.001$	4.60 degrees (3.16, 6.05)	39.39°→44.86°	39.84°→49.91°
Note: Table 3 presents the supplementary analysis in the 42 participants who completed all observations from W0 to W4, with 21 participants in each group and 210 longitudinal observations in total. The supplementary analysis also used linear mixed-effects models. Because this table is based on the complete-case set, the corresponding baseline means are not exactly the same as those in the 50 randomized participants shown in Table 1. The interaction P value indicates the overall test of the group-by-time interaction. The beta value for W4 relative to baseline indicates the between-group difference in change from baseline to week 4. CI indicates confidence interval.					

Table 4
Gait speed sensitivity analysis.

Analysis	chi-square	P value (group x time)	Between-group difference in change, beta (95% CI)
Gait speed	74.52	< 0.001	0.07 m/s (0.05, 0.08)
Stance-phase percentage (unadjusted for gait speed)	35.23	< 0.001	-3.34 percentage points (-4.45, -2.24)
Stance-phase percentage (adjusted for concurrent gait speed)	15.79	0.003	-2.67 percentage points (-3.96, -1.37)
Note: Table 4 includes the 42 participants who completed all observations from W0 to W4. All analyses used linear mixed-effects models, with time treated as a categorical variable. Gait speed refers to the complete-case supplementary gait speed analysis. Stance-phase percentage (unadjusted for gait speed) refers to the complete-case supplementary model. Stance-phase percentage (adjusted for concurrent gait speed) refers to the exploratory sensitivity analysis in which concurrent gait speed was included as a covariate in the model. The beta value indicates the between-group difference in change at W4 relative to baseline. Percentage points are abbreviated as pp. CI indicates confidence interval.			

Exploratory changes in functional and cognitive outcomes

Among the 42 participants who completed all observations from W0 to W4, TUG decreased from 33.64 s to 23.61 s in the stimulation group and from 36.02 s to 29.28 s in the control group, with a greater improvement observed in the stimulation group. The between-group difference in change was -3.29 s (95% CI, -6.47 to -0.11 ; $P = 0.043$).

Over the same period, MoCA increased from 20.76 ± 3.90 to 23.24 ± 4.00 in the stimulation group and from 19.43 ± 3.19 to 20.81 ± 2.94 in the control group, with a greater increase in the stimulation group. The between-group difference in change was 1.10 points (95% CI, 0.63 to 1.56; $P < 0.001$) (Fig. 5).

These findings suggest that improvements in gait phase and kinematics may be accompanied by improvements in overall functional mobility and cognitive status. However, because TUG and MoCA were measured only at W0 and W4 and the related analyses were based on complete cases, these results should be interpreted as exploratory auxiliary outcomes.

Discussion

This study showed that, among inpatient rehabilitation stroke survivors with cognitive impairment, 4 weeks of dorsal foot sensory electrical stimulation combined with conventional rehabilitation further improved abnormal paretic stance phase during normal walking, and this change was not fully explained by increased gait speed. Together with the increased single-support phase percentage and improved knee flexion peaks, these findings suggest that dorsal foot sensory input may promote paretic weight-bearing participation and reorganization of gait phase control, which represents the main clinical significance of this study.

Post-stroke gait asymmetry usually reflects not only insufficient motor output but also reduced paretic weight-bearing capacity and impaired dynamic stability control [5, 23]. Previous studies have suggested that patients with stroke often maintain walking stability by prolonging double-support time and reducing paretic single-limb weight-bearing time [23, 24]. Therefore, abnormal paretic stance time represents a stability-prioritizing compensatory strategy rather than a simple change in a temporal parameter. In this study, baseline paretic stance-phase percentages were 68.85% and 69.46% in the two groups, both higher than the reference range of approximately 60% during comfortable walking in healthy adults. After 4 weeks, the value decreased to 60.59% in the stimulation group but remained around 64.78% in the control group. Although stance-phase percentage is influenced by gait speed, individual compensatory strategies, and balance strategies and should not be directly equated with normalization, these results at least suggest that paretic weight bearing and gait phase distribution in the stimulation group became closer to a healthy gait pattern during overall walking recovery.

Compared with most previous functional electrical stimulation (FES) studies that used gait speed as the primary endpoint, the present study focused more specifically on changes in the temporal structure within the gait cycle. Gait speed reflects overall walking ability but cannot easily distinguish whether faster walking is achieved through improved paretic control or compensatory gait strategies [4, 25, 26]. In this study, stance-phase percentage remained significantly different after adjustment for concurrent

gait speed, even while gait speed improved. This finding is important because it suggests that the effect of dorsal foot sensory electrical stimulation may not be limited to enabling patients to walk faster but may also influence the internal phase organization of gait. In other words, patients may have changed their gait control strategy after the intervention, rather than simply increasing overall motor output.

This interpretation is consistent with recent views on post-stroke gait control. Increasing evidence suggests that walking after stroke is not a fully automatic rhythmic activity but depends substantially on sensorimotor integration and allocation of attentional resources [7–12, 24]. Particularly in stroke survivors with cognitive impairment, insufficient paretic limb participation may arise not only from muscle weakness but also from impaired attentional allocation and reduced efficiency in using sensory input [7–12]. The dorsal foot sensory stimulation used in this study did not directly induce ankle dorsiflexion but primarily provided peripheral sensory input above the perceptual threshold. Stimulation was mainly delivered during the swing phase, based on the rationale that sensory input in late swing may influence subsequent foot contact and early-stance loading patterns [13, 14, 19, 24, 27]. From this perspective, the observed change in stance phase may reflect enhanced paretic limb participation and sensorimotor regulation.

Notably, stance-phase percentage decreased while single-support phase percentage increased from 23.74% to 31.06% in this study. Although these changes appear to occur in opposite directions, they may jointly reflect a redistribution of the internal temporal structure of the paretic stance phase. An increase in single-support phase suggests a longer duration of independent paretic single-limb weight bearing, whereas a decrease in overall stance-phase percentage suggests that double-support time may have shortened. Because double-support time is generally considered a compensatory period for maintaining stability [3, 6, 23, 24, 28], its reduction may indicate improved capacity to rely on paretic limb loading. However, because double-support phase percentage was not directly collected in this study, this interpretation should be treated with caution.

In addition to temporal variables, this study observed concurrent increases in first and second peak knee flexion. The first peak mainly reflects shock absorption during loading response, whereas the second peak is related to lower-limb propulsion and flexion preparation during the transition from terminal stance to swing [20, 21]. Previous studies have suggested that insufficient paretic propulsion and restricted knee motion after stroke can impair swing initiation [22, 29]. In the present study, increases in knee flexion peaks occurred in parallel with changes in stance phase, suggesting that the intervention was accompanied not only by changes in temporal structure but also by improved dynamic kinematic performance of the paretic lower limb. This concurrent temporal-kinematic change indicates a pattern closer to improved gait coordination control than to a simple increase in speed.

Results from previous FES-related randomized controlled trials have not been entirely consistent. van Bloemendaal et al. [30] did not observe an improvement in step-length symmetry after multichannel FES training, and the PLEASURE trial [31] also reported similar improvements in walking ability between FES and control conditions. These differences may partly relate to outcome selection. Most previous studies

focused mainly on gait speed, step length, or functional scales, with less detailed analysis of temporal changes within the gait cycle [3, 4, 26, 32]. In the present study, gait speed increased, stance-phase percentage decreased, single-support phase increased, and knee kinematics improved, collectively supporting a pattern of improved walking ability accompanied by reorganization of gait phase. This also suggests that macro-level walking indicators alone may be insufficient to fully capture changes in post-stroke gait control.

This study specifically enrolled stroke survivors with cognitive impairment, which differs from most studies that primarily use motor impairment as the main inclusion criterion. In this population, gait control may be more vulnerable to limitations in attentional resources and cognitive load [9]. Although MoCA improved more in the stimulation group than in the control group in the complete-case analysis, this study did not analyze specific cognitive domains, and MoCA was not included in the imputed longitudinal primary analysis. Therefore, the findings cannot currently be interpreted as evidence that dorsal foot sensory electrical stimulation has an independent cognitive benefit. A more appropriate interpretation is that the change in cognitive score may reflect an accompanying improvement in overall functional status. Future studies should further examine this issue using dual-task gait assessment, attention-network measures, and executive function testing.

This study has several limitations. First, the sample size was relatively limited, and all participants were within 6 months after stroke onset; therefore, it remains unclear whether patients at different stages after stroke would show different treatment responses. Second, although missing data were handled using multiple imputation and complete-case supplementary analyses were also performed, the results may still be affected by model specification if the missingness mechanism was not random. In particular, the analysis adjusted for gait speed was based only on complete cases and cannot fully represent the primary imputed full-sample analysis; therefore, it is more appropriately interpreted as exploratory mechanistic evidence. Third, double-support phase, surface electromyography, joint kinetics, and cortical activity were not collected simultaneously. Thus, the relative contributions of enhanced sensory input, changes in weight-bearing strategy, and repeated training-related learning effects cannot yet be determined.

Conclusions

This study demonstrated that, among inpatient rehabilitation stroke survivors with cognitive impairment, 4 weeks of dorsal foot sensory electrical stimulation combined with conventional rehabilitation further improved abnormal paretic stance phase, and this change was not fully explained by increased gait speed. Concurrent improvements in single-support phase percentage and knee flexion peaks suggest that dorsal foot sensory input may contribute to the reorganization of paretic weight bearing and gait phase control after stroke. These findings support sensory-driven intervention as a potential complementary strategy for gait asymmetry rehabilitation in stroke survivors with cognitive impairment.

Abbreviations

CI
confidence interval
FES
functional electrical stimulation
ITT
intention-to-treat
LMM
linear mixed-effects model
MoCA
Montreal Cognitive Assessment
NIHSS
National Institutes of Health Stroke Scale
PT
perceptual threshold
RCT
randomized controlled trial
TUG
Timed Up and Go test
W0
baseline
W4
week 4.

Declarations

Ethics approval and consent to participate

The study protocol was approved by the Ethics Committee of University-Town Hospital of Chongqing Medical University on January 31, 2024. The trial was registered with the Chinese Clinical Trial Registry on March 5, 2024, before participant enrollment (ChiCTR2400081558). All participants provided written informed consent.

Consent for publication

Not applicable.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding authors on reasonable request. The full trial protocol is available from the corresponding authors on reasonable request.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

Yang Zhou and Chuanchuan Bai contributed equally to this work. Yang Zhou, Chuanchuan Bai, Xiu-Qing Yao, and Ya-Xi Luo conceived and designed the study. Yang Zhou, Chuanchuan Bai, Qiu-Yang Zhang, Ming-Zhu Yin, Jia-Hui Chen, Ya-Xing Li, and Dan Song contributed to participant recruitment, intervention implementation, outcome assessment, or data acquisition. Yang Zhou and Chuanchuan Bai performed the data analysis and drafted the manuscript. Xiu-Qing Yao and Ya-Xi Luo supervised the study and critically revised the manuscript. All authors interpreted the data, reviewed the manuscript, and approved the final version for submission.

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Authors' information

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Use of artificial intelligence tools

During manuscript preparation, ChatGPT was used for language polishing. The authors reviewed and edited all AI-assisted text and take full responsibility for the final content of the manuscript.

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Figures

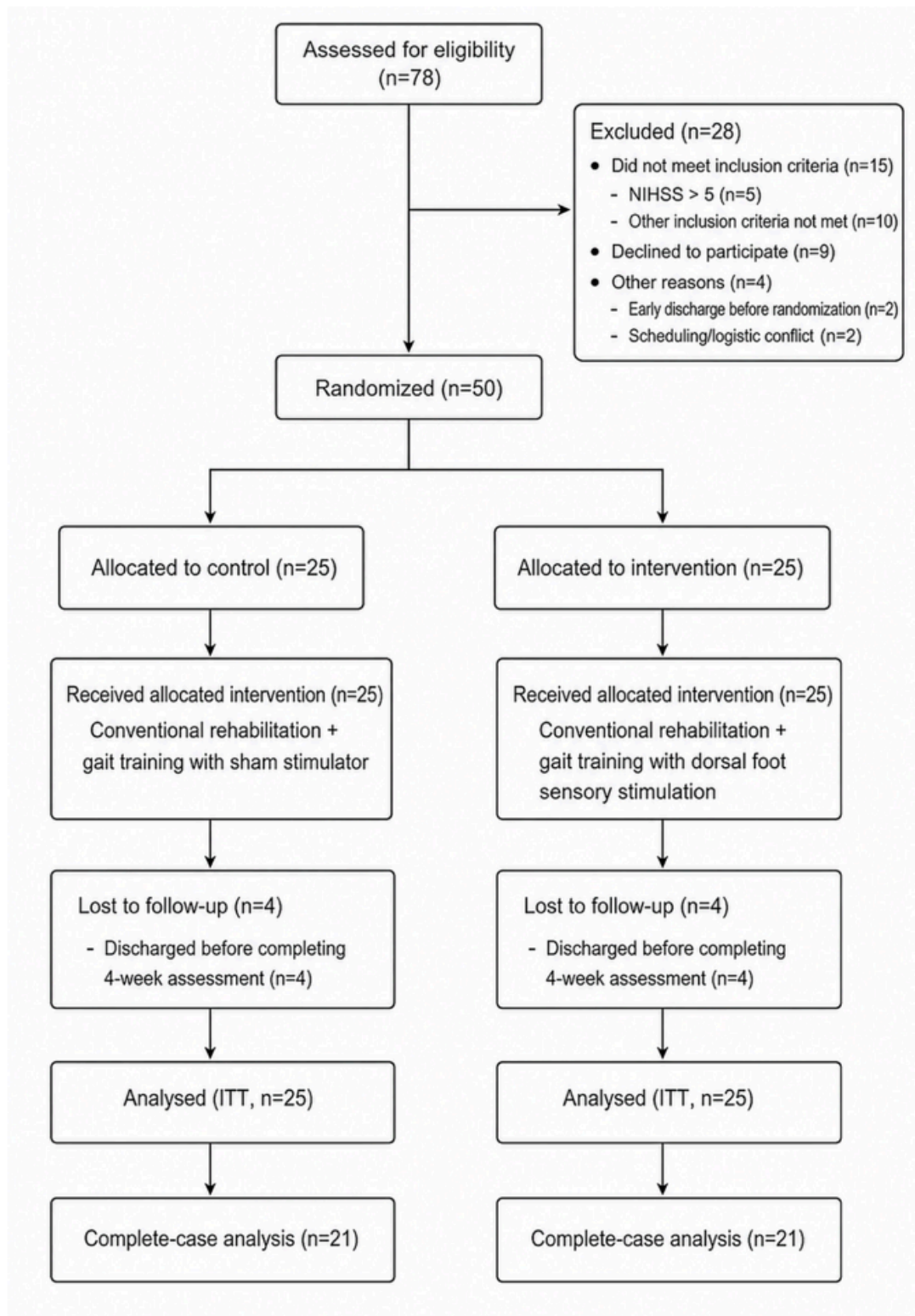


Figure 1

Participant flow diagram.

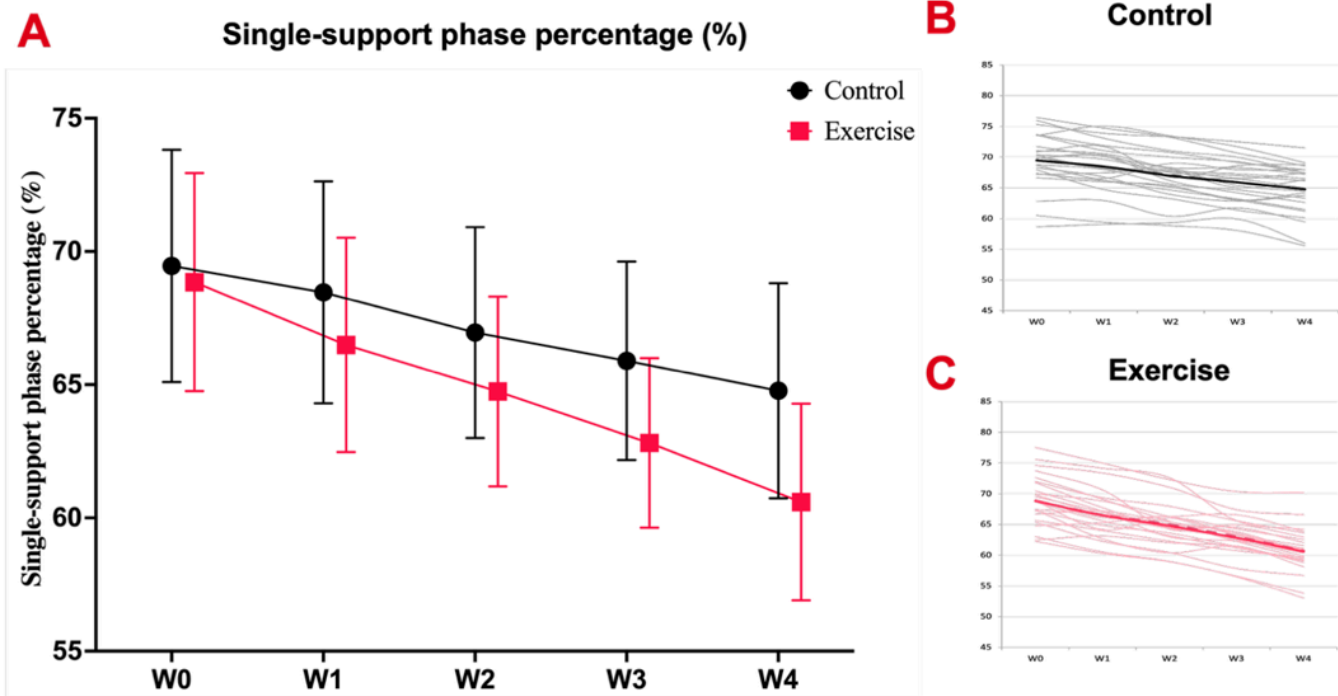


Figure 2

Stance-phase percentage from W0 to W4. Panel A shows group means and standard deviations, and panels B and C show fitted trajectories for all individuals in each group. Black circles indicate the control group and red squares indicate the stimulation group; error bars indicate standard deviations.

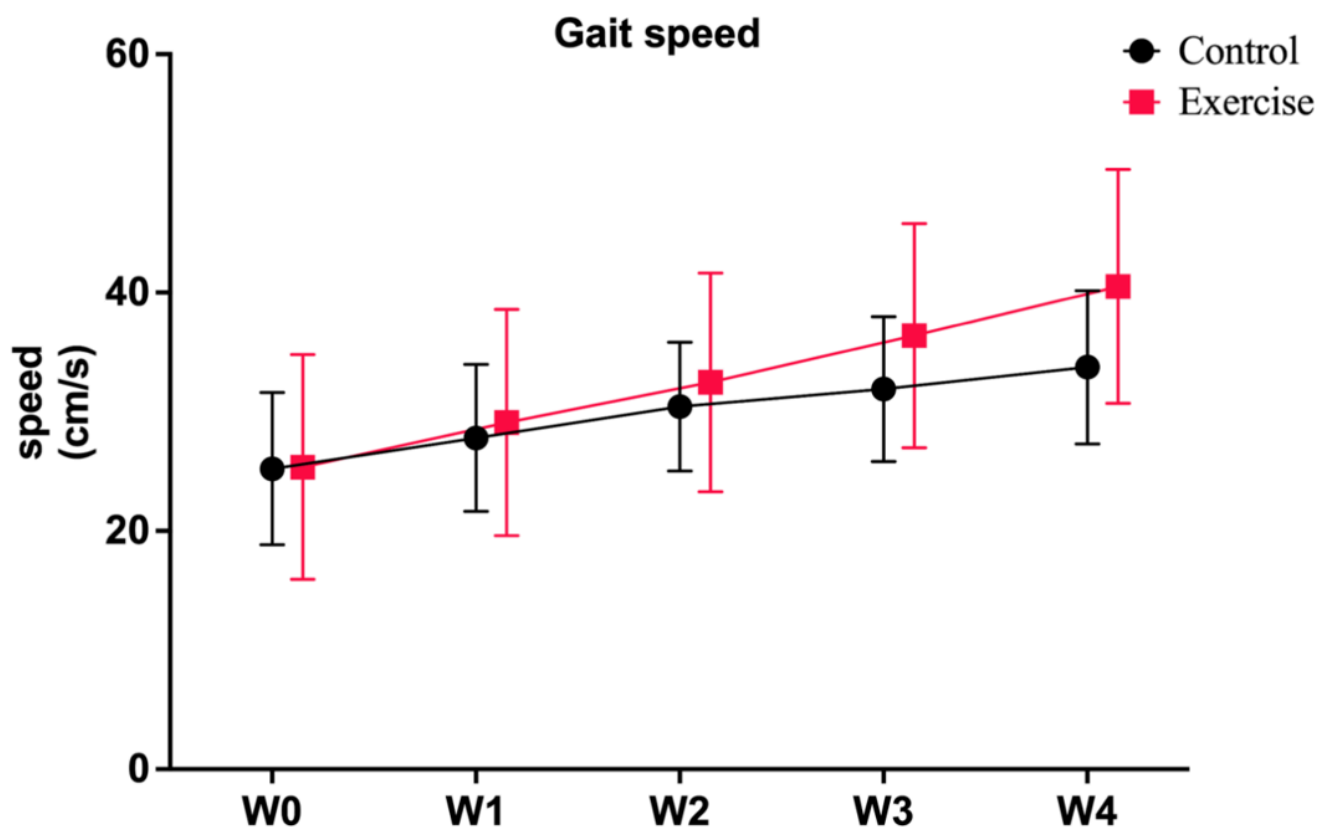


Figure 3

Gait speed from W0 to W4. Black circles indicate the control group and red squares indicate the stimulation group; error bars indicate standard deviations. The unit is cm/s.

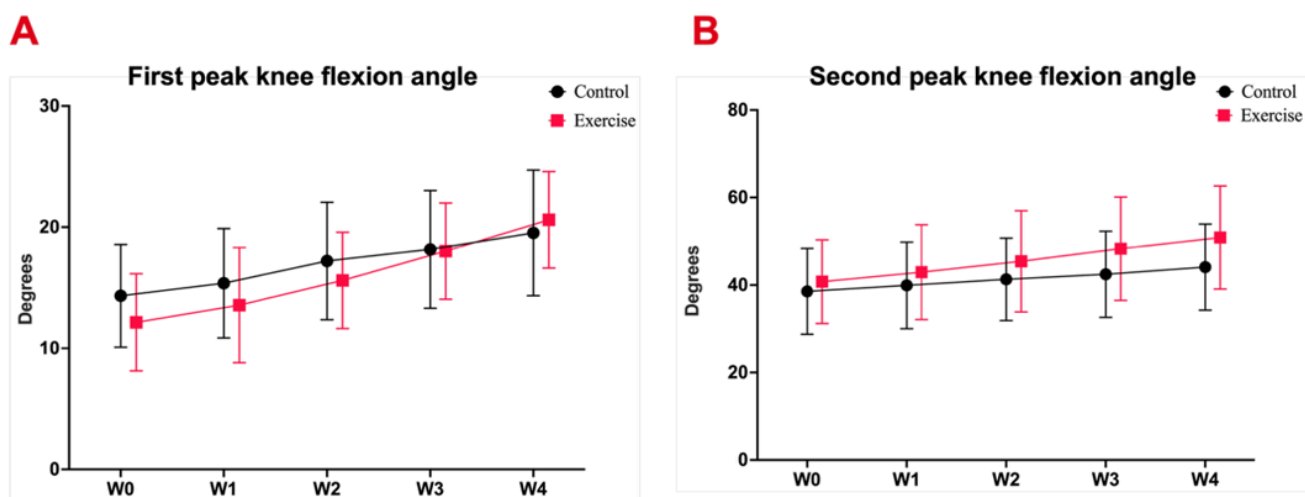


Figure 4

Knee flexion peaks from W0 to W4. Panel A shows first peak knee flexion and panel B shows second peak knee flexion. Black circles indicate the control group and red squares indicate the stimulation

group; error bars indicate standard deviations. The unit is degrees.

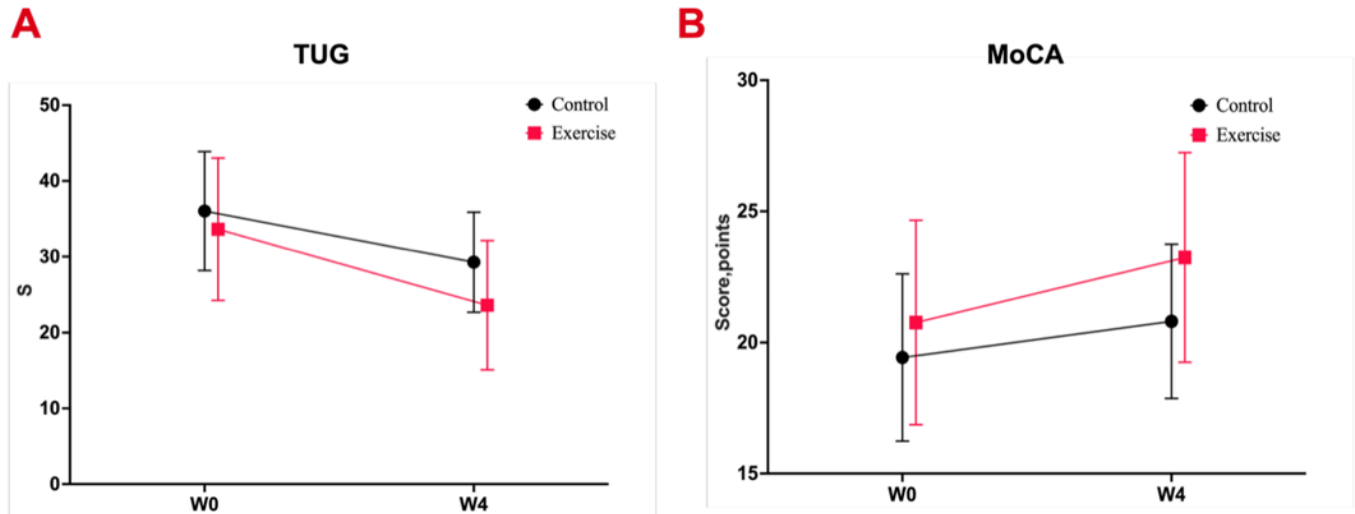


Figure 5

TUG and MoCA changes from W0 to W4. Panel A shows the Timed Up and Go test (TUG), in seconds (s), and panel B shows the Montreal Cognitive Assessment (MoCA), in points. Black circles indicate the control group and red squares indicate the stimulation group; error bars indicate standard deviations. Lower TUG values indicate better functional mobility. The total MoCA score is 30; 1 point was added for participants with ≤ 12 years of education, and higher scores indicate better global cognitive status. These results are interpreted as auxiliary observational outcomes and do not represent confirmatory primary outcomes.

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