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Transcranial Direct Current Stimulation Over the Motor or Frontal Cortex in Patients With Chronic Ankle Instability

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Context: Growing evidence has suggested clinical efficacy for the use of anodal transcranial direct current stimulation (atDCS) when combined with motor interventions in patients with chronic ankle instability (CAI). However, no studies have compared multiple approaches for improving motor function with atDCS in patients with CAI.

Objective: We therefore aimed to determine the efficacy of atDCS over the motor or frontal cortex when combined with a 4-week motor-planning intervention on neural function, performance, and patient-reported outcomes in patients with CAI.

Design: Double-blind, sham-controlled, parallel randomized controlled trial.

Main Outcome Measure(s): Participants (n = 44, 15 males, 29 females, age = 23.6 ± 6.1 years) were assessed for outcome measures of cortical and reflexive excitability; performance measures of dynamic balance, muscle activation, reaction times, and cognitive performance on a dual-task balance test; and patient-reported outcome measures at baseline, midtraining (week 2), posttraining (week 4), and retention (week 6). After baseline testing, participants were randomized to receive atDCS over the motor cortex or frontal cortex or a sham current during rehabilitation exercises over 4 weeks. Participants reported for 8 training sessions, where they were instrumented for atDCS while performing obstacle walking, dual-task balance, and agility exercises. Analyses between groups and time points were performed with mixed linear models ($\alpha = .05$).

Results: Forty-six individuals were recruited and randomized, with 37 completing the investigation (motor = 14, frontal = 11, sham = 12). No differences across groups or times were observed in neural excitability or muscle activation variables ($P > .05$). Significant improvements in dynamic postural stability indices were observed from baseline across all groups ($P < .05$). Improvements were observed for foot and ankle function, perceived disablement, and the Global Rating of Change at posttraining and retention ($P < .001$).

Conclusions: Improvements in patient function were observed across all groups, suggesting the motor-planning intervention improved function regardless of atDCS application. Observing benefits from atDCS may depend on proper pairing of rehabilitation exercise with electrode location.

Key Words: ankle sprain, neuromodulation, dual tasking, rehabilitation

Key Points

- Patient-reported function, functional performance, and balance improved after a 4-week rehabilitation intervention emphasizing motor planning in patients with chronic ankle instability.
- Participants received transcranial direct current stimulation over the motor or frontal cortex or a sham current; however, improvements were not tied to whether participants received an active brain stimulation current.
- Rehabilitation emphasizing motor planning may be beneficial for individuals with limited participation restrictions; however, further research is needed to understand mechanistic changes.

Ankle sprains are an often-confounding injury to clinicians due to high reinjury rates.[1] Reinjury rates up to 70% have been reported, with recurrent sensations of rolling termed *chronic ankle instability* (CAI) developing in approximately half of these patients.[2] Patients with CAI report impairments that include decreased balance, diminished neuromuscular control, and altered reaction times.[3] Collectively, these impairments lead to decreased health-related quality of life and physical activity across the lifespan, contributing to increased risk of long-term health complications.[4–6] This negative symptom progression originating from an ankle sprain suggests that current rehabilitation protocols that emphasize the minimization of impairment may need to be reconsidered to better address underlying factors contributing to decreased function.

In patients with ligamentous pathology at the knee and ankle, emerging evidence supports the presence of maladaptive neuroplasticity that may undermine rehabilitation efforts.[7,8] Current models suggest that both acute and chronic sensory changes contribute to inhibition at the cortical and segmental

levels, yielding decreased neural excitability. Although these inhibitory changes can hinder the activation of stabilizing musculature and potentially leave the joint vulnerable, muscle function is often regained through increased neural activation from extraneous areas, such as the cerebellum, contralateral motor cortex, somatosensory cortex, and the frontal cortex, as observed in individuals with CAI.[9,10] Therefore, muscle function appears largely restored following the injury rehabilitation process, but is being achieved with less efficient neural activation that can lead to degraded movement strategies in complex and unconstrained environments.[11] These models suggest the need to consciously address maladaptive neuroplasticity throughout rehabilitation efforts for CAI and other ligament pathologies in order to adequately restore patient function.[7]

Many common clinical therapies have neuromodulatory effects that may positively affect neuroplasticity in patients with musculoskeletal injury.[12] Recently, transcranial direct current stimulation (tDCS) has been implemented in patients with chronic and acute ligament pathology to directly address neuroplasticity.[7,12,13] Transcranial direct current stimulation implements a direct current across the brain to modify synaptic plasticity.[14] In most rehabilitation research contexts, anodal tDCS (atDCS) has been implemented to facilitate the primary motor cortex (M1) while individuals perform exercises emphasizing muscle strength. In patients with CAI and after anterior cruciate ligament reconstruction, this protocol has been tied to improved postural control, neural excitability, and health-related quality of life after a 2- to 4-week intervention.[15–17] However, these improvements are compared with a sham current rather than alternate cortical targets. The dorsolateral prefrontal cortex (DLPFC) has been targeted with atDCS in individuals with neurologic pathologies (eg, stroke, Parkinson disease) and healthy adults to evoke improvements in motor planning and affect motor cortex activity and performance.[18,19] Given the recruitment of extraneous cortical areas in patients with CAI, such an intervention may be effective in this population.

The current study aimed to investigate the comparative effects of rehabilitative exercises when combined with atDCS over the M1, atDCS over the DLPFC, and sham tDCS on neural excitability, dynamic balance, neuromuscular control, and patient-reported function in patients with CAI. We hypothesized that stimulation over the M1 would yield the greatest improvements in neural excitability and muscle activation, whereas stimulation over the DLPFC would yield the greatest improvements in reaction times and dynamic balance, indicating enhanced motor planning.[20,21] We further hypothesized that both groups receiving tDCS would improve patient-reported function to a greater degree than those receiving sham stimulation and maintain those changes after cessation of the rehabilitation program.

METHODS

Study Design

The present study implemented a double-blind parallel randomized controlled trial. Participants performed a 4-week intervention consisting of 8 rehabilitation sessions that emphasized muscle activation and motor planning. Participants were tested for outcome measures at week 0 (baseline), week 2 (midtraining), week 4 (posttraining), and week 6 (retention). Independent variables included group (motor, frontal, sham) and time (baseline, midtraining, posttraining, retention). Dependent variables included measures of neural excitability, dynamic postural control, lower-leg muscle activation and reaction times during a reactive hop test, and patient-reported outcome measures (modified Disablement in the Physically Active Scale [mDPAS], Foot and Ankle Ability Measure [FAAM], Tampa Scale of Kinesiophobia [TSK], and Global Rating of Change [GROC]). Participants were masked to group allocation, and both therapists and assessors were masked to whether participants received an active or sham current, although they were aware of electrode location (motor or frontal). This clinical trial was registered on clinicaltrials.gov (NCT06024720).

Participants

Forty-six individuals with CAI were recruited for the present study. Classification with CAI followed guidelines from the International Ankle Consortium,[22] including experiencing their first ankle sprain more than 1 year before study enrollment and scoring above a 10 on the Identification of Functional Ankle Instability instrument.[23] Participants had no history of foot, ankle, or lower leg fractures or surgery or injuries restricting physical activity in the 3 months before study enrollment and reported no red-green color vision deficiency. Additionally, participants met criteria for the safe practice of transcranial magnetic stimulation (TMS) and tDCS, including no personal or immediate family history of seizure or epilepsy, metallic implants or medication infusion devices, skull abnormalities, frequent headaches or migraines, concussion within 6 months, or medications that raised the risk of seizure.[24–26] Sample size was based on preliminary data, which reported effect sizes of η_p^2 between 0.07 and 0.14.[16] To achieve statistical power ($1 - \beta$) of .95 with the previously observed effect ($f = 0.27$) and a level of significance (α) at .05, 11 participants were required per group. To account for potential attrition of up to 25%, we aimed to recruit 45 total participants. All participants provided informed consent as approved by the Appalachian State University Institutional Review Board (22-0050).

Outcome Measures

Neural excitability was assessed using the Hoffmann reflex (H-reflex) and TMS for segmental and corticospinal excitability, respectively. For these measurements, participants were instrumented with electromyography (EMG) electrodes over the tibialis anterior (TA), peroneus longus, and soleus. The area over each muscle was palpated, shaved if necessary, cleaned with isopropyl alcohol, and abraded, and an active electrode connected to an amplifier (B&L Engineering) was placed along the muscle.[27] The H-reflex was assessed first with the patient in a prone position. A bar electrode connected to a constant current stimulator (DS7R; Digitimer LTD) set to 300 V was placed in the popliteal fossa. To assess H-reflex across all target muscles, the location of the sciatic nerve before its bifurcation was identified using 10-mA pulses and used for subsequent testing.[28] Pulses (1 millisecond duration) were applied every 10 seconds beginning at a stimulation of 0 and increasing by 2 mA each pulse until a plateau response was noted across all recorded muscles. The ratio of the maximal reflexive response (H_{max}), occurring 40 to 80 ms from stimulus, was compared with the maximal motor response (M_{max}), occurring from 10 to 30 ms from stimulus, to derive $H_{max} : M_{max}$ for each muscle.[28]

After H-reflex assessment, cortical excitability was assessed simultaneously across all muscles using TMS. Participants were seated and familiarized with TMS before the M1 was located by providing submaximal pulses and observing the location yielding the largest response in the TA.[29] The TA was selected due to its greater cortical representation relative to the peroneus longus and soleus.[30,31] Following these procedures, 40 to 50 stimuli, ranging from below the previously noted motor threshold to above a maximal response that would be expected from each muscle, were applied to obtain a stimulus-response curve from each muscle.[32] The stimulus-response curve was used to estimate the resting motor threshold (RMT) and maximum motor evoked potential (normalized to M_{max}) for each muscle.[29] All neural outcomes were assessed in an electromagnetically-shielded laboratory.

Dynamic balance, muscle activation, and reaction times were assessed using a reactive hop. Participants were reinstrumented with EMG as described above using a system that allowed for free movement (Bagnoli-4; Delsys Inc). Participants were positioned in unipedal stance in the middle of 3 in-ground force plates (60 × 90 cm; Bertec), with a 5-cm vertical hurdle placed between each force plate. Three reactive lights (ROXProX; A-Champs) were placed on tripods surrounding the participant, with one 10 feet directly in front of the participant (memory light), and the other 2 placed at the front outside corners of the adjacent force plates (trigger lights). Participants were familiarized with the task, consisting of monitoring the memory light, which flashed 1 of 4 colors every 2 seconds, as participants were instructed to recall the previous 3 colors in order. At a random time in a 15-second interval, the trigger lights illuminated, and participants were instructed to hop toward the green-lit light. When participants were within 50 cm of the trigger light, it would deactivate and send a reaction time (ie, time between trigger light illumination and deactivation) to the linked software via Bluetooth connection (ROXPro Android application; A-Champs). Participants were instructed to land on the affected side, regain balance for 15 seconds, and recite the 3 colors that occurred before the trigger light. To offset fatigue, participants did 3 consecutive trials and then were provided a 1- to 2-minute rest period. Three trials were provided for familiarization and practice, and then a minimum of 5 hops to each side were collected. If participants did not successfully complete the task (eg, did not clear the hurdle, touched opposite limb down within 15 seconds, hopped in the wrong direction), an error was recorded on the data collection sheet and the trial was not included in the 5 that needed to be completed to each side. This method provided strong reliability for reaction time and color memory across multiple trials (intraclass correlation coefficient [1,k] between 0.707 and 0.828). Force plate and EMG data were collected in custom LabVIEW software (National Instruments) at 1000 Hz.

Dynamic balance was quantified from force plate data using dynamic postural stability indices (DPSIs), including anteroposterior (APSI), mediolateral (MLSI), and vertical (VSI) components.[20] Electromyography data were band-pass filtered (20–400 Hz), rectified, and low-pass filtered (10 Hz) to create a complete linear envelope, and normalized to peak activation across all trials. Average activation for each muscle was extracted in the 500 ms before and after landing on each force plate. Reaction times were recorded from the reactive lights using the ROXPro Android application, and color memory was written down noting the number of correct colors out of the 3 displayed. Trials were further stratified into medial and lateral hops, relative to the test leg.

Patient-reported outcome measures were collected in the Research Electronic Data Capture (REDCap) tools hosted by Appalachian State University.[33] Foot and ankle function was assessed using the FAAM, including Activities of Daily Living (ADL) and Sport subscales.[34] Health-related quality of life was assessed using the mDPAS.[35] Kinesiophobia related to sport activity was assessed using the TSK 17-item scale.[36] Lastly, global changes from baseline were assessed at midtraining, posttraining, and retention using the GROC, which consists of a 15-point scale, where 0 indicated no change (*about the same*) from baseline, 7 indicated *a very great deal better* and −7 indicated *a very great deal worse*.[37] As a performance-based metric of patient function, participants also performed a side-hop test, consisting of 10 side-to-side hops over 2 parallel lines placed 30 cm apart.[38] Participants were provided a full practice trial, and the time to complete a single trial was extracted. To test fidelity of masking efforts, at the conclusion of the retention visit, participants were asked whether they felt they received the real or sham current.

Intervention

After the baseline test session, participants were randomized into either motor, frontal, or sham groups using a block-randomization scheme (randomized block sizes of 3–9). To maintain masking, individuals were assigned a 6-digit code that was entered into a stimulator (1 × 1 CT; Soterix) that corresponded with an active current (2.0 mA provided over 18 minutes) or a sham current to mimic some sensation while maintaining participant blinding (2.0 mA provided over 1 minute).[16,19] The codes were accompanied by a stimulus location, instructing researchers where to place electrodes (frontal or motor location). This allowed for participants in the sham group to be near-equally allocated to have electrodes placed at motor or frontal locations.

Participants reported to a separate laboratory and were instrumented with tDCS. For participants receiving the motor location, a 5 × 3 anode (EASYPad; Soterix) was placed at the C3/C4 location of the International 10-20 System, corresponding with the hemisphere contralateral to the test ankle, and the same-sized cathode was placed at the opposite supraorbital area (ipsilateral to the test ankle).[16] For participants receiving the frontal location, a 5 × 7 EASYPad anode was placed over the F3/F4 location of the International 10-20 System, corresponding with the hemisphere contralateral to the injured ankle, and the same-sized cathode was placed at the opposite F3/F4 location.[18] Electrodes were saturated with 6 to 8 mL of saline before placement on the individual, and electrodes were secured using an elastic fastener set provided by the manufacturer. Good electrode impedance was assured before beginning the stimulation. Participants were instructed to sit for 2 minutes after beginning stimulation, and then proceed through the exercise protocol.

Full details of the rehabilitation progression, including stages of progressions achieved by each group, are included in the Supplemental Material. First, participants performed hurdle walking, performed laterally and at oblique angles. Second, participants performed a unipedal stand-and-reach task with a go/no-go component, in which participants balanced on the test side and reached with the nontest side toward a light trigger placed at 50% of leg length in front of the participant.

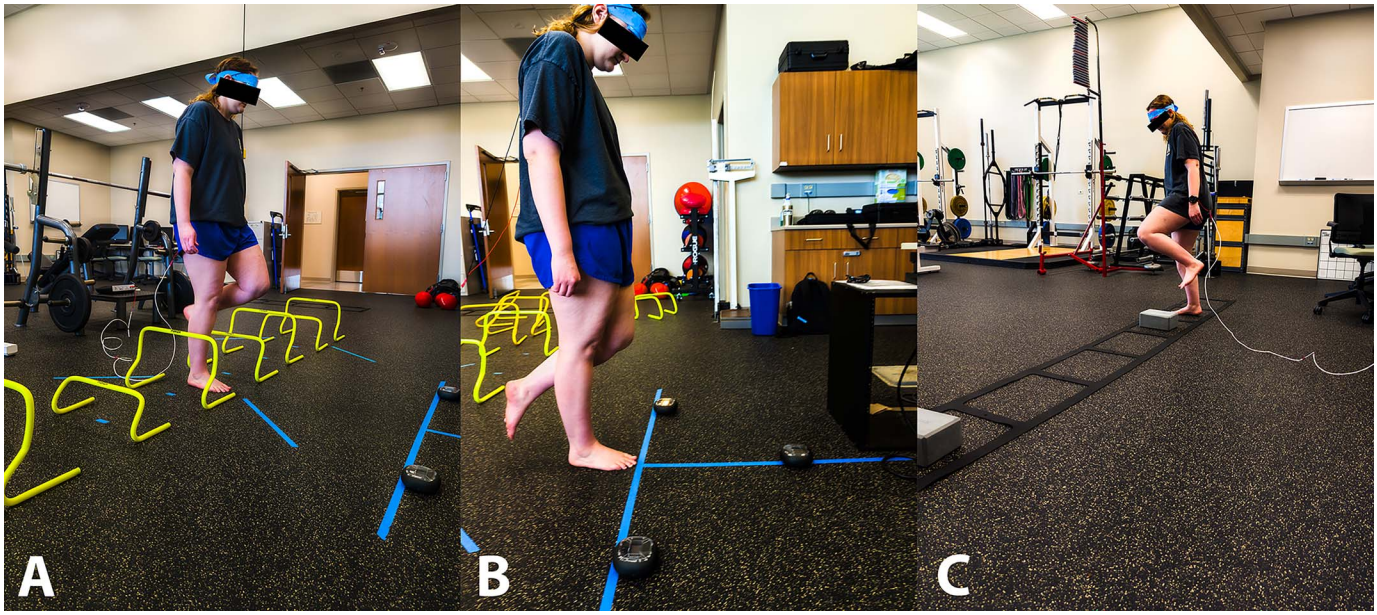

Figure 1. Exercises included in the rehabilitation progression. A, Lateral and oblique hurdle walking. B, Go/no-go unipedal balance. C, Agility ladder. All exercises were performed with transcranial direct current stimulation instrumented (frontal montage pictured).

Lastly, participants performed 6 agility ladder exercises consisting of 3 rounds of lateral stepping and 3 rounds of a shuffle step (Figure 1). Treatment sessions were designed to take 18 to 20 minutes, aligning with the duration of tDCS administration. In all cases, tDCS remained through the entire 18-minute timer.

Data Analysis

All data were assessed using linear mixed models to account for the nested nature of the data and allow for the inclusion of partial data in an intention-to-treat analysis. Descriptives were examined and baseline characteristics were compared for a group effect. Each variable was assessed for fixed effects of group (motor vs frontal vs sham), time (baseline, midtraining, posttraining, and retention), and group-by-time interaction effect. In the case of a significant effect on omnibus tests for fixed effects, we reported the marginal (R^2_M) and conditional (R^2_C) R^2 values to estimate the contribution of fixed and random effects on the linear mixed models.[39] Further, for significant fixed effects, parameter estimates were examined post hoc to assess the sources of significant differences. To allow for understanding the magnitude of the observed changes, 95% CIs were reported surrounding mean differences (MDs). An a priori level of significance (α) was set at .05.

RESULTS

Demographics

Forty-six individuals were recruited for this study, with 37 completing all testing sessions and considered compliant ($\geq$6 out of 8 training sessions completed) (Figure 2). There were no significant differences in attrition across groups ($\chi^2_2 = 1.342$, $P = .511$), with only the frontal group having retention below 80%. Demographic information for all groups is presented in Table 1. No significant differences were observed between groups for sex ($\chi^2_2 = 5.004$, $P = .082$), age ($F_{2,41} = 0.658$, $P = .523$), height ($F_{2,41} = 0.963$, $P = .392$), mass ($F_{2,41} = 0.254$, $P = .777$), or Identification of Functional Ankle Instability scores ($F_{2,41} = 0.880$, $P = .422$). A χ^2 analysis was conducted to explore whether individuals in each group perceived themselves to have received an active or sham current. In the motor, frontal, and sham groups, 72.7%, 75.0%, and 72.7% of participants thought they had received an active current, respectively ($\chi^2_2 = 0.015$, $P = .992$).

Neural Excitability

Means, SDs, and omnibus effects for $H_{max}:M_{max}$, RMT, and maximum motor evoked potential are presented in Table 2. No significant main effects of time, group, or group-by-time interactions were observed for neural excitability variables within any muscle.

Dynamic Postural Control and Muscle Activation

Means, SDs, and omnibus effects for DPSI, APSI, MLSI, and VSI during medial and lateral hops are presented in Table 3. No significant group or group-by-time interaction effects were noted for any dynamic postural control variables. Significant main effects of time were observed for DPSI and VSI on the lateral hops (DPSI: $F_{3,100.2} = 4.389$, $P = .006$, $R^2_M = 0.120$, $R^2_C = 0.559$; VSI: $F_{3,100.2} = 4.225$, $P = .007$, $R^2_M = 0.118$, $R^2_C = 0.555$). Significant improvements in postural stability from baseline were noted at midtraining (DPSI: MD = −0.04; 95% CI = −0.06, −0.02; $P < .001$; VSI: MD = −0.04; 95% CI = −0.06, −0.02; $P < .001$), posttraining (DPSI: MD = −0.03; 95% CI = −0.05, 0.00; $P = .020$; VSI: MD = −0.03; 95% CI = −0.05, −0.00; $P = .023$), and retention (DPSI: MD = −0.03; 95% CI = −0.06, −0.01; $P = .014$; VSI: MD = −0.03; 95% CI = −0.05, −0.01; $P = .017$) for lateral hops.

Means, SDs, and omnibus effects for muscle activation during medial and lateral hops are presented in Table 4. No significant main effects of time, group, or group-by-time interaction effects were observed for average activation within any muscle before or after landing.

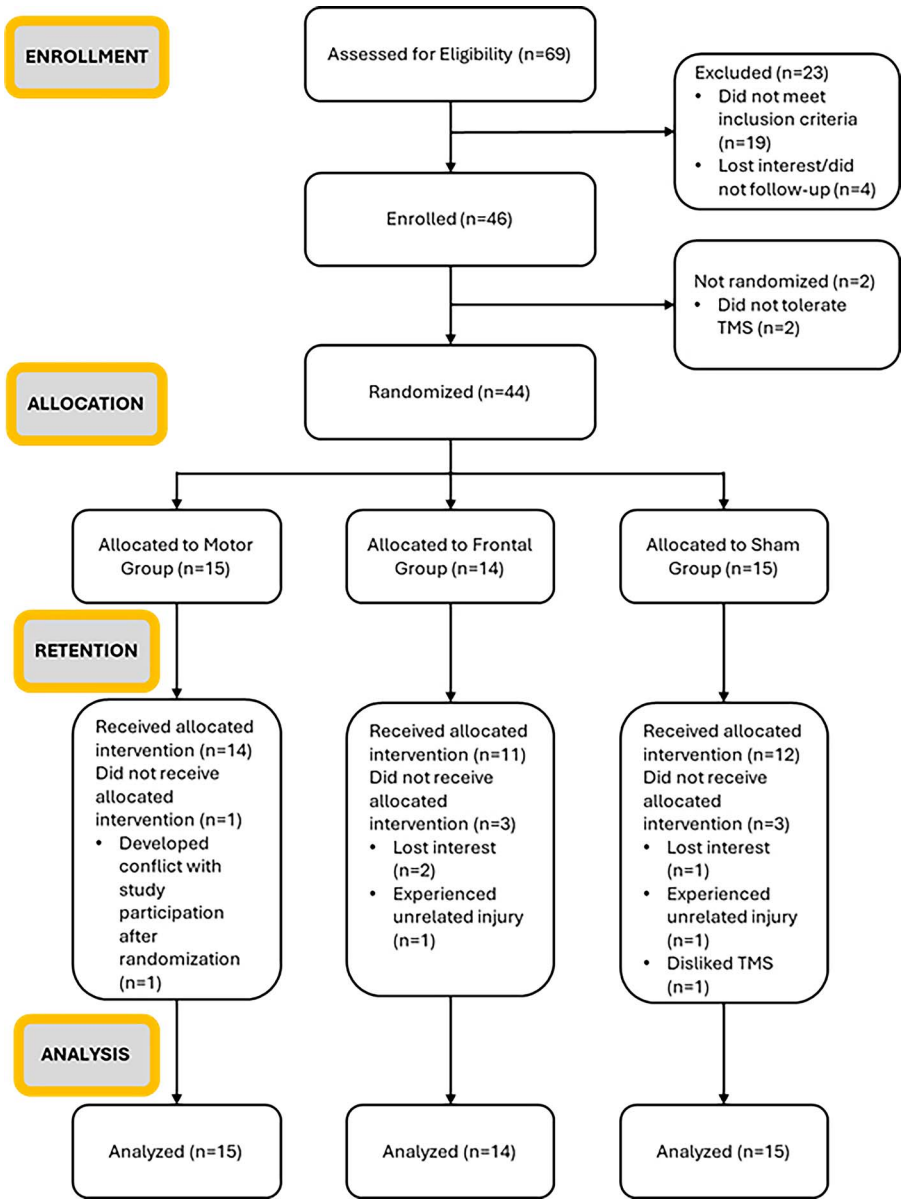

Figure 2. Consolidated Standards of Reporting Trials diagram depicting the flow of participants through the study. Abbreviation: TMS, transcranial magnetic stimulation.

Reaction Times and Cognitive Performance

Reaction times and cognitive performance are presented in Table 5. No significant changes in reaction times on medial or lateral hops were observed for the main effects of group, time, or group-by-time interaction effect. Improvements were observed for the number of colors correctly recalled over time ($F_{3,108.0} = 9.83$, $P < .001$, $R^2_M = 0.146$, $R^2_C = 0.478$), with significant differences from baseline noted at midtraining (MD = 0.2; 95% CI = 0.1, 0.3; $P < .001$), posttraining (MD = 0.2; 95% CI = 0.1, 0.4; $P < .001$), and retention (MD = 0.3; 95% CI = 0.2, 0.4; $P < .001$).

Table 1. Participant Characteristics

	Motor	Frontal	Sham	*P* Value
No. recruited (M/F)	15 (3/12)	14 (8/6)	15 (4/11)	.082
Age, mean ± SD, y	22.6 ± 2.7	22.9 ± 5.6	25.2 ± 8.7	.523
Height, mean ± SD, cm	169.1 ± 12.9	175.3 ± 12.6	180.4 ± 9.1	.392
Mass, mean ± SD, kg	69.3 ± 14.0	73.2 ± 12.5	72.4 ± 16.6	.777
IdFAI Score, mean ± SD	22.9 ± 5.5	20.2 ± 5.8	20.9 ± 5.8	.422
No. (%) completing study	14 (93.3)	11 (78.5)	12 (80.0)	.511

Abbreviation: IdFAI, Identification of Functional Ankle Instability Instrument.

Table 2. Neural Excitability Means ± SDs Across Groups and Time Points and F Values for Main Effect of Time and Group-by-Time Interaction Effect for Each Variable

Variable	Group	Week 0	Week 2	Week 4	Week 6	Time Effect (*P* Value)	Interaction Effect (*P* Value)
Tibialis anterior							
$H_{max}:M_{max}$	Motor	0.227 ± 0.169	0.168 ± 0.082	0.199 ± 0.125	0.215 ± 0.152	1.39 (.249)	1.41 (.216)
	Frontal	0.170 ± 0.115	0.156 ± 0.078	0.305 ± 0.254	0.242 ± 0.263		
	Sham	0.157 ± 0.073	0.198 ± 0.147	0.170 ± 0.102	0.192 ± 0.134		
Resting motor threshold, %2T	Motor	33.5 ± 9.0	27.6 ± 12.2	33.8 ± 10.4	28.3 ± 8.4	2.22 (.091)	0.45 (.845)
	Frontal	32.3 ± 9.6	32.9 ± 10.1	34.4 ± 12.6	29.2 ± 13.4		
	Sham	35.7 ± 12.5	36.8 ± 12.6	39.9 ± 12.5	34.3 ± 15.9		
$MEP_{max}:M_{max}$	Motor	0.165 ± 0.140	0.202 ± 0.168	0.139 ± 0.064	0.162 ± 0.145	3.83 (.012)	1.23 (.298)
	Frontal	0.141 ± 0.116	0.117 ± 0.084	0.092 ± 0.064	0.165 ± 0.140		
	Sham	0.121 ± 0.090	0.144 ± 0.101	0.096 ± 0.058	0.124 ± 0.104		
Peroneus longus							
$H_{max}:M_{max}$	Motor	0.251 ± 0.165	0.258 ± 0.133	0.290 ± 0.141	0.259 ± 0.169	0.77 (.515)	0.95 (.466)
	Frontal	0.382 ± 0.230	0.326 ± 0.194	0.303 ± 0.169	0.269 ± 0.184		
	Sham	0.307 ± 0.147	0.296 ± 0.188	0.314 ± 0.195	0.286 ± 0.203		
Resting motor threshold, %2T	Motor	29.9 ± 11.6	28.4 ± 11.2	31.8 ± 10.8	30.8 ± 13.8	0.24 (.867)	0.28 (.947)
	Frontal	32.5 ± 11.4	34.2 ± 13.1	35.8 ± 13.8	34.7 ± 17.7		
	Sham	36.8 ± 9.8	35.4 ± 14.2	39.3 ± 10.2	38.3 ± 16.1		
$MEP_{max}:M_{max}$	Motor	0.078 ± 0.067	0.058 ± 0.032	0.078 ± 0.055	0.047 ± 0.031	0.48 (.699)	1.56 (.167)
	Frontal	0.065 ± 0.060	0.077 ± 0.076	0.042 ± 0.043	0.092 ± 0.092		
	Sham	0.071 ± 0.089	0.077 ± 0.051	0.050 ± 0.043	0.059 ± 0.052		
Soleus							
$H_{max}:M_{max}$	Motor	0.552 ± 0.185	0.505 ± 0.210	0.442 ± 0.176	0.427 ± 0.132	1.35 (.264)	1.13 (.351)
	Frontal	0.560 ± 0.213	0.542 ± 0.220	0.491 ± 0.191	0.558 ± 0.193		
	Sham	0.516 ± 0.212	0.473 ± 0.193	0.505 ± 0.215	0.507 ± 0.216		
Resting motor threshold, %2T	Motor	27.6 ± 11.0	29.4 ± 10.3	27.3 ± 17.1	31.2 ± 6.5	0.32 (.812)	0.51 (.802)
	Frontal	34.1 ± 12.1	35.4 ± 13.0	36.3 ± 15.0	33.2 ± 12.6		
	Sham	35.5 ± 12.1	37.7 ± 13.8	37.8 ± 11.4	34.6 ± 17.6		
$MEP_{max}:M_{max}$	Motor	0.026 ± 0.018	0.026 ± 0.019	0.023 ± 0.017	0.023 ± 0.020	0.69 (.562)	0.08 (.998)
	Frontal	0.017 ± 0.012	0.029 ± 0.029	0.023 ± 0.026	0.019 ± 0.016		
	Sham	0.027 ± 0.017	0.029 ± 0.023	0.022 ± 0.016	0.025 ± 0.016		

Abbreviations: $H_{max}:M_{max}$, ratio of maximum reflexive response to maximum motor response; $MEP_{max}:M_{max}$, ratio of maximum motor evoked potential to maximum motor response.

Side Hop Test

Significant improvements were observed in side-hop test performance over time ($F_{3,104.5} = 11.004$, $P < .001$, $R^2_M = 0.096$, $R^2_C = 0.674$), but no group or group-by-time interaction effect was observed (Table 5). Significant differences from baseline indicating improvement performance were noted at midtraining (MD: −3.2; 95% CI = −4.7, −1.6, $P < .001$), posttraining (MD: −3.9; 95% CI = −5.5, −2.3; $P < .001$), and retention (MD: −4.2; 95% CI = −5.9, −2.6; $P < .001$).

Patient-Reported Outcomes

Means, SDs, and omnibus test effects are presented in Table 6. The FAAM-ADL revealed no significant group or group-by-time interaction effects, but the time effect was significant ($F_{3,105.7} = 6.775$, $P < .001$, $R^2_M = 0.036$, $R^2_C = 0.846$). Parameter estimates were significant at posttraining (MD = 3.3; 95% CI = 1.3, 5.3; $P = .002$) and retention (MD = 4.36; 95% CI = 2.3, 6.4; $P < .001$) but not at midtraining (MD = 1.7; 95% CI = −0.3, 3.7; $P = .091$). The FAAM-Sport revealed no significant group or group-by-time interaction effects, but the time effect was significant ($F_{3,106.5} = 13.58$, $P < .001$, $R^2_M = 0.062$, $R^2_C = 0.830$). Parameter estimates were significant at midtraining (MD = 3.8; 95% CI = 0.7, 6.9; $P = .019$), posttraining (MD = 8.1; 95% CI = 4.9, 11.3; $P < .001$) and retention (MD = 9.35; 95% CI = 6.1, 12.6; $P < .001$). Both scales followed the same pattern of increased perceived function over time.

The mDPAS Physical subscale revealed no significant group or group-by-time interaction effect, but a significant effect of time was observed ($F_{3,106.9} = 8.049$, $P < .001$, $R^2_M = 0.045$, $R^2_C = 0.791$). Parameter estimates showed significant differences from baseline at posttraining (MD = −3.8; 95% CI = −4.8, −1.8; $P < .001$) and retention (MD = −4.7; 95% CI = −5.7, −2.6; $P < .001$), but not at midtraining (MD = −1.8; 95% CI = −3.8, 0.2; $P = .083$). The mDPAS Mental subscale also demonstrated no significant group or group-by-time interaction effect, but did demonstrate a significant effect of time ($F_{3,107.5} = 3.43$, $P = .020$, $R^2_M = 0.045$, $R^2_C = 0.791$). Parameter estimates showed significant differences at posttraining (MD = −0.8; 95% CI = −1.5, −0.1; $P = .034$) and retention (MD = −0.9; 95% CI = −1.6, −0.2; $P = .014$), but not at midtraining (MD = −0.0; 95% CI = −0.7, 0.6; $P = .919$). A similar pattern was observed for the TSK, as there was no significant group or group-by-time interaction effect, but a significant effect of time was observed ($F_{3,107.7} = 2.719$, $P = .048$, $R^2_M = 0.040$, $R^2_C = 0.772$). However, post hoc parameter estimates revealed no significant differences at any given time for the TSK.

The GROC followed a similar trend, demonstrating no significant group or group-by-time interaction effect, but a significant effect of time ($F_{2,69.6} = 20.046$, $P < .001$, $R^2_M = 0.129$,

Table 3. Means ± SDs for Balance Variables on Medial and Lateral Hops Across Groups and Time Points and *F* Values for Main Effect of Time and Group-by-Time Interaction Effect for Each Variable

Variable	Group	Week 0	Week 2	Week 4	Week 6	Time Effect (*P* Value)	Interaction Effect (*P* Value)
Medial DPSI	Motor	0.329 ± 0.072	0.342 ± 0.068	0.335 ± 0.065	0.340 ± 0.085	2.38 (.074)	0.66 (.68)
	Frontal	0.324 ± 0.069	0.276 ± 0.033	0.283 ± 0.044	0.326 ± 0.068		
	Sham	0.332 ± 0.113	0.281 ± 0.058	0.282 ± 0.063	0.313 ± 0.102		
Medial APSI	Motor	0.074 ± 0.009	0.072 ± 0.009	0.070 ± 0.009	0.070 ± 0.008	0.92 (.437)	0.63 (.703)
	Frontal	0.069 ± 0.009	0.072 ± 0.005	0.069 ± 0.006	0.069 ± 0.008		
	Sham	0.068 ± 0.008	0.067 ± 0.007	0.066 ± 0.007	0.066 ± 0.006		
Medial MLSI	Motor	0.037 ± 0.011	0.038 ± 0.008	0.036 ± 0.008	0.038 ± 0.007	0.64 (.591)	1.09 (.371)
	Frontal	0.036 ± 0.009	0.033 ± 0.007	0.033 ± 0.007	0.038 ± 0.008		
	Sham	0.035 ± 0.010	0.033 ± 0.008	0.032 ± 0.007	0.031 ± 0.007		
Medial VSI	Motor	0.317 ± 0.074	0.331 ± 0.070	0.324 ± 0.066	0.329 ± 0.088	2.38 (.074)	0.66 (.679)
	Frontal	0.314 ± 0.069	0.263 ± 0.034	0.272 ± 0.045	0.315 ± 0.069		
	Sham	0.321 ± 0.115	0.270 ± 0.059	0.264 ± 0.065	0.303 ± 0.104		
Lateral DPSI	Motor	0.363 ± 0.077	0.319 ± 0.046	0.333 ± 0.083	0.307 ± 0.054	4.39 (.006)[a]	1.00 (.431)
	Frontal	0.312 ± 0.059	0.275 ± 0.042	0.268 ± 0.045	0.291 ± 0.056		
	Sham	0.311 ± 0.096	0.270 ± 0.043	0.291 ± 0.068	0.288 ± 0.093		
Lateral APSI	Motor	0.066 ± 0.007	0.067 ± 0.006	0.067 ± 0.007	0.066 ± 0.006	2.26 (.086)	0.96 (.456)
	Frontal	0.068 ± 0.007	0.065 ± 0.004	0.065 ± 0.005	0.062 ± 0.004		
	Sham	0.066 ± 0.006	0.064 ± 0.008	0.062 ± 0.005	0.064 ± 0.006		
Lateral MLSI	Motor	0.037 ± 0.009	0.037 ± 0.005	0.038 ± 0.009	0.039 ± 0.006	0.71 (.550)	0.89 (.508)
	Frontal	0.036 ± 0.006	0.030 ± 0.006	0.032 ± 0.006	0.031 ± 0.004		
	Sham	0.034 ± 0.009	0.034 ± 0.007	0.032 ± 0.003	0.034 ± 0.008		
Lateral VSI	Motor	0.354 ± 0.079	0.309 ± 0.047	0.322 ± 0.085	0.297 ± 0.055	4.23 (.007)[a]	1.01 (.424)
	Frontal	0.301 ± 0.060	0.265 ± 0.042	0.258 ± 0.045	0.282 ± 0.057		
	Sham	0.301 ± 0.098	0.259 ± 0.043	0.282 ± 0.069	0.278 ± 0.095		

Abbreviations: APSI, anteroposterior stability index; DPSI, dynamic postural stability index; MLSI, mediolateral stability index; VSI, vertical stability index.
[a] Significant at .05 level.

$R^2_C = 0.747$). Parameter estimates showed significant differences at posttraining (MD = 1.3; 95% CI = 0.7, 1.8; $P < .001$) and retention (MD = 1.6; 95% CI = 1.1, 2.1; $P < .001$).

DISCUSSION

This study aimed to explore whether a tDCS over the motor or frontal cortex improved patient function when combined with rehabilitative exercises emphasizing motor planning. Although patients demonstrated improvements in patient-reported function and some performance measures over the course of the intervention, changes were not tied to the use of tDCS at either location. These data potentially highlight the effectiveness of motor-planning interventions, while suggesting caution in the implementation of tDCS to augment rehabilitation efforts in patients with musculoskeletal injury.

Functional Improvements After the Intervention

Across all groups, participants displayed improvements in patient-reported outcome measures, side-hop test performance, and dynamic balance during lateral hops. These improvements were observed after the 4-week intervention, regardless of tDCS application, suggesting improvements were tied to the exercises performed. The exercises in the rehabilitation intervention—consisting of stepping over obstacles, reactive semi-dynamic balance exercises, and agility—were selected to demand activation of motor-execution areas of the cortex while simultaneously challenging planning areas, which may be less efficient in individuals with CAI.[7] Implementing motor-learning strategies and dual-task focused interventions has recently grown in patients with musculoskeletal injury,[40,41] with improvements generally observed in balance performance.[42] Given deficits in motor planning in patients with CAI, it is possible that incorporating motor-planning–focused exercises into rehabilitation was sufficient to improve patient-reported function, supporting the implementation of these exercises in rehabilitation efforts for CAI.[40]

Although the observed improvements in patient-reported function were statistically significant, caution should be exercised when interpreting the clinical significance of these data. Many of the outcome measures did not exceed published minimum clinically important differences (MCIDs). Improvements did not exceed the MCID for the FAAM-ADL (MCID = 8),[43] TSK (MCID = 4),[44] and mDPAS (MCID = 9),[35] with only the FAAM-Sport (MCID = 9)[43] having confidence intervals that exceeded this threshold. For the GROC, indicating participants' perceived improvement from the intervention, scores observed were in line with the MCID of 2; however, a score of 5 has been recommended to gauge the success of an intervention.[45] Although we observed improvements from our intervention, it would be important to compare an intervention emphasizing motor planning with a more typical rehabilitation protocol that may emphasize balance and strength training.[46]

Participants were recruited from a general population rather than those specifically seeking care for CAI. As such, although similar across groups, these individuals may have had higher baseline function, limiting the magnitude of potential improvements. However, the observed increases in patient-reported function represent a potentially meaningful change. Clinically, this suggests that bringing patients who exhibit impairments with few participation restrictions through continued rehabilitation emphasizing motor planning and dual tasking may improve function. The responsiveness to the intervention seen across

Table 4. Means ± SDs for Electromyography Variables on Medial and Lateral Hops Across Groups and Time Points and *F* Values for Main Effect of Time and Group-by-Time Interaction Effect for Each Variable; All Units Are Percentages of Ensemble Peak

Variable	Group	Week 0	Week 2	Week 4	Week 6	Time Effect (*P* Value)	Interaction Effect (*P* Value)
Medial hops							
Tibialis anterior							
Prelanding	Motor	0.402 ± 0.117	0.367 ± 0.106	0.422 ± 0.121	0.337 ± 0.125	0.366 (.778)	1.980 (.074)
	Frontal	0.381 ± 0.126	0.418 ± 0.137	0.408 ± 0.132	0.405 ± 0.126		
	Sham	0.463 ± 0.139	0.386 ± 0.126	0.344 ± 0.114	0.411 ± 0.141		
Postlanding	Motor	0.481 ± 0.098	0.433 ± 0.069	0.530 ± 0.194	0.447 ± 0.156	1.574 (.200)	10.109 (.361)
	Frontal	0.473 ± 0.099	0.521 ± 0.075	0.548 ± 0.115	0.474 ± 0.104		
	Sham	0.528 ± 0.139	0.478 ± 0.075	0.474 ± 0.076	0.478 ± 0.142		
Peroneus longus							
Prelanding	Motor	0.510 ± 0.078	0.482 ± 0.103	0.575 ± 0.183	0.559 ± 0.134	1.051 (.373)	0.720 (.634)
	Frontal	0.510 ± 0.102	0.483 ± 0.095	0.533 ± 0.094	0.517 ± 0.171		
	Sham	0.554 ± 0.162	0.533 ± 0.107	0.509 ± 0.129	0.562 ± 0.140		
Postlanding	Motor	0.486 ± 0.120	0.450 ± 0.141	0.498 ± 0.236	0.540 ± 0.215	1.735 (.164)	0.562 (.760)
	Frontal	0.475 ± 0.069	0.423 ± 0.067	0.483 ± 0.093	0.459 ± 0.150		
	Sham	0.463 ± 0.119	0.423 ± 0.109	0.425 ± 0.086	0.473 ± 0.110		
Soleus							
Prelanding	Motor	0.373 ± 0.044	0.385 ± 0.104	0.419 ± 0.136	0.465 ± 0.166	1.895 (.135)	0.997 (.431)
	Frontal	0.382 ± 0.097	0.349 ± 0.072	0.369 ± 0.127	0.476 ± 0.209		
	Sham	0.415 ± 0.159	0.449 ± 0.174	0.383 ± 0.087	0.416 ± 0.150		
Postlanding	Motor	0.295 ± 0.074	0.296 ± 0.076	0.318 ± 0.146	0.376 ± 0.156	2.119 (.102)	0.800 (.572)
	Frontal	0.300 ± 0.083	0.270 ± 0.057	0.279 ± 0.093	0.356 ± 0.142		
	Sham	0.310 ± 0.125	0.299 ± 0.106	0.326 ± 0.088	0.303 ± 0.101		
Lateral hops							
Tibialis anterior							
Prelanding	Motor	0.398 ± 0.155	0.426 ± 0.114	0.411 ± 0.157	0.422 ± 0.125	1.185 (.319)	0.267 (.951)
	Frontal	0.443 ± 0.083	0.437 ± 0.155	0.411 ± 0.123	0.396 ± 0.114		
	Sham	0.461 ± 0.117	0.441 ± 0.076	0.375 ± 0.094	0.420 ± 0.132		
Postlanding	Motor	0.484 ± 0.164	0.544 ± 0.123	0.525 ± 0.155	0.500 ± 0.145	0.498 (.685)	0.410 (.871)
	Frontal	0.532 ± 0.116	0.521 ± 0.070	0.500 ± 0.082	0.526 ± 0.099		
	Sham	0.528 ± 0.103	0.521 ± 0.105	0.505 ± 0.074	0.488 ± 0.116		
Peroneus longus							
Prelanding	Motor	0.515 ± 0.128	0.477 ± 0.114	0.565 ± 0.222	0.523 ± 0.110	0.228 (.877)	1.263 (.280)
	Frontal	0.502 ± 0.098	0.562 ± 0.103	0.524 ± 0.103	0.507 ± 0.090		
	Sham	0.518 ± 0.116	0.461 ± 0.121	0.476 ± 0.092	0.494 ± 0.092		
Postlanding	Motor	0.429 ± 0.135	0.430 ± 0.163	0.481 ± 0.238	0.457 ± 0.164	0.591 (.622)	1.125 (.352)
	Frontal	0.455 ± 0.082	0.439 ± 0.089	0.399 ± 0.09	0.448 ± 0.141		
	Sham	0.455 ± 0.135	0.377 ± 0.115	0.437 ± 0.073	0.401 ± 0.116		
Soleus							
Prelanding	Motor	0.452 ± 0.140	0.363 ± 0.066	0.447 ± 0.173	0.382 ± 0.066	1.243 (.298)	1.854 (.095)
	Frontal	0.396 ± 0.104	0.399 ± 0.067	0.351 ± 0.071	0.465 ± 0.130		
	Sham	0.401 ± 0.138	0.398 ± 0.091	0.427 ± 0.119	0.461 ± 0.151		
Postlanding	Motor	0.339 ± 0.719	0.274 ± 0.437	0.309 ± 0.484	0.307 ± 0.536	2.400 (.072)	0.970 (.449)
	Frontal	0.304 ± 0.487	0.291 ± 0.447	0.257 ± 0.318	0.340 ± 0.509		
	Sham	0.337 ± 0.760	0.285 ± 0.435	0.330 ± 0.492	0.329 ± 0.510		

groups may be evidence of unfulfilled potential in traditional rehabilitation, where patients often resume activity and discontinue care for a variety of reasons before advanced, dynamic rehabilitation strategies can be implemented. However, the observed small effect sizes for these clinical improvements may be due to a lack of concurrent neural changes. That is, although individuals gained confidence in their ankle over the course of the intervention, the absence of neural changes (eg, improvements in neural excitability) potentially limited the magnitude of these effects and may reflect a lack of durability to these changes. Overall, these results suggest a degree of effectiveness to the intervention, but a failure to optimally restore patient function.

Efficacy of tDCS

Our a priori hypotheses were that the implementation of tDCS would enhance rehabilitation by driving mechanistic changes that would subsequently improve function. However, nearly all measures tied to mechanistic changes in individuals with CAI did not demonstrate improvements over the course of the intervention. Notably, improvements in segmental or corticospinal excitability—a consistent finding in tDCS-related research—were not observed in this study.[47] Similarly, no improvements were observed in muscle activation or reaction times during the reactive hop task.

These data are in contrast to previous studies implementing tDCS in individuals with musculoskeletal injuries.[15,16,48] In individuals with CAI, tDCS has been implemented with eccentric strengthening exercises,[16] foot intrinsic muscle strengthening,[15] and balance training,[48] yielding improvements in neural excitability, muscle activation, and dynamic balance outcomes as well as patient function when compared with a similar sham group.[15,16] Although no evidence exists for the use of DLPFC stimulation in individuals with musculoskeletal injury, improvements have been seen in motor performance of complex walking

Table 5. Means ± SDs for the Side Hop Test, Choice Reaction Time, and Cognitive Performance; *F* Values for the Main Effect of Time and the Group-by-Time Interaction Effect for Each Variable

Variable	Group	Week 0	Week 2	Week 4	Week 6	Time Effect (*P* Value)	Interaction Effect (*P* Value)
Side hop test, s	Motor	17.0 ± 8.4	15.1 ± 8.9	15.0 ± 9.8	15.1 ± 11.6	11.00 (<.001)[a]	0.49 (.812)
	Frontal	16.1 ± 4.8	12.6 ± 3.4	11.8 ± 3.9	11.5 ± 3.8		
	Sham	16.8 ± 10.5	12.2 ± 3.8	11.2 ± 2.3	10.8 ± 3.1		
Medial reaction time, s	Motor	1.95 ± 0.45	1.92 ± 0.54	1.98 ± 0.63	1.84 ± 0.50	0.46 (.714)	1.20 (.310)
	Frontal	2.00 ± 0.38	1.73 ± 0.37	1.88 ± 0.26	1.79 ± 0.24		
	Sham	1.83 ± 0.55	1.96 ± 0.62	1.81 ± 0.45	1.86 ± 0.35		
Lateral reaction time, s	Motor	1.73 ± 0.33	1.80 ± 0.66	1.91 ± 0.94	2.11 ± 0.90	0.56 (.645)	1.40 (.220)
	Frontal	1.68 ± 0.38	1.58 ± 0.31	1.72 ± 0.38	1.62 ± 0.21		
	Sham	1.72 ± 0.39	1.93 ± 0.68	1.82 ± 0.49	1.77 ± 0.43		
Colors correct, No.	Motor	2.3 ± 0.6	2.6 ± 0.3	2.7 ± 0.3	2.7 ± 0.4	9.82 (<.001)[a]	0.92 (.484)
	Frontal	2.5 ± 0.4	2.7 ± 0.3	2.7 ± 0.2	2.8 ± 0.2		
	Sham	2.6 ± 0.4	2.7 ± 0.2	2.7 ± 0.3	2.8 ± 0.3		

[a] Significant at .05 level.

in healthy individuals.[49] Alternatively, stimulation over the supplementary motor area, similarly used to improve motor planning, has not demonstrated improvements on motor-planning outcomes in individuals with CAI.[50] A key difference between the current investigation and these previous studies is the intervention paired with tDCS. Here, the intervention was selected to create both motor execution and planning demands, allowing for all participants to do the same exercise sets despite the intention of their tDCS application (motor execution versus planning). However, our results and prior investigations suggests that the exercise selection may be of utmost importance in the treatment of joint instability. Transcranial direct current stimulation over the M1 may need to be implemented with direct strengthening exercises that have disinhibitory effects (eg, eccentric exercise, plyometrics), whereas frontal cortex stimulation may require interventions that more intentionally create demand on the DLPFC, such as motor imagery or action-observation interventions.[7]

Although early evidence has supported the use of M1 stimulation in patients with CAI, the use of DLPFC stimulation has been less explored.[15,16] The aim of frontal cortex stimulation would be to improve neural efficiency in feed-forward motor planning by improving the brain's ability to anticipate outcomes; however, patients with CAI may have increased dependence on these frontal areas and facilitation here may not be warranted.[10]

Limitations

Certain limitations in study design limit our ability to draw finite conclusions based on our intervention. All participants received exercises for CAI, potentially generating biases to improve scores on patient-reported outcome measures, reflecting a potential Hawthorne effect.[51] A true control group receiving no intervention could have alleviated this concern. Similarly, physical and cognitive performance improvements, such as that on the side-hop test, VSI, or cognitive correct letters, may have been tied to learning effects that may explain some of the improvements we observed. Alternatively, as approximately 75% of participants across all groups felt they had received an active tDCS current, a placebo effect may have aided

Table 6. Means ± SDs for Patient-Reported Outcome Measures and *F* Values for the Main Effect of Time and the Group-by-Time Interaction Effect for Each Variable

Variable	Group	Week 0	Week 2	Week 4	Week 6	Time Effect (*P* Value)	Interaction Effect (*P* Value)
Global Rating of Change	Motor		1.36 ± 1.60	3.00 ± 1.96	3.36 ± 1.99	20.05 (<.001)[a]	0.759 (.555)
	Frontal		0.77 ± 1.96	2.33 ± 2.27	2.45 ± 2.62		
	Sham		1.15 ± 2.54	1.83 ± 2.25	2.42 ± 2.39		
FAAM: ADL subscale	Motor	86.9 ± 14.2	87.4 ± 12.0	90.7 ± 10.7	92.5 ± 9.7	6.77 (<.001)[a]	1.95 (.080)
	Frontal	90.7 ± 9.5	90.4 ± 12.9	90.3 ± 14.2	90.4 ± 14.0		
	Sham	85.6 ± 11.9	92.0 ± 6.5	93.5 ± 7.3	94.3 ± 6.9		
FAAM: Sport subscale	Motor	75.3 ± 18.6	77.8 ± 15.2	83.9 ± 13.4	82.4 ± 14.7	13.58 (<.001)[a]	0.50 (.808)
	Frontal	75.5 ± 20.8	77.8 ± 21.4	81.3 ± 18.9	81.8 ± 16.8		
	Sham	68.7 ± 17.4	76.5 ± 17.7	80.7 ± 12.2	83.0 ± 12.8		
mDPAS: Physical subscale	Motor	12.7 ± 12.1	12.1 ± 10.3	9.7 ± 9.7	8.5 ± 8.7	8.05 (<.001)[a]	1.03 (.409)
	Frontal	12.1 ± 12.3	11.0 ± 10.7	9.8 ± 11.3	11.1 ± 12.0		
	Sham	15.8 ± 6.7	10.8 ± 8.1	8.4 ± 7.5	6.6 ± 7.0		
mDPAS: Mental subscale	Motor	3.3 ± 3.1	3.5 ± 5.0	2.5 ± 3.4	1.9 ± 2.8	3.43 (.020)	0.73 (.624)
	Frontal	3.0 ± 3.7	2.6 ± 4.1	2.0 ± 2.9	1.8 ± 3.0		
	Sham	1.9 ± 2.4	1.8 ± 2.5	1.3 ± 2.6	1.6 ± 2.9		
Tampa Scale of Kinesiophobia	Motor	35.5 ± 6.1	35.9 ± 7.8	35.2 ± 7.4	34.6 ± 7.4	2.72 (.048)[a]	0.24 (.96)
	Frontal	32.6 ± 3.9	33.9 ± 5.1	33.0 ± 7.1	32.3 ± 5.9		
	Sham	34.1 ± 6.1	36.0 ± 9.4	33.8 ± 6.4	33.1 ± 6.3		

Abbreviations: ADL, Activities of Daily Living; FAAM, Foot and Ankle Ability Measure; mDPAS, modified Disablement in the Physically Active Scale.
[a] Significant at .05 level.

outcome scores. Inclusion of longer follow-up might have provided a better indication of whether the improvements in patient-reported outcomes were durable beyond 2 weeks after cessation of training. Although efforts were made to control for as many variables at baseline, there is of course a great deal of variability among the deficits observed in patients with CAI that could have been mitigated by controlling for factors such as sex and baseline function before randomization; however, doing so often limits the clinical applicability of the data observed.

Clinical and Research Implications

Recent findings related to the use of brain stimulation in treating musculoskeletal injuries reflect the need for neuromodulatory interventions in the treatment of joint instability. Although it is unclear whether the increase in brain stimulation research in sports medicine has yielded its increased use in clinical practice, the results of this study provide some pause toward tDCS implementation. Placing this investigation in the context of the existing evidence, it appears that the impacts of tDCS are optimized when motor cortex stimulation is combined with direct motor interventions such as eccentric strengthening or foot intrinsic strengthening.[7,15] Conversely, the evidence does not support the use of tDCS over motor-planning areas in these patients.

Our rehabilitation intervention, emphasizing motor planning and dual-tasking components, did yield improvements in function. This emphasizes the need to incorporate these components into rehab, especially in continuing rehabilitation for individuals with few participation restrictions. These findings can be further enhanced by considering individualized changes to mechanical, sensorimotor, and psychological function, reflecting the varied clinical presentation of CAI.[3]

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SUPPLEMENTAL MATERIAL

Supplemental Material. Rehabilitation protocol.
Supplemental Table. Completion statistics for the training protocol, including the total number of training sessions completed by participants within each group, the time between training sessions, and the highest progression reached on each exercise.

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