COMBINED EFFECTS OF TRANSCRANIAL DIRECT-CURRENT STIMULATION AND STATIC STRETCHING ON HIP RANGE OF MOTION

A Thesis
by
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Abstract

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Static stretching is commonly used to increase joint range of motion (ROM). These changes are due to increases in the participant's stretch tolerance. Recent studies have shown that cathodal transcranial direct-current stimulation (c-tDCS) can decrease the excitability of the somatosensory cortex (S1) and improve joint ROM due to changes in pain perception. The aim of this investigation was to determine if c-tDCS of S1 combined with static stretching is more effective at improving and maintaining flexibility than stretching alone.

Twenty-eight healthy adult participants (age 22.0 ± 3.0 years) were assigned to receive active c-tDCS or a sham c-tDCS. In both groups, the cathode was placed over S1 of the dominant leg and the anode (reference electrode) over the ipsilateral eyebrow. The active group received a current of 2.0 mA for 20 minutes. In the sham group the current was turned off after 60 seconds. Following c-tDCS, all participants underwent a passive hamstring stretch consisting of three 30-second holds at the maximum angle tolerated. Hip flexion range of motion (HFROM), passive torque, and pain perception were assessed pre- and post-

stimulation and at 0, 5, 15, 30, and 60 minutes after stretching. An a priori significance level of $p < 0.05$ was adopted for the investigation. Outcome variables were compared using a 2x7 mixed-model ANOVA.

In the active group, HFROM and passive torque significantly increased after stretching (9.51° and 8.8 Nm, respectively) and remained elevated for 60 minutes (9.0° and 10.3 Nm, respectively). Improvements to HFROM and passive torque were significantly greater in the active group than in the sham group. Pain perception did not vary by group or by time.

The use of c-tDCS to S1 before stretching appears to be more effective at improving and maintaining flexibility for at least 60 minutes than stretching alone. Because participants tolerated a higher degree of stretch without an increase in pain perception, the improvements were likely due to a change in stretch tolerance. Though c-tDCS enhances the effects of stretching, more research is needed to determine if these improvements correlate with functional outcomes in clinical settings.

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First, I would like to offer my sincerest gratitude to Dr. Travis Triplett, Dr. Alan Needle, and Dr. Jared Skinner. This would not have been possible without your willingness to take me on as a student researcher, your constant encouragement, and invaluable and timely feedback and suggestions throughout this process. I truly appreciate your patience and mentorship in all of this.

I would also like to thank Dr. Andrew Shanely for your support and advocacy during this endeavor. Some of us need to occasionally be reminded why we are here and that there is a time to put on our buttons.

And thank you Dr. Marco Meucci, for coming through for me in two tight spots.

Thank you lastly Dr. Kym Fasczewski, for some early encouragement and advice and for having an open office.

Dedication

I would like to dedicate this to my grandparents, Carolyn and Bill Schwartz. I cannot imagine beginning this project, much less finishing it, without their perpetual love and support. Thank you for absolutely everything.

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Chapter 1: Introduction

Adequate joint range of motion (ROM) of the lower body is necessary for activities of daily living such as squatting, kneeling, and sitting cross-legged.[1] An even greater joint ROM is required for athletic activities such as gymnastics, ballet, and martial arts.[2] At the extremes, gymnasts require sufficient spine, hip, and shoulder ROM to perform back-bends during practice and competition,[3] and ballet dancers are expected to perform 180° splits in the air and ideally to have perfect turnout (external hip rotation).[2] Some evidence suggests that pre-activity stretching may reduce the risk of musculotendinous injuries,[4,5] though there is a paucity of research on the effects of a single acute bout of stretching on injury risk. Joint ROM may be limited due to injury to the joint or muscle tendon unit or prolonged immobilization.[6] And it is accepted that joint ROM declines with age,[7–9] though it can be maintained and even improved with training at any age[10]

Several stretching methods, including passive static and proprioceptive neuromuscular facilitation (PNF) have been shown to be effective at improving joint ROM[11,12] Passive static stretching is the most well-studied of these.[13] A common passive static stretching intervention involves rotating a joint to extend the muscle to a new length and maintaining this position for 10-60 seconds[12]. The mechanism by which stretching improves joint ROM is not well understood.[14] Mechanical, neural, and sensory explanations have been proposed, though growing evidence suggests that changes to joint ROM following static stretching are due primarily to a change in stretch tolerance rather than changes in muscle length or activation, and that greater applied torque is associated with greater maximum joint ROM.[15] Recent studies have shown that cathodal transcranial direct-current

stimulation (c-tDCS), a non-invasive neuromodulation treatment, can also acutely improve joint ROM. By inhibiting areas of the cerebral cortex associated with pain, c-tDCS has been shown to reduce pain perception in clinical and healthy populations.[16] This reduction in pain perception is believed to be the reason c-tDCS improves stretch tolerance.[17–20]

Given that static stretching improves joint ROM by increasing stretch tolerance, and c-tDCS can induce a centrally oriented reduction in pain perception, it follows that combing the two treatments should yield greater improvements to joint ROM and passive torque. This study aimed to investigate whether 20 minutes of c-tDCS applied to the somatosensory cortex (S1) combined with an acute bout of static stretching yields greater improvements in hip flexion range of motion (HFROM), passive torque at maximum angle, and pain perception than stretching alone. It was hypothesized that HFROM and passive torque would increase more following the combined treatment and that pain perception would remain unchanged despite these improvements. Further, it was expected that improvements would be retained longer following the combined treatment than following stretching alone.

Chapter 2: Literature Review

Passive Static Stretching

One of the most common and effective ways to improve a joint's range of motion is static stretching, which involves rotating a joint to the end of its range of motion and maintaining this position for a period, typically between 10 and 60 seconds.[12,13] An acute bout of static stretching can significantly increase joint ROM for as long as 24 hours,[21] though more typical retention times for improvements to ROM are 20 or 30 minutes.[22,23]

The magnitude of improvement following static stretching depends on the intensity[24] and duration of the stretch.[25,26] The intensity of a static stretch can be described using the point of discomfort,[27] point of pain,[28] or maximum tolerable stretch without pain[21] of each subject as an endpoint. For instance, performing a static stretch at the maximum joint angle without pain is a typical stretch intensity, and maintaining this position for 180 seconds has been shown to be sufficient to significantly increase knee extension ROM.[25] Another study found significant increases in knee extension ROM after five sets of 30-second static stretching to a point "without pain or discomfort."[26] High-intensity (maximum point of discomfort) static stretching has been shown to increase knee extension ROM in as few as 10, 15, and 20 seconds.[27] Takeuchi and colleagues compared the effects of jack-knife stretching at two different intensities for three sets of 20 seconds and found that stretching to the point of discomfort and stretching to the maximum tolerated angle both significantly increased joint ROM and passive torque.[29]

Several studies have investigated the time course of changes to joint ROM after static stretching.[22,23,30] When passively stretched to the point of discomfort for five one-minute

holds, ankle ROM and passive torque remain elevated for at least 30 minutes.[30] Hatano and colleagues investigated hamstring stretching, and found that after a single stretch of 300 seconds at maximum tolerable intensity, ROM and passive torque remained elevated for at least 20 minutes.[22] Another study looked at hip ROM after three 30-second passive hamstring stretches at the maximum angle without pain and found that though improvements in ROM began to decline after 15 minutes, ROM remained significantly higher than baseline for 24 hours.[21]

How Stretching Improves Joint ROM

It is accepted that stretching can increase ROM,[31] but the mechanism by which stretching increases muscle extensibility is still debated.[14] Proposed explanations for the acute changes in ROM following a bout of stretching include mechanical changes, in which there is a decrease in the muscle's resistance to stretch, due to changes in mechanical properties or architecture[14,32] and neuromuscular relaxation, in which the stretch reflex is altered.[33] Another explanation for the increases in muscle length after stretching is sensory theory, which posits that subjective tolerance to passive stretch is increased after intervention, but without a change in tension at a given muscle length.[14,15]

Several studies support a mechanical theory for explaining the increase in ROM seen following stretching.[14,34,35] One explanation for the increase in muscle extensibility after stretching is viscoelastic deformation.[21] Skeletal muscle behaves as a viscoelastic material.[36] This means that muscle, like a liquid, exhibits resistance to flow (viscosity) and the ability to deform under stress and return to its original shape once the stress is removed (elasticity).[37] It has been demonstrated that when a muscle is held in an elongated position, its resistance to stretch decreases with time under tension. The decrease in resistance to stretch is called

"viscoelastic stress relaxation."[36] Given a constant load the muscle will deform and lengthen in a time-dependent manner. This deformation is referred to as "creep."[38]

In a widely cited study on animals,[39–41] Taylor and colleagues showed an increase in rabbit hindlimb muscle length immediately after repeated cyclic and static stretches, indicating creep.[42] However, this increase in length has been shown to be transient. A study examining human hamstrings found that, following 3 sets of 45-second hamstring stretches, despite an immediate decrease in passive torque of 18-20%, indicating a change in the mechanical properties of the muscles, by the end of the 30-second rest period, this had returned to baseline.[43]

Some literature suggests that a neuromuscular stretch reflex may limit muscle extensibility during static stretching.[33,44] Following this, it has been proposed that a slowly applied stretch may allow for relaxation of muscles under stretch,[32,33] and that long term increases in extensibility are due to adaptation of the neuromuscular stretch reflexes[33] However, there is no evidence to support this explanation.[36,45,46] Stretch reflexes occur during rapid muscle lengthening and produce an immediate, short-lived contraction,[45]. Studies using a slow, passive stretch found no significant stretch reflex activation,[36,45,47,48] and even studies using high velocity stretching showed no significant activation of stretch reflexes.[49] Furthermore, it has been shown that electromyography (EMG) activity is not increased significantly above baseline in the passively lengthening muscle.[50–52] Therefore, increases in muscle extensibility cannot be attributed to neuromuscular relaxation.[13,14,36]

According to sensory theory, the increases in ROM following stretching are ultimately due to increased stretch tolerance,[14,52,53] and the maximum angle achieved during stretching depends on the amount of torque applied to the joint.[13,15] The amount of torque

required to move a joint to a certain angle is determined by the passive torque, or involuntary resistance to stretch, which depends on the joint and its surrounding structures, such as the joint capsule, muscles, and tendons. That sensation may be the limiting factor in flexibility was first described in a study by Magnusson and colleagues. They examined knee extension ROM following a static stretch of 90 seconds and found that neither stiffness nor EMG changed despite an increase in both passive torque at maximum angle and ROM.[52] In a similar study examining straight-leg raise ROM after 10 minutes of stretching, ROM and passive torque significantly increased, while stiffness remained the same.[54] In both studies, the endpoint for the ROM test was determined by the subject's sensation. It was therefore concluded that the subject's stretch tolerance was the primary limit to ROM, rather than mechanical (viscoelastic) properties.[52,54] More recent studies found similar results. Following five static stretches of one minute each with the ankle in maximum dorsiflexion, ROM, passive torque, and stiffness were significantly increased. However, the stiffness returned to baseline within 15 minutes, while the increases in ROM and passive torque persisted for 30 minutes. It was concluded that at 15 minutes increases in ROM could be attributed to both stiffness and stretch tolerance, but at 30 minutes the increase in ROM was due solely to increased stretch tolerance.[55] These studies suggest that the ROM achieved after static stretching is dependent on the applied torque, and that only the stretch tolerance of the participants changed.

The mechanism for how stretching increases stretch tolerance remains unclear.[15,56] Proposed explanations include alterations to nociceptive nerve endings,[52] mechanoreceptors, or proprioceptors.[57] Another potential mechanism is that afferent input from muscles and joints during stretch may interfere with signals from nociceptive fibers relaying stretch

discomfort, thus reducing the perception of pain.[56] Finally, there could be psychological changes to perception of stretch discomfort. Because participants cannot be blinded to the stretching procedure, they may have the preconceived expectation that they will become more flexible after stretching, and therefore tolerate more discomfort.[21,58]

Transcranial Direct-Current Stimulation

Transcranial direct-current stimulation (tDCS) is a safe, non-invasive, neuromodulation technique that runs a weak direct current through the cerebral cortex that flows from a positively charged anode to a negatively charged cathode.[59,60] Research shows that tDCS can offer clinical benefits for conditions such as major depression,[61] improving working memory in stroke[62] and Parkinson's disease patients,[63] and improving visual memory in Alzheimer's patients.[64] Studies show that tDCS can also improve chronic pain associated with fibromyalgia[16] and traumatic spinal cord injury[65] and can reduce pain perception in of experimentally induced pain.[66] Though currently not FDA-approved, tDCS devices are widely available to consumers, who primarily use it for cognitive enhancement or self-treatment.[67]

The primary mechanism of action appears to be a subthreshold modulation of neuronal membrane potentials,[68] which changes the cortical excitability of target neurons.[59,69] This modulation occurs due to the local extracellular electric field that is generated in the neuronal membranes when direct current is applied through the electrodes attached to the scalp. The increase or decrease in cortical excitability due to tDCS is polarity dependent. Anodal tDCS tends to have an excitatory (depolarizing) effect on the neuronal membrane of a given population of neurons, and cathodal tDCS tends to have an inhibitory (hyperpolarizing) effect.[59,70,71] Further, it has been shown that the changes in cortical

excitability induced by a single treatment of tDCS can last for at least 90 minutes following cessation of stimulation.[72]

Transcranial Direct-Current Stimulation, Pain Perception, and ROM

Imaging studies have shown that the primary somatosensory cortex (S1) is involved in pain perception.[73] Though the effects of tDCS on pain are considered to be polarity-dependent, the effects of polarity and the brain regions involved in relation to pain perception are still unclear. Both anodal and cathodal tDCS over S1 have been shown to decrease pain perception. In one study, cathodal tDCS applied over S1 decreased laser-stimulated pain perception.[73] A review by Vaseghi and colleagues concluded that anodal tDCS over either the primary motor cortex (M1) or S1 increased pain thresholds.[74] In another study, cathodal tDCS applied over S1, M1, or the dorsolateral prefrontal cortex all increased pain thresholds in healthy adults.[66] These findings suggest that modulation of S1 or M1 by c-tDCS is likely to affect pain perception.[73,74]

Recent studies have examined the effects of c-tDCS on passive joint ROM in healthy adult males[17–19] and females.[20](Table 1) The first study examined ankle dorsiflexion. Cathodal and anodal tDCS were applied over the sensorimotor region corresponding to the foot (Cz) for 10 minutes at a current of 2.0 mA. Ankle ROM increased significantly following the cathodal but not the anodal or sham conditions. The submaximal passive torque, a measure of joint mechanical properties, showed no changes in the anodal, cathodal, or sham conditions. Further, because participants stopped the measure at the same level of discomfort both before and after tDCS, despite the increase in joint angle, it was concluded that the improvement in ROM was likely due to inhibition of pain perception rather than changes in the mechanical properties of the muscle.[19] Three studies have investigated the

effects of 20 minutes of tDCS with a current of 2.0 mA on HFROM. In one study, c-tDCS was applied over Cz,[18] and in the others c-tDCS was applied over M1.[17,20] All found significant increases in hip ROM and decreased pain compared to sham conditions.[17,18,20] These findings support previous studies in which c-tDCS over M1 or Cz were able to modulate pain perception,[66,73] and those in which pain perception was determined to be a limiting factor for joint ROM.[30,55,75]

Table 1. Summarized results of previous studies on transcranial direct-current stimulation (tDCS) and joint range of motion (ROM).

Study	ROM	tDCS (2.0mA)	Time (min)	Effects	Notes
Mizuno & Aramaki, 2017	AD WD	c/Cz, a/forehead a/Cz, c/forehead sham: c/Cz, a/forehead	10	ROM increased ROM unchanged ROM unchanged ROM unchanged	males only
Lins, et al, 2019	PSLR	c/Cz, a/Oz a/Cz, c/Oz sham: a/Cz, c/forehead	20	ROM increased, pain decreased ROM decreased, pain unchanged ROM unchanged, pain unchanged	males only
Henriques, et al, 2019	PSLR	c/M1, a/DLPFC a/M1, c/ DLPFC sham: c/M1, a/ DLPFC	20	ROM increased, pain decreased ROM decreased, pain unchanged ROM unchanged, pain unchanged	males only
Rodrigues, et al, 2022	PSLR	c/M1, a/OC	20	ROM increased	females only

Abbreviations: AD, ankle dorsiflexion; WD, wrist dorsiflexion; PSLR, passive straight leg raise; c, cathodal; a, anodal; Cz, vertex; Oz, occipital, central; M1, primary motor cortex; DLPFC, dorsolateral prefrontal cortex

Current Study

Previous studies have shown that c-tDCS combined with an active warm-up can improve HFROM acutely,[17,18,20]. However, none have investigated the duration of these improvements. Furthermore, tDCS is most often used as an adjunctive to another form of treatment such as complex walking,[76] the stand-and-walk test,[77] a serial reaction time task,[78] or resistance training.[79] Therefore, it seems likely that combining tDCS with a task such as static stretching would yield greater improvements to HFROM, and that these improvements would be retained longer than without tDCS. To the author's knowledge, this is the first study to investigate the effects of cathodal tDCS combined with an acute bout of static

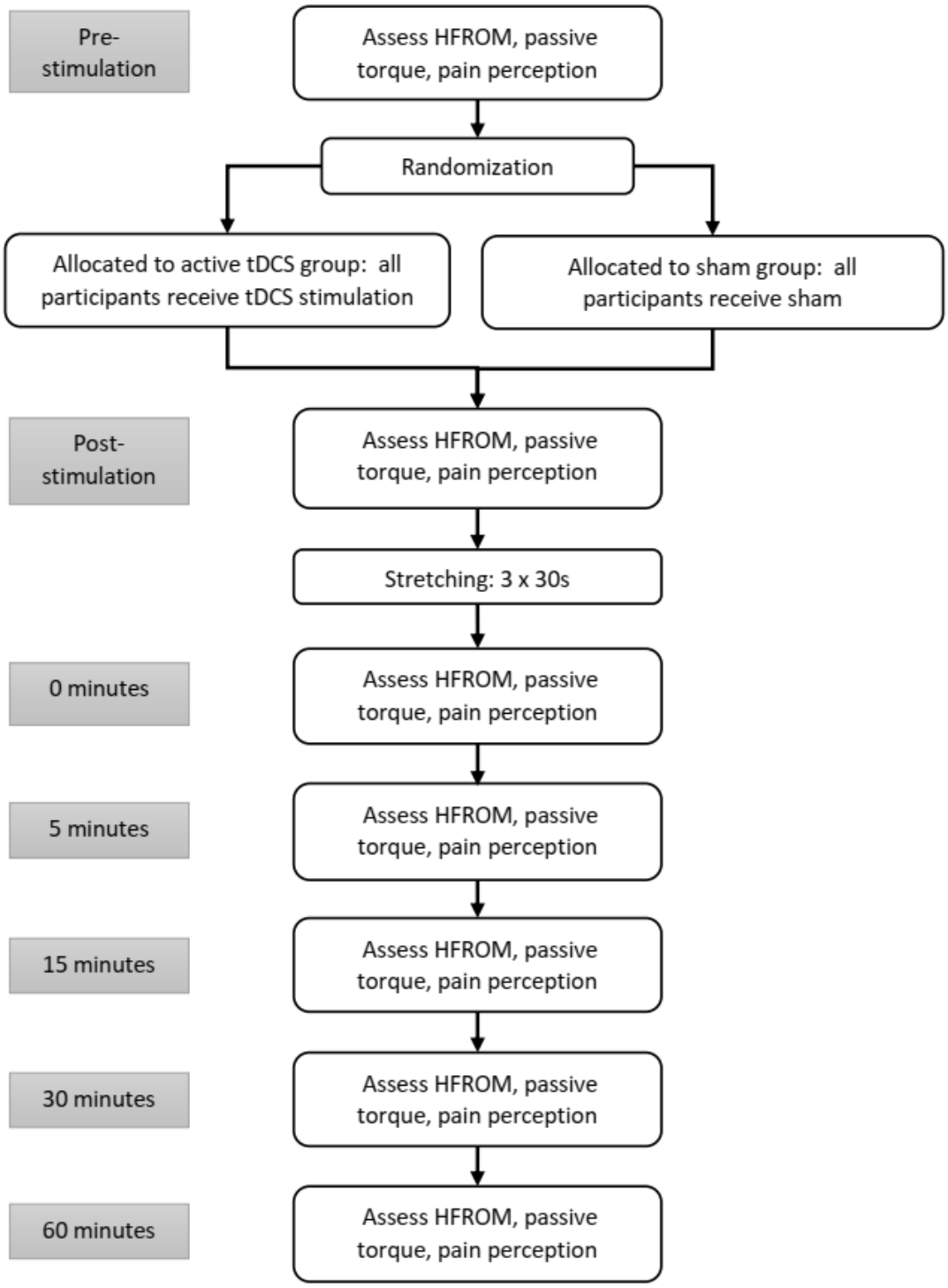
stretching. It has been shown that an acute bout of static stretching can improve joint ROM for up to 60 minutes[21]. It has also been shown that tDCS can improve joint ROM.[17–20] Therefore, the purpose of this study is to determine cathodal stimulation to S1 before an acute static stretching intervention can increase the effectiveness and retention of stretching. It is hypothesized that cathodal stimulation of S1 followed by an acute bout of static stretching will yield greater increases in hip ROM, greater increase in passive torque, and no change in pain perception compared to stretching alone, and that these improvements will persist for at least 60 minutes.

Chapter 3: Methods

Study Design

The experiment was conducted as a single-session, double-blind, randomized, sham-controlled trial. The independent variables were group assignment (active vs. sham) and time (pre-stimulation, post-stimulation, 0 minutes, 5 minutes, 15 minutes, 30 minutes, and 60 minutes). The dependent variables were HFROM, passive torque, and pain perception. Figure 1 shows the experimental design of the study. All testing was performed in the Neuromuscular Lab at Appalachian State University.

Figure 1: Experimental Design



Participants

Twenty-eight healthy men and women aged 18-31 were recruited (12 males, 16 females, age = 22.0 ± 3.0 years, height = 173.5 ± 9.0 cm, body mass = 76.2 ± 21.1 kg). No participants were engaged in a specific flexibility training program at the time of this study. Participants had no history or current symptoms of neurological or psychiatric disorders. Participants were excluded if they had a lower body or back musculoskeletal injury in the past six months. They also had no history or current symptoms of neurological or psychiatric disorders. Participants with contraindications to tDCS (e.g.: history of stroke or seizure, pacemaker, or intracranial metal implantation) were excluded.[68] Participants were informed of all risks and provided written informed consent at the time of enrollment, and the Appalachian State University Institutional Review Board approved the experiment. A previous study that examined the effects of c-tDCS on HFROM was used to estimate the sample size.[17]

Procedures

Participants completed the tDCS screening questionnaire, signed the informed consent form, and had anthropometric measurements taken. Maximum passive hip range of motion and passive torque of each participant were measured, and pain perception at the maximum joint angle were assessed using the Borg CR-10 scale.[80] Each participant was then randomly assigned to either the active or sham group. A member of the research team who was blinded to participants and sessions provided a randomized code to the principal investigator. Thus, neither participants nor the investigator were aware of group assignments. Participants underwent tDCS or sham protocol for 20 minutes. During treatment time and in the periods between measurements, participants lay supine on the table

in the lab. Immediately following treatment time, HFROM, passive torque, and pain perception were assessed. The lead researcher then led the participants through the static stretching protocol—described below.[21] Immediately after stretching and 5, 15, 30, and 60 minutes after, HFROM, passive torque, and pain perception were again evaluated. At the end of all procedures, participants were asked if they believed they received the active or sham tDCS.

Passive Hip Flexion Range of Motion

Maximum HFROM was assessed using a passive straight leg raise until the point of maximum tolerated stretch.[29] Participants lay supine on a padded table in the lab with the non-dominant leg flat on the table while the researcher passively stretched the dominant leg (preferred ball-kicking leg of subject).[29] Participants were fitted with a knee immobilizer on the dominant leg to prevent knee flexion during the measurement. A digital inclinometer (Johnson, model 1886-0000, USA), was attached to the knee immobilizer. The researcher slowly (approx. 5°/sec) moved the participants' leg through hip flexion to the farthest point the participants tolerated by applying force perpendicular to the leg. A previous study demonstrated that there is no significant reflex activity at this velocity.[14] After the subject indicated the maximum stretch they can tolerate [29] or posterior rotation of the pelvis was observed,[81] the researcher lowered the leg to the resting position. The researcher measured the maximum joint angle achieved with the digital inclinometer and the force applied with a handheld dynamometer (Baseline, model 12-0342, USA). During HFROM assessment, participants were asked to close their eyes to prevent visual input from affecting the results.[49] HFROM was defined as the angle α with the active leg's resting position at 0° represents the active leg being parallel to the table (no flexion or extension) and 90° represents the active

leg being perpendicular to the table (Figure 2). Intra-rater reliability of this measure was determined using intraclass correlation coefficients (ICC 3,1) of 0.951.

Hip Passive Torque

Passive torque of the hip at the maximum angle tolerated by the participant was assessed similarly to a previous study.[82] The force required to hold the leg at HFROM was assessed using the handheld digital dynamometer, which was attached distally to the knee immobilizer. Force was applied by the researcher perpendicularly to the leg. Figure 2 shows the experimental setup, and Figure 3 shows the biomechanical model and acting forces. The applied pushing force F_a consists of a weight component F_w, due to the force of gravity acting on the leg, and an elastic component F_e, due to the passive resistance of the hamstrings. Therefore:

$$F_a = F_w + F_e \quad (1)$$

The passive torque T_e was calculated by subtracting the torque caused by the weight of the leg, T_w from the applied torque T_a according to:

$$T_e = T_a - T_w \quad (2)$$

The equation for passive torque T_e at any angle α is:

$$T_e = (F_a - F_0 \cdot \cos\alpha)HA \quad (3)$$

F_0 was the force applied to the dynamometer at angle α = 0°. The moment arm, *HA*, was the distance between the palpated distal edge of the greater trochanter (fulcrum) and the point of applied force on the dynamometer. Intra-rater reliability of this measure was determined using intraclass correlation coefficients (ICC 3,1) of 0.821.

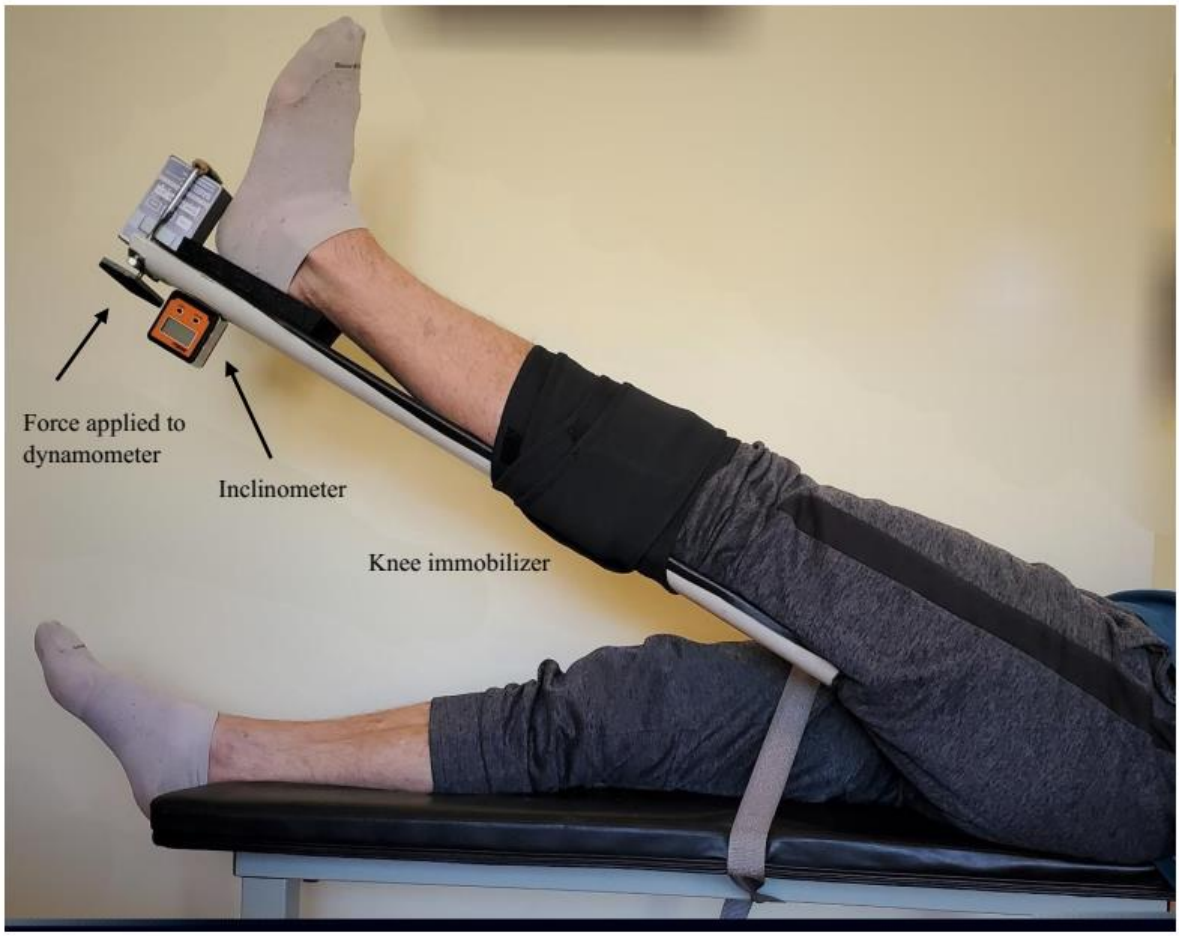


Figure 2. Experimental setup for HFROM and passive torque measures.

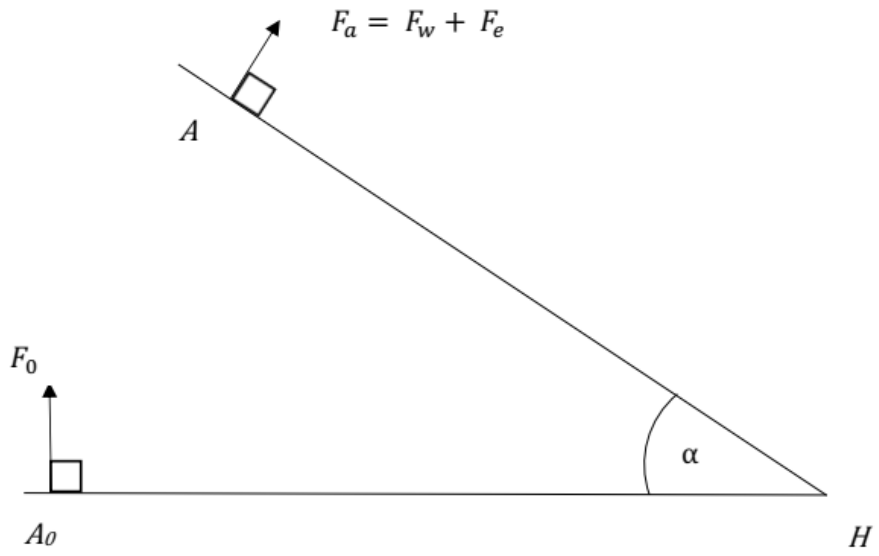


Figure 3. Biomechanical model with acting forces. F_0, force due to gravity of leg at α=0°; F_a = total force applied at ankle; F_w, weight of leg due to force of gravity acting on leg; F_e, elastic component; HA, moment arm.

Pain Perception

Pain perception was assessed immediately following each measure of HFROM and passive torque using a verbal Borg CR-10 scale. Participants were asked to rate the level of pain that occurred at the maximum angle achieved. The scale was anchored by "no pain" (score of zero) and "worst pain experienced" or "worst imaginable pain" (score of 10).

Transcranial Direct-Current Stimulation (tDCS) Intervention

Participants lay on the table in the laboratory for the application of the electrodes for the tDCS procedure. Each subject's head was measured according to the International 10-20 system to locate S1 for the lower body (Cp). Electrodes were applied using two 5 cm x 7 cm (35 cm^2) sponges (EasyPad, Soterix Medical Systems, New York, NY), each soaked in 8 mL saline solution (NaCl 0.9% dissolved in water). The electrodes were connected to the tDCS unit (1x1 tDCS-CT, Soterix Medical Systems, New York, NY) and held in position by specialized headgear. The cathode was positioned over S1 corresponding to the active limb, and the anode (reference electrode) was placed over the ipsilateral eyebrow[73]. The total time

of tDCS stimulation was 20 minutes at 2.0 mA of current, corresponding to the parameters used in previous studies.[17,18,20] The sham group received 30 seconds of ramping up in the beginning, 30 seconds of active current, then 30 seconds of ramping down.[17] This short duration stimulation protocol has been shown to be an effective sham procedure as participants become desensitized to the sensation after about 60 seconds of stimulation.[83] During the 20 minutes of stimulation (or sham) and in the periods between measurements, the participant lay on the padded table in the laboratory. Pilot studies have shown that this position is comfortable for the participant and maintains the hamstrings in a neutral position (zero degrees of flexion or extension).

Transcranial Direct-Current Stimulation Safety

When used in accordance with established protocols and common exclusion criteria (e.g.: no intracranial metal implants or pacemakers) adverse effects due to tDCS are rare and generally mild.[68,84] Adverse effects reported include itching, tingling, discomfort, and burning sensation of the skin beneath the electrodes, which dissipated after stimulation.[85] These effects may result from faults in the protocol, such as drying of the contact media between the scalp and the electrode or using tap water rather than saline solution as the contact medium.[68] Headache (11.8%), nausea (2.9%), and insomnia (0.98%) have also been reported after tDCS.[86] A recent review found no instances of serious adverse effects or irreversible injury in over 33,200 sessions and 1000 participants.[84]

Static Stretching Protocol

The stretching intervention was identical for the active tDCS and sham groups. No warm-up exercises were performed, to avoid possible interactions between the warm-up and treatments.[87,88] During the static stretching exercise, the researcher passively stretched the dominant leg to the maximum angle tolerated by the participant. The participant was positioned supine on a padded table as for the initial HFROM assessment. The researcher slowly (approx. 5°/sec)[49] moved the participant's leg through hip flexion to the maximum angle the subject can tolerate, even with pain[29] by applying a force perpendicular to the leg. After the participant indicated the maximum angle they can tolerate, the researcher maintained the position of the leg for 30 seconds beginning when the participant indicated their limit. Increasing force was applied as the participant allowed, so that there was a constant maximal stretch on the hamstrings. After 30 seconds, the leg was slowly lowered to the resting position, where it remained for 10 seconds. This stretch and relax pattern was repeated two more times, for a total of 3 bouts of 30 seconds each.[21] A stopwatch was used to time stretches and rest periods. Participants were vocally encouraged to relax, breathe, and not to contract the leg muscles during the stretching. All stretching was performed by the same researcher.

Statistical Analysis

All variables are reported as mean ± SD. A 2x7 mixed-model ANOVA was used to examine the effects of the group (active tDCS vs. sham) and time (Pre-stimulation vs. post-stimulation vs. post-stretching vs. 5 minutes vs. 15 minutes vs. 30 minutes vs. 60 minutes) on HFROM, passive torque, and pain perception. Post hoc analyses using Fisher's LSD were

performed to maintain an alpha level of $p < 0.05$ for all pairwise comparisons. Statistical analyses were performed using SPSS version 28 (SPSS, Inc., Chicago, IL, USA).

Chapter 4: Results

Twenty-eight participants were randomly assigned to either the active c-tDCS group (n = 14) or the sham group (n = 14). There were no significant differences between groups for age, height, or mass (Table 2).

Table 2. Anthropometrics of participants (means ± SD).

Group	Male:Female Ratio	Age (y)	Height (cm)	Mass (kg)
Active tDCS	7:7	22.1±3.3	176.2±8.9	82.3±25.7
Sham tDCS	5:9	21.9±2.8	170.9±8.9	70.2±14.9

No significant differences were found across groups for age (p=0.855), height (p=0.125), or mass (p=0.142).

Hip Flexion Range of Motion

Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(20) = 43.800$, $p = 0.002$), therefore the Huynh-Feldt correction was used ($\varepsilon = 0.782$). There was a significant two-way interaction effect (group x time, $F = 2.469$, $p = 0.039$, $\eta^2 = 0.087$). There was a significant difference in pre-stimulation HFROM between treatment groups (active tDCS: 75.0 ± 19.0°; sham: 92.7 ± 24.2°, $p = 0.041$)(Figure 3). In the active tDCS group there was no significant difference in HFROM between pre- and post-stimulation (75.0° to 78.3°, $p = 0.104$), however, there was a significant difference in HFROM between pre-stimulation and 0 minutes (75.0° to 84.5°, $p < 0.001$), 5 minutes (75.0° to 83.7°, $p = 0.002$), 15 minutes (75.0° to 83.2°, $p = 0.002$), 30 minutes (75.0° to 85.8°, $p < 0.001$), and 60 minutes after stretching (75.0° to 84.0°, $p = 0.001$). In the sham group, no significant differences were found between pre-stimulation HFROM and any subsequent time ($p = 0.355$).

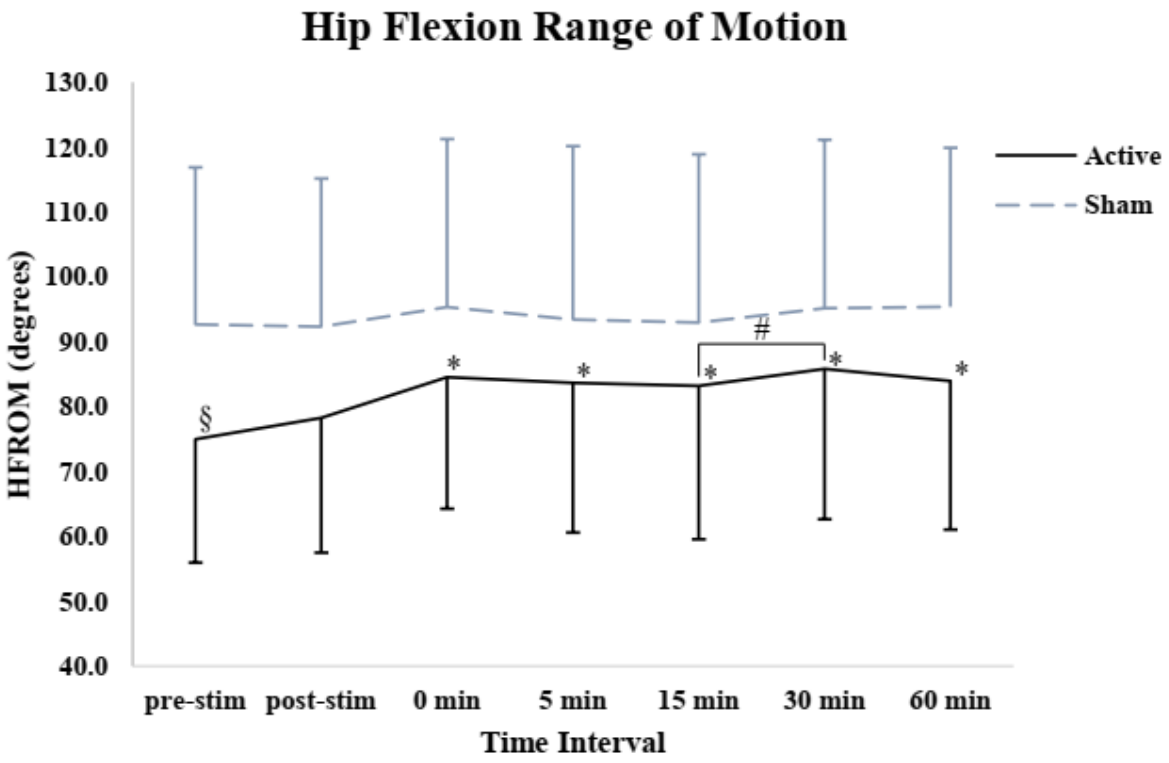


Figure 4. Effects of c-tDCS on HFROM. *Significant difference from pre- and post-stimulation ($p<0.05$). §Significantly different from sham ($p=0.041$). # Statistical difference ($p=0.028$). Error bars: Standard deviation.

Hip Passive Torque

Mauchly's test indicated that the assumption of sphericity had been violated ($\chi^2(20) = 62.558$, $p < 0.001$), therefore the Huynh-Feldt correction was used ($\varepsilon = 0.594$). There was a significant two-way interaction effect (group x time, $F(6, 21) = 3.225$, $p = 0.020$, $\eta^2 = 0.110$). In the active group there was no significant difference in passive torque at maximum hip flexion between pre- and post-stimulation (40.3 Nm to 41.1 Nm, $p = 0.753$), however, there was a significant difference in passive torque between pre-stimulation and 0 minutes (40.3 Nm to 49.1 Nm, $p = 0.002$), 5 minutes (40.3 Nm to 50.0 Nm, $p = 0.002$), 15 minutes (40.3 Nm to 51.2 Nm, $p = 0.003$), 30 minutes (40.3 Nm to 53.1 Nm, $p < 0.001$), and 60 minutes

after stretching (40.3 Nm to 50.6 Nm, p = 0.008)(Figure 4). In the sham group, no significant differences were found between pre-stimulation passive torque and any subsequent time (p = 0.415).

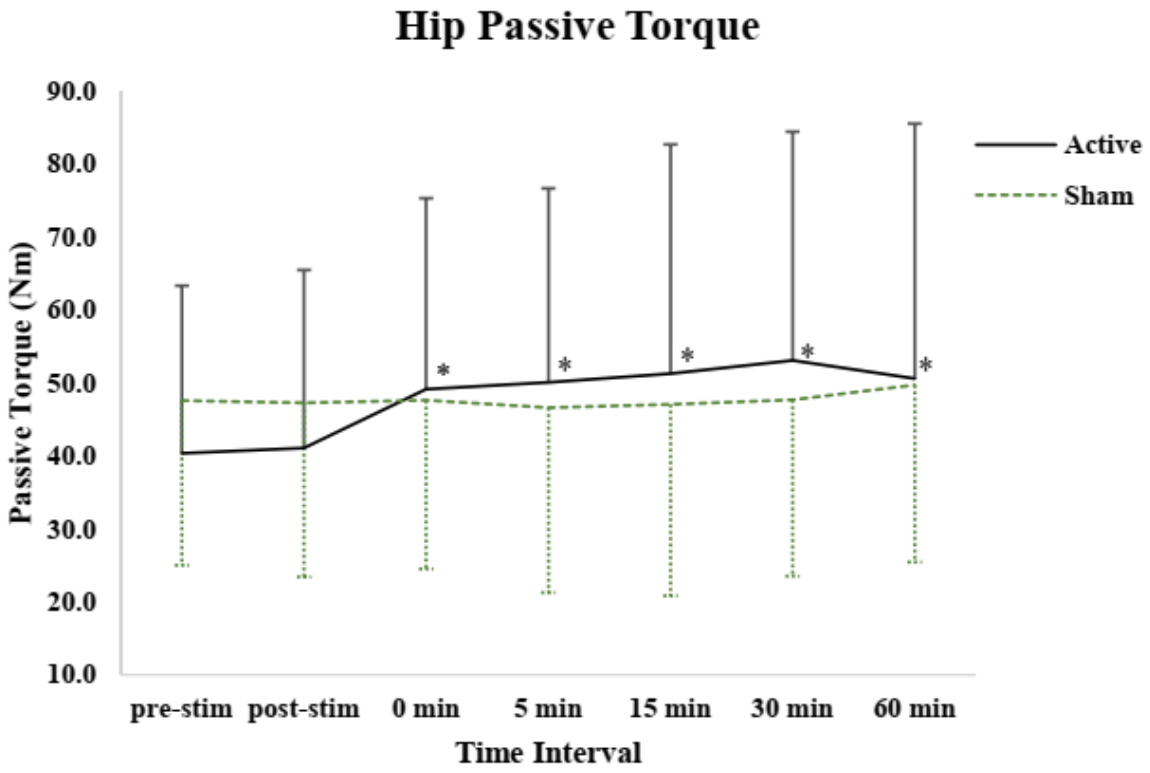


Figure 5. Effects of c-tDCS on passive torque. *Significant difference from pre- and post-stimulation ($p<0.05$). Error bars: Standard deviation.

Pain Perception

There was no significant two-way interaction (group x time, $F(6,21) = 1.158$, $p = 0.332$, $\eta^2 = 0.043$). There were no significant main effects for time ($F(6,21) = 1.671$, $p = 0.132$, $\eta^2 = 0.060$) or group ($F = 3.850$, $p = 0.061$, $\eta^2 = 0.129$). See Table 3 for details.

Table 3. Pain perception values (means±SD)

Treatment	Pre-Stim	Post-Stim	0 min	5 min	15 min	30 min	60 min
Active	4.2±2.2	4.2±2.1	4.6±2.4	4.3±2.0	4.3±2.2	4.1±2.4	3.9±2.1
Sham	5.5±1.8	4.8±2.0	5.7±2.0	5.7±1.8	5.6±1.9	5.9±1.8	5.5±1.8

No significant differences were found at any time ($p \geq 0.05$).

Perceived Treatment

Participants in the active tDCS group correctly perceived their treatment 71.4% of the time. Those in the sham tDCS group correctly perceived their treatment 35.7% of the time. In total, participants correctly perceived their treatments 53.6% of the time.

Chapter 5: Discussion

This study examined changes in HFROM and passive torque and the time course of those changes following either active c-tDCS or sham stimulation and a bout of high-intensity passive static stretching. In line with the initial hypothesis, the active c-tDCS group showed an increase in HFROM and passive torque following the stimulation and stretch, and these increases were maintained for at least 60 minutes. No changes in HFROM or passive torque were found in the sham group. Pain perception did not change at any time for either group. This was the first study to combine static stretching with c-tDCS and the first to investigate the time course of changes in flexibility in males and females following static stretching combined with c-tDCS.

Hip Flexion Range of Motion

In the current study, c-tDCS was applied over the lower limb region of S1. Contrary to the findings of previous studies,[17,18,20] HFROM did not improve following active c-tDCS without stretching. This finding conflicts with the initial hypothesis but may be explained by the lack of a warm-up in the current study. An active warm-up on its own has not been shown to alter ROM,[21,89] but an interaction between the active warm-up and c-tDCS could explain the changes found in studies on tDCS and ROM. In the current, c-tDCS without stretching was insufficient to elicit changes in HFROM in healthy adults.

At 0 minutes after static stretching, the active group showed a significant increase in HFROM ($p = 0.005$), and this increase was maintained for at least 60 minutes ($p = 0.021$). In the active group, it is likely that stretching elicited both viscoelastic and sensory (stretch tolerance) changes.[15] Changes to passive mechanical properties of muscles typically last up

to 10[30] or 15 minutes[21], therefore, changes to the mechanical properties of the muscle may explain the changes to HFROM up to 15 minutes, but beyond this time improvements are likely due to increased stretch tolerance alone. It is possible that the improvements in HFROM at 0 minutes after stretching and up to 60 minutes were due to the combination of altered stretch tolerance after stretching and reduced S1 excitability after c-tDCS. Typically, ROM improvements following static stretching last up to 30 minutes.[22,55] In the current study, greater improvement in HFROM and greater retention of improvements were found in the active group compared to the sham group. It is possible that the retention of HFROM improvements up to 60 minutes was due to the cortical excitability induced by c-tDCS. However, the sham group showed no improvement in HFROM at any time after stretching ($p \geq 0.05$), despite receiving the same stretch as the active group, so it may be misleading to compare the active group (c-tDCS plus stretching) to the sham group (only stretching).

It was unexpected to find no improvement in HFROM in the sham group. A possible explanation is that because the pre-stimulation HFROM of the sham group was significantly higher than that of the active group (17.7°, $p < 0.001$), a greater stretching stimulus may have been required to elicit improvements in HFROM. However, 9 out of 14 participants, including those with the highest pre-stimulation HFROM, did improve. Furthermore, studies have found similar improvements to joint ROM following acute[90] or long-term static stretching[91] regardless of baseline ROM. Therefore, baseline HFROM seems an unlikely explanation. There were several participants in the sham group who either did not improve with stretching and who worsened, reducing the apparent effects of the stretching.

Hip Passive Torque

As with HFROM, passive torque did not change following c-tDCS without stretching in the active group ($p = 0.753$), indicating that c-tDCS alone had no effect. One explanation for this, as with HFROM, was the use of an active warm-up in previous studies. There was a significant difference between 0 minutes after stretching and all other times in the active group ($p \leq 0.05$). The simultaneous increase in passive torque and HFROM after stretching was expected, based on previous studies.[49,52,54,92] That changes in passive torque coincided with changes in HFROM in the current study suggests that the static stretching improved participant stretch tolerance and that the maximum HFROM is dictated by the amount of external torque applied to the joint. Further, this finding offers support for a sensory theory as an explanation for the increases in joint ROM after stretching. Treatment with c-tDCS alone does not appear to be sufficient to elicit changes in passive torque in healthy adults.

Pain Perception

No changes in pain perception were found between groups at any time ($p \geq 0.05$). This was expected because the endpoint was determined by the participant's subjective stretch (pain) tolerance, and it was assumed they would stop the measurement at the same level of pain perception. Therefore, despite an increase in HFROM and passive torque in the active group, participants stopped the stretch at the same level of pain. It may be inferred that participants experienced a lower pain perception at submaximal angles than at the maximum angle, which was observed in a previous study[19].

Limitations

There were methodological limitations to this study that must be addressed. First, treatment groups were not stratified by pre-stimulation HFROM. Indeed, the sham group

baseline HFROM was 17.7° greater than that of the active group ($p = 0.041$). However, a study involving gymnasts showed that baseline joint ROM does not affect the changes elicited by stretching.[90] Likewise, a study on adults aged 50-75 years with presumably lower joint ROM than younger individuals found that improvements in joint ROM after stretching were comparable to younger individuals.[93] Another limitation is that true blinding may not be possible when using a current intensity of 2.0 mA, as in the current study.[74] One study found that investigators and participants were able to correctly distinguish active from sham conditions at a rate greater than expected by chance, though never above 65%.[94]

Applications and Future Studies

That c-tDCS and static stretching were more effective at improving HFROM and prolonging those improvements could have important clinical applications. Enhanced treatment results at a similar level of pain may benefit patients for whom stretching causes undue pain. Pain itself is a barrier that prevents patients from performing physical therapy exercises[95] and reduced pain perception during rehabilitation exercises may increase adherence to those exercises and ultimately improve rehabilitative outcomes.

Future studies should examine the long-term changes elicited by combined c-tDCS and stretching. The combined intervention appears to be effective acutely, but it should show a clear advantage in effectiveness over chronic static stretching. Also, studies investigating the effects of tDCS on passive ROM have focused on healthy populations, so evaluating the effects of combined treatment in clinical populations would help determine the clinical value of the treatment. The stretching protocol in this study was deliberately intense. For athletic populations this may be acceptable, but for clinical populations this level of intensity may preclude the usefulness of the treatment. A less intense stretching protocol, using a stretch

intensity that has been reported to elicit improvements in joint ROM, such as "onset of discomfort,"[96] following c-tDCS should be investigated to determine if the combined treatment is still superior to stretching alone. Finally, future studies should examine the unintended effects of inhibiting S1 using c-tDCS, alone or in combination with stretching. Despite relatively small changes in sensation, complications could arise due to decreased sensory or pain perception during dynamic activities such as athletics, dance, or manual labor. Therefore, use of c-tDCS prior to static stretching before such activities is not yet recommended.

Conclusion

Twenty minutes of cathodal transcranial direct-current stimulation at a current of 2.0 mA applied over the somatosensory cortex of healthy adults can acutely improve passive hip range of motion following a bout of static stretching for up to 60 minutes. Though c-tDCS alone may not be sufficient to elicit significant changes in HFROM, a combination of c-tDCS and static stretching appears to be more effective at improving HFROM for at least 60 minutes than stretching alone.

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Vita

Kenneth “Bryan” Taylor grew up in North Carolina and graduated from Appalachian State University in 2010 with a Bachelor of Science in Anthropology. He went on to complete coursework for a Bachelor of Science in Biology in 2012. In the spring semester of 2021 Bryan returned to Appalachian State to begin working toward his Master of Science degree in Exercise Science. He is currently working as faculty at Appalachian State teaching Parkour, Kung Fu, and Self-Defense. After a year off from school, he intends to continue his education in a Ph.D. program related to research in Exercise Science.