

Sensory electrical stimulation improves foot placement during targeted stepping post-stroke

Eric R. Walker · Allison S. Hyingstrom ·
Brian D. Schmit

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Abstract Proper foot placement is vital for maintaining balance during walking, requiring the integration of multiple sensory signals with motor commands. Disruption of brain structures post-stroke likely alters the processing of sensory information by motor centers, interfering with precision control of foot placement and walking function for stroke survivors. In this study, we examined whether somatosensory stimulation, which improves functional movements of the paretic hand, could be used to improve foot placement of the paretic limb. Foot placement was evaluated before, during, and after application of somatosensory electrical stimulation to the paretic foot during a targeted stepping task. Starting from standing, twelve chronic stroke participants initiated movement with the non-paretic limb and stepped to one of five target locations projected onto the floor with distances normalized to the paretic stride length. Targeting error and lower extremity kinematics were used to assess changes in foot placement and limb control due to somatosensory stimulation. Significant reductions in placement error in the medial–lateral direction ($p = 0.008$) were observed during the stimulation and post-stimulation blocks. Seven participants, presenting with a hip circumduction walking pattern, had reductions ($p = 0.008$) in the magnitude and duration of hip abduction during swing with somatosensory stimulation. Reductions in circumduction correlated with both functional and clinical measures, with larger improvements observed in participants with greater impairment. The results of this

study suggest that somatosensory stimulation of the paretic foot applied during movement can improve the precision control of foot placement.

Keywords Stroke · Stepping · Electrical stimulation · Foot · Hip circumduction · Balance · Motor control

Introduction

The precision control of foot placement location is an important component of locomotion. For example, step-by-step modification of foot placement is important for dynamic balance control during walking (Hof et al. 2007, 2010), and much of the focus of this control is centered upon the frontal plane (O'Connor and Kuo 2009). Additionally, accurate control of foot placement is important for adapting the walking pattern to environmental conditions, such as when stepping over obstacles. This control of foot placement requires the integration of visual and proprioceptive feedback signals and involves brain structures such as the primary motor cortex (Bretzner and Drew 2005) and posterior parietal cortex (Marigold et al. 2011). After stroke, damage to these and other brain structures can disrupt sensorimotor integration, impairing the control of foot placement during stepping.

Impairment in sensorimotor control of foot placement might substantially impact walking function in stroke survivors. Walking dysfunction post-stroke includes slower walking speeds (Turnbull et al. 1995), decreased walking endurance (Michael et al. 2005), and increased risk of falls (Mackintosh et al. 2005). Impairments in control of foot placement appear to contribute to these functional losses. For example, foot placement asymmetries in both the frontal and sagittal plane during walking correlate with functional impairments post-stroke (Balasubramanian et al.

E. R. Walker · B. D. Schmit (✉)
Department of Biomedical Engineering, Marquette University,
P.O. Box 1881, Milwaukee, WI 53201-1881, USA
e-mail: brian.schmit@marquette.edu

A. S. Hyingstrom
Department of Physical Therapy, Marquette University,
Milwaukee, WI, USA

2010). Additionally, stroke survivors modify foot placement location relative to an obstacle, providing additional time for the paretic limb to clear the obstacle, but also potentially compromising balance (Said et al. 2001). Stroke survivors also have difficulty making medial foot placement adjustments mid-step; however, their ability to make these adjustments improves when balance assistance is provided during the task (Nonnekens et al. 2010). These studies demonstrate that the control of foot placement is associated with balance control and walking function. Therefore, increased walking function might be achieved through techniques aimed at improving foot placement control in stroke survivors.

Augmenting sensory feedback provides a potential mechanism to improve foot placement. Somatosensory electrical stimulation applied to the paretic wrist improves hand function for a period of time after stimulation in stroke survivors (Wu et al. 2006). Applying vibratory stimulation to the paretic wrist during movement improves endpoint stability during both planar reaching (Conrad et al. 2011a) and tracking tasks (Conrad et al. 2011b). Sensory stimulation has also been used in the lower extremity to improve standing and walking function. Increased plantar sensory feedback, through the use of a textured insole, improves standing balance in neurologically intact individuals when visual feedback is removed (Corbin et al. 2007). Additionally, sub-sensory threshold vibration of the plantar surface of the foot improves standing balance control in stroke participants, with the largest improvements observed in participants with the greatest balance impairments (Priplata et al. 2006). Foot sole vibration also improves walking function in Parkinson's patients when applied during stance (Novak and Novak 2006). Delivering electrical stimulation to the paretic foot and ankle during movement improves both walking speed and standing balance in chronic stroke survivors (Tyson et al. 2013). These studies demonstrate that augmented sensory feedback, through various techniques, can improve the control of upper and lower extremity movements. In this study, we used electrical stimulation to augment sensory feedback from the paretic foot, which might be useful for improving foot placement control post-stroke.

The purpose of this study was to quantify the effects of sensory stimulation, provided by an electrical stimulus applied to the paretic foot, on foot placement during a stepping task. We hypothesized that electrical stimulation of the paretic foot would decrease foot targeting error and improve lower extremity kinematics.

Methods

Participant information

Twelve chronic (>6 months) stroke participants (age 47–63) with unilateral brain injury participated in this study.

All twelve participants reported a vascular origin of their injury. Exclusion criteria included inability to obtain informed consent, diagnosis of other neurologic disorders or cognitive deficits, recent (<3 months) use of botulinum toxin, and inability to walk independently (with or without the use of an assistive device). A licensed physical therapist conducted a clinical evaluation of each individual consisting of the lower extremity Fugl-Meyer test (Fugl-Meyer et al. 1975), Berg balance assessment (Berg et al. 1992), and 10 m walking test (Mudge and Stott 2009). Participant characteristics are summarized in Table 1. All procedures were approved by the Institutional Review Board at Marquette University, and all participants provided written informed consent.

Data collection

Kinematic data from the lower extremities were collected using a six camera Vicon Mx motion capture system (Vicon Motion Systems Ltd, Oxford, UK). Fifteen passive infrared reflective markers were placed at anatomical locations according to the Plug-In-Gait model (Davis et al. 1991). All signals were collected using the Vicon Nexus software at 100 Hz.

Experimental protocol

Participants were placed in a ceiling-mounted fall arrest system. Participants started from a standing position, aligning both feet with two lines projected onto the floor to keep the starting location consistent across trials. One line aided in aligning the paretic foot in the medial lateral direction, while the other line aided in positing both feet in the anterior posterior direction. Participants initiated each trial with the non-paretic limb, stepped to the projected target with the paretic limb, and then completed one more step each with the non-paretic and paretic limb. This sequence produced one complete goal directed stride for each limb. During each trial, a circular target ($r = 20$ mm) was projected onto the floor 500 ms after a buzzer sounded, indicating the start of the trial. Target locations were normalized to a percentage of the participant's paretic limb stride length, determined at the beginning of the session. Close, normal, and far targets were located in line with the paretic limb at a distance of 80, 100, and 120 % of the paretic limb stride length, respectively. The last two targets were located 20 % of the paretic stride length medial or lateral to the paretic limb starting location, at an anterior–posterior distance equal to the paretic stride length (Fig. 1). Participants performed one practice trial to each target location to ensure they could complete the stepping sequence and to reduce possible practice effects.

The testing was conducted in three blocks. During each block, targets were presented in a randomized order, and each target location was repeated four times, resulting in

Table 1 Clinical data for stroke participants

Participant	Age (years)	Sex	Time since stroke (months)	Affected side	Stroke location	LE FM ^a	Berg ^b	Self-selected walking speed (m/s)	Paretic limb monofilament threshold ^c
S01	48	M	77	R	Cort	22	51	1.173	3.84
S02	61	M	101	L	Subcort	21	39	0.502	5.07
S03	60	F	61	R	Cort	32	49	1.270	5.07
S04	63	F	236	L	n/a	32	55	1.298	3.61
S05	49	M	26	L	Cort	24	45	0.743	5.18
S06	58	M	55	R	Subcort ^d	29	46	1.361	5.88
S07	53	M	72	L	Cort	24	49	0.988	4.56
S08	54	F	90	R	Subcort	31	56	1.271	3.61
S09	64	M	30	R	Subcort	28	52	1.043	4.31
S10	55	M	194	R	Cort	32	54	1.576	4.31
S11	61	F	60	L	Cort ^e	19	46	0.837	4.17
S12	58	F	285	L	Cort	23	38	0.626	3.22

^a Lower extremity Fugl-Meyer, maximum score 34

^b Berg Balance Score, maximum score 56

^c Normal ≤ 3.61 , loss of protective sensation ≥ 5.07

^d Carotid stroke; all others middle cerebral artery

^e Hemorrhagic stroke; all others ischemic

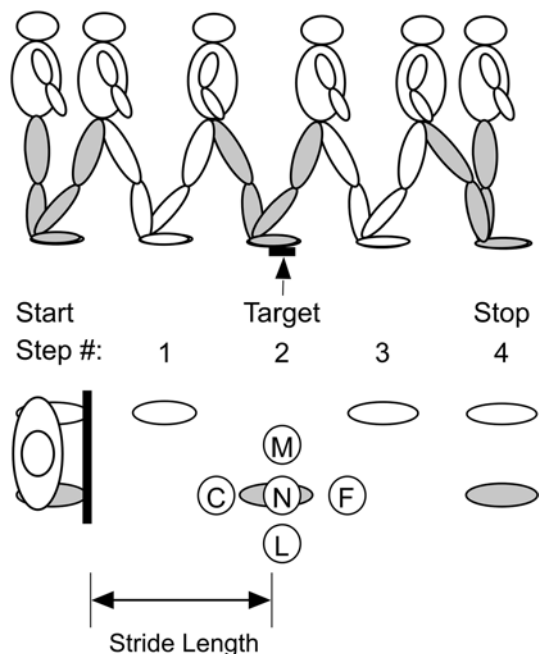


Fig. 1 Diagram of targeted stepping task. Participant started from rest, initiated movement with the non-paretic limb, stepping to the projected target with the paretic limb, finishing the sequence stepping the non-paretic then paretic limbs. Steps one and three were completed with the non-paretic limb, while steps two and four were completed with the paretic limb. *Top view* of experiment depicting target locations, a single-target location was projected for each trial. Shaded limb/foot represents the paretic limb

20 trials in each experimental block. During the second of the three blocks, a 30 Hz electrical stimulation was applied to the medial plantar nerve of the paretic limb, providing evaluation of stepping before, during, and after stimulation. The stimulation began one second before target projection and remained on for the duration of the trial (6 s). A constant current stimulator (DigitimerDS7A, Digitimer Ltd, Hertfordshire, England) delivered biphasic pulses to two surface electrodes (Vermed Inc, Bellows Falls, VT, USA) placed posterior to the medial malleolus on the paretic foot. Stimulation intensity was set to 95 % of motor threshold of the abductor hallucis. This intensity produced a tactile sensation on the plantar surface of the foot, without producing a palpable contraction in the foot. The final, third, experiment block was conducted without stimulation to evaluate any potential aftereffects from the stimulation. A custom LabVIEW (National Instruments, Austin, TX, USA) program was used to control timing of the Vicon data collection, target presentation, and electrical stimulation.

Data analysis

Processing of the marker trajectories was completed using the Plug-In-Gait model in Vicon Nexus to obtain lower extremity kinematics and kinetics. Further data analysis was completed in MATLAB (Mathworks, Natick, MA, USA). Marker trajectories were low-pass filtered at 15 Hz

prior to analysis. The analysis produced joint angles for each joint in three planes (sagittal, frontal, and transverse), foot placement locations, stance and swing timing, and stride and step lengths. Initially, stepping performance was assessed by the error magnitude between the projected target location and the toe marker location during paretic limb stance. Targeting error measures were calculated separately for the anterior–posterior and medial–lateral directions. Hip frontal plane motion during swing was quantified further by integrating the paretic limb frontal plane angle, while the limb was in abduction during swing. The area of the frontal plane hip angle provided a measure of limb circumduction during swing and was sensitive to changes in both the magnitude and duration of abduction. A measure of swing time symmetry was obtained by dividing the paretic by the non-paretic swing duration. A value of one indicated perfect swing time symmetry between the two limbs, and a value greater than one indicated that the paretic limb spent more time in swing compared with the non-paretic limb.

Separate univariate ANOVAs were completed to assess the effect of the electrical stimulation on error magnitude and frontal plane hip motion. Bonferroni post-hoc tests were used to examine differences between pre-stimulation, stimulation, and post-stimulation blocks. Pearson correlation analyses were completed to examine the relationships between the changes in hip frontal plane motion, lower extremity Fugl-Meyer, Berg Balance Score, self-selected walking speed, and swing time symmetry. A correlation analysis between targeting error and trial number was performed for each participant to test for the presence of learning effects in the pre-stimulation block. All statistical tests were conducted with a significance level of $\alpha = 0.5$ and were completed using SPSS 16.0 software (IBM, Endicott, NY, USA).

Results

Targeting error

Changes in the control of foot placement due to electrical stimulation were quantified by the targeting error magnitude in both the medial–lateral and anterior–posterior directions (Fig. 2). A significant main effect of stimulation condition ($p = 0.008$) was observed across all targets for targeting error in the medial–lateral direction, while no significant effect was observed in the anterior–posterior direction. Post-hoc analyses indicated that medial–lateral targeting error was significantly greater in the pre-stimulation block compared with the stimulation ($p = 0.006$) and post-stimulation blocks ($p = 0.035$), as shown in Fig. 2a. No significant correlations between targeting error and trial number were observed for any of the 12 participants, indicating that the decrease in targeting error was not due to a learning effect.

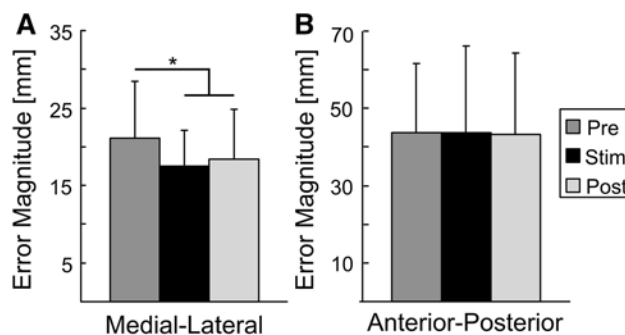


Fig. 2 Group average (+std) targeting error magnitude in medial–lateral (a) and anterior–posterior (b) directions across all targets. Medial–lateral targeting error was significantly reduced during the stimulation and post-stimulation trials (Bonferroni post hoc, $p < 0.05$)

Joint kinematics

In addition to reductions in medial–lateral targeting error, 7/12 participants displayed decreases in magnitude and duration of hip abduction during swing (mean trajectories for participants S04 and S05 are shown in Fig. 3). These seven participants demonstrated sustained hip abduction through late swing during the pre-stimulation block (Fig. 3a) that was not present in the other five participants (Fig. 3b). The presence of increased hip abduction during late swing is indicative of a hip circumduction compensatory strategy (Kerrigan et al. 2000). When sensory stimulation was applied to the paretic limb, we observed decreases in this circumduction pattern that remained in the post-stimulation trials (Fig. 3a).

To evaluate the differential effects of stimulation on frontal plane hip motion, we correlated changes in frontal plane hip area from the pre-stimulation to stimulation block with clinical and functional measures. This change in hip abduction area significantly correlated with lower extremity Fugl-Meyer score ($r = 0.752$, $p = 0.005$), self-selected walking velocity ($r = 0.642$, $p = 0.024$), and swing time asymmetry ($r = -0.702$, $p = 0.011$) (Fig. 4). No significant correlations were observed for either the Berg Balance Score or paretic limb monofilament perception threshold. Reductions in hip abduction area during swing were observed in individuals with lower Fugl-Meyer scores (<29) and slower self-selected walking speeds (<1.2 m/s). These seven participants also presented with hip circumduction movement patterns during the pre-stimulation block, which were not observed in the other five participants. These seven individuals (circumduction group) showed a significant effect of stimulation condition ($p = 0.008$), and post-hoc analyses indicated that there was a significant decrease in the stimulation and post-stimulation blocks compared with the pre-stimulation block ($p < 0.001$) (Fig. 5). There were no significant effects of stimulation condition for the non-circumducting group ($n = 5$).

Fig. 3 Frontal plane hip motion of the paretic limb from two representative participants when stepping to the normal target location (**a** S05, **b** S04). Shaded region represents swing phase. The somatosensory stimulation reduced the amplitude and duration of hip abduction during late swing for individuals presenting with a circumduction movement pattern (**a**), but had no effect on hip abduction for the non-circumduction group (**b**)

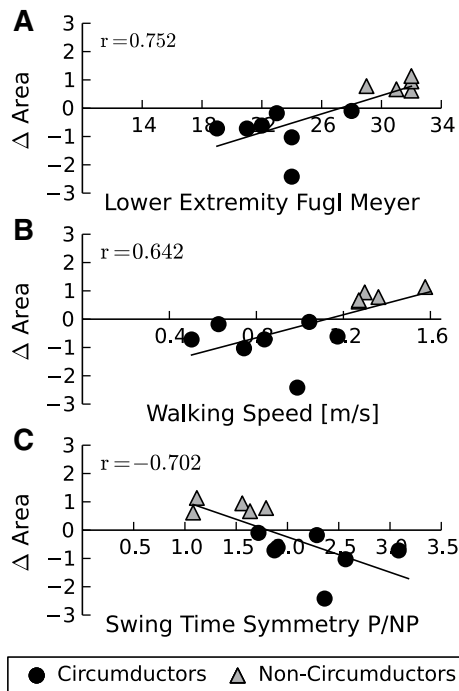
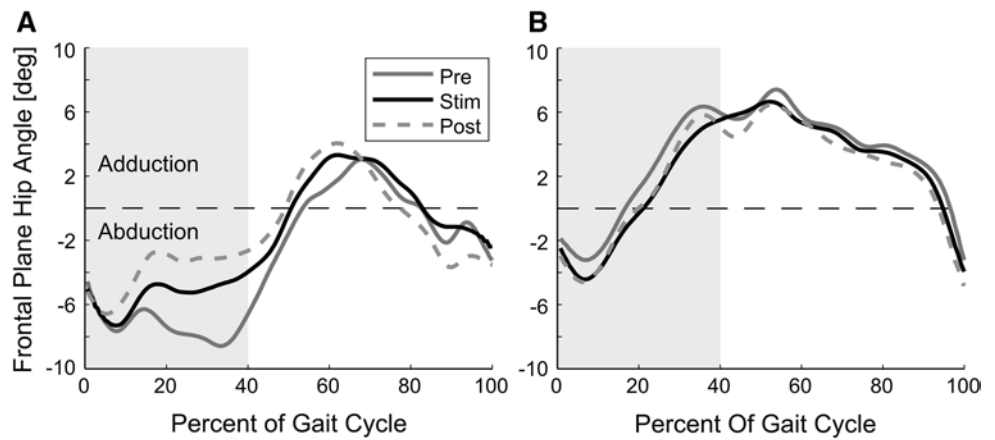


Fig. 4 Correlation of average change in abduction area from stimulation to pre-stimulation block (open triangle area) with lower extremity Fugl-Meyer (**a**), self-selected walking speed (**b**), and swing time symmetry ratio (**c**). A negative value represents a decrease in circumduction when stimulation was applied. The change in area significantly correlated with all three metrics, with reductions in circumduction area observed in patients with lower Fugl-Meyer scores, slower walking speeds, and more swing time asymmetry

Discussion

Application of somatosensory, electrical stimulation to the paretic foot produced improvements in frontal plane control of the paretic leg during a targeted stepping task. Specifically, we observed significant reductions in medial-lateral targeting error during the stimulation and post-stimulation blocks (Fig. 2), suggesting improvement in the

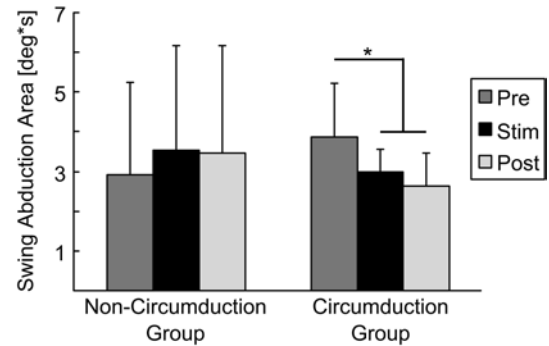


Fig. 5 Average hip abduction area during swing for the two participant groups: those presenting with hip circumduction movement pattern ($n = 7$), and those without hip circumduction movement pattern ($n = 5$). Swing abduction area significantly decreased in both the stimulation and post-stimulation block compared with the pre-stimulation trials only for the circumduction group

control of foot placement post-stroke. Somatosensory stimulation of the paretic limb also reduced hip abduction area during swing for participants presenting with a circumduction walking pattern (7/12), suggesting changes in frontal plane limb control. These results indicate that somatosensory stimulation might provide a mechanism to improve walking function post-stroke, especially in more impaired individuals.

The observation of locomotor changes in the frontal plane may be attributed to the manner in which supraspinal structures actively control walking. During walking, leg movement is inherently stable in the sagittal plane, and therefore, supraspinal resources are likely focused upon control of frontal plane motion to optimally ensure balance and stability while walking (O'Connor and Kuo 2009). Similarly, somatosensory electrical stimulation applied to the paretic wrist improves hand function by inducing changes at the cortical level (Kaelin-Lang et al. 2002). It is plausible that our somatosensory stimulation paradigm activated a similar cortical mechanism, despite being

applied to the lower extremity. Somatosensory stimulation of the paretic foot may be acting to enhance sensorimotor integration in areas such as the posterior parietal cortex, which are important to the execution of visually guided locomotor movements (Marigold et al. 2011). Further research is needed to understand the potential mechanisms behind these improvements in locomotor control in order to maximize its effect for stroke survivors.

The observed improvements in paretic leg control might also be associated with stimulation-induced changes in hip and knee synergy patterns that reduce circumduction. After stroke, increased multi-joint coupling between the paretic hip and knee (Lewek et al. 2007) contributes to both reduced gait speeds as well as increased pelvic compensatory movements (Cruz et al. 2009). The persistence of abnormal hip abduction movements during robot-assisted gait (Neckel et al. 2008; Sulzer et al. 2010) suggests that measures must be taken to reduce this coupling in order to restore normal kinematic patterns. The observed decreases in hip abduction area during swing in this study may represent changes in functional coupling of the hip and knee muscles due to the somatosensory stimulation. This reduced frontal plane hip movement could contribute to observed reductions in targeting error by enabling participants to take a more direct path to the target location. However, we did not observe any significant correlations between hip abduction area and frontal plane targeting error. Reductions in hip circumduction were only observed for individuals presenting with a hip circumduction movement pattern, while all participants showed improvements in foot placement control. Therefore, we do not attribute reduced targeting error solely to reductions in hip circumduction. Improved frontal plane biomechanics, especially in more impaired stroke survivors, and improved locomotor planning likely act together to enhance foot placement control during the task.

It is important to note that the targeted stepping task used in this study is somewhat different from continuous walking. In our task, participants initiated gait with the non-paretic limb, stepped to a projected target with the paretic limb, and finished with a series of two more steps. This design ensured that participants completed this goal directed movement within the context of a walking task. Unlike previous studies that have evaluated foot placement during obstacle avoidance (Said et al. 2001) or targeted foot placement during walking (Alexander et al. 2011), which allowed for modification of the walking pattern over a series of steps, we wanted to evaluate the ability of stroke survivors to execute a targeted movement within a single-gait cycle of the paretic limb. However, due to the fact that participants started this task from rest, larger demands were placed on the paretic limb to generate forward momentum to initiate walking (Hesse et al. 1997), which has

been shown to have reduced propulsive output post-stroke (Bowden et al. 2006). The increased propulsive demands placed on the paretic limb during the step to the target, relative to normal walking, may result in larger improvements than those expected during continuous walking. We were unable to obtain ground reaction forces in this experiment to quantify the role of paretic limb propulsion during the baseline task performance, or the influence of the somatosensory stimulation on paretic propulsion. However, it is unlikely that the improvements in targeting error were due to changes paretic propulsion, since deficits in the frontal plane control of foot placement were also observed during a step of the paretic limb only (Nonnekes et al. 2010). In addition to differences in the biomechanical demands of the targeted stepping task, the goal directed nature of this task might also involve different neural control elements compared with continuous walking. Recordings from the cat motor cortex have demonstrated higher firing rates during targeted stepping movements compared with normal locomotion (Beloozerova et al. 2010). Since the neural mechanism behind these improvements with somatosensory stimulation is unclear, it is unknown how these improvements in frontal plane foot placement transfer to continuous walking with somatosensory stimulation. However, it is likely that the greatest benefits will be observed when continual adjustments are needed during walking, such as walking over an uneven surface or through a cluttered environment.

The results of this study demonstrate the potential for including somatosensory stimulation of the paretic foot into traditional rehabilitation techniques to further improve walking function in stroke survivors. Stroke survivors possess the ability to produce symmetric walking patterns (Reisman et al. 2009), but the prevalence of asymmetries in the walking pattern post-stroke suggests a significant contribution of abnormal control mechanisms. Applying somatosensory stimulation to the paretic foot during the walking task improved the precision control of paretic foot placement, as well as reducing hip circumduction in more impaired individuals. Furthermore, these reductions in hip abduction correlated with both clinical and functional metrics, suggesting that somatosensory stimulation will likely have the largest effect in individuals with the most impaired walking function. Similarly, a ceiling effect was observed when somatosensory stimulation was applied to the paretic wrist (Kaelin-Lang et al. 2002), supporting the use of somatosensory stimulation with more impaired patients. Additionally, these improvements in frontal plane control remained when the stimulation was removed, suggesting at least a short-term (20 stepping trials) changes in locomotor control (i.e., aftereffects). Further research is needed to determine the duration of these plastic changes in stepping function, as well as to identify the impact of somatosensory stimulation of the paretic foot on continuous walking.

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