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Symmetry of corticomotor input to plantarflexors influences the propulsive strategy used to increase walking speed post-stroke

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HIGHLIGHTS

- Corticomotor asymmetry to plantarflexors influences the propulsive strategy used to increase post-stroke walking speed.
- Individuals with good corticomotor symmetry improve propulsive symmetry to achieve their fastest walking speeds.
- Rehabilitation strategies that promote corticomotor symmetry to plantarflexors may improve post-stroke gait mechanics and enhance functional walking outcomes.

ABSTRACT

Objective: A deficit in paretic limb propulsion has been identified as a major biomechanical factor limiting walking speed after stroke. The purpose of this study was to determine the influence of corticomotor symmetry between paretic and nonparetic plantarflexors on the propulsive strategy used to increase walking speed.
Methods: Twenty-three participants with post-stroke hemiparesis underwent transcranial magnetic stimulation and biomechanical testing at their self-selected and fastest walking speeds. Plantarflexor corticomotor symmetry (CS_{PF}) was calculated as a ratio of the average paretic versus nonparetic soleus motor evoked potential amplitude. The ratio of the paretic and nonparetic peak ankle plantarflexion moments (PF_{sym}) was calculated at each speed.
Results: CS_{PF} predicted the ΔPF_{sym} from self-selected and fastest speeds ($R^2 = .629$, $F(1,21) = 35.56$, $p < .001$). An interaction between CS_{PF} and ΔPF_{sym} ($\beta = .596$, $p = .04$) was observed when predicting Δspeed ($_{adj}R^2 = .772$, $F(3,19) = 20.48$, $p < .001$). Specifically, the ΔPF_{sym} with speed modulation was positively related to the Δspeed ($p = .03$) in those with greater CS_{PF}, but was not related in those with poor CS_{PF} ($p = .30$).
Conclusions: Symmetry of the corticomotor input to the plantarflexors influences the propulsive strategy used to increase post-stroke walking speed.
Significance: Rehabilitation strategies that promote corticomotor symmetry may positively influence gait mechanics and enhance post-stroke walking function.

1. Introduction

Following stroke, the majority of survivors are unable to regain sufficient walking function to allow for ambulation at speeds that are safe and effective for community function and participation (van de Port et al., 2008). In addition to typically slower walking speeds compared to neurologically-intact individuals, persons post-stroke are left with a reduced capacity to increase walking speeds (van de Port et al., 2008). The ability to modulate walking speed is clinically meaningful because it underlies an individual's capacity for safe and effective community function (Jonkers et al., 2009; van de Port et al., 2008). Altered muscular strength and

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coordination leads to asymmetrical gait patterns that underlie post-stroke hemiparesis and limit walking function (Nadeau et al., 1999; Olney et al., 1994; Den Otter et al., 2007; Turns et al., 2007). In addition to biomechanical impairments, neurophysiologic measures of corticomotor pathway integrity to the lower extremity muscles have been shown to be related to lower extremity strength (Beaulieu et al., 2014) and walking function post-stroke (Hendricks et al., 2003; Steube et al., 2001; Palmer et al., 2016).

A critical factor in producing functional walking speeds is the ability to generate sufficient propulsion to advance the body's center of mass forward (Neptune et al., 2001). In fact, the most significant biomechanical contributor to limited post-stroke walking speeds has been identified as a deficiency in propulsive force generated by the paretic limb (Bowden et al., 2006; Nadeau et al., 1999; Neptune et al., 2001; Peterson et al., 2010). Knowledge of an individual's paretic plantarflexor contribution to forward propulsion can distinguish him or her between functional ambulation classifications of limited versus unlimited community ambulators (Bowden et al., 2008; Peterson et al., 2010). Further, rehabilitation strategies that improved paretic limb propulsion have also improved post-stroke walking function (Awad et al., 2014; Bowden et al., 2013). The two main contributors to forward propulsion are trailing limb angle and ankle plantarflexion moment (Hsiao et al., 2015a,b). The plantarflexion moment represents the net torque generated by the plantarflexor muscles that cross the ankle joint. Thus, the ability to activate the plantarflexor muscles plays a critical role in generating propulsion to attain and increase gait speeds in both neurologically-intact (Hsiao et al., 2015b) and stroke populations (Olney et al., 1994; Hsiao et al., 2015a; Peterson et al., 2010).

Although impaired paretic propulsion has been shown to be related to post-stroke walking function, analyzing the biomechanical strategies that individuals use to *increase* their gait speed reveals important impairments of walking function (Jonkers et al., 2009). In the presence of an inability to recruit the paretic plantarflexors, persons with post-stroke hemiparesis utilize a variety of compensatory strategies to achieve faster walking speeds. These include utilization of the paretic hip flexors (Nadeau et al., 1999; Jonkers et al., 2009) and compensation with the nonparetic limb (Jonkers et al., 2009). Amongst a heterogeneous stroke patient population, individuals may utilize different mechanisms (e.g. increase paretic plantarflexion moment or increase reliance on nonparetic plantarflexion moment) to achieve similar walking speeds (Allen et al., 2014). Previously, Jonkers et al. (2009) found that individuals who walked at slower speeds did not use paretic plantarflexion power to increase gait speed, but instead relied on increased nonparetic plantarflexion power (Jonkers et al., 2009). In contrast, individuals who walked at faster speeds increased both paretic and nonparetic plantarflexion power to increase gait speed, which is the strategy expected in neurologically-intact individuals (Jonkers et al., 2009). However, other studies have found that propulsion asymmetry between paretic and nonparetic legs is only weakly related to walking speed and that individuals walking at the same speed exhibit varying degrees of asymmetry, with some individuals improving paretic leg propulsion contribution (improved symmetry) and others relying heavily on the nonparetic leg (worse symmetry) (Bowden et al., 2006; Allen et al., 2014). Although it is clear that individuals utilize different biomechanical strategies to increase walking speed post-stroke and that such strategies are associated with the level of functional recovery, *previous research has failed to identify the underlying factors that determine the biomechanical strategy used to increase gait speed.*

Following stroke, disuse of the paretic limb coupled with heavy reliance on the nonparetic limb for functional activities have been shown to induce major cortical neuronal reconstruction (Kleim and Jones, 2008) and influence corticomotor input to affected muscles (Harris-Love, 2013). Transcranial magnetic stimulation (TMS) is a noninvasive brain stimulation tool that is used to investigate the neurophysiologic components underlying post-stroke motor function and recovery by quantifying the strength of corticomotor input to specific muscles (Harris-Love, 2013). In the upper extremity, the strength of corticomotor input to the paretic arm and hand has been shown to be related to muscle activation, strength and function in individuals post-stroke (Dimyan and Cohen, 2010; Harris-Love, 2013) and can be used to predict an individual's ability to regain activation of those muscles and functional outcomes in response to a rehabilitation intervention (Koski et al., 2004). Additionally, abnormally increased corticomotor input to the nonparetic limb has been observed following stroke (Harris-Love, 2013; Traversa et al., 1998). The resulting corticomotor asymmetry between the paretic and nonparetic limbs has been shown to be related to poor upper extremity motor recovery (Koski et al., 2004). Though limited at this time, evolving research in the lower extremity has indicated that decreased corticomotor input to paretic leg muscles is related to poor function (Hendricks et al., 2003; Steube et al., 2001; Palmer et al., 2016; Beaulieu et al., 2014). Additionally, a recent study from our laboratory showed that contraction of the nonparetic tibialis anterior and activation of the nonlesioned hemisphere facilitated corticomotor input to the paretic tibialis anterior in those with slow walking speeds post-stroke (Palmer et al., 2016). This suggests that there may be a maladaptive influence of the nonparetic lower extremity on paretic limb walking function. However, little is known about the role of corticomotor input to the nonparetic leg and the influence of corticomotor asymmetry between paretic and nonparetic legs on *biomechanical walking function*. Further, studies to date have failed to investigate the role of corticomotor input to the *ankle plantarflexor muscles*, the primary contributors to forward propulsion during walking.

A better understanding of possible interactions between biomechanical and neurophysiologic factors that affect walking function post-stroke could be crucial for the development of effective rehabilitation approaches. The purpose of this study was to investigate the influence of lower extremity corticomotor input on the propulsive strategy used to modulate walking speed post-stroke. Specifically, we aimed to determine (1) the relationship between the symmetry of corticomotor input to the plantarflexor muscles versus the changes in plantarflexion moment symmetry and (2) if symmetry of corticomotor input to the plantarflexors moderates the relationship between change in plantarflexion moment symmetry and change in walking speed. We hypothesized that there would be a positive relationship between plantarflexor corticomotor symmetry and changes in plantarflexion moment symmetry with increases in walking speed. Additionally, there will be an interaction between change in ankle plantarflexion moment and plantarflexion corticomotor symmetry, with individuals with the most symmetrical corticomotor input to paretic and nonparetic plantarflexors improving relative paretic ankle moment contribution with increases in walking speed.

2. Methods

Twenty-three individuals with chronic stroke (>6 mo.) (15 males, mean time since stroke 50 ± 59 mo., mean age 61.5 ± 8.4 years) and hemiparesis were recruited. All participants gave written informed consent and the protocol was approved by the University of Delaware's Institutional Review Board. Participants sustained a single cortical or subcortical stroke, were able to walk for at least 1 min without an orthotic and without the assistance of another person, and had sufficient ankle passive range of motion to allow the paretic ankle joint to reach the neutral