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The effect of post-stroke lower-limb spasticity on the control of standing balance: Inter-limb spatial and temporal synchronisation of centres of pressure

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ABSTRACT

Background: Challenges in stability control are common post-stroke. Although lower-limb spasticity is a common sensorimotor consequence post-stroke, its potential to further complicate stability control among stroke-survivors remains largely unknown. Advancing such understanding can help inform strategies to reduce fall risk and increase independence among these individuals. The purpose of this study was to characterise the extent of limb-specific dyscontrol among individuals with spasticity.
Methods: A retrospective analysis of 131 patients assessed for spasticity was performed. Patients selected for inclusion were categorised into two groups, with ($n = 19$) or without ($n = 63$) unilateral lower-limb spasticity. Two force platforms were used to determine the individual-limb and net centres of pressure in both anteroposterior and mediolateral directions during 30 s of quiet standing. Limb-specific dyscontrol was assessed by calculating weight-bearing symmetry ratios, cross-correlation coefficients at zero phase-shift (temporal synchrony) and ratios of individual-limb root-mean-square displacements (spatial symmetry). Total body postural control was assessed by examining the root-mean-square of the net centre of pressure displacement.
Findings: The group with spasticity bore less weight on the affected limb and exhibited reduced temporal synchrony of centre of pressure displacements. There were no differences in inter-limb root-mean-square centre of pressure ratios or in the root-mean-square of the net centre of pressure displacement.
Interpretation: Individuals with lower-limb spasticity may have additional challenges with stability control, specifically linked to the ability to modify the location of the centre of pressure beneath the affected limb, in a time-sensitive manner so as to contribute beneficially to the control of whole body stability.

1. Introduction

Deficits in balance control post-stroke are problematic since they may greatly influence mobility and independence in activities of daily living (Fong et al., 2001), patient-perceived disability (Desrosiers et al., 2002) and fall risk (Nyberg and Gustafson, 1995, 1997). Advancing the understanding of the challenges in stability control faced by stroke-survivors can inform strategies to reduce fall risk and increase independence among these individuals.

Lower-limb spasticity is a prominent secondary sensorimotor consequence of stroke, which may have considerable potential to impact stability control. Spasticity has been defined as a velocity-dependent increase in tonic stretch reflexes, is often discernible as increased resistance to passive limb movement and has been estimated to occur in approximately 30% of stroke-survivors (Lundstrom et al., 2008, 2010; Sommerfeld et al., 2012; Soyuer and Ozturk, 2007; Urban et al., 2010; Watkins et al., 2002). In addition to the velocity-dependent effects of spasticity, concomitant problems with the generation and modulation of muscle force include the disruption of agonist-antagonist sequencing, passive tissue stiffness and co-contraction (Gracies, 2005). Despite the well-documented challenges in stability control (de Haart et al., 2004; Geurts et al., 2005; Lakhani et al., 2011; Mansfield et al., 2012a,b; Weerdesteyn et al., 2008) and increased fall risk among stroke-survivors (Forster and Young, 1995), the influence of post-stroke spasticity on stability control has not been well-established empirically. Indeed, studies examining the determinants of falls and those utilising quantitative force platform measures have revealed conflicting results. Specifically, while both Pang and Eng (2008) and Soyuer and Ozturk

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(2007) employed logistic regression models to determine the relationship between spasticity and fall risk, only Soyuer and Ozturk (2007) found spasticity to be a significant predictor of falls. In addition, Cakar et al. (2010) employed an ankle-foot orthosis to alter the mechanics of the affected limb among patients with spastic hemiparesis and suggested that reductions in spasticity may translate to improvements in standing balance control. In contrast, Nardone et al. (2001) measured centre of pressure (COP) displacements during both quiet- and perturbed-standing and found insufficient evidence to suggest that spasticity contributed to postural dyscontrol during quiet standing. Interestingly, spasticity has also been suggested to be functionally beneficial for stroke-survivors with lower-limb paresis, as lower-limb spasticity may aid in limb extension and body weight support during standing (Berger et al., 1984; Dietz and Sinkjaer, 2007). The present work seeks to better understand the influence of lower-limb spasticity on standing balance control by characterising inter-limb temporal and spatial synchronisation of COP displacements.

Discrepancy regarding the effect of spasticity on stability control may result from the fact that previous studies examining the relationship between lower-limb spasticity and standing balance used either observational balance scales or stabilometric analyses using only a single force platform. These particular measures of balance control may have lacked the sensitivity to reveal the specific control challenges faced by stroke-survivors with lower-limb spasticity. Given that stroke and its consequent effects tend to be lateralised, we believe the need exists to quantify the extent of limb-specific dysconstrol. Previous work employing dual force platforms to quantify individual limb COP has revealed that the non-paretic limb bears more weight and has a larger contribution to postural control than the paretic limb (de Haart et al., 2004; Genthon et al., 2008; Mansfield et al., 2011; Mizrahi et al., 1989; Roerdink et al., 2009). Cross-correlational analysis has also been used to understand the inter-limb temporal synchrony of COP displacements. Among healthy individuals, high direct and inverse correlations have been observed in the anteroposterior (AP) and mediolateral (ML) directions, respectively, indicating that the two limbs work synergistically to regulate stability during quiet standing (Mochizuki et al., 2005; Winter et al., 1993). Relative to healthy controls, a reduction in inter-limb temporal synchrony has been noted among stroke-survivors (Mansfield et al., 2011), which has subsequently been suggested to be a salient marker of limb impairment and balance control (Mansfield et al., 2012b). As previous work has shown measures of limb-specific dyscontrol to be capable of revealing balance control challenges among stroke-survivors, analyses of both the inter-limb temporal synchrony and spatial symmetry of COP displacements may possess the sensitivity to discriminate the additional challenges to stability control faced by stroke-survivors with lower-limb spasticity. This work sought to characterise the extent of limb-specific dyscontrol among individuals with spasticity as an initial step in advancing the understanding of the role of spasticity in the regulation of stability following stroke. While we recognise that the balance control system has been suggested to be a time-varying nonlinear dynamical system (Cavanaugh et al., 2005; Ting et al., 2009), this initial paper focussed primarily on three clinically applicable measures of standing balance control, which we believed would be influenced by the presence of spasticity: a) weight-bearing symmetry; b) inter-limb COP temporal synchrony; c) inter-limb COP spatial symmetry.

Given previous work suggesting that lower-limb spasticity may facilitate body weight support during standing (Berger et al., 1984; Dietz and Sinkjaer, 2007), it was hypothesized that patients with spasticity would exhibit improved weight-bearing symmetry relative to individuals without spasticity, expressed as the ratio of the average vertical force between the affected and less-affected limbs.

Despite potential improvements in weight-bearing symmetry, we believed spasticity and associated difficulties that influence muscle force control (i.e. disruption of agonist-antagonist sequencing, passive tissue stiffness or co-contraction) would impede the typical modulation of net intersegmental moments and consequent COP displacements necessary for stability control, thereby interfering with inter-limb COP temporal synchrony. As such, it was hypothesized that lower-limb spasticity would further interfere with the temporal synchronisation of individual limb AP and ML COP displacements relative to individuals without spasticity, as quantified by reduced cross-correlation coefficients at zero phase-lag. We also hypothesized that lower-limb spasticity would induce a phase-delay in the COP displacement of the affected limb, as quantified by the peak of the cross-correlation function and its associated timing. We expected to observe no significant differences between groups in the peak of the cross-correlation function, while we expected to observe increased phase-lag among individuals with spasticity, suggesting that the challenges to stability control associated with lower-limb spasticity are reflected primarily by difficulties in altering COP position with appropriate timing.

Finally, pertaining to inter-limb COP spatial symmetry, we believed that lower-limb spasticity would reduce the magnitude of COP displacements from the affected limb, thereby diminishing its contribution to standing balance control. We expected to observe reduced AP and ML root-mean-square (RMS) ratios among individuals with spasticity, expressed as the ratio of the RMS COP displacements between affected and less-affected limbs. Consistent with previous work (Cakar et al., 2010; Nardone et al., 2001), we expected to observe no differences between groups when examining the RMS of the net-COP displacements in either AP or ML directions, highlighting the potential for balance compensations by the less-affected limb and the necessity for dual force platforms to detect the specific balance control challenges associated with spasticity. Portions of this study have been previously presented as an abstract (Mochizuki et al., 2012).

2. Methods

2.1. Participants

Data were obtained from the Heart and Stroke Foundation Centre for Stroke Recovery Longitudinal Database. This database amalgamates data collected from patients assessed at one of four hospitals, as part of a larger examination of the recovery of sensorimotor and cognitive function post-stroke. Participants were included in the current analysis if they had been assessed for spasticity using the Modified Ashworth Scale (MAS) (Bohannon and Smith, 1987), were assessed 90 days or more after first-ever stroke, could stand independently (for 30 s) and could understand and follow instructions. Each lower-limb was assessed with the MAS and the 'leg' and 'foot' components of the Chedoke-McMaster Stroke Assessment (CMSA) (Gowland et al., 1993) — participants were excluded if they had bilateral spasticity, or bilateral motor impairment, respectively. None of the participants were using an ankle-foot orthoses at the time of testing. None of the participants without spasticity exhibited contracture at any of the joints of the lower-limb, although one participant exhibited clonus of the soleus and gastrocnemius. Of the 131 stroke-survivors assessed with the MAS, 85 met these criteria. Given the primary role of the ankle musculature in regulating anteroposterior standing balance, an additional 3 participants with spasticity were excluded, as they exhibited only knee extensor spasticity, without spasticity of the soleus or gastrocnemius, leaving a total participant pool of 82 individuals. Individuals were categorised into two groups: those with lower-limb spasticity ($n = 19$) and those without lower-limb spasticity ($n = 63$). The group of patients with lower-limb spasticity exhibited ankle plantarflexor (gastrocnemius/soleus) spasticity (MAS > 0), either with or without knee extensor (rectus femoris/vasti) spasticity. The group of patients without spasticity had MAS scores of 0 for all lower-limb muscles tested. This study received ethical approval from the Research Ethics Board at each institution. All participants provided informed consent in accordance with established institutional policies. Table 1 provides detailed participant characteristics.