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Coactivation of ankle muscles during stance phase of gait in patients with lower limb hypertonia after acquired brain injury

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HIGHLIGHTS

- After stroke or traumatic brain injury, the magnitude of coactivation between tibialis anterior (TA) and medial gastrocnemius (MG) muscles is increased in the more-affected limb during initial and late double support.
- The duration of TA–MG coactivation is prolonged in the less-affected limb also during initial and late double support.
- The correlation between coactivation and stretch velocity-dependent increase in MG activity in TBI patients suggests central contribution to MG output during its lengthening in stance phase of gait.

ABSTRACT

Objective: Examine (1) coactivation between tibialis anterior (TA) and medial gastrocnemius (MG) muscles during stance phase of gait in patients with moderate-to-severe resting hypertonia after stroke or traumatic brain injury (TBI) and (2) the relationship between coactivation and stretch velocity-dependent increase in MG activity.
Methods: Gait and surface EMG were recorded from patients with stroke or TBI (11 each) and corresponding healthy controls ($n = 11$) to determine the magnitude and duration of TA–MG coactivation. The frequency and gain of *positive* (>0) and *significant positive* ($p < 0.05$) EMG–lengthening velocity (EMG–LV) slope in MG were related to coactivation parameters.
Results: The magnitude of coactivation was increased on the more-affected (MA) side, whereas the duration was prolonged on the less-affected (LA) side of both stroke and TBI patients. The difference reached significance during the initial and late double support. The magnitude of coactivation positively correlated with the gain of *significant positive* EMG–LV slope in TBI patients.
Conclusions: Increased coactivation between TA and MG during initial and late double support is a unique feature of gait in stroke and TBI patients with muscle hypertonia.
Significance: Increased coactivation may represent an adaptation to compensate for impaired stability during step transition after stroke and TBI.

1. Introduction

Spasticity is commonly defined as 'a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex as one component of the upper motor neuron syndrome' (Lance, 1980). Other features of spasticity as a movement disorder include abnormal muscle coactivation, clonus, muscle spasms and loss of coordination.

In the preliminary study (Chow et al., 2009), we examined how closely an increase in electromyographic (EMG) activity of the medial gastrocnemius (MG) is related to lengthening velocity (LV) during stance phase of gait in chronic stroke and traumatic brain injury (TBI) patients with moderate-to-severe resting hypertonia. We found that EMG–LV relation, as one component of spastic movement disorder, is not exaggerated in patients during stance or related to resting muscle hypertonia. In the current study, we focus on coactivation between tibialis anterior (TA) and MG muscles during stance phase of gait.

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Increased coactivation in the lower limb muscles during gait is well-documented in cerebral palsy (Shiavi et al., 1987; Berger et al., 1982; Unnithan et al., 1996; Damiano et al., 2000; Manganotti et al., 2007) and stroke (Knutsson and Richards, 1979). Lamontagne et al. (2000) found that the duration of coactivation between TA and MG was shorter on the paretic side during single support and longer on the non-paretic side during initial and late double support within six months of stroke compared to healthy controls. However, Den Otter et al. (2007) did not find a significant difference in the duration of TA–MG coactivation between patients and controls, or between two sides in patients 3–21 months after stroke. Addressing this controversy is of interest since increased coactivation during gait may be viewed as a persistent impairment or useful adaptation (Arene and Hidler, 2009). While some coactivation of ankle muscles during stance may stiffen a joint and provide stability, excess coactivation can lead to inefficient gait (Falconer and Winter, 1985).

The first objective of this study was to examine coactivation between TA and MG muscles during stance phase of gait in chronic stroke and TBI patients with moderate-to-severe resting hypertonia. TA–MG coactivation was chosen over TA-soleus coactivation so we could compare our findings with published results (Lamontagne et al., 2000, 2002; Den Otter et al., 2007). We hypothesized that the magnitude and duration of TA–MG coactivation will be increased in patients compared to controls. The second objective was to determine whether TA–MG coactivation is related to the EMG–LV relation in MG muscle and resting muscle hypertonia (Ashworth score). Preliminary results have been reported in abstract form (Stokic et al., 2009).

2. Methods

2.1. Subjects

The sample included 11 stroke patients (mean ± SD age 41 ± 9 years, height 169 ± 13 cm, body mass 95 ± 15 kg, time post-onset 45 ± 46 months, three males), 11 TBI patients (27 ± 11 years, 175 ± 114 cm, 79 ± 24 kg, 38 ± 29 months, seven males), and two groups of age- and sex-matched healthy control subjects ($n = 11$ each). The inclusion criteria for patients were: (1) significant increase in muscle tone that impairs function or care, (2) ability to walk safely for at least 10 m at a speed faster than 10 cm/s with or without assistive devices, and (3) consent to undergo laboratory gait evaluation. Eight stroke and four TBI patients wore a short, non-rigid polypropylene ankle–foot orthosis on their more-affected side to prevent foot drop during gait. Patients with evidence of ankle clonus during the stance phase of gait were excluded to eliminate confounding effect of rhythmic EMG pattern to MG activity. Each subject attended one data collection session and signed the informed consent approved by the institutional review board for human research.

2.2. Experimental setup and protocol

Wearing their own footwear, patients walked at a self-selected *free* speed whereas controls walked at a self-selected *very slow* speed 8–10 times along a 7 m-long walkway equipped with five imbedded forceplates (Type 4060, 60 × 40 cm, Bertec Corp., Columbus, OH 43229). This protocol provides more appropriate comparison between patients with acquired brain injury and controls (Chow et al., 2010) and differentiates coactivation associated with impairments from walking at a slow speed (Van Hedel et al., 2006). Gait kinematics were collected with eight digital Hawk cameras at 60 Hz (Motion Analysis Corporation, Santa Rosa, CA). Passive spherical reflective markers were affixed to body landmarks according to the Helen Hayes marker system (Kadaba et al., 1990).

Bipolar EMG electrodes with built-in preamplification (model MA411, gain 20, 2 cm center-to-center distance, input impedance $>10^{10}\ \Omega$, CMRR >100 dB, Motion Lab Systems, Inc., Baton Rouge, LA) were attached to bilateral MG and TA (Cram and Kasman, 1998). Signals were further amplified by an EMG system (model MA300, input impedance 31 KΩ, CMRR >50 dB, Motion Lab Systems, Inc.) before 12-bit A/D conversion (sampling rate 1200 Hz). Resting EMG was collected for 5 s in sitting position before walking trials.

Before recording gait, a physical therapist assessed muscle hypertonia in patients in the bilateral hip flexors and extensors, knee flexors and extensors, and ankle plantar flexors, using the modified Ashworth scale (AS) (Bohannon and Smith, 1987) (Table 1). The average AS was used to identify more-affected (MA) and less-affected (LA) sides. When the difference was less than 0.4 or data were not available, the side with longer stance time was considered the LA side (Ochi et al., 1999).

2.3. Data reduction

Kinematic data were processed with OrthoTrak Gait Analysis software (Motion Analysis Corporation) and initial foot contact and toe-off events were identified for each gait cycle. A gait cycle was defined by two consecutive initial contacts of the same foot. Footfall instants were used to compute durations of different phases of a gait cycle. Raw EMG signals were baseline-adjusted, filtered (recursive digital Matlab Elliptic filter, band pass 10–500 Hz), and full-wave rectified.

The magnitude of MG–TA coactivation was quantified with coactivation index (CoI). The average rectified EMG at rest was subtracted from the rectified EMG during each gait cycle for MG and TA muscles, respectively. The adjusted EMG was then normalized to the average amplitude of each muscle over the entire gait cycle (Yang and Winter, 1984). CoI (AvgEMG) was calculated from the amplitude-normalized EMG by dividing the area of MG–TA overlap by the overlap duration (Unnithan et al., 1996) (Fig. 1a). Since CoI represents a relative magnitude of EMG overlap, it allows comparison of coactivation between different muscles and across subjects (Granata et al., 2000).

The duration of coactivation (CoD) between TA and MG was computed after the EMG signals were rectified and smoothed with a low pass bi-directional 2nd order Butterworth filter (cut-off frequency 10 Hz). A muscle was considered active during gait when the EMG exceeded 3 SDs of the resting average (Pierce et al., 2007). CoD was calculated as the duration of overlap between active TA and MG muscles and expressed as a percent of the phase duration (Fig. 1b).

CoI and CoD were derived for the entire stance and its components, namely, initial double support, single support, and late double support, as well as for the segments of MG lengthening during stance. The respective values for CoI and CoD were averaged across all gait cycles for each subject and submitted to statistical analysis.

Using the approach proposed by Lamontagne et al. (2001), EMG–LV relationship of MG during stance was determined for each gait cycle (Chow et al., 2009). The muscle–tendon length of MG during gait was derived from knee and ankle joint angles (Hawkins and Hull, 1990). Using changes in muscle–tendon length to define shortening/lengthening portions of stance was deemed appropriate since in this phase of gait the profiles of MG muscle–tendon length and fascicle length are largely similar (Ishikawa et al., 2005). MG shortening/lengthening velocities were computed as the first derivatives of length and EMG levels of MG were normalized to its peak value during stance. Linear regression was fitted between normalized EMG and the corresponding LV data points for stance phase of each gait cycle. The gain (steepness) of EMG–LV slope and goodness of linear fit (p-value) were determined. Both