Clinical Biomechanics 30 (2015) 960–965

Contents lists available at ScienceDirect

Clinical Biomechanics

journal homepage: www.elsevier.com/locate/clinbiomech

Ankle anticipatory postural adjustments during gait initiation in healthy and post-stroke subjects

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ARTICLE INFO

Article history:
Received 27 January 2015
Accepted 5 July 2015

Keywords:
Gait initiation
Anticipatory postural adjustments
Stroke
Ankle muscles
Centre of pressure

ABSTRACT

Background: Anticipatory postural adjustments during gait initiation have an important role in postural stability but also in gait performance. However, these first phase mechanisms of gait initiation have received little attention, particularly in subcortical post-stroke subjects, where bilateral postural control pathways can be impaired. This study aims to evaluate ankle anticipatory postural adjustments during gait initiation in chronic post-stroke subjects with lesion in the territory of middle cerebral artery.

Methods: Eleven subjects with post-stroke hemiparesis with the ability to walk independently and twelve healthy controls participated in this study. Bilateral electromyographic activity of tibialis anterior, soleus and medial gastrocnemius was collected during gait initiation to assess the muscle onset timing, period of activation/deactivation and magnitude of muscle activity during postural phase of gait initiation. This phase was identified through centre of pressure signal.

Findings: Post-stroke group presented only half of the tibialis anterior relative magnitude observed in healthy subjects in contralesional limb ($t = 2.38$, $P = 0.027$) and decreased soleus deactivation period (contralesional limb, $t = 2.25$, $P = 0.04$; ipsilesional limb, $t = 3.67$, $P = 0.003$) as well its onset timing (contralesional limb, $t = 3.2$. $P = 0.005$; ipsilesional limb, $t = 2.88$, $P = 0.033$) in both limbs. A decreased centre of pressure displacement backward ($t = 3.45$, $P = 0.002$) and toward the first swing limb ($t = 3.29$, $P = 0.004$) was observed in post-stroke subjects.

Interpretation: These findings indicate that chronic post-stroke subjects with lesion at middle cerebral artery territory present dysfunction in ankle anticipatory postural adjustments in both limbs during gait initiation.

1. Introduction

Gait initiation can be considered a unique and challenging task. The central nervous system uses stable, efficient mechanisms for dealing with the inherent instability during the transition from quiet standing, were all body segments possess only potential energy, to a steady state gait, where the body segments contain not only potential energy, but also kinetic energy, and thus a higher energy state (Miller and Verstraete, 1999). In fact, the initiation of gait is considered to be governed by a motor programme, as stereotyped patterns of activity and invariant relative timing have been demonstrated (Brenière et al., 1987; Brunt et al., 1991; Brunt et al., 1999; Crenna and Frigo, 1991; Elble et al., 1994; Fiolkowski et al., 2002; Shapiro et al., 1981). Inhibition of the tonically active soleus (SOL) followed by activation of the tibialis anterior (TA) early in gait initiation, with invariant relative timing between SOL inhibition and TA activation, has been described in healthy subjects (Crenna and Frigo, 1991; Elble et al., 1994; Jian et al., 1993). These first phase mechanisms of gait initiation, namely Anticipatory Postural Adjustments (APA) (Brenière et al., 1987), enable centre of pressure (CoP) backward displacement (Brunt et al., 1991; Crenna and Frigo, 1991), contributing to postural stability (Massion, 1992; McIlroy and Maki, 1999) and enable the optimum generation of momentum to reach the steady-state gait at the end of the first step (Lepers and Brenière, 1995).

Unlike steady-state gait, gait initiation requires an asymmetric lower limbs role. While the first swing limb is responsible for applying a large vertical force to lift its foot from the ground (Patchay and Gahéry, 2003), the contralateral limb (stance limb) is responsible for body support and for a greater forward propulsion (Brunt et al., 1991; Nissan and Whittle, 1990). These asymmetrical limb requirements may thus provide additional insight about gait impairments in pathologies with asymmetric distribution like stroke. However, gait initiation has received little attention in post-stroke subjects see references (Brunt et al., 1995; Hesse et al., 1997; Kirker et al., 2000; Hwang et al., 2009; Melzer et al., 2009). The few studies available showed impairments in contralesional limb (CONTRA) that lead to a reduced step length and gait velocity and

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http://dx.doi.org/10.1016/j.clinbiomech.2015.07.002
0268-0033/

increased duration of postural phase during gait initiation in acute post-stroke subjects (Kirker et al., 2000; Tokuno and Eng, 2006). Such impairments involve a reduction of the propulsion forces (Tokuno and Eng, 2006), decreased TA (Brunt et al., 1995), adductors and abductors muscle activity associated to later onset latencies (Kirker et al., 2000). Despite a delay in the body's forward acceleration associated to an increased forward push from ipsilesional limb to initiate gait (Hesse et al., 1997), post-stroke subjects prefer the CONTRA limb as the starting leg in most cases (Hesse et al., 1997). Initiating with their CONTRA limb enables these individuals to use the IPSI limb as the main propulsion generator helped by the acceleration of the CONTRA swing limb, leading to a higher speed (Tokuno and Eng, 2006; Gillet et al., 2003). Despite research has been more focused on CONTRA limb, IPSI deficits were also demonstrated in gait initiation both when this limb was the stance limb or the first swing limb (Hesse et al., 1997; Tokuno and Eng, 2006). When post-stroke subjects initiate gait with this limb, the centre of mass (CoM) move forward prior to the initial toe-off (Hesse et al., 1997), when it is used as stance limb it develops a lower anteroposterior force (Tokuno and Eng, 2006).

It has been demonstrated that subjects with stroke in subcortical areas in the territory of the middle cerebral artery (MCA) present dysfunction in the modulation process of CONTRA SOL muscle in various functional tasks (Silva et al., 2012a; Silva et al., 2012b; Cheng et al., 2004) in both limbs, possible as a result of impairment of bilateral ventromedial disposed pathways, and failure in CONTRA TA activation, resultant from lesion in the unilateral disposed lateral cortico-spinal system (Capaday et al., 1999). These deficits could explain bilateral impairments in post-stroke subjects during gait initiation. However, to the best of our knowledge no study evaluated APAs during gait initiation in chronic post-stroke subjects with lesion in the territory of the MCA.

Stroke in this territory typically involve cortical and subcortical areas, or their axons, responsible for the control of APAs (Massion, 1992). The supplementary motor area (Yoshida et al., 2008; Jacobs et al., 2009), premotor cortex (Chang et al., 2010) and pontomedullary reticular formation through brain stem–spinal pathways that may be engaged through motor corticofugal connections (Drew et al., 2004; Kably and Drew, 1998; Matsuyama et al., 2004; Prentice and Drew, 2001; Schepens and Drew, 2004), have an important role in APAs generation.

This study aims to evaluate ankle APAs during gait initiation in chronic post-stroke subjects with lesion in the territory of MCA. Based on neuroanatomic and neurophysiological foundations it can be hypothesised that post-stroke subjects present bilateral decreased modulation of ankle plantar flexors and CONTRA TA activation failure during postural phase of gait initiation.

2. Methods

2.1. Subjects

Eleven patients who had suffered a stroke at least 6 months earlier (6 females, 5 males) and 12 healthy subjects (5 females, 7 males) participated in this study (Table 1). For the subjects with stroke, the mean time between their stroke and the time of inclusion in this study was 26.0 months (SD = 11.3). All subjects suffered an ischemic stroke: 3 of them had suffered an infarction in their left hemisphere, whereas 8 had suffered an infarction in their right hemisphere. To be included, patients were required to: (Miller and Verstraete, 1999) have suffered a first-ever ischemic stroke involving the MCA territory, as revealed by computed tomography, resulting in hemiparesis; (Brenière et al., 1987) have a Fugl–Meyer *(Assessment of* Sensorimotor *Recovery After Stroke* scale) score in the motor subsection below 34; (Brunt et al., 1999) have the ability to walk, with close supervision if necessary, but without physical assistance, as judged by the treating physiotherapist; (Brunt et al., 1991) have the ability to stand with feet apart for 30 s or more; and (Elble et al., 1994) have provided written or verbal informed consent. Patients were excluded for one or more of the following reasons: (Miller and Verstraete, 1999) cognitive deficit that could hinder communication and cooperation (assessed by the Mini-Mental State Examination); (Brenière et al., 1987) history of orthopaedic or neurological (other than stroke) disorders, known to affect walking performance and quiet standing position; (Brunt et al., 1999) history of stroke involving the brainstem or cerebellar areas; and (Brunt et al., 1991) taking medication such has antispasticity medication that could affect motor performance and balance. Gait data of the group of subjects with stroke were compared with data obtained from healthy control subjects. All control group subjects were selected according to the same exclusion criteria applied to the stroke group, as well as being excluded if they had suffered any neurological disorder. The study was approved by the local ethics committee and implemented according to the Declaration of Helsinki.

Table 1
Mean and standard deviation (SD) values of age, height and weight of healthy and post-stroke groups.

Variables	Post-stroke group	Healthy group	p-Value
	Mean (SD)	Mean (SD)	
Age (years)	53.10 (7.58)	44.78 (10.85)	0.054
Height (m)	1.65 (0.11)	1.66 (0.10)	0.775
Body weight (kg)	76.72 (10.29)	68.26 (12.43)	0.102
Self-selected gait speed ($m\ s^{-1}$)	0.42 (0.09)	0.77 (0.10)	<0.001
	$n = 11$	$n = 12$	

2.2. Instrumentation

The values of the vertical (F_z), anteroposterior (F_x) and mediolateral (F_y) components of GRF, as well as the values of the moments of GRF in the frontal (My) and sagital (Mx) planes, were acquired using a force plate[a] at a sampling rate of 100 Hz (FP4060-08 model from Bertec Corporation (USA), connected to a Bertec AM 6300 amplifier [a] and to an analogue board [b], from Qualysis, Inc. (Sweden)).

The activity of Gastrocnemius Medialis (GM), Soleus (SOL) and Tibialis Anterior (TA) of both lower limbs was assessed through electromyography (EMG). The bilateral EMG signal of these muscles was monitored using a bioPLUX[c] research wireless signal acquisition system (Plux Ltda, Portugal). The signals were collected at a sampling frequency of 1000 Hz and were pre-amplified in each electrode and then fed into a differential amplifier with an adjustable gain setting (25–500 Hz; common-mode rejection ratio (CMRR): 110 dB at 50 Hz, input impedance of 100 MΩ and gain of 1000). Self-adhesive silver chloride EMG electrodes were used in a bipolar configuration and with a distance of 20 mm between detection surface centres. The skin impedance was measured with an Electrode Impedance Checker[d] (Noraxon USA, Inc.).

The force plate signals were analysed with the Acqknowledge software (Biopac Systems, Inc., USA). All subjects used standard tennis footwear (1.5 cm heel), in their adequate size, as different kind of footwear leads to different levels of postural stability reflected in centre of pressure oscillation (Nag et al., 2011).

2.3. Procedures

2.3.1. Skin preparation and placement of electrodes

The skin surface of selected muscles of the midbelly and patella was prepared (shaved and then the dead skin cells and non-conductor elements were removed with alcohol and with an abrasive pad) to reduce the electrical resistance to <5000 Ω, the electromyographic electrodes were placed according to anatomic references (Table 2).

2.3.2. Data acquisition

GRF and EMG data were acquired during gait initiation. All individuals were asked to stand as still as possible (Zok et al., 2008), with feet