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

Table 2
Anatomical references to electrode placement. Electrode locations were confirmed by palpation of the muscular belly with the subject in the test position, being the electrodes placed on the most prominent area.

Muscle	Electrode placement
TA	1/3 on the line between the tip of the tibia and the tip of the medial malleolus
GM	Most prominent bulge of the muscle
SOL	2 cm distal to the lower border of the medial gastrocnemius muscle belly and 2 cm medial to the posterior midline of the leg
Ground electrode	Patella centre

at pelvis width, keeping their arms by their sides and to focus on a target 2 m away and at eye level during 30 s (Le Clair and Riach, 1996). After this interval subjects were asked to walk at self-adopted speed over a 5 m walkway, without explicit instructions. If a subject asked which leg to start with, the researcher replied "whatever feels natural for you", as lower limb preference plays an influential role in the control of frontal plane body motion during gait initiation (Dessery et al., 2011). However, subjects were asked to keep the starting leg consistent over all trials (Miller and Verstraete, 1999). A trial was considered valid when the subject performed at least three steps (Mann et al., 1979; Miller and Verstraete, 1996). Each subject performed three trials with rest periods of 60 s between trials (Kitabayashi et al., 2003). All participants from post-stroke group initiated gait with their CONTRA limb.

2.3.3. Data processing

GRF data were low-pass filtered using a fourth-ordered Butterworth filter by using a zero-phase lag with a cutoff frequency of 20 Hz (Cau et al., 2014). The acquired force and moment of force time series of each trial were used to calculate the CoP fluctuation in the AP and ML directions using the following approximation:

$$CoP_{AP} = \frac{M_y}{F_z}, \quad (1)$$

$$CoP_{ML} = \frac{M_x}{F_z} \quad (2)$$

where M_y and M_x are the moments of GRF in the frontal and sagital planes, respectively, and F_z the vertical components of GRF collected with a force plate.

In all subjects the beginning of CoP displacement was observed in the AP direction. As a consequence, time series of CoP displacement in AP direction was used to assess the onset of gait initiation (T_0). The CoP_{AP} backward displacement onset was defined as the beginning of an interval lasting for at least 50 ms when its value was higher than the mean plus 3 SD of CoP_{AP} displacement obtained during upright standing. CoP displacement in AP and ML directions, during postural control phase, was calculated through the difference between maximum CoP backward (first inflection of CoP_{AP}) and toward the swing limb (first inflection of CoP_{ML}) positions and T_0.

The electromyographic signals were filtered using a zero-lag, second-order Butterworth filter with an effective band pass of 20 to 450 Hz, and the root mean square was calculated. The muscle latency was detected in a time window from −450 in relation to T_0 (Santos et al., 2009) to the end of postural phase using a combination of computational algorithms and visual inspection (Di Fabio, 1987). The latency for a specific muscle was defined as the instant lasting for at least 50 ms when its EMG amplitude was higher (activation) or lower (inhibition) than the mean of its baseline value plus 3 standard deviation (SD) (Hodges and Bui, 1996), measured from −500 to −450 ms (Santos et al., 2009). For each TA activation and SOL and GM deactivation periods, the magnitude of electromyographic signal was normalised by baseline values to assess the degree of magnitude modulation of each muscle during APAs in relation to upright standing. The limb that performed the first step was designed as first swing limb and the contralateral limb was designed as stance limb.

2.3.4. Statistical analysis

The acquired data were analysed using the Statistic Package Social Science (SPSS)[e] software from *IBM Company* (USA). Mean and standard deviation were used for descriptive analysis. The Independent Sample T-test was used to compare CoP displacement and bilateral lower limb muscle onset/offset timings, muscle activation/deactivation duration and magnitude between healthy and post-stroke participants. Shapiro–Wilk test and histogram analysis indicated that data was normally distributed. A 0.05 significance level was used for inferential analysis.

3. Results

Generally, lower magnitude levels of activity were observed in both TA and SOL and higher GM activity in post-stroke group regarding the first swing limb and stance limb (Fig. 1). Statistically significant differences were observed in TA of first swing limb ($t = 2.38$, $p = 0.027$) where post-stroke group presented only half of the relative magnitude observed in healthy subjects. Because the magnitude of electromyographic activity during APAs was normalised to values obtained during upright standing, we have compared upright standing SOL, TA and GM

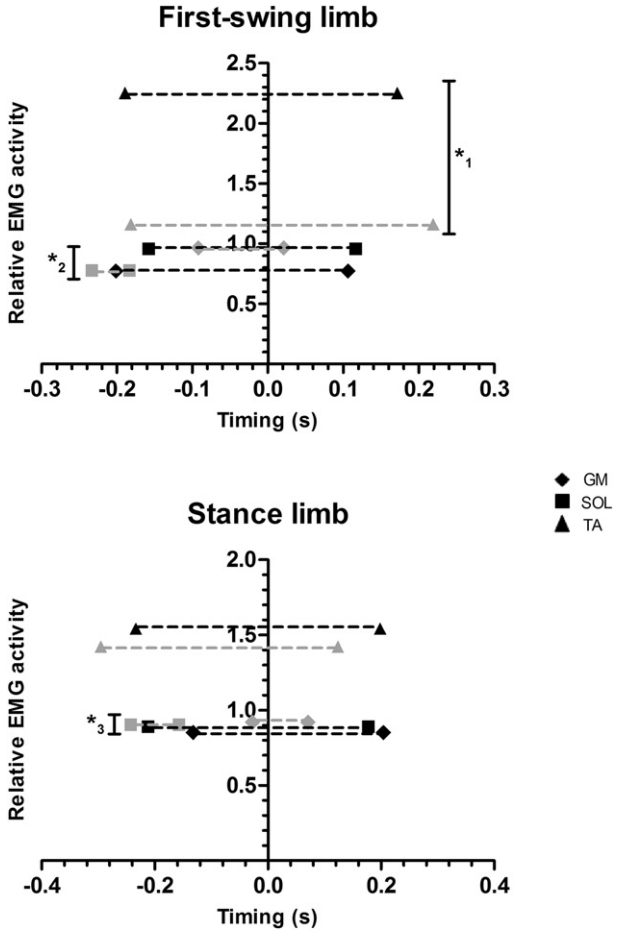

Fig. 1. Representation of activation periods of TA and deactivation periods of SOL and GM calculated from −450 ms in relation to T_0 to the final of postural phase. Gray dashed lines represent values obtained in post-stroke subjects while dark dashed lines represent values obtained in healthy subjects. Statistically significant differences obtained between post-stroke subjects and healthy subjects in TA relative magnitude (*1), in SOL deactivation duration and onset timing in the first swing limb (*2), and in SOL deactivation duration and onset timing in the stance limb (*3) are represented.

magnitude between groups. No significant differences occurred between the IPSI and the CONTRA limbs of post-stroke subjects and healthy subjects.

A tendency to a later onset timing of TA was also observed in post-stroke subjects in the first swing limb and the opposite was observed in stance limb. However, no significant differences were observed in temporal analysis of TA muscle. The differences between groups were more notorious in SOL muscle (Fig. 1), as statistical significant differences occurred in SOL deactivation duration (first swing limb, $t = 2.25$, $p = 0.04$; stance limb, $t = 3.67$, $p = 0.003$) and in its onset timing in both limbs (first swing limb, $t = 3.2$. $p = 0.005$; stance limb, $t = 2.88$, $p = 0.033$).

The results obtained in muscle timing and magnitude were accompanied by a decreased CoP displacement backward and toward the first swing limb in post-stroke subjects compared to healthy subjects. The post-stroke group presented only about half of the CoP displacement observed in healthy subjects for both directions (Fig. 2).

4. Discussion

The purpose of this study was to evaluate ankle APAs during gait initiation in chronic post-stroke subjects with lesion in the territory of MCA. The results obtained confirm our hypothesis that this group of subjects present bilateral SOL modulation impairment and CONTRA TA activation failure during gait initiation. These changes in muscle activation patterns lead to decreased CoP displacement backward and toward the first swing limb. This decreased CoP displacement leads to decreased CoM forward momentum (Jian et al., 1993) and ultimately to a reduction of gait velocity and step length (Brenière et al., 1987; Bensoussan et al., 2006; Cook and Cozzens, 1976). In fact, it has been reported that, in healthy subjects, the amplitude of CoP displacement backward and toward the first swing limb, as well TA magnitude, increases with an increased speed of the intended gait to generate higher forward CoM propulsion (Brenière et al., 1987; Crenna and Frigo, 1991; Lepers and Brenière, 1995; Tokuno and Eng, 2006; Caderby et al., 2014). In the present study, participants were instructed to walk at their comfortable speed. As a consequence, post-stroke participants performed gait with lower speed when compared to healthy participants (Table 1). Based on previous studies, it can be argued that this can result from impairments in APAs during gait initiation. However, no differences in CoP displacement were previously found between healthy and post-stroke participants when healthy participants were instructed to walk with a speed close to the one chosen by the post-stroke group (0.73 m s^{-1}) (Tokuno and Eng, 2006). Based on this, it would be hypothesised that the diferences observed in APAs could result from the lower speed adopted by the post-stroke group and not the reverse. Despite the post-stroke participants of the presented study walked at a slower speed than the post-stroke participants of Tokuno and Eng (2006) study, similar values of CoP displacement were observed. Also, although healthy participants of the present study adopted a walking speed (0.77 m s^{-1}) similar to the slower speed adopted by participants of Tokuno and Eng (2006) study (0.73 m s^{-1}), CoP displacement was close to the one obtained when participants from the latter study walked at their comfortable speed (1.07 m s^{-1}). These findings indicate that more similar CoP displacement values are obtained when subjects walk at their self-selected speed, than when subjects are asked to walk at the same speed. Based on this, it is reasonable to suggest that changes observed in APAs in post-stroke subjects contribute to the decreased gait speed and not the reverse, as they walked at their self-selected speed.

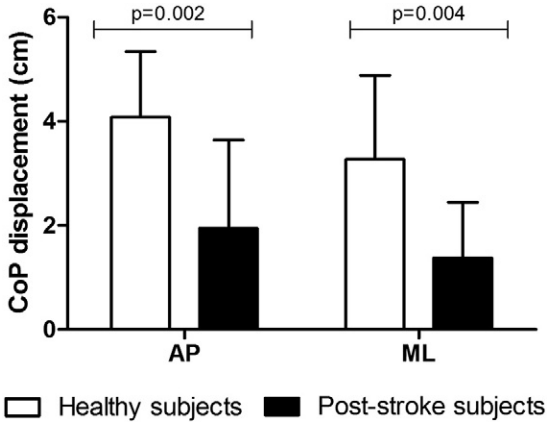

Fig. 2. Mean (bars) and standard deviation (error bars) of CoP displacement backward and toward the first swing limb in healthy and post-stroke subjects.

It should be noted that participants were instructed to initiate gait with their preferential limb and as a result post-stroke subjects initiated gait with their CONTRA limb. This preference has been interpreted as an adaptative strategy to increase forward propulsion (Tokuno and Eng, 2006). The results of the present study demonstrate that post-stroke subjects present not only half of the TA magnitude observed in healthy subjects, as well a decreased SOL inhibition in CONTRA limb. It has been demonstrated that the CoM movement forward and toward the initial stance leg during gait initiation, occurs approximately 300 ms after activation of the tibialis anterior muscle and that, backward CoP displacement begins with an increase in the TA muscles (Elble et al., 1994). The similar TA activation timings obtained are post-stroke subjects and healthy controls in our study, are in accordance with other studies (Ko et al., 2011). The lower magnitude levels of CONTRA TA observed in the present study, together with decreased CONTRA plantarflexor, hip flexor and hip extensor strength (Nadeau et al., 1999; Lindmark and Hamrin, 1995), can explain the reduction of propulsion forces (Tokuno and Eng, 2006), as well the increased duration of postural phase in post-stroke subjects (Kirker et al., 2000; Tokuno and Eng, 2006). This difficulty in modulating activity from quiet standing to gait initiation in CONTRA limb probably results from a deregulation of supplementary motor area (Yoshida et al., 2008; Jacobs et al., 2009) and premotor cortex (Chang et al., 2010). The decreased TA activity can also result from reduced SOL deactivation period through reciprocal inhibition mechanism.

It should be noted that a lesion in the premotor cortex affects the APAs of bilateral lower extremities in step initiation (Chang et al., 2010).These neuroanatomical foundations help understanding the modulation deficit over IPSI SOL muscle observed in the present study. Since forward propulsion is controlled by the unimpaired dorsolateral system, the deficits demonstrated in IPSI anteroposterior force (Tokuno and Eng, 2006) are probably related from impairments in APAs in IPSI SOL muscle during gait initiation. Postural control dysfunction of the IPSI limb has been demonstrated in other functional tasks (Lamontagne et al., 2002; Peterson et al., 2010) and particularly in subjects with sub-cortical injuries located at the internal capsule level (Silva et al., 2012a; Silva et al., 2012b; Sousa et al., 2013). In fact, injuries located at this region are typically associated with dysfunction of the ventral–medial systems, like corticoreticular pathway, and may justify changes in the activity of the IPSI SOL muscle (Matsuyama et al., 2004).

As only one force plate was used and the degree of weight distribution asymmetry was not assessed (Tokuno and Eng, 2006), it would be questioned the possible influence of it on bilateral impairments obtained. A decrease of CoP displacement has been demonstrated in healthy subjects, in the first swing limb, when there is a reduced loading over this limb (Azuma et al., 2007). It has been argued that this asymmetrical weight bearing leads to change in proprioceptive information from cutaneous receptors and Golgi tendon organs, which in turn leads to reduced ankle muscle activity (Hiebert and Pearson, 1999; Dietz et al., 1992; Kavounoudias et al., 1998). The non-existence of significant differences in SOL, TA and GM muscle activity during upright standing between post-stroke and healthy subjects, in the present study, supports the argumentation that changes observed in APAs result

from a dysfunction of ventromedial disposed pathways and not from weight bearing asymmetry. This is also supported by the results obtained by Ko et al. (2011) in post-stroke subjects, as APAs during gait initiation were observed in both asymmetric and symmetric weight bearing conditions (Ko et al., 2011).

5. Conclusion

The results obtained in this study indicate that chronic post-stroke subjects with lesion at MCA territory present dysfunction in ankle APAs in both limbs during gait initiation. CONTRA limb presents failure in modulating SOL inhibition and in activating TA, while IPSI limb presents failure in modulating SOL inhibition. These impairments lead to decreased bilateral backward CoP displacement compromising stability and performance of gait initiation. From a clinical point of view, the results obtained in this study indicate that attention should be given to the postural phase of gait initiation in the rehabilitation of post-stroke subjects in both the IPSI and the CONTRA limbs.

Suppliers

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d. Noraxon USA Inc., 15770 North Greenway-Hayden Loop, Ste 100, Scottsdale, AZ 85260.
e. SPSS Inc., 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.

Conflict of interest

The authors report no conflict of interest.

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