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

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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.

Keywords: gait initiation; anticipatory postural adjustments; stroke; ankle muscles;

1 centre of pressure

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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 (1). In fact, the initiation of gait is considered to be governed by a motor program, as stereotyped patterns of activity and invariant relative timing have been demonstrated (2-8). 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 (5, 7, 9). These first phase mechanisms of gait initiation, namely Anticipatory Postural Adjustments (APA) (2), enable centre of pressure (CoP) backward displacement (4, 7), contributing to postural stability (10, 11) and enable the optimum generation of momentum to reach the steady-state gait at the end of the first step (12).

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 (13), the contralateral limb (stance limb) is responsible for body support and for a greater forward propulsion (4, 14). 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 (15-19)). The few studies available showed impairments in contralesional limb (CONTRA) that lead to a reduced step length and gait velocity and increased duration of postural phase during gait initiation in

acute post-stroke subjects (17, 20). Such impairments involve a reduction of the propulsion forces (20), decreased TA (15), adductors and abductors muscle activity associated to later onset latencies (17). Despite a delay in the body's forward acceleration associated to an increased forward push from ipsilesional limb to initiate gait (16), post-stroke subjects prefer the CONTRA limb as the starting leg in most cases (16). 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 (20, 21). 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 (16, 20). When post-stroke subjects initiate gait with this limb, the center of mass (CoM) move forward prior to the initial toe-off (16), when it is used as stance limb it develops a lower anteroposterior force (20).

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 (22-24) 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 (25). 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 (11). The supplementary motor area (26, 27), premotor cortex (28) and pontomedullary reticular formation through brain stem–spinal

pathways that may be engaged through motor corticofugal connections (29-33), 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: (1) have suffered a first-ever ischemic stroke involving the MCA territory, as revealed by computed tomography, resulting in hemiparesis; (2) have a Fugl-Meyer (Assessment of Sensorimotor Recovery After Stroke scale) score in the motor subsection below 34; (3) have the ability to walk, with close supervision if necessary, but without physical assistance, as judged by the treating physiotherapist; (4) have the ability to stand with feet apart for 30 seconds or more; and (5) have provided written or verbal informed consent. Patients were excluded for one or more of the following reasons: (1) cognitive deficit that could hinder communication and cooperation (assessed by the Mini-Mental State Examination); (2) history of orthopaedic or

neurological (other than stroke) disorders, known to affect walking performance and quiet standing position; (3) history of stroke involving the brainstem or cerebellar areas; and (4) 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.

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 100Hz (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 (1.5cm heel), in their adequate size, as different kind of footwear leads to different levels of postural stability reflected in centre of pressure oscillation (34).

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 <5000Ω, 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 (35), with feet at pelvis width, keeping their arms by their sides and to focus on a target 2 meters away and at eye level during 30 seconds (36). 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 (37). However, subjects were asked to keep the starting leg consistent over all trials (1). A trial was considered valid when the subject performed at least three steps (38, 39). Each subject performed three trials with rest periods of 60 seconds between trials (40). 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 (41). 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:

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

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

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 450Hz, and the root mean square was calculated. The muscle latency was detected in a time window from -450 in relation to T_0 (42) to the end of postural phase using a combination of computational algorithms and visual inspection (43). 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) (44),

measured from -500 to -450 ms (42). 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.

2.3.3 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 (Figure 1). Statistical 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 magnitude between groups. No significant differences occurred between the IPSI and the CONTRA limbs of post-stroke subjects and heathy 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 (Figure 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 (Figure 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 lead to decreased CoM forward momentum (9) and ultimately to a reduction of gait velocity and step length (2, 45, 46). In fact, it has been reported that, in healthy subjects, the amplitude of CoP displacement backwards and towards the first swing limb, as well TA magnitude, increase with an increased speed of the intended gait to generate higher forward CoM propulsion (2, 7, 12, 20, 47). In the present study, participants were instructed to walk at their confortable 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 where instructed to walk with a speed close to the one chosen by the post-stroke group (0.73 $m.s^{-1}$) (20). 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 et al (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 et al (2006) study (0.73 $m.s^{-1}$), CoP displacement was close to the one obtained when participants from the latter study walked at their confortable 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.

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 (20). 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 towards 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 (5). The similar TA activation timings obtained is post-stroke subjects and healthy controls in our study, are in accordance with other studies (48). The lower magnitude levels of CONTRA TA observed in the present study, together with decreased CONTRA plantarflexor, hip flexor and hip extensor strength (49, 50), can explain the reduction of propulsion forces (20), as well the increased duration of postural phase in post-stroke subjects (17, 20). This difficulty in modulating activity from quiet standing to gait initiation in CONTRA limb probably results from a deregulation of supplementary motor area (26, 27) and premotor cortex (28). 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 (28).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 (20) 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 (51, 52) and particularly in subjects with sub-cortical injuries located at the internal capsule level (22, 23, 53). 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 (31).

As only one force plate was used and the degree of weight distribution asymmetry was not assessed (20), 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

(54). 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 (55-57). 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 (48).

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 present 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.

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Suppliers

a. Bertec Corp, 6171 Huntley Rd, Ste J, Columbus, OH 43229.

b. Qualysis AB, Packhusgatan 6, 411 13 Gothenburg, Sweden.

c. Plux wireless biosignals S.A., Av. 5 de Outubro, 70-8_, 1050-059 Lisboa, Portugal.

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.

Figure Captions

Figure 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.

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