

1 **Ankle anticipatory postural adjustments during gait**

2 **initiation in healthy and post-stroke subjects**

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1 **Abstract**

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

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

15 *Findings:* Post-stroke group presented only half of the tibialis anterior relative
16 magnitude observed in healthy subjects in contralesional limb ($t=2.38$, $p=0.027$) and
17 decreased soleus deactivation period (contralesional limb, $t=2.25$, $p=0.04$; ipsilesional
18 limb, $t=3.67$, $p=0.003$) as well its onset timing (contralesional limb, $t=3.2$, $p=0.005$;
19 ipsilesional limb, $t=2.88$, $p=0.033$) in both limbs. A decreased centre of pressure
20 displacement backward ($t=3.45$, $p=0.002$) and toward the first swing limb ($t=3.29$,
21 $p=0.004$) was observed in post-stroke subjects.

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

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

1 centre of pressure

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1. INTRODUCTION

4 Gait initiation can be considered a unique and challenging task. The central
5 nervous system uses stable, efficient mechanisms for dealing with the inherent
6 instability during the transition from quiet standing, where all body segments possess
7 only potential energy, to a steady state gait, where the body segments contain not only
8 potential energy, but also kinetic energy, and thus a higher energy state (1). In fact, the
9 initiation of gait is considered to be governed by a motor program, as stereotyped
10 patterns of activity and invariant relative timing have been demonstrated (2-8).
11 Inhibition of the tonically active soleus (SOL) followed by activation of the tibialis
12 anterior (TA) early in gait initiation, with invariant relative timing between SOL
13 inhibition and TA activation, has been described in healthy subjects (5, 7, 9). These first
14 phase mechanisms of gait initiation, namely Anticipatory Postural Adjustments (APA)
15 (2), enable centre of pressure (CoP) backward displacement (4, 7), contributing to
16 postural stability (10, 11) and enable the optimum generation of momentum to reach the
17 steady-state gait at the end of the first step (12).

18 Unlike steady-state gait, gait initiation requires an asymmetric lower limbs role.
19 While the first swing limb is responsible for applying a large vertical force to lift its foot
20 from the ground (13), the contralateral limb (stance limb) is responsible for body
21 support and for a greater forward propulsion (4, 14). These asymmetrical limb
22 requirements may thus provide additional insight about gait impairments in pathologies
23 with asymmetric distribution like stroke. However, gait initiation has received little
24 attention in post-stroke subjects (see references (15-19)). The few studies available
25 showed impairments in contralesional limb (CONTRA) that lead to a reduced step
26 length and gait velocity and increased duration of postural phase during gait initiation in

1 acute post-stroke subjects (17, 20). Such impairments involve a reduction of the
2 propulsion forces (20), decreased TA (15), adductors and abductors muscle activity
3 associated to later onset latencies (17). Despite a delay in the body's forward
4 acceleration associated to an increased forward push from ipsilesional limb to initiate
5 gait (16), post-stroke subjects prefer the CONTRA limb as the starting leg in most cases
6 (16). Initiating with their CONTRA limb enables these individuals to use the IPSI limb
7 as the main propulsion generator helped by the acceleration of the CONTRA swing
8 limb, leading to a higher speed (20, 21). Despite research has been more focused on
9 CONTRA limb, IPSI deficits were also demonstrated in gait initiation both when this
10 limb was the stance limb or the first swing limb (16, 20). When post-stroke subjects
11 initiate gait with this limb, the center of mass (CoM) move forward prior to the initial
12 toe-off (16), when it is used as stance limb it develops a lower anteroposterior force
13 (20).

14 It has been demonstrated that subjects with stroke in subcortical areas in the
15 territory of the middle cerebral artery (MCA) present dysfunction in the modulation
16 process of CONTRA SOL muscle in various functional tasks (22-24) in both limbs,
17 possible as a result of impairment of bilateral ventromedial disposed pathways, and
18 failure in CONTRA TA activation, resultant from lesion in the unilateral disposed
19 lateral cortico-spinal system (25). These deficits could explain bilateral impairments in
20 post-stroke subjects during gait initiation. However, to the best of our knowledge no
21 study evaluated APAs during gait initiation in chronic post-stroke subjects with lesion
22 in the territory of the MCA.

23 Stroke in this territory typically involve cortical and subcortical areas, or their
24 axons, responsible for the control of APAs (11). The supplementary motor area (26, 27),
25 premotor cortex (28) and pontomedullary reticular formation through brain stem–spinal

1 pathways that may be engaged through motor corticofugal connections (29-33), have an
2 important role in APAs generation.

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

8 **2. METHODS**

9 *2.1 Subjects*

10 Eleven patients who had suffered a stroke at least 6 months earlier (6 females, 5
11 males) and 12 healthy subjects (5 females, 7 males) participated in this study (Table 1).
12 For the subjects with stroke, the mean time between their stroke and the time of
13 inclusion in this study was 26.0 months (SD = 11.3). All subjects suffered an ischemic
14 stroke: 3 of them had suffered an infarction in their left hemisphere, whereas 8 had
15 suffered an infarction in their right hemisphere. To be included, patients were required
16 to: (1) have suffered a first-ever ischemic stroke involving the MCA territory, as
17 revealed by computed tomography, resulting in hemiparesis; (2) have a Fugl-Meyer
18 (Assessment of Sensorimotor Recovery After Stroke scale) score in the motor
19 subsection below 34; (3) have the ability to walk, with close supervision if necessary,
20 but without physical assistance, as judged by the treating physiotherapist; (4) have the
21 ability to stand with feet apart for 30 seconds or more; and (5) have provided written or
22 verbal informed consent. Patients were excluded for one or more of the following
23 reasons: (1) cognitive deficit that could hinder communication and cooperation
24 (assessed by the Mini-Mental State Examination); (2) history of orthopaedic or

1 neurological (other than stroke) disorders, known to affect walking performance and
2 quiet standing position; (3) history of stroke involving the brainstem or cerebellar areas;
3 and (4) taking medication such as antispasticity medication that could affect motor
4 performance and balance. Gait data of the group of subjects with stroke were compared
5 with data obtained from healthy control subjects. All control group subjects were
6 selected according to the same exclusion criteria applied to the stroke group, as well as
7 being excluded if they had suffered any neurological disorder. The study was approved
8 by the local ethics committee and implemented according to the Declaration of
9 Helsinki.

10 *2.2 Instrumentation*

11 The values of the vertical (F_z), anteroposterior (F_x) and mediolateral (F_y)
12 components of GRF, as well as the values of the moments of GRF in the frontal (M_y)
13 and sagittal (M_x) planes, were acquired using a force plate^a at a sampling rate of 100Hz
14 (FP4060-08 model from Bertec Corporation (USA), connected to a Bertec AM 6300
15 amplifier^a and to an analogue board^b, from Qualysis, Inc. (Sweden)).

16 The activity of Gastrocnemius Medialis (GM), Soleus (SOL) and Tibialis Anterior
17 (TA) of both lower limbs was assessed through electromyography (EMG). The bilateral
18 EMG signal of these muscles was monitored using a bioPLUX^c research wireless signal
19 acquisition system (Plux Ltda, Portugal). The signals were collected at a sampling
20 frequency of 1000 Hz and were pre-amplified in each electrode and then fed into a
21 differential amplifier with an adjustable gain setting (25 - 500 Hz; common-mode
22 rejection ratio (CMRR): 110 dB at 50 Hz, input impedance of 100 M Ω and gain of
23 1000). Self-adhesive silver chloride EMG electrodes were used in a bipolar
24 configuration and with a distance of 20 mm between detection surface centres. The skin

1 impedance was measured with an Electrode Impedance Checker^d (Noraxon USA, Inc.).

2 The force plate signals were analysed with the Acqknowledge software (Biopac
3 Systems, Inc., USA). All subjects used standard tennis footwear (1.5cm heel), in their
4 adequate size, as different kind of footwear leads to different levels of postural stability
5 reflected in centre of pressure oscillation (34).

6 *2.3 Procedures*

7 2.3.1 Skin preparation and placement of electrodes

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

12 2.3.2 Data acquisition

13 GRF and EMG data were acquired during gait initiation. All individuals were
14 asked to stand as still as possible (35), with feet at pelvis width, keeping their arms by
15 their sides and to focus on a target 2 meters away and at eye level during 30 seconds
16 (36). After this interval subjects were asked to walk at self-adopted speed over a 5 m
17 walkway, without explicit instructions. If a subject asked which leg to start with, the
18 researcher replied “whatever feels natural for you”, as lower limb preference plays an
19 influential role in the control of frontal plane body motion during gait initiation (37).
20 However, subjects were asked to keep the starting leg consistent over all trials (1). A
21 trial was considered valid when the subject performed at least three steps (38, 39). Each
22 subject performed three trials with rest periods of 60 seconds between trials (40). All
23 participants from post-stroke group initiated gait with their CONTRA limb.

24 2.3.3 Data processing

1 GRF data were low-pass filtered using a fourth-ordered Butterworth filter by
2 using a zero-phase lag with a cutoff frequency of 20 Hz (41). The acquired force and
3 moment of force time series of each trial were used to calculate the CoP fluctuation in
4 the AP and ML directions using the following approximation:

$$5 \quad \text{CoP}_{\text{AP}} = \frac{M_y}{F_z}, \quad (1)$$

$$6 \quad \text{CoP}_{\text{ML}} = \frac{M_x}{F_z} \quad (2)$$

7
8 where M_y and M_x are the moments of GRF in the frontal and sagittal planes,
9 respectively, and F_z the vertical components of GRF collected with a force plate.

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

18 The electromyographic signals were filtered using a zero-lag, second-order
19 Butterworth filter with an effective band pass of 20 to 450Hz, and the root mean square
20 was calculated. The muscle latency was detected in a time window from -450 in relation
21 to T_0 (42) to the end of postural phase using a combination of computational algorithms
22 and visual inspection (43). The latency for a specific muscle was defined as the instant
23 lasting for at least 50 ms when its EMG amplitude was higher (activation) or lower
24 (inhibition) than the mean of its baseline value plus 3 standard deviation (SD) (44),

1 measured from -500 to -450 ms (42). For each TA activation and SOL and GM
2 deactivation periods, the magnitude of electromyographic signal was normalised by
3 baseline values to assess the degree of magnitude modulation of each muscle during
4 APAs in relation to upright standing. The limb that performed the first step was
5 designed as first swing limb and the contralateral limb was designed as stance limb.

6 *2.3.3 Statistical Analysis*

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

14 **3. RESULTS**

15 Generally, lower magnitude levels of activity were observed in both TA and SOL
16 and higher GM activity in post-stroke group regarding the first swing limb and stance
17 limb (Figure 1). Statistical significant differences were observed in TA of first swing
18 limb ($t=2.38$, $p=0.027$) where post-stroke group presented only half of the relative
19 magnitude observed in healthy subjects. Because the magnitude of electromyographic
20 activity during APAs was normalised to values obtained during upright standing, we
21 have compared upright standing SOL, TA and GM magnitude between groups. No
22 significant differences occurred between the IPSI and the CONTRA limbs of post-
23 stroke subjects and healthy subjects.

1 A tendency to a later onset timing of TA was also observed in post-stroke subjects
2 in the first swing limb and the opposite was observed in stance limb. However, no
3 significant differences were observed in temporal analysis of TA muscle. The
4 differences between groups were more notorious in SOL muscle (Figure 1), as statistical
5 significant differences occurred in SOL deactivation duration (first swing limb, $t=2.25$,
6 $p=0.04$; stance limb, $t=3.67$, $p=0.003$) and in its onset timing in both limbs (first swing
7 limb, $t=3.2$, $p=0.005$; stance limb, $t=2.88$, $p=0.033$).

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

12 **4. DISCUSSION**

13 The purpose of this study was to evaluate ankle APAs during gait initiation in
14 chronic post-stroke subjects with lesion in the territory of MCA. The results obtained
15 confirm our hypothesis that this group of subjects present bilateral SOL modulation
16 impairment and CONTRA TA activation failure during gait initiation. These changes in
17 muscle activation patterns lead to decreased CoP displacement backward and toward the
18 first swing limb. This decreased CoP displacement lead to decreased CoM forward
19 momentum (9) and ultimately to a reduction of gait velocity and step length (2, 45, 46).
20 In fact, it has been reported that, in healthy subjects, the amplitude of CoP displacement
21 backwards and towards the first swing limb, as well TA magnitude, increase with an
22 increased speed of the intended gait to generate higher forward CoM propulsion (2, 7,
23 12, 20, 47). In the present study, participants were instructed to walk at their comfortable
24 speed. As a consequence, post-stroke participants performed gait with lower speed
25 when compared to healthy participants (Table 1). Based on previous studies, it can be

1 argued that this can result from impairments in APAs during gait initiation. However,
2 no differences in CoP displacement were previously found between healthy and post-
3 stroke participants when healthy participants were instructed to walk with a speed
4 close to the one chosen by the post-stroke group ($0.73 \text{ m}\cdot\text{s}^{-1}$) (20). Based on this, it
5 would be hypothesised that the differences observed in APAs could result from the lower
6 speed adopted by the post-stroke group and not the reverse. Despite the post-stroke
7 participants of the presented study walked at a slower speed than the post-stroke
8 participants of Tokuno et al (2006) study, similar values of CoP displacement were
9 observed. Also, although healthy participants of the present study adopted a walking
10 speed ($0.77 \text{ m}\cdot\text{s}^{-1}$) similar to the slower speed adopted by participants of Tokuno et al
11 (2006) study ($0.73 \text{ m}\cdot\text{s}^{-1}$), CoP displacement was close to the one obtained when
12 participants from the latter study walked at their comfortable speed ($1.07 \text{ m}\cdot\text{s}^{-1}$). These
13 findings indicate that more similar CoP displacement values are obtained when subjects
14 walk at their self-selected speed, than when subjects are asked to walk at the same
15 speed. Based on this, it is reasonable to suggest that changes observed in APAs in post-
16 stroke subjects contribute to the decreased gait speed and not the reverse, as they
17 walked at their self-selected speed.

18 It should be noted that participants were instructed to initiate gait with their
19 preferential limb and as a result post-stroke subjects initiated gait with their CONTRA
20 limb. This preference has been interpreted as an adaptative strategy to increase forward
21 propulsion (20). The results of the present study demonstrate that post-stroke subjects
22 present not only half of the TA magnitude observed in healthy subjects, as well a
23 decreased SOL inhibition in CONTRA limb. It has been demonstrated that the CoM
24 movement forward and towards the initial stance leg during gait initiation, occurs
25 approximately 300 ms after activation of the tibialis anterior muscle and that, backward

1 CoP displacement begins with an increase in the TA muscles (5). The similar TA
2 activation timings obtained in post-stroke subjects and healthy controls in our study, are
3 in accordance with other studies (48). The lower magnitude levels of CONTRA TA
4 observed in the present study, together with decreased CONTRA plantarflexor, hip
5 flexor and hip extensor strength (49, 50), can explain the reduction of propulsion forces
6 (20), as well the increased duration of postural phase in post-stroke subjects (17, 20).
7 This difficulty in modulating activity from quiet standing to gait initiation in CONTRA
8 limb probably results from a deregulation of supplementary motor area (26, 27) and
9 premotor cortex (28). The decreased TA activity can also result from reduced SOL
10 deactivation period through reciprocal inhibition mechanism.

11 It should be noted that a lesion in the premotor cortex affects the APAs of bilateral
12 lower extremities in step initiation (28). These neuroanatomical foundations help
13 understanding the modulation deficit over IPSI SOL muscle observed in the present
14 study. Since forward propulsion is controlled by the unimpaired dorsolateral system, the
15 deficits demonstrated in IPSI anteroposterior force (20) are probably related from
16 impairments in APAs in IPSI SOL muscle during gait initiation. Postural control
17 dysfunction of the IPSI limb has been demonstrated in other functional tasks (51, 52)
18 and particularly in subjects with sub-cortical injuries located at the internal capsule level
19 (22, 23, 53). In fact, injuries located at this region are typically associated with
20 dysfunction of the ventral–medial systems, like corticoreticular pathway, and may
21 justify changes in the activity of the IPSI SOL muscle (31).

22 As only one force plate was used and the degree of weight distribution asymmetry
23 was not assessed (20), it would be questioned the possible influence of it on bilateral
24 impairments obtained. A decrease of CoP displacement has been demonstrated in
25 healthy subjects, in the first swing limb, when there is a reduced loading over this limb

1 (54). It has been argued that this asymmetrical weight bearing leads to change in
2 proprioceptive information from cutaneous receptors and Golgi tendon organs, which in
3 turn leads to reduced ankle muscle activity (55-57). The non-existence of significant
4 differences in SOL, TA and GM muscle activity during upright standing between post-
5 stroke and healthy subjects, in the present study, supports the argumentation that
6 changes observed in APAs result from a dysfunction of ventromedial disposed
7 pathways and not from weight bearing asymmetry. This is also supported by the results
8 obtained by Ko et al (2011) in post-stroke subjects, as APAs during gait initiation were
9 observed in both asymmetric and symmetric weight bearing conditions (48).

10 **5. CONCLUSION**

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

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46 **Suppliers**

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

- 1 b. Qualysis AB, Packhusgatan 6, 411 13 Gothenburg, Sweden.
- 2 c. Plux wireless biosignals S.A., Av. 5 de Outubro, 70-8_, 1050-059 Lisboa, Portugal.
- 3 d. Noraxon USA Inc, 15770 North Greenway-Hayden Loop, Ste
- 4 100, Scottsdale, AZ 85260.
- 5 e. SPSS Inc, 233 S Wacker Dr, 11th Fl, Chicago, IL 60606.
- 6
- 7

1

2 **Figure Captions**

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

10

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