Communication

Antagonist Coactivation of Muscles of Ankle and Thigh in Post-Stroke vs. Healthy Subjects during Sit-to-Stand Task

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Abstract: This study aims to analyse the coactivation of antagonist muscles of the thigh and ankle during the sit-to-stand task in post-stroke subjects, specifically during forward and antigravity sub-phases. A group of 18 healthy subjects and another with 18 subjects with a history of stroke participated voluntarily in this study. Bilateral surface electromyography (EMGs) of the soleus, gastrocnemius medialis, tibialis anterior, rectus femoris and biceps femoris muscles were collected synchronously with ground reaction forces (GRF) during the sit-to-stand task. The magnitude of electromyographic (EMG) activity was analysed during forward translation and antigravity sub-phases which were determined through GRF signals. The coactivation was calculated to quantify the degree of antagonist coactivation according to the role of the muscles during the task. Statistically significant values were found between antagonist coactivation on both sub-phases of the sit-to-stand task when comparing healthy and post-stroke subjects (healthy with ipsilesional (IPSI); healthy with contralesional (CONTRA); and healthy with IPSI and with CONTRA limbs) in all muscle pairs analysed ($p < 0.01$), except on thigh muscles ($p > 0.05$), in the antigravity sub-phase. When comparing IPSI with CONTRA sides in post-stroke subjects, no statistically significant differences were found. Increased values of antagonist coactivation were observed in post-stroke subjects compared to healthy subjects (both IPSI and CONTRA limb) in the two sub-phases analysed. The forward sub-phase CONTRA limb showed higher antagonist coactivation compared to IPSI, while in the antigravity sub-phase, IPSI antagonist coactivation was higher than in the CONTRA. In conclusion, post-stroke subjects presented an antagonist coactivation more dysfunctional at the ankle joint muscles compared to the thigh segment. So, it seems that the distal segment could express more accurately the central nervous system dysfunction in post-stroke subjects, despite the need for further studies to achieve a better spatiotemporal understanding of the variability on coactivation levels.

Keywords: stroke; electromyography; antagonist coactivation; postural control; postural tone

1. Introduction

Coordinated muscular activity is essential for postural control to achieve a dynamic interplay between postural orientation and stability. The complex synchronization of muscle activity in a coactivation pattern to provide antigravity postural control during centre-of-mass forward and upward translation has been demonstrated in the sit-to-stand (SitTS) task [1–6]. Muscle coactivation, its variation and its expression [7], is the phenomenon by which the central nervous system coordinates the activity of the antagonist muscle during an agonist muscle action, through the simultaneous activation of agonist and antagonist muscles around a joint [8–11]. The level of muscular coactivation, one part of postural tone expression [7], to be constantly adjusted to different tasks and the related phases depends on reticulospinal output due to their role on postural stability demands. Impaired motor control due to nervous system injury, such as that which occurs in strokes, shows increased levels of muscle coactivation particularly in relation to functional tasks and in body segments that assist in stability [12,13], and it has been expressed by high levels of antagonist coactivation [14].

In the SitTS task, the distal muscles in the leg assume a greater role in the control of ankle postural stability than do the thigh muscles [2,15]. Therefore, the assessment of this functional task would help to confirm the hypothesis that antagonist coactivation will result in more dysfunction at the distal level compared to the thigh level in post-stroke subjects. It can also be expected that both sides would present postural control impairment due to the bilateral disposal of the reticulospinal system [16,17] and its influence on neuromodulation of antagonist coactivation. The agonist role of anterior distal leg muscles in the forward translation sub-phase and its reversal role in the antigravity phase makes these two phases, in a more specific way, crucial to understanding the lack of neuromodulation to ensure antigravity postural stability in post-stroke subjects. Consequently, this study aims to analyse thigh and ankle muscles antagonist coactivation during the SitTS task in post-stroke subjects, specifically during forward and antigravity sub-phases.

2. Materials and Methods

2.1. Participants

A total of 18 post-stroke subjects with a first ischemic stroke episode were recruited for this study (age 71 ± 11.51 years, height 169 ± 9.10 cm, weight 53 ± 9.92 kg, 5 females, time post-stroke 26.7 ± 12.10 months, 10 with the contralesional limb (CONTRA) at the right). The inclusion criteria were: (1) the presence of a lesion in the territory of the middle cerebral artery at the sub-cortical level, confirmed by computerized axial tomography of the brain, (2) a score below 34 in the motor subsection of the Fugl-Meyer Assessment of Sensorimotor Recovery After Stroke Scale and (3) the ability to perform the SitTS sequence independently without losing stability. All subjects with previous history of other neurologic diseases, lower limb surgery or any orthopaedic or rheumatoid conditions that would interfere with the SitTS task were excluded. The 18 healthy subjects, age- and sex-matched, were compared with the control group (age 74 ± 11.81 years, height 168 ± 11.45 cm, weight 53 ± 12.41 kg, 5 females). These subjects were considered sedentary according to the Center for Disease Control for the American College Sports Medicine [18]. In both groups, subjects who did not have sufficient cognitive functioning to understand orders (assessment using the Mini-Mental State Examination) were excluded.

This study was approved by the local ethics committee of the Health School of Porto. All subjects gave their informed consent according to the Declaration of Helsinki.

2.2. Instruments

Ground reaction forces (GRF) were collected from one force platform (FP4060-108; Bertec, Columbus, OH, USA) connected to a BERTEC AM6300 signal amplifier. The bilateral EMG signals from the soleus (SOL), gastrocnemius medialis (GM), tibialis anterior (TA), rectus femoris (RF) and biceps femoris (BF) muscles were monitored using a wireless TrignoTM acquisition system (Delsys Inc., Natick, MA, USA). Pre-amplified bipolar differ-

ential electrodes (Trigno Avanti Sensor model) with a rectangular configuration of two Ag bars in parallel (inter-electrode distance of 10 millimetres) and a gain of 1000 were used to collect the EMGs signal at an acquisition frequency of 1000 Hz. EMGworks software was used to analyse the EMG signal quality. Skin impedance was measured with an electrode impedance checker (Noraxon, USA; Scotsdale, AZ, USA). The EMGs and force platform signals were analysed with MATLAB R2001a (Mathworks^{TM}, Natick, MA, USA).

2.3. Procedures

The skin of both IPSI and CONTRA limbs was prepared through standard procedures (shaving, removing dead skin cells and non-conductor elements with alcohol and with an abrasive pad) to reduce the electrical resistance to a level equal to or less than 5 KΩ, monitored with the electrode impedance checker, before electrode placement in the muscle's mid-belly according to anatomical references [19,20].

All individuals maintained a sitting position with 2/3 of the femur supported on the seat, whose height was adjusted to 100% of the lower leg length. Then, they were asked to stand up at a self-selected speed without using upper limbs, maintaining them comfortably along the body, or moving the feet [21]. At the end of the task, they remained quietly standing. A one-minute rest period was provided between each trial, and sufficient repetitions were performed to obtain three valid trials. In the post-stroke subjects' group, both lower limbs were analysed, while in the control group, just one lower limb was randomly selected.

The raw EMG signal was filtered with a band-pass filter (20 Hz–500 Hz), and the root mean square was calculated with a sliding window of 100 ms. The EMG signals have been normalized to the maximum voluntary contraction of the subjects' different muscles. The signal from the force plate was also filtered using a low pass filter of 10 Hz, and the values were normalized to the weight of each subject. The onset of the task, defined as time zero (T0), and the definition of forward translation and antigravity sub-phases were performed through GRF signals. These events were identified through the antero-posterior component of the GRF (FAP) and the vertical component of the GRF (FV) as represented by the schematic strategy illustrated in Figure 1 [3,22–24].
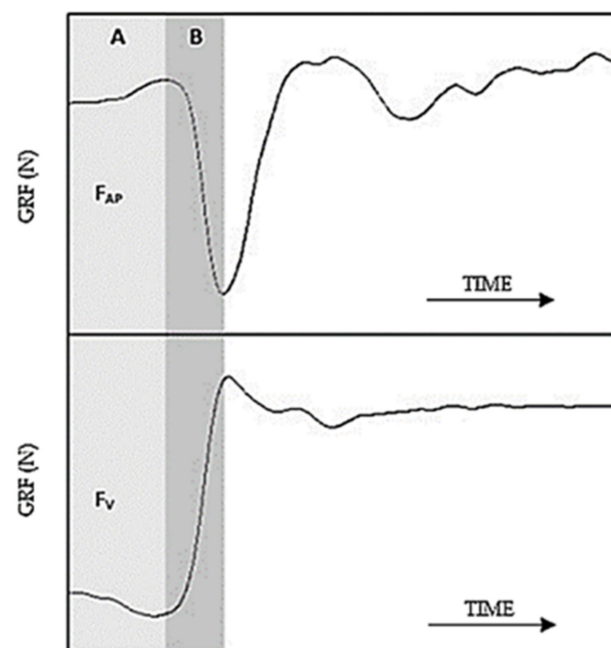

Figure 1. Illustrative identification of forward translation (A) and antigravity sub-phases (B) of the SitTS task through FAP and FV signals.

T0 was defined as the instant when the FAP value was greater or less than the mean of its baseline value plus 2 standard deviations (SD) for at least 50 milliseconds (ms). The forward translation sub-phase was identified in the interval between the end of the previous phase and the maximum value of the FV. As an example, Figure 2 demonstrates a force plate and EMG signals in a 2 s representative window of the SitTS task from two (healthy and stroke) analysed participants.
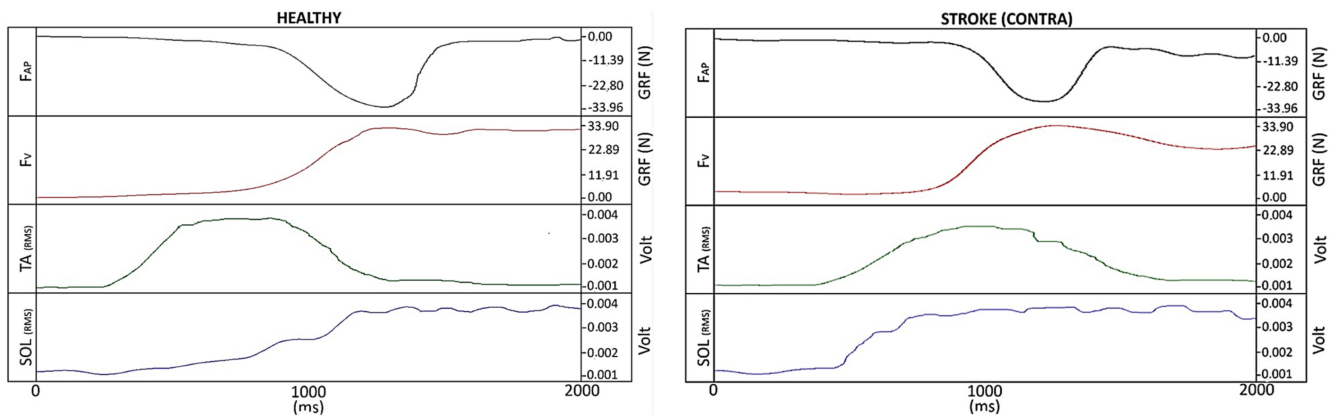

Figure 2. Healthy and Stroke 2 s window force plate and EMG signals. First signal corresponds to FAP, second to FV, third to TA EMG activity and, finally, fourth to SOL EMG activity.

In each sub-phase, the mean of the EMG signal was used to assess antagonist coactivation through the following Formula (1) [25]:

$$C (\%) = \text{antagonist activity} / \text{agonist} + \text{antagonist activity} \times 100 \qquad (1)$$

Muscles were classified according to their role during the task. Specifically, during the forward translation sub-phase, the TA and BF muscles were considered the agonists, while in the antigravity sub-phase, this role was attributed to the SOL, GM and RF muscles. The TA muscle is likely to be the most representative for postural adjustments considering that it is activated early on to ensure foot stability and it assists in centre-of-mass forward displacement [4,26,27]. When acting in a synergistic pattern with coactivation, the more proximal muscles, despite their secondary role in the task [28], play an important role in thigh stability during trunk-forward translation and forward rotation of the tibia over the foot [29]. In the antigravity sub-phase, the SOL and GM muscles play an agonist role in body support and stability against gravity [30], while the proximal muscles behave as prime movers [27].

2.4. Statistics

The data were analysed using MATLAB R2001a (Mathworks™, Natick, MA, USA). To ensure that there were no significant differences between groups (stroke vs. healthy) regarding age, height and weight, the independent *t*-test was used. Since a normal distribution was not verified on all the coactivation variables, the Mann–Whitney test was used to compare antagonist coactivation levels between healthy and IPSI, healthy and CONTRA, and IPSI and CONTRA limbs. The Kruskal–Wallis test, with the Dunn-Bonferroni post hoc test, was applied to compare antagonist coactivation levels between healthy, CONTRA and IPSI limbs. A significance of 0.05 was considered for analysis.

3. Results

The characteristics of the participants are summarized on Table 1.

Table 1. Participants characteristics: mean and standard deviation (SD) values of age, height and weight of control and post-stroke groups, as well as side lesion and evolution time of the post-stroke group.

	Mean (SD)		*p*-Value [1]
	Control Group	**Post-Stroke Group**	
Age (years)	74 (11.81)	71 (11.51)	0.325
Height (cm)	168 (11.45)	169 (9.10)	0.702
Weight (kg)	53 (12.41)	53 (9.92)	0.896
Gender	Female: *n* = 5 Male: *n* = 13	Female: *n* = 5 Male: *n* = 13	
Contralesional side	--	Left: *n* = 8 Right: *n* = 10	--
Time since stroke (months)	--	26.7 (12.10)	--

[1] Independent *t*-test.

No statistically significant values were found; therefore, both groups were comparable (Table 1).

Statistically significant values were found between antagonist coactivation on both sub-phases (A and B) of the SitTS task, when comparing healthy with IPSI, healthy with CONTRA, and healthy, IPSI and CONTRA in all muscle pairs analysed, except on thigh muscles (BF-RF), in the antigravity sub-phase (B). When comparing IPSI with CONTRA in post-stroke subjects, no statistically significant differences were found (Table 2).

Table 2. Median (MED), 25th (P25) and 75th (P75) percentiles of antagonist coactivation (%) in TA-SOL, TA-GM and BF-RF muscle pairs, in the forward (A) and antigravity (B) sub-phases of the SitTS task, in healthy, IPSI and CONTRA.

	SitTS Sub-Phase	Antagonist Coactivation (%)			*p*-Values [#]			*p*-Values [$]
		Healthy MED (P25–P75)	**IPSI MED (P25–P75)**	**CONTRA MED (P25–P75)**	**Healthy vs. IPSI**	**Healthy vs. CONTRA**	**IPSI vs. CONTRA**	**Healthy vs. IPSI vs. CONTRA**
TA-SOL	A	24.06 (19.33; 27.43)	51.91 (42.54; 61.35)	55.69 (51.11; 60.28)	<0.001 *	<0.001 *	0.335	<0.001 *
	B	34.68 (24.22; 44.50)	60.05 (53.17; 65.34)	53.54 (45.57; 64.33)	<0.001 *	<0.001 *	0.788	0.020 *
TA-GM	A	21.47 (15.09; 24.60)	47.81 (41.61; 51.33)	47.84 (46.82; 55.95)	<0.001 *	<0.001 *	0.289	<0.001 *
	B	42.22 (38.77; 48.42)	62.44 (55.92; 68.47)	58.87 (55.87; 63.20)	<0.001 *	0.010 *	0.211	<0.001 *
BF-RF	A	38.90 (31.95; 41.17)	53.08 (49.67; 54.86)	54.29 (50.10; 59.12)	<0.001 *	<0.001 *	0.275	<0.001 *
	B	40.95 (32.65; 50.25)	50.72 (40.03; 57.66)	43.82 (37.36; 65.98)	0.111	0.402	0.477	0.273

* Significative differences. [#] Mann–Whitney Test. [$] Kruskal–Wallis Test.

It stands the fact that, increased values of antagonist coactivation were observed in post-stroke subjects compared to healthy, both on IPSI and CONTRA, in the two sub-phases analysed (Figure 3). In the forward sub-phase (A), CONTRA showed higher antagonist coactivation compared to IPSI, while in the antigravity sub-phase (B), IPSI antagonist coactivation was higher than CONTRA (Figure 3).
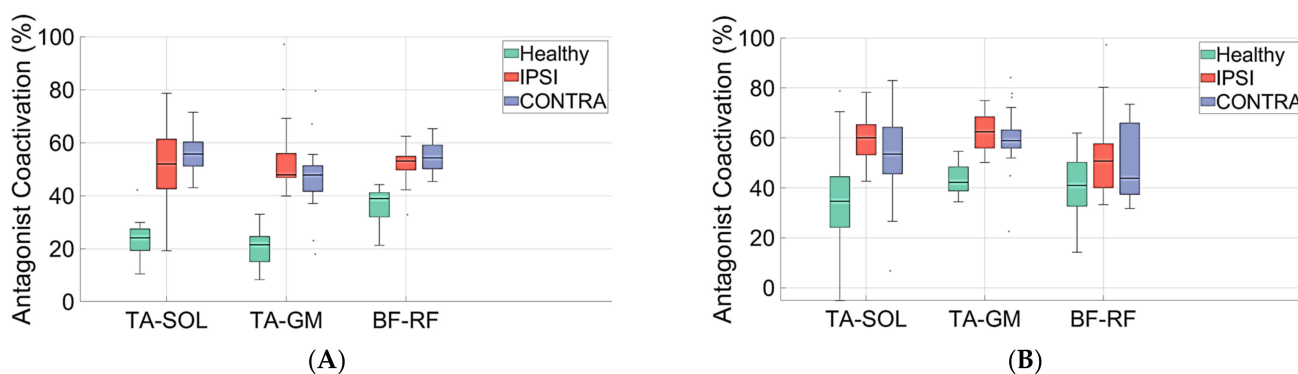
Figure 3. Antagonist coactivation (%) in healthy and post-stroke subjects (CONTRA and IPSI) in both forward (**A**) and antigravity (**B**) sub-phases of the SitTS task in ankle (TA-GM and TA-SOL) and thigh (RF-BF).

4. Discussion

Ankle (TA-SOL and TA-GM muscle pairs) and thigh (RF-BF muscle pairs) antagonist coactivation in forward translation and antigravity sub-phases in the SitTS task in post-stroke subjects exhibit a specific trend toward higher antagonist coactivation. This finding agrees with previous studies involving walking, specifically in the double-support phase of walking [31]. On both, in SitTS and in the double-support phase of walking, antagonist coactivation observed in the two lower limbs seems to reflect a bilateral postural control dysfunction in these subjects. The influence of supra-spinal structures on antagonist coactivation [32] reinforces the value of this synergy in joint stability [33] through precise control of joint position [15].

Previous studies have demonstrated that ankle muscles play a determinant role in influencing proximal muscles activity for stability in both healthy [34,35] and post-stroke subjects [36,37]. Like in walking, SitTS involves sub-phases with specific coactivation patterns across joints and the role of ankle muscles in stability has also been highlighted in elders [38]. The results obtained in the present study showed that it is possible to explore post-stroke subjects' incapacity in performing the SitTS task [39] based on the impairment in the antagonist muscle coactivation modulation against the environment. The results also revealed that during the antigravity sub-phase, IPSI presented the most relevant alteration of antagonist coactivation. These findings are in accordance with other studies in SitTS muscle synergies [37], and also during double support in walking [31], and may be related to the ipsilateral postural control dysfunction often evidenced in lesions affecting the middle cerebral artery territory [26]. In this sub-phase, the plantar flexors act as agonists for stability, and, particularly, the motoneurons of the SOL are more dependent on output from the ventromedial systems than from the reticulospinal system [26,40,41]. Given these assumptions, it is important to think about the mechanisms underlying IPSI vs. CONTRA weakness, while suggesting that IPSI presents the most relevant alteration in antagonist coactivation. Future studies could improve the clarity regarding this area.

Globally, the more variable pattern of ankle muscles antagonist coactivation compared to the thigh in both IPSI and CONTRA legs in post-stroke subjects seems to suggest that the distal ones may present with more accuracy in the postural control impairment in both sides of these subjects when performing functional tasks with higher postural control demand.

However, as no significant differences were observed in the antigravity sub-phase, in the antagonist coactivation of the thigh, it may be thought that during double support, similarly to studies carried out in gait, the role of ankle coactivation for stabilization during movement may have a major influence on the proximal adjustment [34,36]. This might explain the differences obtained where a higher performing postural control demand was expressed. The non-existence of proximal statistically significant differences may demonstrate the lower requirement of postural performance during the task when compared to

distal, which was observed in the results. It would be interesting to complement the linear analysis performed with the combination of a non-linear treatment of the variables under study to explore their behaviour. This might show the trend of existing behaviours in a more detailed spatiotemporal way, describing the variability inherent in human movement in order to better characterize it [11]. The variations expressed during coactivations may also suggest improvement in knowledge regarding the biomechanical characteristics of postural tone and its evaluation [7]. Although considering that all post-stroke subjects analysed presented a lesion in the territory of the middle cerebral artery at the sub-cortical level, the correspondence between coactivation mechanisms and the impairment in specific neurophysiologic regions and related pathways cannot be confirmed in the present study. Future studies are required to confirm this association.

5. Conclusions

Considering that the values of the muscle antagonist coactivation demonstrated a bilateral increased ankle (TA-SOL and TA-GM) antagonist coactivation in both forward and antigravity sub-phases of SitTS tasks in post-stroke subjects, it seems possible to confirm the hypothesis that post-stroke subjects present an antagonist coactivation more dysfunctional distally compared to the thigh segment (BF-RF). So, the distal segment could more accurately express the central nervous system dysfunction in post-stroke subjects despite the need for further studies to achieve a better spatiotemporal understanding of the variability of coactivation levels.

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