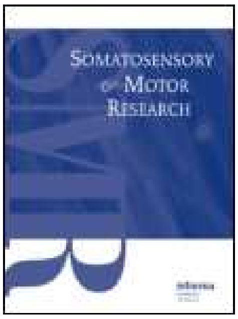

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ORIGINAL ARTICLE

Ankle antagonist coactivation in the double-support phase of walking: Stroke vs. healthy subjects

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Abstract

Introduction: Lesions in ipsilateral systems related to postural control in the ipsilesional side may justify the lower performance of stroke subjects during walking.
Purpose. To analyze bilateral ankle antagonist coactivation during double support in stroke subjects.
Methods: Sixteen (8 females; 8 males) subjects with a first isquemic stroke and 22 controls (12 females; 10 males) participated in this study. The double-support phase was assessed through ground reaction forces and the electromyography of ankle muscles was assessed in both limbs.
Results: The ipsilesional limb presented statistically significant differences from the control when assuming specific roles during double support. The tibialis anterior and soleus pair was the one in which this atypical behavior was more pronounced.
Conclusion: The ipsilesional limb presents a dysfunctional behavior when a higher postural control activity was demanded.

Keywords

Antagonist coactivation ratio, double support, leading limb, trailing limb, stroke, walking

History

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Introduction

Walking performance can be substantially affected after a stroke (Milot et al. 2006; Achache et al. 2010), decreasing the ability to return to work, participate in the community, or perform other daily activities (Higginson et al. 2006).

Most of the literature emphasizes the contralesional deficits in the contralesional side in relation to a hemispheric lesion (CONTRA). It is well known that the CONTRA paretic muscles contribute differently in gait sub-phases when compared to healthy subjects. It has been recently hypothesized that stroke subjects may have postural control impairment also in the ipsilesional side (IPSI), based on the possibility of lesion involving a cortico-reticular system in a stroke involving the territory of the middle cerebral artery (Silva et al. 2012a, 2012b). However, few studies analyzed the ipsilesional limb performance (Peterson et al. 2010; Rosa et al. 2014). This recent hypothesis justifies the possibility of bilateral involvement in stroke subjects.

During walking, a consistent interlimb coordination has been demonstrated in subjects without neurological problems during step-to-step transition (double support) (Sousa et al. 2012a, 2012b). This functional connection between limbs (Anderson and Pandy 2003; Hall et al. 2011; Sousa et al. 2012a; Sousa and Tavares 2012) is supported by studies that found strong crossed effects of group II fibers in motorneuron pools (Corna et al. 1996) and by the role of the reticulospinal system (with IPSI disposal) (Schepens and Drew 2004). Changes in the function of the reticulospinal system during walking can be analyzed through postural control behavior of soleus muscle (SOL), when this muscle acts to provide body support. Actually, it has been demonstrated that subjects with middle artery territory stroke present dysfunctional behavior of this muscle in the IPSI side when its action is related to body support (Silva et al. 2012b). In fact, in a study developed by Sousa et al. (2013), a negative influence of IPSI SOL during loading response, as leading limb (LEAD), over forward propulsive, as trailing limb (TRAIL), muscles of the CONTRA was found (Sousa et al. 2013).

The dynamic relationship between limbs during walking may also be analyzed through the levels of the antagonist coactivation ratio, related to functional position of each limb (TRAIL vs. LEAD) and the subsequent role of each. In fact, during the double-support phase, TRAIL plantar flexors assume mainly the function of body support by SOL (McGowan 2008) and forward progression by gastrocnemius medialis action (Neptune et al. 2001; Anderson and

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Pandy 2003; Hall et al. 2011). The LEAD limb is more related with smooth ground contact and weight acceptance, where the dorsi flexors are the main agonists (Winter 1983). In this sense, it is important to evaluate the ankle antagonist coactivation when the LEAD limb is the IPSI and when the TRAIL is the CONTRA, but also when these roles are reversed. The present study aims to understand the behavior of each limb concerning the ankle antagonist coactivation between muscle pairs when assuming the role of TRAIL and LEAD.

Methods

Participants

Sixteen (8 females; 8 males) subjects with a first isquemic subcortical stroke and 22 healthy subjects (12 females; 10 males) participated in this study (Table I).

For the stroke subjects, the mean time since the injury was 26 months (SD = 9). All subjects suffered an injury in the region of the middle cerebral artery, more specifically in the internal capsule, which was confirmed by brain computerized axial tomography. All subjects included had a score lower than 34 on the Fugl–Meyer Assessment of Sensoriomotor Recovery After Stroke scale (Lamontagne et al. 2002a) and the capacity to perform gait without the use of orthoses.

The stroke subjects have also preserved the cognitive function to understand orders, which was confirmed by assessment using the Mini-Mental State Examination. All potential subjects with previous history of neurologic pathology (e.g., Parkinson, pontine and cerebellar lesions), sensory impairment, diabetes, thrombophlebitis, history of lower limb surgery, and any orthopedic or rheumatoid conditions interfering with walking capacity were excluded, as well as subjects under medication that could affect the motor performance.

Signals collected in the stroke group were compared with those obtained from sedentary healthy subjects, selected according to the same exclusion criteria applied to the stroke group. In addition, potential healthy subjects that had suffered any neurological disorder were excluded.

All participants gave their informed consent according to the Declaration of Helsinki.

Instruments

Ground reaction forces were collected from two force plates (models FP4060-10 and FP4060-08; BERTEC, Columbus, OH, USA) connected to a signal amplifier (model AM6300; BERTEC). The activity of the ankle agonist muscles of TRAIL (namely, gastrocnemius medialis (GM), SOL (Neptune et al. 2001)) and LEAD (namely, tibialis anterior (TA) (Cappellini et al. 2006; Bonell et al. 2007)) were assessed through electromyography. The bilateral electromyographic signal of these muscles was monitored using a bioPLUX research wireless signal acquisition system (PLUX Wireless Biosignals, Arruda dos Vinhos, Portugal). The signals were collected at a sampling frequency of 1000 Hz and were preamplified in each electrode and then fed into a differential amplifier with an adjustable gain setting (25–500 Hz; common-mode rejection ratio: 110 dB at 50 Hz, input impedance of 100 MΩ and gain of 1000). Self-adhesive silver chloride electromyographic electrodes (Dahlhausen®, Köln, Germany) were used in a bipolar configuration with a distance of 20 mm between detection surface centers. The skin impedance was measured with an Electrode Impedance Checker (Noraxon USA, Scottsdale, AZ, USA). The electromyography and force platform signals were analyzed with the Acqknowledge software (Biopac Systems, Goleta, CA, USA).

Procedures

Preparation

Immediately before the electrode placement, the skin was prepared to reduce the impedance to a level equal or inferior to 5 kΩ.

Electrodes were placed in muscles midbelly according to anatomical references (Cheng et al. 2004; Klein et al. 2010). The ground electrodes were placed over each patella.

Measurement

After an explanation about the procedures, subjects were instructed to walk at their self-selected speed on an 8-m walkway after a voice command. A self-selected walking speed was adopted since ankle plantar flexor muscles develop higher activity at this speed (Milot et al. 2008). To prevent fatigue, a 1-min rest between each trial was provided and the necessary repetitions were performed in order to obtain three valid trials. A trial was considered valid when at least one complete stance phase was collected by each force plate during the task. The electromyographic data were acquired from both lower limbs simultaneously.

Data analysis

The raw electromyographic signal and the ground reaction forces signal were processed using the Acqknowledge software. The raw electromyographic signal was filtered using a band-pass filter of 20 and 450 Hz, processed using a root mean square (RMS) procedure (Lamontagne et al. 2002a; Billot et al. 2010), and normalized to mean signal over the entire gait cycle.

The signals from the force plates were also filtered, using a low-pass filter of 8 Hz, and the force values were normalized to the weight of each subject (Turns et al. 2007). The double-support phase was assessed through ground reaction forces. The beginning of double support during the stance phase was defined as the interval where Fz of LEAD presents a value equal or higher than 7% of body weight, until the initiation of the TRAIL swing phase (Sousa et al. 2012a, 2013). The EMG activity of each muscle was assessed during double support in two conditions: (a) when the IPSI limb was the TRAIL and the CONTRA was the LEAD, and (b) when the CONTRA was the TRAIL and the IPSI limb was the LEAD. The ankle antagonist coactivation was calculated according to the following formula:

$$\begin{aligned}&\text{Antagonist coactivation}(\%)\\&\quad=\frac{\text{antagonist activity}}{\text{agonist}+\text{antagonist activity}}\times 100.\end{aligned}\qquad(1)$$

Statistics

Using descriptive statistics, measures of central tendency (mean) and dispersion (standard deviation) for the magnitude of TA and SOL's electromyographic activity were calculated. Taking into account the small sample size of both groups ($n < 10$), it was assumed that the variables did not follow a normal distribution. Thus, the Wilcoxon Signed Rank Test was used to compare the magnitude of the muscles' activity and antagonist coactivation between both lower limbs. The confidence interval used was equal to 95%, with a significance level of 0.05.

Results

Ankle antagonist coactivation in the IPSI limb changes according to its role in walking, LEAD or TRAIL. In fact, non-significant differences were observed in relation to a healthy control when the IPSI limb assumes the role of LEAD (Table II). However, the antagonist coactivation between SOL and TA presents statistical significant differences in relation to a healthy control when in the TRAIL position (Table II).

Contrary to what was expected, the CONTRA limb presented values more similar to the healthy controls when assuming the TRAIL position (Table II).

Discussion

The choice to study plantar flexor muscles (SOL and GM), instead of other proximal muscles, was based on the knowledge that, through sensorial feedback, they act as one functional unit (Cappellini et al. 2006) and are the main contributors to move the body forward (Lin et al. 2006; Grey et al. 2007; Neptune et al. 2008). However, while SOL contributes mostly to body support (McGowan 2008), GM acts mainly at providing forward progression (Neptune et al. 2001; Anderson and Pandy 2003; Hall et al. 2011). Based on the neurophysiologic mechanism of reciprocal enervation, TA was the antagonist selected to study the coactivation process. It is also important to note that this study focuses on the relation between pairs of muscles. Consequently, the results obtained could not reflect changes in individual muscle activity. In this sense, when both muscles present activation impairments, the antagonist coactivation in stroke subjects can be similar to that obtained in healthy subjects. However, this did not mean that recruitment failure is not present, since this particular aspect was not evaluated.

Table I. Mean and standard deviation (SD) values of age, height, and weight of the healthy and stroke groups. The values of the self-selected walking speed adopted by each group are also indicated.

	Control group	Stroke group	
Variables	Mean ± SD	Mean ± SD	p Value
Age (years)	49.24 ± 7.69	53.87 ± 7.17	0.070
Height (m)	1.66 ± 0.09	1.65 ± 0.10	0.942
Body weight (kg)	67.40 ± 8.76	75.29 ± 7.03	0.006
Self-selected gait speed (m/s)	1.00 ± 0.03	0.57 ± 0.13	<0.001

Table II. Antagonist coactivation ratio of TRAIL and LEAD limb of stroke and healthy subjects.

			Stroke group		Healthy group	
Limb action	Muscle pairs	Limb	Mean	p Value	Mean	p Value
LEAD	SOL/TA	CONTRA	51.2	0.09	58.5	0.452
		IPSI	61.3			0.713
	GM/TA	CONTRA	52.4	**0.04**	62.9	0.163
		IPSI	63.3			0.946
TRAIL	SOL/TA	CONTRA	35.1	**0.02**	39.7	0.359
		IPSI	54.2			**0.005**
	GM/TA	CONTRA	31.4	**0.02**	36.1	0.309
		IPSI	47.5			0.062

Bold values are statistically significant p values.

CONTRA limb antagonist coactivation

Globally the results of the present study reveal that non-significant differences were observed between the CONTRA limb and CONTROL, contradicting previous studies reviewed in Rosa et al. (2014). However, these results do not exclude possible muscle activation impairments in this limb (Olney and Richards 1996; Lamontagne et al. 2002b). Based on neurophysiology, it would be expected that when assuming the LEAD position, the CONTRA limb would present ankle antagonist coactivation dysfunction as a result of a higher impairment in TA recruitment (agonist role) subsequent from the corticospinal lesion. However, the possible dysfunction of the postural control system, also described in stroke subjects in this limb, can lead to a decreased activity of the antagonist activity (SOL and GM) (Sousa et al. 2013). This can explain the lack of differences observed in ankle coactivation compared to CONTROL, when assuming the LEAD position. The same reasoning may explain findings obtained in the CONTRA while assuming the TRAIL position. In fact, both TA and GM are predominantly phasic muscles, and so both may express an atypical behavior in subcortical stroke in the territory of the middle cerebral artery by the dependence of these muscles on the dorsolateral system. The SOL as a tonic muscle (Neptune et al. 2001; Anderson and Pandy 2003; Hall et al. 2011), depends on ventromedial system enervation. The possible lesion of both systems in this kind of stroke subject's can justify the lack of differences in coactivation between these muscles compared to CONTROL.

IPSI limb antagonist coactivation

Despite the postural control demand associated with initial contact and loading response, non-significant differences were observed between IPSI limb and CONTROL. These results can be explained by the fact that TA have a major role in stability during this phase (Chow et al. 2012) whose activity is dependent mostly from unimpaired dorsolateral system control. When this limb was the TRAIL, this behavior was no longer similar to those evidenced by the CONTROL, the TA/SOL being the pair in which this atypical behavior was more pronounced. This result can be explained by the knowledge of the ipsilateral disposal ventromedial system that has a strong influence over predominantly postural muscles like SOL (Figure 1). Taking this into consideration, the results of the present study seem to indicate that the IPSI limb postural control dysfunction, associated with a possible

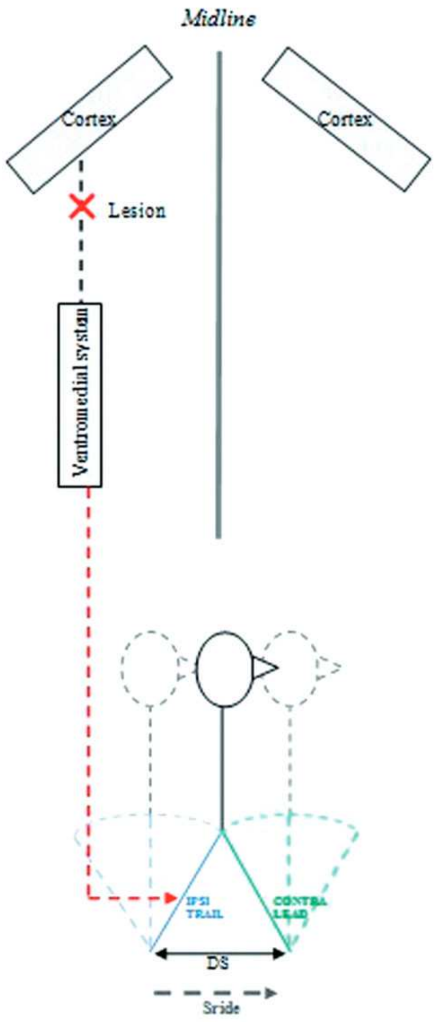

Figure 1. Representation of the influence of the ventromedial disposed system on ipsilesional (IPSI) limb action when assuming the TRAIL position during the double-support (DS) phase of walking, in subjects with subcortical stroke in the territory of the middle cerebral artery.

lesion of ventromedial systems, interferes with the body support function during forward propulsion. This hypothesis hasn't been questioned in previous studies about antagonist coactivation in stroke subjects as the changes observed in the IPSI limb have been interpreted as a compensatory adaptative strategy (Lamontagne et al. 2000; Chow et al. 2012; Rosa et al. 2014). It is important to note that, since our study is dedicated to antagonist coactivation and not to individual muscle activity, future studies are required to confirm this possibility. On the other hand, it should be highlighted that our criteria for selecting participants for our study based on vascular territory and lesion area has not been frequently considered in previous studies. Based on this, the differences obtained in the present study in relation to previous studies (Lamontagne et al. 2000; Chow et al. 2012) as to ankle coactivation can be related to this aspect.

Our findings present a novel insight into post-stroke neuromotor impairment, since they also point out difficulties in the IPSI limb possibly related to the vascular lesion.

Conclusions

The ipsilesional limb presents a dysfunctional behavior when a higher postural control activity was demanded (as when it assumed the trailing position). This dysfunction was more pronounced in the soleus and tibialis anterior muscle pair as a result of a possible higher relation of soleus muscle with postural control demand.

Declaration of interest

The authors report no conflicts of interest.

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