Bilateral compensatory postural adjustments to a unilateral perturbation in subjects with chronic ankle instability

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A B S T R A C T

Background: To evaluate the magnitude of bilateral compensatory postural adjustments in response to a unilateral sudden inversion perturbation in subjects with chronic ankle instability.
Methods: 24 athletes with chronic ankle instability (14 with functional ankle instability, 10 with mechanical ankle instability) and twenty controls participated in this study. The bilateral electromyography of ankle muscles was collected during a unilateral sudden ankle inversion to assess the magnitude of subcortical and voluntary compensatory postural adjustments in both the perturbed and the contralateral limb (support limb). *Findings:* In the support position, compared to the control group, the group with functional ankle instability presented decreased compensatory postural adjustments of the tibialis anterior in both the injured and the uninjured limbs in the support position and of the soleus in the uninjured limb. In the side of the perturbation, participants with functional ankle instability presented decreased soleus compensatory postural adjustments in the uninjured limb when compared to the control group. Increased values of soleus and peroneal brevis com-pensatory postural adjustments were observed in the group with mechanical instability when compared to the control group and to the group with functional ankle instability.
Interpretation: Subjects with functional ankle instability present bilateral impairment of compensatory postural adjustments of the tibialis anterior in a support position and of the soleus of the uninjured limb regardless of the position. Subjects with mechanical instability present bilateral increase of these adjustments in the peroneal brevis regardless of the position and in the soleus muscle in the side of the perturbation.

Keywords: Chronic ankle instability; Compensatory postural adjustments; Ankle sprain

1. Introduction

It is well known that postural control is successfully maintained using visual, vestibular and somatosensory information. Proprioceptive information originating from sensory receptors in the lower limb has been identified as a key source of triggering information needed to initiate directionally specific, automatic postural responses following an unexpected postural perturbation (Horak, 1996). The determinant role of proprioceptive information provided by the ankle segment (Fitzpatrick et al., 1994) highlights the importance of understanding postural control dysfunction following the most common ankle injury – ankle sprain (Yeung et al., 1994).

It has been argued that patients suffer partial deafferentation following ankle sprain (Freeman, 1965) and that this could chronically suppress gamma activation and desensitize the muscle spindle (Khin Myo et al., 1999). This mechanism, together with the decreased agonist and increasing antagonist muscle activity in response to pain (Lund et al., 1991), has been interpreted as the basis of chronic ankle instability (CAI) (Khin Myo et al., 1999; Riemann, 2002). The evidence demonstrating contralateral healthy limb pain adaptation in other anatomic regions (Falla et al., 2007) suggest that the presence of pain after ankle sprain would lead to impaired muscle responses also in the contralateral limb.

Chronic ankle instability may englobe mechanical and functional deficits (Delahunt et al., 2010) and has been characterized by the presence of impaired proprioception (Docherty and Arnold, 2008; Forkin et al., 1996; Glencross and Thornton, 1981; Konradsen, 2002) and a related delayed activation timing of peroneal muscles during

short latency compensatory responses (Hoch and McKeon, 2014; Konradsen and Bohsen Ravn, 1991; Lofvenberg et al., 1995; Menacho Mde et al., 2010; Mitchel et al., 2008; Munn et al., 2010). Muscle activation deficits can be related to decreased motoneuron pool excitability (Hertel, 2008; Sefton et al., 2008; Sefton et al., 2009) resultant from deficits in peripheral sensory input after injury (Docherty and Arnold, 2008; Forkin et al., 1996; Glencross and Thornton, 1981; Konradsen, 2002) but also from a dysfunction in supraspinal sensorimotor control (Palmieri-Smith et al., 2009). Therefore, the neuromuscular dysfunction in CAI should not be explored at an individual muscle response level only. Beyond this argument, it should be noted that through a systematic review with meta-analysis, Munn et al. (2010) concluded that peroneal reaction time was not impaired in those with CAI (Munn et al., 2010). The conflicting results regarding the role of delayed peroneal muscle timing in CAI, and the lack of studies regarding the magnitude of postural control adjustments, raise the question whether CAI results from failure in individual muscle responses or from global impaired magnitude modulation of compensatory postural adjustments (CPA) resultant from supraspinal sensorimotor dysfunction (Palmieri-Smith et al., 2009). This hypothesis is sustained by the demonstrated postural control deficits in joints proximal to injured ankles (Bullock-Saxton, 1994; Caulfield and Garrett, 2002; Hertel and Olmsted-Kramer, 2007) in both the injured (Mckeon and Hertel, 2008; Wikstrom et al., 2010) and the uninjured (Hertel and Olmsted-Kramer, 2007) limbs during single leg stance in subjects with CAI. Increased error in the evertors' force sense in both injured and uninjured limbs in CAI (Docherty and Arnold, 2008; Sousa et al., 2017; Wright and Arnold, 2012) can be related to this bilateral dysfunction, as increased error by the Golgi tendon organ leads to decreased accuracy in detecting the projection of the body's centre of mass within the base of support (Dietz, 1998) and in regulating the evertors' force (Proske, 2005) and stiffness (Docherty et al., 2004). A bilateral affection supports the lack of significant differences previously found between the injured and uninjured limbs in subjects with CAI (Mckeon and Hertel, 2008).

It has been argued that when a unilateral sudden inversion perturbation is applied (perturbed limb) in bipedal standing, the contralateral limb (support limb) has an important role in accelerating the centre of pressure in the direction of the support limb to dampen the contralateral ankle sprain mechanism (Mitchel et al., 2008). Consequently, a bilateral postural control deregulation in a support position (Hertel and Olmsted-Kramer, 2007; Mckeon and Hertel, 2008; Wikstrom et al., 2010) could lead to increased risk of contralateral ankle sprain in sudden inversion perturbations. However, to the best of our knowledge no study has assessed the magnitude of postural adjustments in response to a unilateral sudden inversion perturbation in both injured and uninjured limbs while assuming a support position.

The purpose of this study was to evaluate the magnitude of bilateral CPA in response to a unilateral sudden inversion perturbation in subjects with unilateral CAI. A decreased magnitude of CPA would be expected in both the injured and the uninjured limbs while assuming a support position. The results of this study could be used in the development of successful rehabilitation strategies to reduce the residual symptoms related to CAI.

2. Methods

2.1. Design

Cross-sectional study.

2.2. Participants

Twenty four athletes (6 women, 18 men) with unilateral CAI and twenty uninjured athletes (3 women, 17 men) from the target population available at the time and willing to take part participated in this study (Table 1). Participants assigned to the CAI group met the criteria set by the International Ankle Consortium (Gribble et al., 2014). For inclusion in the CAI group, subjects had to follow the following criteria: (Horak, 1996) history of at least one significant unilateral ankle sprain; (Fitzpatrick et al., 1994) the initial sprain must have occurred at least 12 months prior to enrolment in the study; (Yeung et al., 1994) at least one ankle sprain was associated with inflammatory symptoms; (Freeman, 1965) at least one ankle sprain created at least one day of interruption of desired physical activity; (Khin Myo et al., 1999) the most recent injury must have occurred more than three months prior to enrolment in the study; and (Lund et al., 1991) history of the previously injured ankle joint "giving way" (at least 2 episodes of giving way in the 6 months prior to study enrolment) and/or recurrent sprain (two or more sprains in the same ankle) and/or "feelings of instability". To meet this last criterion, individuals must have answered "yes" to question 1 ("Have you ever sprained an ankle?") along with "yes" to at least four questions of the Ankle Instability Instrument (Docherty et al., 2006; Gribble et al., 2014). The CAI group was divided into two subgroups: one was composed by subjects presenting CAI without mechanical ankle instability and was designated by functional instability group (FAI group), while the other was composed of subjects with CAI with MAI. (MAI group). Subjects were included in the MAI group if they presented the previously indicated criteria and one or more of the following conditions: 1) presence of pain or changes in talocrural joint mobility higher that 3 mm in anterior drawer and posterior glide manual stress tests, compared to the uninjured side (Karlsson et al., 1991); and/or 2) talar tilt (in frontal plane) higher than 7° together with a difference higher than 0° in relation to the contralateral (uninjured) ankle (Rosenbaum et al., 2000). The orthopaedic tests were performed by a physical therapist specialised in manual therapy. The anterior drawer displacement was quantified through the double integration of the signal obtained from an accelerometer placed on the talus. The talar tilt was quantified through an electrogoniometer. In all participants the subjective information provided by physical therapists agreed with the quantitative values. Subjects with negative orthopaedic tests were included in the FAI group. The exclusion criteria for the CAI group met the criteria set by the International Ankle Consortium (Gribble et al., 2014) and included: (Horak, 1996) history of previous surgeries to the musculoskeletal structures in either limb of the lower extremity; (Fitzpatrick et al., 1994) history of lower limb fracture requiring realignment; (Yeung et al., 1994) acute injury in the other joints of the lower extremity in the previous three months that resulted in at least

Table 1
Mean and standard deviation (SD) values of age, height and body mass of control and CAI groups.

Variables	Mean (SD)			*p*-Value
	Control	FAI	MAI	
Age (years)	21.8 (2.21)	20.4 (2.92)	20.8 (2.34)	0.078
Height (m)	1.78 (0.09)	1.75 (0.10)	1.77 (0.08)	0.720
Body mass (kg)	73.8 (11.5)	69.0 (12.3)	70.5 (11.1)	0.492
Number of previous ankle sprains	–	3.5 (1.76)	2.7 (1.34)	
Frequency of giving way	–	Rarely, $n = 4$ Frequently, $n = 7$ Often, $n = 3$	Rarely, $n = 4$ Frequently, $n = 3$ Often, $n = 3$	
Severity of ankle sprain	–	Moderate ankle sprain, $n = 13$ Mild ankle sprain, $n = 1$	Severe ankle sprain, $n = 1$ Moderate ankle sprain, $n = 9$	
Time since last sprain (months)	–	7.7 (4.08)	10.4 (1.72)	
	n = 20	*n = 14*	*n = 10*	

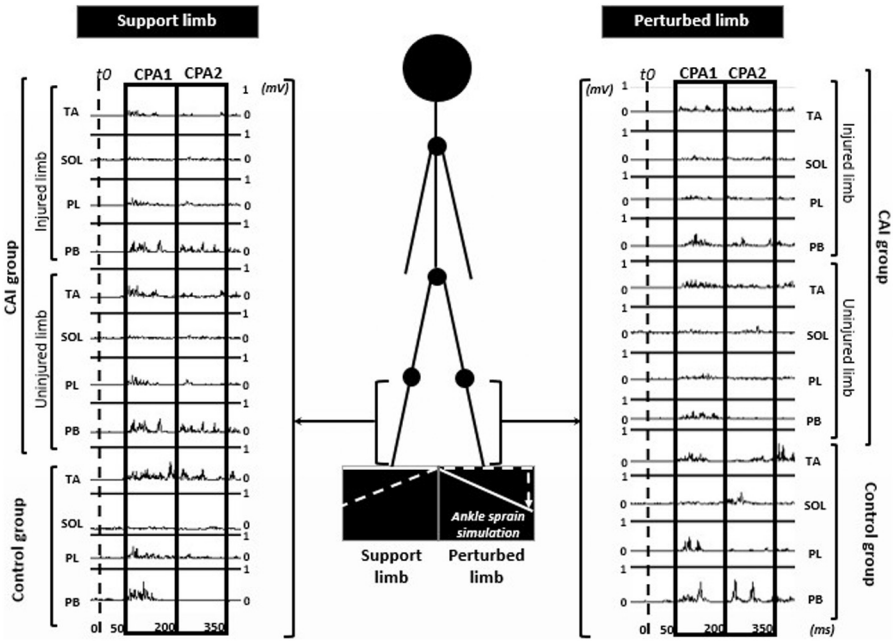

Fig. 1. Representation of the setup adopted and of the EMG signals collected in one participant of CAI group and of control group. The figure illustrates a trial where a sudden ankle inversion was applied in the right limb. The EMG signals from support (left) limb and perturbed (right) limb are provided. The presented signal was previously filtered using a zero-lag, second-order Butterworth filter with an effective band pass of 20 to 450 Hz and full wave rectified.

one day of interruption of desired physical activity; (Freeman, 1965) history of bilateral ankle sprain; and (Khin Myo et al., 1999) neurological impairments. Healthy control participants were selected according to the same exclusion criteria applied to the CAI group and were also excluded if they had history of ankle sprain. All volunteers were athletes practicing sports with high risk of ankle sprain, including soccer, basketball, volleyball and handball. Prior to testing, subjects were asked to identify the dominant limb, which was described as the leg which they would use to kick a ball. As no differences were observed between the dominant and the non-dominant limbs of healthy subjects in a previous study that used a similar protocol to the one used in the present study (Mitchel et al., 2008), in the healthy control group only one limb was selected for evaluation. It should be noted that this limb was evaluated in both support and perturbed positions and the subjects were not informed about the limb that would be exposed to the perturbation in each trial. In the MAI and FAI groups both limbs were evaluated.

The study was approved by the local ethics committee and was implemented according to the Declaration of Helsinki. All subjects gave their written consent.

2.3. Instrumentation

The activity of the agonist muscles for active ankle stability, the peroneus longus (PL), the peroneus brevis (PB), the tibialis anterior (TA) and the soleus (SOL) muscles, was assessed through surface electromyography (EMG). The EMG signal of these muscles was monitored using a bioPLUX[a] research wireless signal acquisition system. 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 (20–500 Hz; common-mode rejection ratio: 110 dB at 50 Hz, input impedance of 100 MU and gain of 1000). Self-adhesive silver chloride EMG electrodes were used in a bipolar configuration with a distance of 20 mm between detection surface centres. The skin impedance was measured with an Electrode Impedance Checker[b] to ensure that skin impedance values were lower than 5000 Ω. The EMG and accelerometer signals were analysed with the Acqknowledge software (Biopac System).

The Ankle Instability Instrument was designed to classify patients with CAI and has been shown to be a reliable and valid tool (Docherty et al., 2006). The instrument presents high values of test-retest reliability (ICC = 0.95). Internal consistency reliability estimates (alpha coefficients) for each factor and the total measure ranged from 0.74 to 0.83.

A tilt platform was used to force 30° of subtalar joint inversion. The platform included two movable plates (trapdoors) so that either foot could be tilted independently, thus removing any subject anticipatory effect (Fig. 1). A triaxial accelerometer sensor (bioPLUX[a] research) connected to the bioPLUX[a] research wireless signal acquisition system was placed in each movable plate to detect the onset of the tilt mechanism (first deflection of the accelerometer signal). The signal was collected with a sampling frequency of 1000 Hz with a range of ± 3.6G and a bandwidth of 0–50 Hz. The EMG and accelerometer signals were integrated and synchronised by the acquisition system. For safety reasons, the tilt platform was surrounded by a handrail to the front and on both sides of the subject and an adhesive, nonslip material was placed to prevent slipping when the trapdoors were dropped and to increase comfort.

2.4. Procedures

2.4.1. Skin preparation and electrode placement

The skin surface of the selected muscles' mid-belly and of the patella was prepared (shaved, 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 EMG electrodes were placed according to anatomical references recommended in Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM): TA (1/3 on the line between the tip of the tibia and the tip of the medial malleolus), 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), PL (1/4 on the line between the tip of the head of the fibula to the tip of the lateral malleolus) and PB (anterior to the tendon of PL at 1/4 of the line from the tip of the lateral malleolus to the fibula-head). The reference electrode was placed on the patella. To assess possible EMG crosstalk, subjects were asked to contract each ankle muscle according to manual muscle testing procedures (Kendall, 2005). In each test the EMG signal of TA, SOL, PL and PB muscles was collected. It was ensured that the signal of these muscles did not increase above the mean of the baseline in unrelated tests.

Table 2
Mean, SEM and MDD values obtained for the relative magnitude of subcortical (CPA1) and voluntary (CPA2) CPAs of TA in control, FAI and MAI groups. Bold values represent $p < 0.05$.

Group	Position	CPA	Uninjured limb			Between-subjects effects			Injured limb			Between-subjects effects			Within-subjects effects		
			Mean (SD)	SEM	MDD	F	*p*	1-β	Mean (SD)	SEM	MDD	F	*p*	1-β	t	*p*	1-β
Control	Support	CPA1	41.94 (20.22)	2.15	5.96	4.689	**0.015**	0.310	–			4.410	**0.019**	0.758	–		
FAI			19.98 (17.56)	2.39	6.62				22.87 (21.62)	2.78	7.70				−1.212	0.253	0.229
MAI			32.16 (17.95)	2.38	6.60				24.83 (17.25)	2.34	6.48				1.466	0.177	0.387
Control	Perturbed		27.62 (16.02)	1.92	5.32	1.450	0.248	0.333	–			4.173	**0.023**	0.780	–		
FAI			20.59 (21.89)	2.63	7.29				15.03 (13.55)	2.13	5.90				0.581	0.576	0.341
MAI			33.99 (15.75)	2.23	6.18				30.20 (10.54)	1.82	5.04				0.629	0.545	0.283
Control	Support	CPA2	79.56 (47.58)	3.30	9.15	3.770	**0.032**	0.806	–			4.213	**0.022**	0.800	–		
FAI			38.84 (32.00)	3.20	8.87				43.48 (41.40)	3.86	10.69				−0.820	0.432	0.122
MAI			57.08 (29.42)	3.05	8.44				41.68 (24.33)	2.77	7.67				1.685	0.126	0.465
Control	Perturbed		33.18 (21.04)	2.20	6.10	1.057	0.358	0.212	–			4.718	**0.007**	0.858	–		
FAI			33.85 (46.49)	3.83	10.62				17.82 (14.98)	2.26	6.26				−1.166	0.274	0.317
MAI			50.79 (34.78)	3.32	9.20				46.41 (24.49)	2.78	7.70				−0.330	0.749	0.090

2.4.2. Data acquisition

All individuals were asked to stand quietly with the support base aligned at shoulder width with one foot in each trapdoor, keeping their arms by their sides, and to focus on a target 2 m away and at eye level for 30 s. The individuals were also instructed to ensure equal weight distribution between the two limbs. One limb at a time was randomly exposed to unilateral sudden ankle inversion and was identified during analysis of each trial as the perturbed limb. The limb which was not exposed to sudden ankle inversion in each trial was identified as the support limb. Each limb was exposed to the sudden ankle inversion three times in a random order after familiarization trials (Fig. 1). In each trial the trapdoor was randomly released by pushing a foot switch not visible to the subject. The subjects did not know the side nor the time of application of the perturbation. Participants were informed that they should maintain stability reacting to the perturbation. In FAI and MAI groups, the EMG signal was collected from both limbs (injured and uninjured limbs) and both where evaluated as support and perturbed limbs. Upon release, the platform fell down through an arch of 30° which was predetermined by a mechanical stop leading to ankle subtalar inversion. Rest periods of 60 s were provided between trials, during which the subjects sat down while maintaining the foot position. When large variations in proximal segment movement occurred between trials, additional trials were performed to guarantee homogeneous responses.

Each participant from the CAI performed 6 valid trials; 3 valid trials were performed with the injured limb in the perturbed position and the uninjured limb in the support position and 3 valid trials were performed with the injured limb in the support position and the uninjured limb in the perturbed position. In the control group 6 valid trials were also performed; 3 valid trials were performed with the dominant limb in the perturbed position and 3 valid trials were performed with the dominant limb in the support position.

2.4.3. Data processing

The EMG signals were filtered using a zero-lag, second-order Butterworth filter with an effective band pass of 20 to 450 Hz, full wave rectified, and the root mean square was calculated. The magnitude of the EMG activity was analysed at two epochs (Fig. 1) in relation to the first deflection of the accelerometer signal (T_0): (Horak, 1996) 50 to 200 ms (subcortical CPA), and (Fitzpatrick et al., 1994) 200 to 350 ms (voluntary CPA) (Latash, 2008). The CPA time window was chosen using the literature data on the timing of corrective reactions observed in the leg muscles in response to external perturbations induced by a platform translation (Henry et al., 1998). The subsequent division of this interval into two sub-windows was performed to differentiate the subcortical responses from the voluntary reactions (41). It was confirmed that no electromyographic responses occurred in the time window of anticipatory postural adjustments. The magnitude of the EMG signal in each interval was normalised by baseline values (from −500 to −350 in relation to T_0) to assess the degree of magnitude modulation of each muscle during compensatory responses in relation to upright standing according to the following formula (42):

$$Normalized\ EMGCPA = \frac{EMGCPA}{EMGbaseline}$$

2.5. Statistical analysis

The acquired data were analysed using the Statistic Package Social Science (SPSS)[c] software from IBM Company (USA). Mean and standard deviation were used for descriptive analysis. The comparisons of the magnitude of subcortical and voluntary CPA, in support and perturbed positions, between the FAI group, the MAI group and the control group, were analysed through the one-way analysis of variance (ANOVA) test. Both injured and uninjured limbs of the FAI and MAI groups were compared to the dominant limb of the control group. Bonferroni

Table 3
Mean, SEM and MDD values obtained for the relative magnitude of subcortical (CPA1) and voluntary (CPA2) CPAs of SOL in control, FAI and MAI groups. Bold values represent $p < 0.05$.

Group	Position	CPA	Uninjured limb			Between-subjects effects			Injured limb			Between-subjects effects			Within-subjects effects		
			Mean (SD)	SEM	MDD	F	*p*	1-β	Mean (SD)	SEM	MDD	F	*p*	1-β	t	*p*	1-β
Control	Support	CPA1	4.76 (3.44)	0.89	2.46	1.186	0.317	0.320	–			2.293	0.114	0.432	–		
FAI			2.46 (2.40)	0.87	2.41				3.18 (2.88)	0.89	2.46				−0.957	0.363	0.174
MAI			5.22 (7.00)	1.57	4.35				6.84 (6.20)	1.44	3.99				−0.600	0.565	0.156
Control	Perturbed		3.22 (1.75)	0.63	1.74	5.439	**0.009**	0.843	–			6.999	**0.002**	0.896	–		
FAI			3.91 (5.47)	1.16	3.21				2.57 (2.14)	0.85	2.35				0.562	0.587	0.153
MAI			9.67 (8.02)	1.63	4.52				6.48 (4.28)	1.17	3.24				0.972	0.359	0.308
Control	Support	CPA2	8.10 (6.68)	1.24	3.43	3.746	**0.033**	0.905	–			3.428	**0.043**	0.631	–		
FAI			2.64 (1.85)	0.76	2.10				4.30 (3.82)	1.12	3.10				2.228	0.053	0.535
MAI			8.10 (5.05)	1.33	3.68				12.48 (11.71)	1.98	5.48				1.025	0.335	0.281
Control	Perturbed		12.32 (8.30)	1.38	3.88	2.594	0.089	0.506	–			6.861	**0.003**	0.957	–		
FAI			7.50 (13.54)	1.46	4.04				3.97 (2.41)	0.93	2.58				−0.766	0.462	0.194
MAI			20.45 (18.56)	2.48	6.87				11.46 (7.34)	0.93	2.58				−1.875	0.098	0.634

Table 4
Mean, SEM and MDD values obtained for the relative magnitude of subcortical (CPA1) and voluntary (CPA2) CPAs of PL in control, FAI and MAI groups. Bold values represent $p < 0.05$.

Group	Position	CPA	Uninjured limb			Between-subjects effects			Injured limb			Between-subjects effects			Within-subjects effects		
			Mean (SD)	SEM	MDD	F	*p*	1-β	Mean (SD)	SEM	MDD	F	*p*	1-β	t	*p*	1-β
Control	Support	CPA1	15.65 (13.79)	1.78	4.93	0.049	0.952	0.058	–			1.328	0.276	0.278	–		
FAI			14.09 (14.47)	2.19	6.07				9.50 (9.72)	1.85	5.12				1.096	0.299	0.362
MAI			14.84 (10.98)	1.86	5.15				16.99 (13.93)	2.10	5.82				−0.571	0.582	0.410
Control	Perturbed		19.80 (10.71)	1.57	4.35	2.008	0.149	0.438	–			3.537	**0.039**	0.612	–		
FAI			16.22 (9.75)	1.75	4.85				11.47 (12.16)	2.10	5.82				1.206	0.259	0.767
MAI			25.78 (11.98)	1.95	5.40				23.71 (13.23)	1.43	3.96				0.501	0.629	0.124
Control	Support	CPA2	18.24 (9.07)	1.44	2.77*	0.309	0.736	0.109	–			3.435	**0.042**	0.630	–		
FAI			16.04 (14.07)	2.15	5.96				10.31 (10.46)	1.92	5.32				−1.522	0.159	0.611
MAI			20.88 (20.85)	2.57	7.12				18.40 (8.19)	1.61	4.46				−0.426	0.680	0.110
Control	Perturbed		24.29 (17.29)	1.78	4.93	0.454	0.639	0.127	–			3.458	**0.041**	0.639	–		
FAI			22.5 (17.91)	2.38	6.59				11.36 (9.16)	1.84	5.10				−2.378	**0.041**	0.864
MAI			29.43 (16.14)	2.26	6.26				26.50 (20.94)	2.57	7.12				−0.447	0.665	0.116

multiple comparison procedures were used to make post hoc comparisons. The comparisons between injured and uninjured limbs in each position were made using the Paired-Samples *t*-test. The Shapiro–Wilk test and histogram analysis indicated that data were normally distributed. A 0.05 significance level was used for inferential analysis. The standard error of measurement (SEM) was calculated by taking the square root of the error variance of each averaged variable. The SEM was used to calculate the minimal detectable difference (MDD). To compute the MDD as the 95% confidence interval limits of the SEM, the SEM had to be multiplied by 1.96 (for the 95% interval) and by the square root of two for the difference scores ($1.96 \times \sqrt{2} \times$ SEM).

3. Results

A decrease of TA CPA in both injured and uninjured limbs in the support position was observed in the FAI group compared to the control group (uninjured limb subcortical CPA, $p = 0.030$, percentage of difference (%DIF) = 47.6; injured limb subcortical CPA, $p = 0.031$, %DIF = 54.5; uninjured limb voluntary CPA, $p = 0.042$, %DIF = 48.8) (Table 2). In the perturbed position the FAI group presented decreased TA subcortical and voluntary CPA in the injured limb compared to the FAI ($p = 0.045$ and $p = 0.005$, respectively).

The FAI group also presented decreased values of voluntary SOL CPA in the uninjured limb compared to the control group ($p = 0.044$, %DIF = 32.6) and in the injured limb compared to the MAI group in the support position ($p = 0.039$) (Table 3). In the perturbed position, the MAI group presented increased values of subcortical SOL CPA compared to the control group (uninjured limb, $p = 0.009$, %DIF = 300; injured limb, $p = 0.012$, %DIF = 201) and the FAI group (injured limb, $p = 0.040$; uninjured limb, $p = 0.003$). In this position, the FAI group presented decreased voluntary SOL CPA in the uninjured limb when compared to the control group ($p = 0.003$, %DIF = 32.2) and the MAI group ($p = 0.031$) (Table 3).

Differences between groups were also observed in the peroneal muscles in both positions. In the perturbed position, the MAI group presented increased activity of subcortical PL CPA compared to the FAI group ($p = 0.049$) (Table 4) and increased PB CPA compared to the control group (subcortical CPA: $p = 0.018$, %DIF = 214 (uninjured

Table 5
Mean, SEM and MDD values obtained for the relative magnitude of subcortical (CPA1) and voluntary (CPA2) CPAs of PB in control, FAI and MAI groups. Bold values represent $p < 0.05$.

Group	Position	CPA	Uninjured limb			Between-subjects effects			Injured limb			Between-subjects effects			Within-subjects effects		
			Mean (SD)	SEM	MDD	F	*p*	1-β	Mean (SD)	SEM	MDD	F	*p*	1-β	t	*p*	1-β
Control	Support	CPA1	15.06 (10.12)	1.56	4.32	3.346	**0.046**	0.714	–			4.248	**0.021**	0.688	–		
FAI			8.77 (5.06)	1.30	3.60				9.86 (9.38)	1.85	5.12				−0.587	0.570	0.104
MAI			19.60 (12.34)	1.97	5.46				25.37 (20.27)	2.53	7.01				−0.899	0.392	0.222
Control	Perturbed		22.70 (12.20)	1.70	4.71	4.946	**0.013**	0.910	–			9.339	< **0.001**	0.963	–		
FAI			15.25 (7.79)	1.57	4.35				13.86 (10.96)	1.97	5.45				−0.260	0.800	0.109
MAI			48.60 (46.50)	3.83	10.61				37.47 (17.37)	2.34	6.48				0.897	0.393	0.221
Control	Support	CPA2	22.64 (12.40)	1.72	4.72	3.898	**0.029**	0.774	–			3.470	**0.041**	0.712	–		
FAI			12.54 (8.15)	1.64	4.54				11.50 (9.82)	1.85	5.12				0.030	0.977	0.098
MAI			25.87 (13.55)	2.07	5.73				45.35 (62.44)	4.44	12.30				−0.955	0.364	0.239
Control	Perturbed		33.82 (18.27)	2.08	5.76	4.896	**0.013**	0.914	–			11.691	< **0.001**	0.622	–		
FAI			21.13 (14.04)	2.11	5.84				15.73 (15.19)	2.33	6.45				−0.689	0.508	0.445
MAI			78.88 (82.33)	4.48	12.41				57.62 (30.55)	3.11	8.61				−0.824	0.431	0.201

limb), $p = 0.022$, %DIF = 165 (injured limb); voluntary CPA: $p = 0.011$, %DIF = 233 (uninjured limb), $p = 0.019$, %DIF = 170 (injured limb)) and to the FAI (subcortical CPA: $p = 0.004$ (uninjured limb), $p < 0.001$ (injured limb); voluntary CPA: $p = 0.001$ (uninjured limb), $p < 0.001$ (injured limb)) (Table 5). In the support position, increased PB activity was also observed in the MAI group when compared to the FAI group in both subcortical (uninjured limb, $p = 0.044$; injured limb, $p = 0.018$) and voluntary CPA (uninjured limb, $p = 0.040$; injured limb, $p = 0.037$) (Table 5).

4. Discussion

The results obtained in the present study are in accordance with the hypothesis stated since bilateral changes in the magnitude modulation of CPA were observed in participants with CAI. The main deficit in the CAI group when compared to the control group was observed in the support limb. In both the injured and the uninjured limbs, decreased TA subcortical and voluntary CPA were observed in the FAI group when compared to the control group, indicating impairments in short, medium and long latency and voluntary responses (Latash, 2008). This is in accordance with previous studies demonstrating an inhibition of the tibialis anterior in subjects with CAI (Klykken et al., 2011), and was accompanied by decreased voluntary responses of the SOL in the uninjured limb. In a closed kinetic chain, the TA together with the SOL muscle has an important role in regulating the projection of the centre of mass on the base of support (Wilkerson et al., 1997; Winter, 1995). The decreased CPA in these muscles could lead to a decreased capacity in accelerating the centre of mass in the direction of the support limb to dampen the contralateral ankle sprain mechanism (Mitchel et al., 2008), increasing the risk of injuring the contralateral (perturbed) limb. Specifically, the decreased activity of TA CPA in both injured and uninjured limbs while assuming the support limb in subjects with FAI could interfere with keeping the centre of plantar pressure from shifting to the lateral aspect, in order to prevent the foot from rolling over the edge to cause an ankle inversion sprain injury in the perturbed limb (Fong et al., 2012). This hypothesis seems to sustain the neuromuscular impairment related to the increased centre of pressure displacement in cases of CAI during single limb stance tasks (dos Santos et al., 2014; Hertel and Olmsted-Kramer, 2007). However, because these tasks are different from that of the present study, future studies are required to confirm this hypothesis.

Differences between groups were observed in the magnitude of peroneal subcortical and voluntary CPA in both support and perturbed limbs. The higher PB CPA observed in both limbs of the MAI group, when compared to the control group in the support position, may indicate that subjects with MAI need higher PB muscle activity to stabilise the calcaneocuboid joint, which improves the efficiency of the PL in working over the cuboid pulley (Andrews et al., 2012). In a closed kinetic chain, the PL stabilises the first ray (the first metatarsal and first cuneiform bones) and everts the foot to transfer body weight from the lateral to the medial side of the foot (Andrews et al., 2012). The need for higher peroneus muscle activity in the MAI group was highlighted in the perturbed position, as higher activity of PB was obtained in this group compared to the FAI group, but also to the control group. It should be noted that the increased activity of peroneal muscles in the MAI group was observed in both injured and uninjured limbs, indicating that a unilateral mechanical instability leads to a reorganisation of the synergy provided by both lower limbs to ensure postural stability. An increased activity of the SOL muscle during subcortical CPA, in both injured and uninjured limbs, in the MAI group when compared to the FAI group and the control group was also observed on the side of the perturbation. This finding seems to reveal a strategy adopted by the MAI group to deal with the mechanical instability, but also that the increased PB activity may not be enough to enhance the PL to work efficiently over the cuboid pulley (Andrews et al., 2012). In fact, in weight-bearing conditions, the PL acts to stabilise the first ray, via the cuboid pulley, creating a rigid lever for push-off by exerting a plantar flexion force at the ankle (Andrews et al., 2012), while the SOL has an important role in controlling the centre of pressure along the foot's longitudinal axis (Gatev et al., 1999; Kim et al., 2003). The increased SOL activity could result from a lower efficiency of the PL for push-off. It should be noted that this strategy was only observed in this group.

Globally, no differences were observed between the injured and the uninjured limbs in both the MAI and the FAI groups. These findings corroborate the idea that the FAI group presents similar postural control deregulation in the injured and the uninjured limbs and that the MAI group presents similar compensatory strategies in the injured and

the uninjured limbs. This bilateral involvement, together with neuromuscular changes previously demonstrated in muscles proximal to injured ankles (Bullock-Saxton, 1994; Caulfield and Garrett, 2002; Hertel and Olmsted-Kramer, 2007) provide evidence that the aetiology of CAI involves alteration in the central nervous system control of lower extremities' neuromuscular function. However, it should be also noted that it has been argued that individuals with CAI compensate for their ankle deficits using proximal muscles to maintain reduced postural sway while kicking a ball (Rios et al., 2015). Because in the present study the activity of the proximal muscle was not evaluated, the results of the present study should not be extrapolated to more proximal segments. Future studies should explore this possibility. Also, future studies exploring if individuals with CAI compensate for the bilateral distal deficits with proximal compensatory strategies are required.

4.1. Limitations

The lack of mechanical variables is the major limitation of the present study. Future studies assessing centre of pressure displacement are needed to evaluate the impact of the postural control impairments of the FAI group and the compensatory postural control strategies of the MAI group on the global mechanical output to assess more accurately their relation with risk of injury. The level of disability of the CAI group was not assessed in the present study, which limits the comparisons of the results obtained in the present study with those obtained in previous studies. Given the low observed power in some comparisons, future studies involving a higher sample are required to confirm the non-existence of significant differences observed in the present study.

4.2. Clinical implications

The results of the present study point to the need for rehabilitation specialists to include both lower limbs in rehabilitation strategies in individuals with unilateral ankle sprain episodes to restore proper motor control. Strategies should be adopted to increase TA CPA in both the injured and the uninjured limbs while assuming a support position in subjects with CAI. Future studies focusing on the rehabilitation of this impairment are required.

5. Conclusion

The results of the present study demonstrate that subjects with FAI present decreased TA CPA in both limbs while assuming a support position, and decreased SOL voluntary CPA in the uninjured limb in both positions. Subjects with MAI revealed in both the injured and the uninjured limbs increased PB CPA in a support position and of PB and SOL CPA in the side of the perturbation.

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