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

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ABSTRACT

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 compensatory 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.

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

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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

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, <i>n</i> = 4 Frequently, <i>n</i> = 7 Often, <i>n</i> = 3	Rarely, <i>n</i> = 4 Frequently, <i>n</i> = 3 Often, <i>n</i> = 3	
Severity of ankle sprain	–	Moderate ankle sprain, <i>n</i> = 13 Mild ankle sprain, <i>n</i> = 1	Severe ankle sprain, <i>n</i> = 1 Moderate ankle sprain, <i>n</i> = 9	
Time since last sprain (months)	–	7.7 (4.08)	10.4 (1.72)	
	<i>n</i> = 20	<i>n</i> = 14	<i>n</i> = 10	

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 than 3mm 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

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