Influence of wearing an unstable shoe construction on compensatory control of posture

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Abstract

This study investigated the influence of wearing unstable shoe construction (WUS) on compensatory postural adjustments (CPA) associated with external perturbations. Thirty two subjects stood on a force platform resisting an anterior-posterior horizontal force applied to a pelvic belt via a cable, which was suddenly released, under two conditions: barefoot and WUS. The electromyographic (EMG) activity of gastrocnemius medialis, tibialis anterior, rectus femoris, biceps femoris, rectus abdominis, and erector spinae muscles and the centre of pressure (CoP) displacement were acquired to study CPA. The EMG signal was used to assess individual muscle activity and latency, antagonist co-activation and reciprocal activation at joint and muscle group levels. Compared to barefoot, WUS led to: (1) increased gastrocnemius medialis activity, (2) increased total agonist activity, (3) decreased antagonist co-activation at the ankle joint and muscle group levels, (4) increased reciprocal activation at the ankle joint and muscle group levels, and (5) decrease in all muscle latencies. No differences were observed in CoP displacement between conditions. These findings demonstrate that WUS led to a reorganization of the postural control system associated to improved performance of some components of postural control responses.

***Key words*:** Posture; External perturbation; Compensatory Postural Adjustments; Electromyography; Centre of pressure; Unstable shoe construction.

1. Introduction

The ability to compensate for external perturbations is important to prevent falls and to ensure safe and independent mobility. Evoked compensatory postural muscle responses are produced when instability occurs (Britton et al., 1993) and are of shorter latency than voluntary activation of the same muscles (Gage et al., 2007; Maki & McIlroy, 1997). Despite a very short latency, the balance-recovery reactions are remarkably complex. Triggered and modulated by multiple sensory inputs, these reactions are highly adaptable to meet functional demands, as defined by the features of the perturbation, the ‘‘central set’’ of the individual, ongoing cognitive or motor activity, environmental constraints on reaction-force generation and limb movement and the postural configuration adopted by the subject (Forssberg & Hirschfeld, 1994; Henry et al., 2001; Horak et al., 1989; Maki & McIlroy, 2007).

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 (Horak & Nashner, 1986; Inglis et al., 1994) has been identified as a key source of triggering information needed to initiate directionally specific, automatic postural responses following an unexpected postural perturbation. It is known that during quiet standing, sway of the entire body is highly correlated with ankle joint rotation, which shows that muscles crossing the ankle joint are able to provide the sensory information necessary to maintain upright standing (Fitzpatrick et al., 1994; Loram et al., 2005a). The ankle joint muscle proprioceptors which might provide this sensory information include those in calf muscles and the tibialis anterior. Ankle plantar flexors act as active agonists and, because the foot is constrained

on the ground, these muscles prevent forward toppling of the body whose centre of mass is maintained in front of the ankle joint (Fitzpatrick et al., 1992; Lakie et al., 2003; Loram & Lakie, 2002; Loram, et al., 2005a; Maki & Ostrovski, 1993). The main antagonist, tibialis anterior, may be a source of muscle proprioceptive input when stretched by body sway. However, these roles are dynamic, and apparently reversible according to the position of the centre of mass in relation to the ankle joint (Di Giulio et al., 2009).

Recently, shoe manufacturers have introduced specific shoes featuring unstable sole constructions to induce neuromuscular stimuli similar to balance training, e.g. Masai Barefoot Technology (MBT) shoes. MBT shoes are characterised by a rounded sole in the anterior-posterior direction with a soft pad underneath the rear foot and are supposed to increase microscopic movement variability during standing (Nigg et al., 2006) and walking (Stöggl et al., 2010), thus enhancing sensory feedback to the locomotor system (Collins et al., 2003). It has been demonstrated that wearing this kind of unstable shoe leads to changes in the ankle control pattern during quiet standing (Landry et al., 2010; Sousa et al., 2012) and gait (Romkes et al., 2006). However, most studies related to postural control have been focused only in centre of pressure (CoP) excursions (Landry, et al., 2010; Ramstrand et al., 2010; Turbanski et al., 2011). To the best of our knowledge, no previous study has analysed the influence of unstable shoe wearing on muscle compensatory responses.

The main purpose of this study was to analyse the influence of wearing unstable shoe construction (WUS) on compensatory postural adjustments (CPA) to an external perturbation. More specifically, the purposes were to evaluate the effect of WUS on 1) muscle latency and activity and 2) CoP

displacement associated with external perturbations. Since postural responses involve activation of muscle synergies throughout the entire body and are also more context-specific, more flexible and adaptable than spinal proprioceptive reflexes (Horak & Macpherson, 1996), muscle activity was analysed not only in terms of individual magnitude but also in terms of degree of antagonist co-activation and reciprocal activation at joint and muscle group levels. As such, a decrease of muscle onset latency, higher level of muscle activity and higher antagonist co-activation level would be expected. The results of this study contribute to understand how WUS affects postural control.

2. Methodology

2.1 Subjects

Thirty-two healthy female subjects (age = 34 ± 9 years, height = 1.61 ± 0.06 m, weight = 63.2 ± 9.3 kg; mean ± SD) took part in the experiment; possible candidates were excluded if they presented a recent osteoarticular and musculotendinous injury or surgery of lower extremities, a background of or signs of neurological dysfunction or medication that could affect motor performance and balance and individuals who had used unstable footwear (specifically Masai Barefoot Technology - MBT, Figure 1) prior to the study. Only female subjects were included because this study is part of a global project whose major goal was related to the study of the influence of unstable shoe construction on venous return in subjects with risk factors for developing venous insufficiency.

The study was conducted according to the ethical norms of the Institutions involved and conformed to the Declaration of Helsinki, with informed consent from all participants.

2.2 Instrumentation

The electromyography (EMG) of *gastrocnemius medialis* (GM), *tibialis anterior* (TA), *rectus femoris* (RF), *biceps femoris* (BF)*, rectus abdominis* (RA), and *erector spinae* (ES) muscles were monitored using the MP 150 Workstation model from Biopac Systems, Inc. (USA), with steel surface electrodes, TD150 model, bipolar configuration, with a 20 mm interelectrode distance and a ground electrode. The selection of these muscles was based on the fact that the unstable shoe construction used in this study have a rounded sole in the anterior-posterior (AP) direction and the perturbation was applied in the same direction.

CoP values were obtained using a force plate, model FP4060-10 from Bertec Corporation (USA), connected to a Bertec AM 6300 amplifier, with default gains and a 1000 Hz sampling rate. The amplifier was connected to a Biopac 16 bit analog-to-digital converter.

2.3 Procedures

2.3.1 Skin preparation and electrode placement

The subjects' lower limb skin surfaces were prepared to reduce electrical resistance to less than 5000 Ω. Measurement electrodes were placed at GM, TA, RF, BF, ES and RA mid-belly according to anatomical references (Table 1)

and fixed with adhesive tape (Basmajian & De Luca, 1985; Hermens et al., 2000).

2.3.2 Experimental setup

Each subject performed two tests in a randomized order: one standing barefoot and another WUS. Subjects were instructed to stand relaxed, with feet comfortably spaced and arms at sides, and to look straight ahead to a target set 2 m away (Fransson et al., 1999). Considering that listening to different types of music does not significantly change the stabilometric variables (Forti et al., 2010), headphones were used to listen to music to mask any auditory cues and to distract the subject from consciously modifying her motion. A horizontal cable was attached to a pelvic belt worn by the subjects while they kept their bodies essentially straight. A backward force of 5% of body weight (Krebs et al., 2001; Wolfson et al., 1986), measured with an isometric dynamometer, was applied to the cable for a random period of 3 to 10 seconds and then the cable was released (time zero, T_0). A 1-minute rest interval was set between each test to prevent fatigue (Maki, 1986). Test instructions to the subject were: “Stand still but compensate the force applied to the belt without moving your feet. I will let go at some point, but you will not know when. Do not move your feet, but keep your balance.” The vertical orientation of the subjects was standardized by visual inspection and using a plumb line, and also trough online assessment of the ground reaction force signal variation. Besides this precaution, the results obtained in a pilot study as to the inclination of the unstable shoe after applying the horizontal force demonstrated that the ankle dorsiflexion angle was not greater than 5º, which is not enough to produce changes in group Ia afferent

feedback or in plantar and dorsiflexor muscle activity levels (Mezzarane & Kohn, 2007). Each subject performed two series, one for each testing condition, being each series comprised by three trials. As no noteworthy differences were verified between the first and the remainder trials, the average values were used for analysis. Measurements were performed on the dominant limb found after asking the subjects to kick a ball. All subjects were right limb dominant. Before data acquisition, all subjects were given time to become familiar with the test environment (Maki, 1986) and were explained by a qualified instructor on how to use the unstable shoe, followed by approximately 10 minutes of walking, until the instructor felt they walked properly and were comfortable using the shoes (Nigg, et al., 2006).

The EMG signals were acquired according to a sample rate of 1000 Hz, pre-amplified at the electrode site, fed into a differential amplifier with an adjustable gain setting (12-500 Hz; Common Mode Rejection Ratio (CMRR): 95 dB at 60 Hz and input impedance of 100 MΩ), digitised and then stored in a computer for subsequent analysis based on the Acqknowledge software (Biopac Systems, Inc. USA). The gain range was set to 1000.

The muscle latency was detected in a time window from -450 to +200 ms in relation to T_0 (Santos et al., 2009) using a combination of computational algorithms and visual inspection to ensure the non-existence of identifications not corresponding to the onset times. The latency for a specific muscle was defined as the instant lasting for at least 50 ms when its EMG amplitude was higher (activation) or lower (inhibition) than the mean of its baseline value plus 1 (one) standard deviation (SD) (Hodges & Bui, 1996), measured from -500 to -450 ms (Santos, et al., 2009). The signal was previously smoothed using a sixth

order elliptical low-pass software filter of 50 Hz, based on the findings described by Hodges & Bui, 1996.

The EMG activity of TA, GM, RF, BF, ES and RA was evaluated at pre-defined epochs. To assess the level of muscle activity, signals were previously band-pass filtered between 20 and 450 Hz and integrated with 150 ms time windows. The integral of EMG activity (Int_{EMGi}) was analysed at two epochs in relation to T_0: 1) 50 to 200 ms (compensatory postural adjustments 1 (CPA1)), and 2) 200 to 350 ms (late compensatory postural adjustments (CPA2)) (Latash, 2008). The integral of EMG activity inside each epoch was corrected by subtracting the calculated value from -500 to -450 ms prior to T_0 multiplied by 3 (Santos, et al., 2009). It should be noted that positive values indicate increased muscle activation, while negative values indicate a decrease in relation to background activity. Then, the Int_{EMGi} data were normalised to maximal isometric contraction for each subject (EMG_{norm}). After a warm-up consisting of 3 submaximal isometric contractions (Lehman & McGill, 1999) the TA and GM maximal isometric contractions were measured with the ankle in a neutral position, for the BF and RF the knee was positioned at 90º and for ES and RA subjects were lying in prone and supine position, respectively. Manual resistance was applied to all muscles. Reciprocal activation and antagonist co-activation were calculated for joint level (i.e., for muscles that span one joint) and muscle group level (group of muscles that span multiple joints). For the joint level, the muscles acting on the ankle (TA/GM pair), on the knee (RF/(GM+BF) pair) and on the trunk (RA/ES pair) were considered. For the muscle group level, the sum of the EMG_{norm} of all the dorsal (GM, BF and ES) and all the

ventral (TA, RF and RA) postural muscles was adopted. Taking into account that the perturbation applied caused a forward oscillation of the subject and the centre of mass position is reestablished through the action of the posterior muscles of the lower limbs and trunk, it was assumed the GM, BF and ES muscles to be the agonists in postural control response and the TA, RF and RA muscles to be their antagonists, respectively.

The antagonist co-activation at joint and muscle group levels during CPA1 and CPA2 were calculated using the following equations (Kellis et al., 2003):

1. Antagonist co-activation at the joint level:

$$Antagonist\ co\text{-}activation_{TA/GM\ pair} = \frac{EMGnorm_{TA}}{EMGnorm_{GM} + EMGnorm_{TA}} \times 100\,, \quad (1)$$

$$Antagonist\ co-activation_{RF/(BF+GM)\ pair} = \frac{EMGnorm_{RF}}{EMGnorm_{(BF+GM)} + EMGnorm_{RF}} \times 100 \quad (2)$$

$$Antagonist\ co-activation_{RA/(ES+RA)\ pair} = \frac{EMGnorm_{RA}}{EMGnorm_{(ES)} + EMGnorm_{RA}} \times 100\,. \quad (3)$$

2. Antagonist co-activation at the muscle group level:

$$Antagonist\ co-activation_{ventral/dorsal\ pair} = \frac{EMGnorm_{(TA+RF+RA)}}{EMGnorm_{(GM+BF+ES)} + EMGnorm_{(TA+RF+RA)}} \times 100\,. \quad (4)$$

This approach provides an estimate of the relative activation of the pair of muscles, as well as the magnitude of the co-activation.

The reciprocal activation at joint and muscle group levels during CPA1 and CPA2 was calculated using the following equations (Slijper & Latash, 2004):

1) Reciprocal activation at the joint level:

$$Reciprocal\ activation_{TA/GM\ pair} = EMGnorm_{GM} - EMGnorm_{TA}\,, \quad (5)$$

$$Reciprocal\ activation_{RF/(BF+GM)\ pair} = EMGnorm_{(BF+GM)} - EMGnorm_{RF}\,, \quad (6)$$

$$Reciprocal\,activation_{RA/ES\ pair} = EMGnorm_{ES} - EMGnorm_{RA}. \quad (7)$$

2) Reciprocal activation at the muscle group level:

$$Reciprocal\,activation_{ventral/dorsal\ pair} = EMGnorm_{(GM+BF+ES)} - EMGnorm_{(TA+RF+RA)}. \quad (8)$$

The acquired force time series of each trial were used to calculate the CoP fluctuation in the AP direction (as the perturbations were induced symmetrically) using the approximation:

$$CoP_{AP} = \frac{M_x}{F_z}, \quad (9)$$

where M_x is the moment of the force in the sagittal plane and F_z is the vertical component of the ground reaction force. A fourth-order, zero phase-lag, low-pass Butterworth filter with a cut-off frequency of 20 Hz was applied to all CoP displacement time series. The AP standard deviation (SD_{AP}) and peak-to-peak ($P\text{-}P_{AP}$) distance of the CoP were measured in the following epochs: (1) +100 to +250 ms (CPA1); (2) +250 to +400 ms (CPA2). These values were selected to compensate for the electromechanical delay and were corrected as to basal values, which were obtained during unperturbed standing (Cavanagh & Komi, 1979; Howatson et al., 2009).

2.4 Statistics

The data were analysed using the software Statistic Package Social Science (SPSS) from IBM Company (USA). Differences in individual muscle activity, antagonist co-activation, reciprocal activation and CoP parameters between WUS and barefoot conditions and between CPA1 and CPA2 were analysed using the Friedman ANOVA test. Muscle onset and offset between the

two conditions were evaluated using the Wilcoxon test. Non-parametric tests were adopted because the Shapiro-Wilk test of normality and the histogram analysis indicated that the variables did not follow a normal distribution.

3. Results

3.1 Influence on EMG activity in CPA at individual, joint and muscle group levels

WUS led to an increased activity of GM and of total agonist activity compared to barefoot (Table 2). The GM activity was higher in CPA1 than in CPA2 for both conditions, while the opposite was verified in TA (Table 3).

Measurements obtained when WUS gave a lower value of antagonist co-activation at the ankle (CPA1, $p<0.0001$; CPA2, $p<0.0001$) and at the knee (CPA2, $p=0.013$) comparing to barefoot condition, Figure 2. In both conditions, antagonist co-activation was higher in CPA2, comparing to CPA1, at the knee and at the ankle only in the barefoot condition. At the muscle group level (Figure 2), the antagonist co-activation was also lower when WUS than in barefoot during CPA2 ($p=0.006$). Additionally, the co-activation at the muscle group level was higher in CPA2 than in CPA1 for both conditions (Table 3, Figure 2).

WUS led to higher values of ankle reciprocal activation (CPA1, $p<0.0001$; CPA2, $p<0.0001$) relatively to the barefoot condition, Figure 3. At this level the comparison of reciprocal activation between CPA1 and CPA2 indicated a higher value in CPA2 when WUS and in CPA1 for the barefoot condition. Reciprocal activation levels were also higher when WUS at muscle group level (CPA1, $p=0.001$; CPA2, $p=0.001$). At this level there was higher reciprocal activation in CPA1 than in CPA2 for both conditions.

3.2 Influence on muscle latency

Average values indicate a distal to proximal activation sequence and a distal to proximal deactivation pattern in barefoot and in WUS. Significant differences occurred between barefoot and when WUS in onset latency of GM and BF ($p=0.001$ and $p=0.016$, respectively) and in offset latency of TA and RF ($p<0.0001$ and $p=0.022$, respectively). In spite of these differences, there were no noteworthy differences in the time between ventral muscle deactivation and dorsal muscle activation in barefoot and in WUS, Figure 4.

3.3 Influence on CoP displacement in CPA

No statistical significant differences were observed in $P\text{-}P_{AP}$ and SD_{AP} between measurements obtained with and without the unstable shoe (Table 2).

4. Discussion

This study aimed to analyse the influence of WUS on CPA at individual muscle, joint and muscle group levels. Starting with the individual muscle level, the results demonstrate that WUS leads to increased GM activity, reflecting ankle strategy use. In fact, the major differences in terms of individual muscle activity occurred at the ankle joint, which is in line with other studies that analised quiet standing (Sousa, et al., 2012), gait (Romkes, et al., 2006) and running (Boyer & Andriacchi, 2009). According to Ivanenko, Levik et al. (1997), when standing on a rocking support, usually humans do not move the CoM, shifting instead the point of contact of the rocking platform with the ground under the CoM, which leads to an increased need of gastrocnemius activation (Ivanenko et al., 1997). Increased GM activity is consistent with values obtained at the muscle group level.

Assuming the hypothesis that the activity of all muscles within the system is interdependent (Feldman & Levin, 1995), values of antagonist co-activation and reciprocal activation were calculated at different joint and at muscle group levels. The decreased antagonist co-activation was a surprising finding since "Freezing degrees of freedom" has been described as a primitive strategy when mastering a new skill (Baratta et al., 1988; De Luca & Mambrito, 1987) and because it has been shown that subjects use co-contraction control to offset the effects of destabilising forces (Burdet et al., 2001). The higher activity of GM when WUS could be associated with the lower levels for leg antagonist co-activation as there is evidence that the level of inhibition of the antagonists increases in proportion to the level of motor activity in the agonists (Lavoie et al., 1997). Evidence suggests that the regulation of antagonist co-activation during a muscle action is continuously controlled by the nervous system (Nielsen & Kagamihara, 1992, 1993), and that it may be centrally mediated by a descending "common drive" (De Luca & Mambrito, 1987). An interesting finding of this study is that in the barefoot condition there was higher leg and muscle group co-activation levels in CPA2, which is consistent with the role of the CNS in controlling co-activation. No statistical significant differences were found between CPA1 and CPA2 at the ankle when WUS. This can be explained by the enhanced reflex excitability that increases the role of the stretch reflex in posture control during standing on an unstable support area (Dietz et al., 1980). In fact, changes in strategy for maintaining the upright posture to adapt postural control to an unstable support area have been demonstrated (Horak & Nashner, 1986). More specifically, it was shown that the antagonist co-activation declines in unstable dynamical tasks (Milner & Cloutier, 1993). The decreased

antagonist co-activation observed when WUS was associated with higher reciprocal activation values for leg and muscle group levels which is consistent with the idea that reciprocal inhibition is stronger in tasks involving joint movement than during voluntary activity of postural maintenance (Lavoie, et al., 1997). Another explanation for the higher level of reciprocal activation and lower level of co-activation for the ankle joint and muscle group levels when WUS could lie in the muscles analysed. The gastrocnemius is a phasic muscle (Di Giulio, et al., 2009) and as a result it developed higher activity to compensate for the external perturbation. Data from the soleus muscle was not acquired in this work and as such the co-activation between soleus and TA can be different from that observed between GM and TA, as the soleus is a tonic muscle (Di Giulio, et al., 2009). However, despite being a phasic muscle, increased activity of the GM muscle has been demonstrated during a co-contraction task, which may indicate that the central command eliciting co-contraction across the ankle joint involves selective activation of this muscle (Nielsen et al., 1994).

Despite the occurrence of different temporal sequences of muscle deactivation/activation, a predominance of a distal to proximal deactivation/activation was observed, suggesting a major use of the ankle strategy. The decrease of agonist onset latency and antagonist offset latency when WUS indicate a higher performance of the postural control system in this condition. Considering the increase of GM activity (Sousa, et al., 2012) and of postural sway while standing with unstable shoes (Landry, et al., 2010), it can be suggested that the higher performance results from a higher neural drive associated to augmented gamamotoneuron activity that leads to a higher sensitivity of the muscles spindles (Ivanenko et al., 1999; Ribot-Ciscar et al.,

2000). Another possibility for the higher postural performance could be an increase of Ia-afferent input onto motoneuron pool of the lower limbs (Loram et al., 2005b) as a result of higher muscle length changes while WUS. To test this hypothesis, it would be relevant to evaluate muscle length changes in triceps surae and TA muscles in this condition in future studies. Another possible source of increased sensory information while WUS may come from the cutaneous afferents of the feet (Kennedy & Inglis, 2002; Wright et al., 2012) as changes in plantar pressure distribution were found in this condition (Stewart et al., 2007). A third possibility could be an increase of vestibular input based on the fact that this afference plays a higher role in situations of increased postural instability (Fitzpatrick and McCloskey 1994) for triggering or coordinating the muscle activation patterns (Horak et al., 1990). However, because almost subjects used ankle strategy, this hypothesis is not sustained by our results (Horak, et al., 1990).

The reorganization of postural control responses observed in the present study resulted from the instability of the support surface provided by the unstable shoe construction adopted (MBT) (Landry, et al., 2010) and the findings should not be generalized to other types of unstable footwear construction. It is important to note that, despite increasing instability, the results obtained as to CoP parameters during CPA1 and CPA2 indicate that WUS did not perturb the performance of the compensatory postural responses.

5. Conclusions

The results obtained reveal that WUS led to a reorganization of postural control responses expressed through a decrease of onset agonist muscle latencies and lower levels of co-activation, higher levels of reciprocal activation

and higher GM and total agonist activity during CPA. The results also demonstrate that the instability provided by the unstable shoe construction adopted did not perturb the performance of the postural control system during CPA. Overall, the findings obtained indicate that the reorganization of the postural system while WUS improved the performance of some components of postural control responses.

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TABLE CAPTIONS

Table 1: Anatomical references used to locate the electrodes. (Electrode locations were confirmed by palpation of the muscular belly with the subject in the test position; the electrodes were placed on the most prominent area.)

Table 2: Mean and standard deviation values for GM, TA, BF, RF, ES and RA EMG activity and total agonist and antagonist muscle activity obtained during CPA1 and CPA2 associated with external perturbations, with and without unstable footwear. (Non-significant values are indicated as ns.)

Table 3: Proof values (p-values) obtained from comparisons of individual muscle activity, reciprocal activation and co-activation at joint and muscle group level, total agonist and antagonist activity and CoP displacement between CPA1 and CPA2. (Non-significant values are indicated as ns.)

FIGURE CAPTIONS

Figure 1: Unstable shoe model used in this study: The Masai Barefoot Technology (MBT) shoe has a rounded sole in the anterior-posterior direction, thus providing an unstable base.

Figure 2: Representation of mean and standard deviation values of co-contraction index at the joint and muscle group levels during CPA in barefoot and wearing unstable shoe.

Figure 3: Representation of mean and standard deviation values of reciprocal activation at the joint and muscle group levels during CPA in barefoot and wearing unstable shoe.

Figure 4: (a) Representation of the organisation of the average postural responses (TA and RF offset, GM and BF onset) activated in response to an external perturbation in barefoot and wearing unstable shoe. Muscle latency of trunk muscles were not analysed as no differences were observed at all levels between the two conditions under comparison. (b) Descriptive analysis on muscle deactivation/activation sequences.

FIGURES

Figure 1

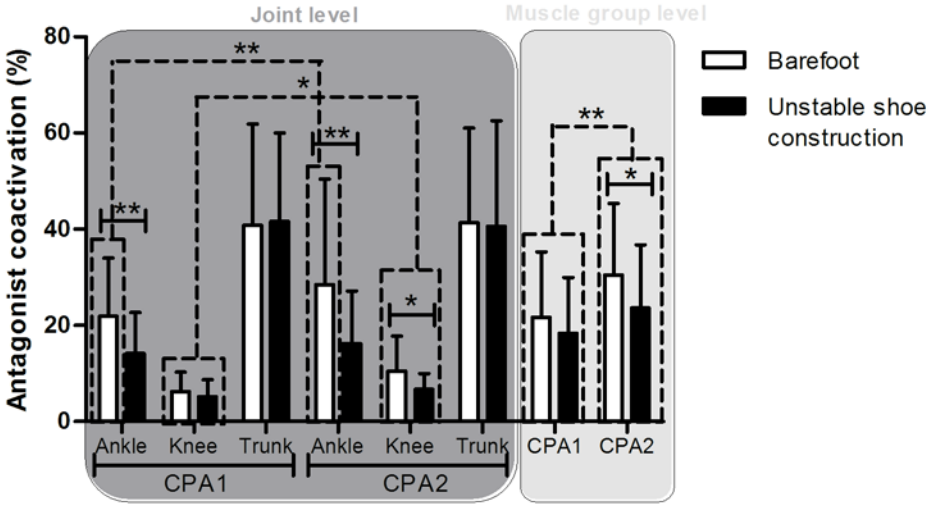


Figure 2

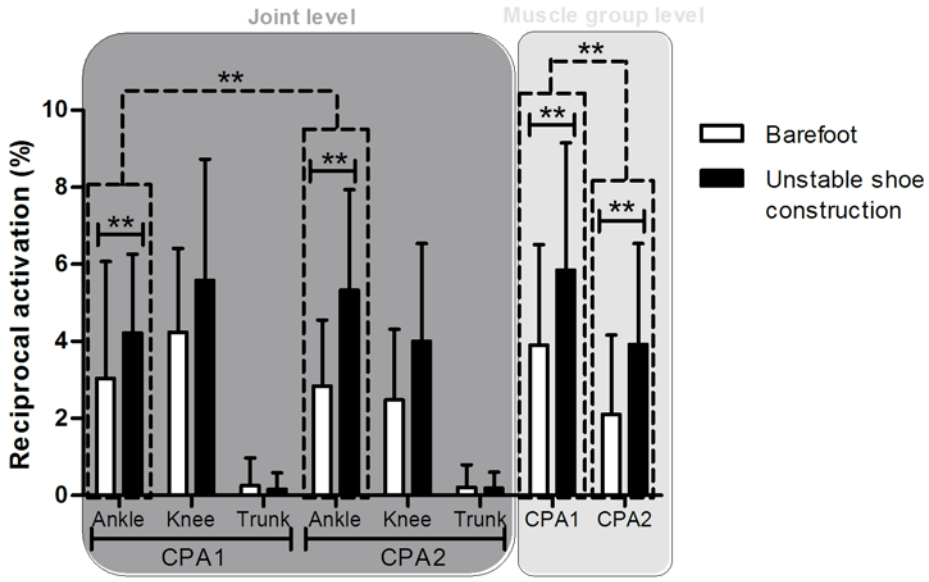
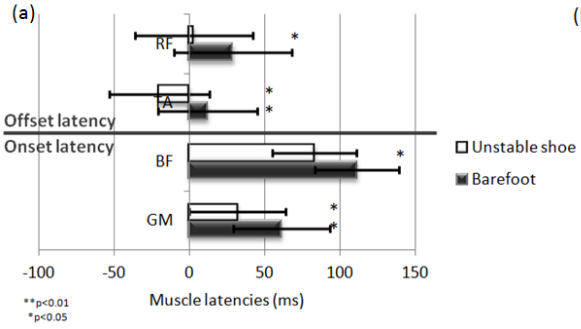


Figure 3



(b)

Condition	Muscle deactivation/activation sequence	Occurrences
Barefoot	TA → RF deactivation / GM → BF activation	75%
	RF → TA deactivation / GM → BF activation	12.5%
	TA → RF deactivation / BF → GM activation	9.38%
	RF → TA deactivation / BF → GM activation	3.12%
Unstable shoe	TA → RF deactivation / GM → BF activation	59.38%
	RF → TA deactivation / GM → BF activation	21.88%
	TA → RF deactivation / BF → GM activation	15.62%
	RF → TA deactivation / BF → GM activation	3.12%

Figure 4

TABLES

Table 1

Muscle	Electrode placement
TA	A third way along the line between the tip of the tibia and the tip of the medial malleolus
GM	Most prominent bulge of the muscle
RF	Halfway along the line from the anterior spina iliaca to the superior border of the patella
BF	Halfway along the line from the ischial tuberosity and the lateral epicondyle of the tibia
RA	Three cm to the right of the umbilicus
ES	Two finger widths lateral from the spinous process of L1
Ground electrode	Patella centre

Table 2

CPA	Muscle	Condition	Mean (%)	Standard Deviation	p-value
CPA1	GM	Barefoot	3.86	2.22	<0.0001
		Unstable shoe	6.33	3.00	
	TA	Barefoot	-0.35	0.77	ns
		Unstable shoe	-0.59	1.25	
	BF	Barefoot	0.70	0.59	ns
		Unstable shoe	0.66	0.52	
	RF	Barefoot	0.29	0.17	ns
		Unstable shoe	0.37	0.26	
	ES	Barefoot	0.62	0.68	ns
		Unstable shoe	0.54	0.36	
	RA	Barefoot	0.36	0.24	ns
		Unstable shoe	0.38	0.28	
	Total agonist	Barefoot	5.17	2.48	P<0.0001
		Unstable shoe	7.52	3.19	
	Total antagonist	Barefoot	0.3	0.87	ns
		Unstable shoe	0.16	1.20	
	CoP	**Condition**	**Mean (m)**	**Standard Deviation**	**p-value**
	$P\text{-}P_{AP}$	Barefoot	0.0395	0.0132	ns
		Unstable shoe	0.0421	0.0132	
	SD_{AP}	Barefoot	0.0118	0.0041	ns
		Unstable shoe	0.0125	0.0042	
CPA2	GM	Barefoot	2.35	1.79	<0.0001
		Unstable shoe	4.56	2.42	
	TA	Barefoot	-0.49	0.89	ns
		Unstable shoe	-0.77	1.36	
	BF	Barefoot	0.58	0.54	ns
		Unstable shoe	0.62	0.52	
	RF	Barefoot	0.28	0.21	ns
		Unstable shoe	0.34	0.23	
	ES	Barefoot	0.54	0.54	ns
		Unstable shoe	0.55	0.32	
	RA	Barefoot	0.33	0.21	ns
		Unstable shoe	0.36	0.26	
	Total agonist	Barefoot	3.47	1.89	P<0.0001
		Unstable shoe	5.73	2.55	
	Total antagonist	Barefoot	0.12	1.02	ns
		Unstable shoe	0.08	1.35	
	CoP	**Condition**	**Mean (m)**	**Standard Deviation**	**p-value**
	$P\text{-}P_{AP}$	Barefoot	0.0039	0.0014	ns
		Unstable shoe	0.0043	0.0025	
	SD_{AP}	Barefoot	0.0010	0.0004	ns
		Unstable shoe	0.0011	0.0008	

Table 3

Level	Variable compared		p-value (CPA1 and CPA2 comparisons)
Individual	TA		Barefoot: p=0.036
			Unstable shoe: p=0.012
	GM		Barefoot: p<0.0001
			Unstable shoe: p<0.0001
	RF		Barefoot: ns
			Unstable shoe: ns
	BF		Barefoot: ns
			Unstable shoe: ns
	RA		Barefoot: ns
			Unstable shoe: ns
	ES		Barefoot: ns
			Unstable shoe: ns
Joint	Reciprocal activation	Ankle	Barefoot: p<0.0001
			Unstable shoe: p<0.0001
		Knee	Barefoot: ns
			Unstable shoe: ns
		Trunk	Barefoot: ns
			Unstable shoe: ns
	Antagonist co-activation	Ankle	Barefoot: p=0.006
			Unstable shoe: ns
		Knee	Barefoot: p<0.0001
			Unstable shoe: p=0.021
		Trunk	Barefoot: ns
			Unstable shoe: ns
Muscle group	Total agonist		Barefoot: ns
			Unstable shoe: ns
	Total antagonist		Barefoot: ns
			Unstable shoe: ns
	Reciprocal activation		Barefoot: p<0.0001
			Unstable shoe: p<0.0001
	Antagonist co-activation		Barefoot: p<0.0001
			Unstable shoe: p<0.0001
CoP	$P\text{-}P_{AP}$		Barefoot: ns
			Unstable shoe: ns
	SD_{AP}		Barefoot: ns
			Unstable shoe: ns