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ORIGINAL ARTICLE

Decreased supraspinal control and neuromuscular function controlling the ankle joint in athletes with chronic ankle instability

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Abstract

Purpose Chronic ankle instability (CAI) alters lower extremity neuromuscular function, associated with a change in corticomotor excitability. The aim of this study was to compare corticomotor excitability and neuromuscular function of the muscles around the ankle between athletes with CAI and without CAI (non-CAI).

Methods Nineteen CAI athletes (15 men and 4 women) and 19 non-CAI athletes (15 men and 4 women) participated (age- and sex-matched). Corticomotor excitability was measured by transcranial magnetic stimulation for the following muscles: the tibialis anterior (TA), peroneus longus (PL) and gastrocnemius medialis (GM). The resting motor threshold (rMT), motor evoked potential (MEP), and latency (Lat) were subsequently measured. Neuromuscular function was assessed with a jump test, using the EMG activity before foot contact, peak torque, and joint position sense.

Results The corticomotor excitability in CAI showed a lower normalized MEP in the TA ($p = 0.026$) and PL ($p = 0.003$), and longer latency in the TA ($p = 0.049$) and GM ($p = 0.027$) compared with non-CAI. The neuromuscular assessment showed CAI had less EMG activity of the PL ($p < 0.001$), less peak torque of the dorsiflexor ($p = 0.019$) muscle compared with non-CAI.

Conclusion Athletes with CAI had lower corticomotor excitability in the TA and PL and a longer latency in the TA and GM muscles. Additionally, CAI demonstrated functional neuromuscular deficits by decreasing EMG activity of the PL muscle and strength of the dorsiflexor muscle. Our findings indicated maladaptation at both cortical and peripheral levels among athletes with CAI.

Keywords Ankle sprains · Chronic ankle instability · Cortical plasticity · Corticomotor excitability · Neuromuscular function · Supraspinal control

Abbreviations

CAI	Chronic ankle instability
Non-CAI	Non-chronic ankle instability
TA	Tibialis anterior
PL	Peroneus longus
GM	Gastrocnemius medialis
EMG	Electromyography
CAIT	Cumberland ankle instability tool
MVC	Maximum voluntary contraction
TMS	Transcranial magnetic stimulation
rMT	Resting motor threshold
MEP	Motor evoked potential
Lat	Latency

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CNS	Central nervous system
ACL	Anterior cruciate ligament
SPM	Statistical parametric mapping
SENIAM	Surface electromyography for the non-invasive assessment of muscles

Introduction

Ankle sprains are one of the most commonly reported sports injuries (Chan et al. 2011). An ankle sprain may lead to a decrease in the neuromuscular function of supporting muscles (Hertel 2000), leading to a condition termed chronic ankle instability (CAI), affecting the performance of athletes (Yeung et al. 1994; Hiller et al. 2011). CAI injuries appear to result in a reduction of sensory input followed by the reorganisation and alteration of motor unit recruitment. Related studies on CAI have frequently used landing skills to analyse the effect of injury on muscle activation (Delahunt et al. 2006; Allet et al. 2017; Brown et al. 2012). However, these studies have not fully addressed the change in corticomotor excitability in CAI. Therefore, the contribution of neural mechanisms to CAI-induced changes in motor control has not been differentiated based on the different levels of the central nervous system (CNS).

Participants with CAI have been shown to have deficits in the ground contact phase during jumping, the point where a number of neuromuscular events are activated in preparation for the ground impact (Neptune et al. 1999). The modified movement in the knee or ankle after injury may result in the adaptation of the CNS (Ward et al. 2015). In particular, studies have reported that an alteration in the CNS after a chronic anterior cruciate ligament (ACL) injury (Kapreli and Athanasopoulos 2006), or a chronic ankle sprain can be associated with supraspinal reorganisation (Needle et al. 2013; Pietrosimone and Gribble 2012).

The study of corticospinal or corticomotor excitability remains limited. Most CAI studies CAI have focused on structure, function, and performance (Allet et al. 2017). Few studies have confirmed the hypothesis that CNS alterations result from CAI adaptations (Sefton et al. 2008). Pietromone and Gribble (2012) observed that with CAI, a deficit in the corticomotor excitability of the peroneus longus (PL) muscle. Nonetheless, the underlying mechanism of how the corticomotor excitability affects neuromuscular function remains unclear. It has been proposed that when a ligament returns to its resting condition, it leads to nervous system alteration and subsequent instability (Needle et al. 2014b).

This study aimed to evaluate both central and neuromuscular control among athletes with functional CAI. The corticomotor excitability of the primary motor areas controlling the muscles around the ankle joint and ankle muscle activity was measured. We hypothesized that differences could be determined regarding corticomotor excitability and neuromuscular function between CAI and non-CAI groups.

Materials and methods

Participants

A matched-group design was used in this study. The sample size calculation was based on a related study (Pietrosimone and Gribble 2012), reporting corticomotor excitability of the PL muscle. The sample size was calculated as 18 participants per group. To obtain this sample size, a total of 116 basketball and volleyball athletes with CAI and without CAI (non-CAI) were screened from the university campus and affiliated sports clubs. A total of 19 athletes with chronic ankle instability (CAI) were subsequently enrolled in the study, and matched to uninjured athletes (non-CAI), based on age and sex (Table 1). The institutional review board of Mahidol University (MU-CIRB 2015/099.2406) approved the current study, and all participants provided their written informed consent.

Participants were classified in CAI and non-CAI groups using an ankle injury history questionnaire and a 30-point ankle instability tool (CAIT). The non-CAI group had no history of ankle sprain or symptoms of instability and recorded a CAIT score ≥ 28. The CAI group had a history of significant right ankle sprain (at least once), a feeling of the ankle, "giving way" and a CAIT score ≤ 24. In addition, the CAI group demonstrated clinically negative anterior drawer and talar tilt during the orthopaedic examination (Hiller et al. 2006). All participants were right-side dominant in controlling the cortical measurement. The dominant upper limb was defined using the Edinburgh Handedness Inventory scale, and the lower limb with which participants reported they would prefer to kick a ball or use to draw a number 8 on the floor (Li et al. 2018). In addition, participants were excluded from the study if they reported a history of any of

Table 1 Participant demographics

Characteristics	Group		p value[a]
	Non-CAI ($n=19$)	CAI ($n=19$)	
Gender (male/female)	15 M/4F	15 M/4F	
Age (years)	20.58 ± 1.30	20.58 ± 1.54	0.839
Height (cm)	175.00 ± 6.89	176.37 ± 9.52	0.179
Mass (kg)	68.18 ± 7.32	69.37 ± 10.55	0.207
Experience in sport			
Competition (years)	7.79 ± 1.55	8.47 ± 1.74	0.831
CAIT score	29.11 ± 1.05	18.47 ± 3.67	0.001*

*Significance was accepted for $p < 0.05$

[a]Independent t test. The p values are of between group comparisons

the following: (1) acute lower extremity injury, (2) fracture or surgery to either lower extremity and (3) failed safety-screening for transcranial magnetic stimulation (TMS) (Rossi et al. 2009).

Instrumentation

Corticomotor excitability was assessed using TMS with a double cone coil (Magstim 2002, Magstim Company; Inc, Morrisville, NC) to demonstrate the corticomotor excitability adaptations underlying the CAI condition (Goodall et al. 2014). A force platform (model 9286B; Kistler, USA) and wireless electromyography (EMG) were integrated with BTS Bioengineering Software (BTS Bioengineering, Italy) to record the activity of the muscle controlling the ankle joint during the initial contact (IC) of the jump-landing test. The EMG recording used dual circular Ag/AgCl disposable electrodes with adhesive areas of 3.3 cm, conductive area of 1.5 cm and an inter-electrode distance of 2 cm (BlueSensor, Ambu® Inc, Malaysia). An isokinetic dynamometer (model System 4 Pro™; Biodex Medical System, Inc, Shirley, NY, USA) was used to evaluate muscle strength, maximal voluntary muscle contraction and joint position sense.

Corticomotor excitability measurement

The assessment of corticomotor excitability required the EMG recording. The Ag/AgCl electrodes were positioned over the muscle belly area on the skin overlying the target muscles: including the tibialis anterior (TA), peroneus longus (PL) and gastrocnemius medialis (GM) muscles. These electrode positions were based on the European recommendations for the surface EMG for non-invasive assessment of the muscles (SENIAM). Once the EMG was configured, the participants sat in the TMS chair, and the hotspot for stimulation was found on the left primary motor cortex. The hotspots of these muscles have been identified in related studies (Fisher et al. 2016; Pietrosimone and Gribble 2012). To determine the hotspot for each muscle, the TMS coil was placed over the scalp at the vertex and moved antero-laterally or postero-laterally approximately in 1-cm increments until the greatest motor evoked potential (MEP) was stimulated (Fig. 1). Next, the resting motor threshold (rMT) was determined over the hotspot using single-pulse TMS. The rMT was found by systematically decreasing the stimulus intensity in 2% steps until no MEP was elicited. The amplitude of MEP was equal to or more than 50 µV in a target muscle in at least 50% of 10 consecutive trials. The peak-to-peak amplitude of MEP and Lat was assessed over the hotspot area using 120% of rMT to stimulate 10 repetitions

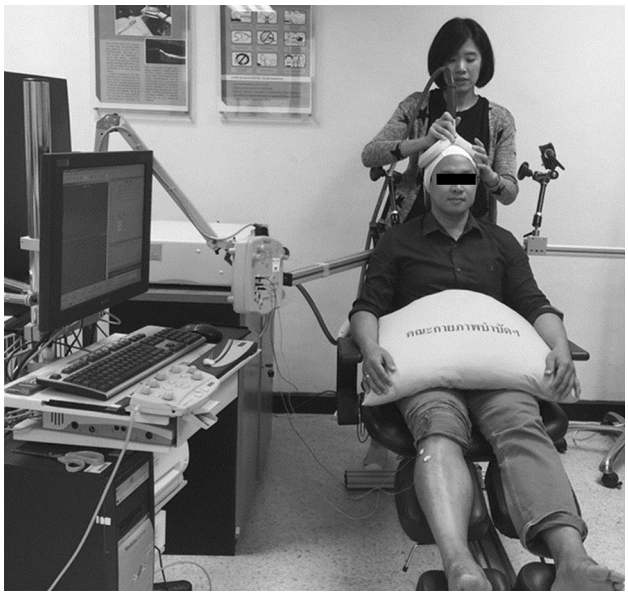

Fig. 1 Setup for corticomotor excitability measurement using transcranial magnetic stimulation

(Mileva et al. 2009). The latency was recorded from the duration between the TMS pulse and the onset of the EMG activity (Maeda and Pascual-Leone 2003). These procedures were repeated for each of the three muscles with a 15-min rest between muscle measurements.

Muscle function measurement

Muscle activity of the TA, PL, and GM muscles was recorded during the jump-landing test to quantify muscle activation before foot contact (Mrdakovic et al. 2008). The electrode was located over the TA, PL and GM muscles as per SENIAM recommendations with respect to muscle motor points and fibre directions (Hermens et al. 2000). Before the actual test, the jump-landing procedures were explained and demonstrated for all participants. The participants were instructed to carefully jump off the wooden platform without any upward jump motion, to look forward throughout the task and to land on their dominant leg. Then all participants completed a stretching warm-up, and were allowed to practice the jump-landing test. The jump-landing method involved three steps as described below. First, the participant stood on a 30-cm wooden platform and placed their hands on their waist. Then the participant changed to a single-leg stance with their non-dominant knee flexed to 90° with their hips in a neutral position after seeing the first light signal. Finally, the participant performed a one-legged jump-landing after seeing the second light signal (Fig. 2). The participants completed three trials with at least 30 s of rest between trials.

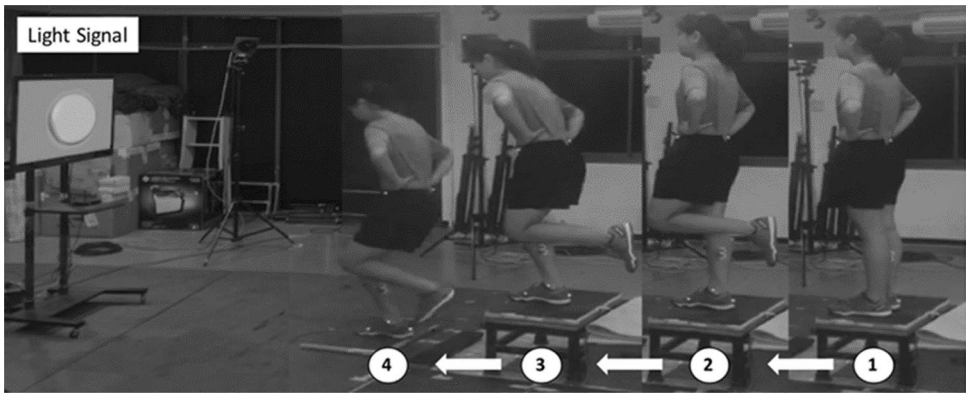

Fig. 2 The jump-landing test procedures were as follows: (1) the participants stood on a 30-cm wooden platform and placed their hands on their waist (2) when the first signal lit up, the participants moved into a single-leg stance with their non-dominant knee flexed to 90° and their hips in neutral (3) upon the second signal, they performed jump-landing tests (4) the participants landed on their dominant leg. They completed three trials with at least 30 s of rest between trials

Strength measurement

Isokinetic strength measurements (concentric/concentric) of ankle dorsiflexion, plantar flexion, inversion, and eversion were determined using a Biodex isokinetic dynamometer at speeds of 120° per second. The participants were positioned on the dynamometer in accordance with the manufacturer recommendations (Sekir et al. 2007). All participants were given the instruction to move their ankle with maximum effort for three repetitions in each of the four directions. Verbal encouragement/motivation was provided. The order of movements was randomised and each test movement was followed by at least 2 min of rest.

Ankle joint position sense

The position sense of the ankle joint was assessed in the four cardinal motions (ankle dorsiflexion, plantar flexion, inversion, and eversion) using the Biodex isokinetic dynamometer in the passive mode. The participants were set up as previously described (Willems et al. 2002) and then given a stop button to hold before having their eyes covered with a blindfold. The ankle was then passively moved by the dynamometer to the target angle, maintained for 10 s and moved back to the starting position. During the 10-s hold, the participants were asked to concentrate on the position of their ankle and foot. Next, the ankle was passively moved in the target direction, and the participants pressed the stop button when they felt that the tested angle had been reproduced.

Three trials for each of the four motions were completed at a velocity of 5° s^{-1}. The target angles were set as follows: inversion at 10°, eversion at 30°, dorsiflexion at 15°, and plantar flexion at 30° (Willems et al. 2002). All position tests were performed in the same manner, and the testing positions were randomly chosen. The error in degrees was recorded for analysis.

Data processing

All MEP amplitudes in each subject were normalized to the rMT. We chose rMT to normalize the MEP because they were measured at rest. The rMT represents resting membrane excitability of the neurons under the stimulating coil (Rossi et al. 2009). Therefore, it could represent the baseline excitability of each individual's brain. The normalized rate in microvolts per % of maximum output was used for the statistical analyses. The muscle onset time was defined as the duration before foot contact when muscle activity surpassed two standard deviations above passive standing baseline activity for at least 25 ms (P. and J 2010). The average amplitude of EMG activity was recorded at 200 ms before foot contact. This period was chosen as it covered the musculature preparation before landing from jumping (Santello and McDonagh 1998). All EMG data from the TA, PL, and GM muscles were detected at a sampling frequency of 1000 Hz and filtered at low-pass frequency (350 Hz) and high-pass frequency (30 Hz) with full wave rectified. The EMG amplitudes were normalized by the maximum voluntary contraction (MVC) of each muscle. The maximum EMG was obtained during the muscle strength test using the isokinetic dynamometer (model System 4 Pro™; Biodex Medical System, Inc, Shirley, NY).

Dependent measures

The corticomotor excitability

The corticomotor excitability of the TA, PL and GM muscles is presented as rMT, MEP and Lat (Fig. 3). These variables

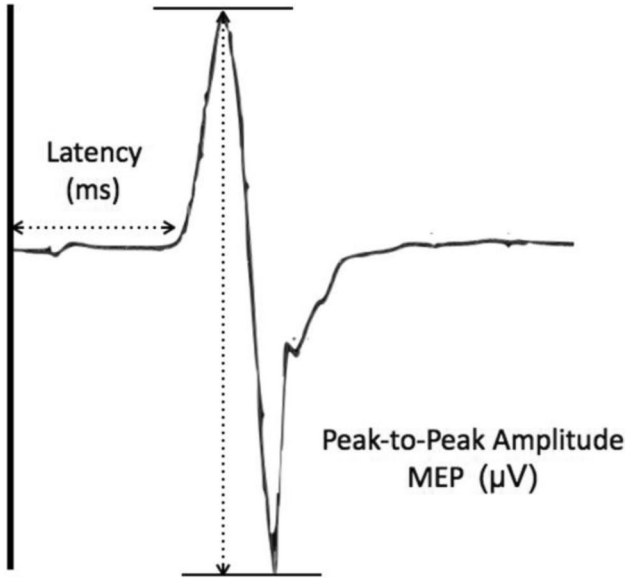

Fig. 3 Corticomotor excitability parameters including amplitude of Motor Evoked Potential (MEP) measured peak-to-peak amplitude (microvolts), and latency, which was defined as the duration between the transcranial magnetic stimulation pulse and the onset of the EMG activity

were recorded at rest. The rMT can represent membrane excitability of the corticospinal neurons and interneurons projecting in the motor cortex, and MEP can reflect the integration of the corticospinal tract and the excitability of the motor cortex (Kobayashi and Pascual-Leone 2003). The latency reveals the corticomotor conduction time of the signals that are sent from the cortical circuits to the peripheral muscle (Maeda and Pascual-Leone 2003).

The neuromuscular function

Muscle function was evaluated via muscle onset time and the average of EMG activity from the jump-landing test, recorded from the TA, PL and GM muscles. The onset and amplitude of the pre-landing EMG activity revealed the preparing strategies that are anticipated by the CNS (Santello and McDonagh 1998). Muscle strength, i.e. peak torque, was normalized with body weight.

Statistical analysis

The normality of data was tested using the Shapiro Wilk test. As the data were not normally distributed, multivariate Kruskal–Wallis test ($p < 0.05$) was performed to test for differences between CAI and non-CAI groups. Independent *t* tests were used to compare the demographic data between CAI and non-CAI groups. The correlation between corticomotor excitability and neuromuscular function was evaluated using Spearman's rank correlation. We classified correlation coefficients of 0–0.4 as weak, 0.41–0.7 as moderate and 0.71–1 as strong (Pietrosimone and Gribble 2012). The correlation statistical significance was set at $p < 0.05$. All statistical analyses were performed using SPSS, Version 18 (IBM, SPSS, Inc, Chicago, IL, USA).

Results

No significant differences were noted in the participant's age, height, mass or sporting experience between groups (Table 1). As anticipated, the CAI group had a significantly lower ($p = 0.001$) CAIT score than the non-CAI group.

Corticomotor excitability, as assessed via TMS, did not differ between groups when rMT for all the muscles was evaluated. The normalized MEP in the CAI group had significantly lower values compared with the non-CAI group regarding the TA ($p = 0.026$) and PL muscles ($p = 0.003$) (Table 2). In addition, the CAI group showed a significantly longer latency duration concerning the TA ($p = 0.049$) and GM ($p = 0.027$) muscles (Table 2).

The EMG, recorded at 200 ms before initial contact during the jump-landing test, showed significantly less activity of the PL ($p < 0.001$) muscles in the CAI compared with the non-CAI group (Table 3). No group differences were found in the time to peak torque or in joint position sense. However, a significantly lower peak torque was found in the dorsiflexor ($p = 0.019$) muscle in the CAI group (Table 4).

The correlations between corticomotor excitability and neuromuscular functions were reported by selecting the same muscle function. The tibialis anterior, dorsiflexor and invertor muscles were reported together; the peroneus longus and evertor muscle and the gastrocnemius medialis and plantar flexor. We found moderate correlations between corticomotor excitability and neuromuscular functions in both groups. While the non-CAI group demonstrated moderate correlations across muscles around the ankle joint, the CAI group had a correlation only in the TA muscle (Table 5).

Discussion

The purpose of this study was to evaluate the corticomotor and neuromuscular changes underlying chronic ankle instability. As anticipated, the athletes with chronic ankle instability (CAI) showed maladaptation in the ability of the CNS to control the musculature around the ankle joint. Additionally, at the peripheral control level, we found decreased muscle performance of the ankle joint.

Table 2 Corticomotor excitability of the muscles around the ankle joint

Corticomotor excitability	Non-CAI (mean ± SD)	CAI (mean ± SD)	*p* value
Resting motor threshold (rMT) (% of maximum output)			
Tibialis anterior (TA)	46.42 ± 7.03	47.84 ± 6.53	0.313
Peroneus longus (PL)	49.21 ± 7.65	49.11 ± 9.49	0.715
Gastrocnemius medialis (GM)	52.95 ± 7.82	51.58 ± 8.74	0.639
Normalized motor evoked potential (MEP) (μV/% of maximum output)			
Tibialis anterior (TA)	10.28 ± 6.76	5.80 ± 2.45	0.026*
Peroneus longus (PL)	4.93 ± 4.09	2.90 ± 1.15	0.003*
Gastrocnemius medialis (GM)	2.30 ± 1.30	2.95 ± 1.49	0.148
Latency (Lat) (ms)			
Tibialis anterior (TA)	29.14 ± 1.50	30.19 ± 1.38	0.049*
Peroneus longus (PL)	29.79 ± 2.18	30.62 ± 2.01	0.220
Gastrocnemius medialis (GM)	30.83 ± 2.48	32.16 ± 3.27	0.027*

*Significance was accepted for $p < 0.05$

Table 3 Results of EMG activity

EMG	Non-CAI (mean ± SD)	CAI (mean ± SD)	*p* value
Onset time (ms)			
Tibialis anterior (TA)	919.52 ± 401.69	942.39 ± 333.64	0.988
Peroneus longus (PL)	871.91 ± 406.16	812.54 ± 317.29	0.589
Gastrocnemius medialis (GM)	760.27 ± 376.65	757.42 ± 257.42	0.895
Average EMG (%MVC)			
Tibialis anterior (TA)	29.94 ± 27.74	21.71 ± 13.64	0.511
Peroneus longus (PL)	86.38 ± 68.39	46.05 ± 18.53	< 0.001*
Gastrocnemius medialis (GM)	102.74 ± 40.23	90.34 ± 54.94	0.184
MVC			
Tibialis anterior (TA)	386.48 ± 132.88	390.94 ± 129.29	0.919
Peroneus longus (PL)	207.96 ± 63.02	236.13 ± 77.49	0.287
Gastrocnemius medialis (GM)	278.08 ± 112.04	273.11 ± 83.49	0.872

*Significance was accepted for $p < 0.05$

Table 4 Results of the neuromuscular function

Neuromuscular function	Non-CAI (mean ± SD)	CAI (mean ± SD)	*p* value
Muscles' strength (peak torque)			
Plantar flexor muscle	74.28 ± 22.38	67.47 ± 19.84	0.405
Dorsiflexor muscle	29.69 ± 6.41	24.75 ± 5.97	0.019*
Evertor muscle	25.52 ± 4.77	22.02 ± 5.37	0.060
Invertor muscle	29.16 ± 5.09	26.94 ± 7.00	0.405
Time to peak torque (ms)			
Plantar flexor muscle	205.79 ± 63.78	204.95 ± 67.51	0.988
Dorsiflexor muscle	446.84 ± 246.96	432.26 ± 206.38	0.895
Evertor muscle	238.95 ± 128.02	330.47 ± 201.54	0.170
Invertor muscle	226.84 ± 81.45	252.21 ± 93.71	0.396
Joint position sense (error degrees)			
Plantar flexor muscle	4.25 ± 2.11	3.24 ± 1.42	0.096
Dorsiflexor muscle	2.23 ± 1.30	2.12 ± 1.28	0.672
Evertor muscle	1.57 ± 0.55	2.31 ± 1.67	0.266
Invertor muscle	2.45 ± 1.66	2.26 ± 1.08	0.804

*Significance was accepted for $p < 0.05$

Table 5 Correlations between supraspinal and neuromuscular function

Muscles	Supraspinal[a]	Neuromuscular function[b]	Correlation (p value)[c]	
			Non-CAI	CAI
Tibialis anterior (TA) Dorsiflexor muscle Invertor muscle	MEP.TA	MVC.TA	(−)	0.581 (0.009)*
		TPT.Dorsiflexor	−0.453 (0.050)*	(−)
		TPT.Inversion	(−)	0.528 (0.020)*
		PT.Invertor	−0.565 (0.012)*	(−)
	Lat.TA	MVC.TA	(−)	0.619 (0.005)*
		Onset-time.TA	−0.431 (0.066)	
Peroneus longus (PL) Evertor muscle	rMT.PL	TPT.Evertor	−0.401 (0.089)	(−)
	Lat.PL	EMG.PL	0.582 (0.009)*	(−)
Gastrocnemius medialis (GM)	MEP.GM	MVC.GM	0.423 (0.071)	(−)
		TPT.Plantarflexor	−0.422 (0.072)	(−)
Plantar flexor muscle	Lat.GM	PT.Plantarflexor	0.515 (0.024)*	(−)

*Significance was accepted for $p < 0.05$

(−) Weak correlation

[a]Supraspinal outcomes consist of resting motor threshold (rMT), motor-evoked potential (MEP), and latency (Lat)

[b]Neuromuscular function outcomes consist of onset-time, peak torque (PT), time to peak torque (TPT), Maximum voluntary contraction (MVC), and average EMG activity (EMG)

[c]Spearman signed rank test. The p values are of intergroup comparisons

Corticomotor excitability

It is hypothesised that a peripheral joint injury could adversely affect the normal functioning of cortical and spinal levels (Ward et al. 2015). Prior work with individuals experiencing an ACL injury has demonstrated alterations in the CNS, via changes in corticomotor excitability (Kapreli and Athanasopoulos 2006). Despite limited available research, similar results have also been demonstrated among subjects with CAI. It has been shown that a decrease in corticomotor excitability can occur in the PL muscle among non-elite participants with CAI (McLeod et al. 2015; Pietrosimone and Gribble 2012). However, we presently observed no differences between the CAI and non-CAI groups in rMT for the TA, PL and GM muscles. Whilst difficulties exist to explain the differences between our findings, the participants in our study were elite athletes who were currently undertaking their daily training and competition as usual. Therefore, any changes in neural plasticity may be partly related to the intensity and protocol of training (Galvan 2010). This may also explain why rMT did not differ between groups in our study; all the participants were currently at a similar level of training intensity and competitive skill. Another possible explanation might be the large variability in rMT because this parameter represents only the local density of excitatory interneurons (Rossini and Rossi 2007). Consequently, rMT only shows the membrane excitability of the neurons (Maeda and Pascual-Leone 2003).

The MEP, which reportedly has less inherent variability than rMT, may represent the integrity of the corticospinal tract and excitability of the corticomotor system (Rossini and Rossi 2007). In the present study, a significant reduction was found in the normalized MEP of TA and PL muscles in the CAI group. Therefore, it appears that these athletes with CAI had deficits in the corticomotor excitability of their TA and PL muscles. Thus, athletes with CAI may exhibit greater difficulty in generating a motor command to their TA and PL muscles. These findings provide insights in the cortical maladaptation, which is associated with CAI. Nevertheless, normalized MEP of the GM muscle was not changed among athletes with CAI. One possibility is that the GM muscle was able to maintain the integrity of corticomotor excitability. Additionally, the adaptation and compensation of the GM muscle may have occurred above the ankle joint, for example, the knee joint and/or hip joint (Brown et al. 2012; Doherty et al. 2016). Indeed, this is in agreement with related studies which have reported no subacute (Allet et al. 2017) or chronic (Suda et al. 2009) change in neuromuscular control of the GM muscle after an ankle sprain.

The duration of the neural signal transmissibility from the motor cortex to the targeted muscle was defined as the latency (Maeda and Pascual-Leone 2003). The current study showed that the latency for the TA and GM muscles was prolonged in the CAI group compared with the non-CAI group. This deficit in corticomotor excitability might contribute to an increased risk of re-injury due to a slowing in motor control, especially during sporting activities. Although the athletes with CAI showed delayed latency, we could not specify the altered part that may have affected the neurological purpose. To provide an in-depth explanation, future

studies are required to support the mechanisms of corticomotor excitability maladaptation with M-wave and H-reflex EMG responses. However, individuals with CAI have been reported to have a decreased ability to modulate the spinal reflexes during testing tasks (Sefton et al. 2008). Therefore, the greater latency with CAI may partly support the previous findings that the corticomotor pathway was altered.

Neuromuscular functions

To assess neuromuscular function, EMG activities during the 200 ms before foot contact in the jump-landing test, isokinetic muscles' strength and joint position sense were evaluated. The EMG data demonstrate the motor unit and spinal motor neuron activity engaged in cortical control, resulting from the recruitment of motor unit action potentials (Mezzarane et al. 2013).

The pre-landing event is anticipated by the CNS, which is the feed-forward control. Before foot contact (200 ms), the CAI group demonstrated a significant decrease in average EMG activity in the PL muscles. This finding is consistent with related work (Delahunt et al. 2006) and may be a factor in recurrent ankle injuries. Since less corticomotor excitability was observed in the CAI group, it may be hypothesised that motor unit recruitment ability was also decreased. This demonstrates a possible relationship between central control measured by MEP and peripheral functions evaluated by EMG. Since athletes with CAI might use segmental spinal reflex modulation in adapting to pathology (Sefton et al. 2008), the compensation movements after ankle sprain might be associated with both central and peripheral controlled alterations.

Before foot contact after the jump, the preparation of the tendon and muscular activities is necessary to provide joint stability during a landing with high-impact forces, such as with an explosive jump landing. Therefore, muscular activation is increased in preparation for landing from jumping (Santello 2005). Moreover, the muscle activity is continually built up to prepare for the jump-landing (Suda et al. 2009). However, the muscle activation-onset times observed in this study were similar between the groups and across the three muscles under investigation. This again might have been due to the effect of the participant sample used in this study (active elite athletes) on corticomotor excitability.

Another component of neuromuscular function is muscle strength, encompassing both motor unit recruitment and firing rate to activate the required muscle contraction, with elaborated information processing taking place in the brain (Kandel et al. 2000). No consistency was found in the literature regarding the strength of the ankle musculature among individuals with CAI. Several studies have reported evertor (Willems et al. 2002; Donnelly et al. 2017), dorsiflexor (Negahban et al. 2013) and plantarflexor (Fox et al. 2008) muscle weakness among participants with CAI. In contrast, other studies have demonstrated no differences in ankle strength between participants with CAI and healthy controls (Kaminski et al. 1999; Munn et al. 2003). In the present study, the strength of the dorsiflexor muscle among participants with CAI was significantly lower than those of non-CAI. Moreover, their evertor muscle showed lower than with non-CAI subjects, but it was not a significant difference ($p = 0.060$). Thus, athletes with CAI exhibited the alterations of muscle performance in evertor and dorsiflexor muscles. The decreased strength of the dorsiflexor muscle was consistent with decreased MEP and longer latency of the TA muscle controlling the ankle dorsiflexion. The decreased dorsiflexor muscle strength in our study might be associated with the corticomotor excitability and the delayed conduction time (longer latency) in sending from the cortical to the target muscle including the TA muscle (Bravo-Esteban et al. 2017). In addition, the tendency of decreased strength of the evertor muscle ($p = 0.060$) may be associated with decreased MEP and EMG of the PL muscle controlling the ankle eversion. It may imply that alterations in the neural pathways contributed to the weakness of the evertor muscle (Lepley et al. 2014). This casual effect is consistent with a study by Kosik et al. (2017). They concluded that CAI had reduced the cortical map area in the PL muscle; thereby, reducing the command sent to the PL muscle (Kosik et al. 2017). In contrast, the unchanged strength of the invertor muscle and EMG of the TA muscle was not aligned with the findings of decreased MEP of the TA muscle in CAI. It might be an effect of the alteration in the spinal loop as in the related study finding (Sefton et al. 2008). An alternative explanation is that CAI had a muscle adaptation to maintain their joint stability by preserving EMG (Li et al. 2018) and the strength of the invertor muscle.

In addition to the association between brain and behaviour, the neuromuscular function, i.e. EMG and muscle strength in CAI remains controversial (Delahunt et al. 2006; Suda et al. 2009; Son et al. 2017; Monaghan et al. 2006; Webster et al. 2016; Allet et al. 2017; Fox et al. 2008; Donnelly et al. 2017; Negahban et al. 2013). The discrepancy of results may be influenced by different measurements method, such as single-muscle versus multi-muscle. The results of MEP and EMG in our study represented corticomotor excitability of the single muscle including the TA, PL and GM muscles. However, the strength measurement represented multi-muscle, for example, ankle dorsiflexion consisting of the tibialis anterior, extensor hallucis longus, extensor digitorum and peroneus tertius muscle. The other possible reason is the difference in resting versus active muscle contraction. The corticomotor excitability in the present study was collected while the participants were at rest, while the muscle strength was measured during active movement.

In line with related published literature (Santos and Liu 2008; Willems et al. 2002), ankle joint position sense did not differ between groups. Despite proprioceptive deficits being suggested as a factor in both injury and re-injury, contradictory results were observed with CAI. Indeed, because the participants in this study had 'functional' CAI and no current anatomical symptoms, their sensory input was intact.

Therefore, quite possibly, CAI occurrence may have resulted from altered corticomotor excitability, especially in relation to the PL muscle. The alteration of neuromuscular function with CAI might be located in the area of supraspinal control. Since the CAI participants in the present study presented functional ankle instability, their sensory information ascending to the cortex was assumed to remain intact.

The relationship between corticomotor excitability and neuromuscular functions

The non-CAI group in the present study demonstrated moderate correlations between corticomotor excitability and neuromuscular functions across muscles around the ankle joint. These associations may indicate an ability to provide appropriate joint stability (Needle et al. 2014a). In comparison, the CAI group exhibited a moderate correlation in the TA muscle only. The weakness or absence of correlation in the PL and GM muscles in the CAI group reveals a desynchronisation between cortical and neuromuscular function in the other muscles around the ankle. This may represent an adaptation of the TA muscle with CAI. Consequently, a sustained alteration of corticomotor excitability and the lack of correlation between corticomotor excitability and neuromuscular activation may reflect cortical reorganisation leading to motor planning disorders (Masse-Alarie et al. 2016).

Our analysis showed poor-to-moderate correlation between corticospinal excitability and neuromuscular functions in both CAI and non-CAI groups. It may have been associated with the different magnitudes of change in corticospinal excitability and neuromuscular functions. Consistent with a related study, the changes were only observed at the cortical level, but not at the peripheral level (McLeod et al. 2015). Other possible reasons include different measurement methods such as single versus multi-muscle, and resting versus active movement as discussed in the above section.

Clinical implication

Our results support the hypothesis that peripheral injury could affect cortical control (Kapreli and Athanasopoulos 2006; Ward et al. 2015). Clinicians can use this information to develop rehabilitation strategies, specifically by introducing specific training programmes that influence both muscle performance and corticomotor excitability to restore regular motor control patterns to prevent recurrent ankle injuries. In particular, motor skill training of the muscles controlling the ankle should be introduced to the rehabilitation protocol as goal-directed training tasks may induce functional–behavioural and neuromodulatory change (Gallasch et al. 2009). Motor skill training is engaged with changes in cortical excitability as well as cortical reorganisation, and it may play an important role in musculoskeletal rehabilitation (Boudreau et al. 2010). Our results demonstrated corticomotor excitability changes including decreased corticomotor excitability in the PL muscle and delayed conduction time in the TA muscle among athletes with chronic ankle instability. Therefore, they are recommended to undergo motor skill training.

Limitations

Our study demonstrated reduced conduction time sending from the brain to the target muscle. However, this study could not specify whether the deficit occurred at the spinal loop or at the neuromuscular junction. Therefore, further work is recommended to measure the H-reflex, representing the motor neuron pool excitability in the spinal loop. This would support our findings and verify the mechanism underlying this adaptation. Furthermore, in the present study, corticomotor excitability data were collected whilst participants were in a resting condition. The data collected during movement would reveal more about cortical control during functional activity. Moreover, the measurement of corticomotor excitability and neuromuscular functions at the hip and knee joints would be an advantage to assess the maladaptation of supraspinal control and behaviour underlying CAI. Additionally, the use of complex EMG analyses such as statistical parametric mapping could extend the analysis regarding the pattern of muscle activities. This method may help eliminate some of the limitations associated with EMG time-series analysis, which tends to involve the extraction of summarising scalar parameters. Statistical Parametric Mapping (SPM) analysis allows inter-muscle and time-dependence to be incorporated in the statistical calculation (Robinson et al. 2015).

Most participants in the present study were male; and might constitute a limitation for generalising to female athletes. However, this factor might not be an influence in our study. When sex was subcategorised; the finding remained the same. Additionally, sex has been reported to have no effect on corticomotor excitability including MEP, motor threshold and the silent period (Matsunaga et al. 1998; Mills and Nithi 1997; Wassermann 2002; van der Kamp et al.

1996). Moreover, we minimised the effect of sex difference using the matched-paired design.

Conclusion

The athletes with CAI demonstrated cortical and neuromuscular deficits that were manifested by decreased corticomotor excitability, some muscle activity deficits, and lack of correlation between cortical and neuromuscular functions. In addition, the results of involuntary (EMG recorded) and voluntary (muscle strength) controls in CAI were correlated. Therefore, deficits in the periphery resulting from CAI affected both neuromuscular control and corticomotor excitability, particularly in the PL and TA muscles. This suggests a maladaptive occurrence among athletes with CAI, which might preserve the homeostasis of body movements; however, this phenomenon might increase the risk of recurrent ankle injury.

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Compliance with ethical standards

Conflict of interest The authors report they have no potential conflict of interest.

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