

Gait Biomechanics in Participants, Six Months after First-time Lateral Ankle Sprain

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Abstract

No research currently exists predicating a link between the injury-affiliated sensorimotor deficits of acute ankle sprain and those of chronic ankle instability during gait. This analysis evaluates participants with a 6-month history of ankle sprain injury to affirm this link. 69 participants with a 6-month history of acute first-time lateral ankle sprain were divided into subgroups ('chronic ankle instability' and 'coper') based on their self-reported disability and compared to 20 non-injured participants during a gait task. Lower extremity kinematic and kinetic data were collected from 200 ms pre- to 200 ms post-heel strike (period 1) and from 200 ms pre- to 200 ms

post-toe off (period 2). The 'chronic ankle instability' subgroup (who reported greater disability) displayed increased knee flexion during period 1. During period 2, this subgroup exhibited greater total displacement at their ankle joint and greater extensor dominance at their knee. That many of these features are present, both in individuals with acute ankle sprain and those with chronic ankle instability may advocate a link between acute deficits and long-term outcome. Clinicians must be aware that the sensorimotor deficits of ankle sprain may persevere beyond the acute stage of injury and be cognizant of the capacity for impairments to pervade proximally.

Introduction

Safe locomotion is dependent on the synchronous interaction of pre-programmed sensorimotor efferents and afferent feedback mechanisms [1]. Disturbance of this synchrony, which may occur due to disruption of sensory afferents, may have the capacity to distort the pre-programmed gait coordination strategies 'stored' by the sensorimotor system. For example, the damage to the lateral ligamentous complex that is typical of an acute lateral ankle sprain (LAS) injury has been theorized to "interrupt the flow of impulses from the mechanoreceptors in the injured ligament and associated joint capsule to the central nervous system" [35]. This, in turn, may trigger adaptive patterns of sensorimotor reorganization, possibly "resetting" previously established coordination strategies in the adoption of new motor control patterns [30]. These alterations may then manifest either in a continuum of residual symptoms that lead towards ankle instability and thus re-injury (collectively referred to as "chronic ankle instability" (CAI) [5]), or recovery/compensation (these individuals have been termed

"copers"), in the year following the initial LAS [30].

Movement strategy anomalies during gait are apparent immediately following first-time LAS [8], lending weight to the hypothesis that this injury corrupts the sensorimotor accuracy of lower limb motor control and hence the foot-surface interaction, which is essential for safe locomotion [32]. It is unclear, however, based on the current literature, what patterns of movement and/or motor control differentiate individuals from developing CAI compared with LAS copers after sustaining a first-time, LAS. Recently published research from our laboratory was designed to tackle this issue; individuals were recruited soon after sustaining an acute first-time LAS and underwent biomechanical evaluation to discern their movement patterns during gait [8], while internal joint moments were calculated to identify the predominant motor control underlying these patterns [21]. It emerged that individuals with a current acute LAS exhibited some kinematic and energetic patterns previously observed in CAI populations, while others were contextually unique [8].

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The current investigation is a follow-up analysis of the aforementioned cohort [8]. The same individuals who completed the gait task in the acute phase of their LAS injury returned to our laboratory 6 months later to complete the same protocol. In this way, we sought to evaluate the motor control and coinciding movement patterns that characterize these individuals, now 6 months into their recovery/compensation and prior to the establishment of CAI or copers status at the 1-year time-point [30]. This study is exploratory in nature, and stands to advance current understanding of the potential for persistence of movement pattern anomalies following an acute first-time LAS.

Methods



Design

The Human Research Ethics Committee of University College Dublin approved this study. All participants signed an informed consent form prior to testing. The protocol for this study meets the ethical standards of the journal [13].

A convenience group of 69 participants who were recruited from a university-affiliated hospital emergency department (ED) within 2 weeks of sustaining an acute first-time LAS injury attended our laboratory 6 months later for the evaluation detailed in this report. All injured participants were provided with basic advice on applying ice and compression for the week on discharge from the hospital ED: they were each encouraged to weight-bear and walk within the limits of pain. Activities in daily life were encouraged. Whether the patient sought additional medical treatment was not controlled in the current study. An additional convenience sample of 20 participants with no prior history of LAS were recruited from the hospital catchment area population using posters and flyers to act as a control group.

The participant exclusion criteria have previously been described [8]. The diagnosis of acute LAS was made by the attending physician in the ED and confirmed by the principal investigator, who was a chartered physiotherapist (CD). This diagnosis was made at the time of recruitment following physical examination and on the basis of the patient-reported mechanism of injury. The Ottawa ankle rules [26] were followed in the event the attending physician considered the patient to have a risk of fracture or osteochondral defect. Patients were not recruited for the current study in the presence of any abnormality when further investigation was considered warranted.

Questionnaires

2 assessment tools were used to quantify ankle function [3] and perceived instability [15]: the activities in daily life and sport subscales of the FAAM (FAAMadl and FAAMsport), and the Cumberland Ankle Instability Tool (CAIT), respectively. Questionnaires were completed by all participants on arrival to the testing laboratory prior to completion of the gait task as detailed in this report.

Experimental procedures

Collection methods for this study have been previously documented [8]. Briefly, following completion of the questionnaires, anthropometric data were obtained and each participant was then instrumented with the Codamotion bilateral lower limb gait set-up (Charnwood Dynamics Ltd, Leicestershire, UK), according to manufacturer guidelines by the same investigator

[20]. A neutral stance trial was used to align the participant with the laboratory coordinate system and to function as a reference position for subsequent kinematic analysis [34].

Gait analysis data acquisition was made using 3 Codamotion cx1 units. This system was fully integrated with 2 AMTI walkway-embedded force plates (Watertown, MA); the Codamotion cx1 units were time-synchronized with the force plates. Kinematic data acquisition was made at 250 Hz and kinetic data at 1 000 Hz. Joint moments at the hip, knee and ankle were calculated from the force plate, lower extremity kinematic, and anthropometric data using an inverse dynamics procedure [11].

During testing, participants walked barefoot across a 10 m walkway at a self-determined speed. Each participant was instructed to look at a distant mark to inhibit them from looking down at the floor. 5 'clean' gait cycles, defined by both the participant's feet landing fully on each of the force plates, were identified and saved for future analysis. Any data obtained whereby the participant did not strike the force plate fully was discarded. Participants were familiarized with testing procedures prior to commencement. Prior to data analysis all values of force were normalized with respect to each subject's body mass (BM).

Data analysis

A vertical component GRF threshold of 10 N was used to identify foot contact with the force plate [24]. Kinematic and kinetic data relating to 2 periods for both limbs were analyzed using the Codamotion software: period 1 extended from 200 ms pre-heel strike (HS) to 200 ms post-HS and period 2 extended from 200 ms pre-toe off (TO) to 200 ms post-TO. These time windows were chosen for analysis as they have previously been used to investigate both acute LAS and CAI-associated movement pattern anomalies during gait [6, 7, 8, 20, 25], thus fulfilling our primary experimental objectives.

The kinematic dependent variables were sagittal (hip, knee, ankle) and frontal plane (ankle) joint angles at HS/TO, at maximum and total displacement in the 400 ms window for each period [2]. The kinetic dependent variables were maximum sagittal plane (hip, knee, ankle) and frontal plane (ankle) internal joint moments in the 200 ms time windows following HS and prior to TO. This resulted in 16 variables for each limb in each time period.

Statistical analyses

The LAS cohort was split into subgroups on the basis of the CAIT: individuals with a score of <24 were labelled 'CAI' and those with a score of ≥ 24 were labelled 'copers'. Note that these were subgroups by label only; current consensus recommends that this stratification can only be confirmed 12 months following a first-time LAS [12]. For both LAS groups, the limb injured at the time of recruitment was labelled as 'involved' and the non-injured limb as 'uninvolved'. Limbs in the control group were side-matched to limbs in the injured group as 'involved' and 'uninvolved'.

A 2-way MANOVA model was adopted for each gait period (period 1/period 2). The independent variables were group ('CAI', 'coper', control) and limb ('involved', 'uninvolved'). The dependent variables were the 16 variables previously outlined. In the event of a main effect for group, post-hoc comparisons were completed using a Tukey HSD test where appropriate. The significance level for all analyses was set at a more conservative level of $p < 0.01$ to reduce the risk of familywise error [16].

Results



Of the 69 participants in the LAS cohort, 33 were labelled 'CAI' and 36 as 'copers' on the basis of the CAIT. Demographics, self-reported function and perceived instability scores on the CAIT and FAAM respectively are presented for the LAS (including the 'CAI' and 'coper' subgroups) and control groups in **Table 1**.

Regarding the biomechanical dependent variables, there were no interactions between 'group' and 'limb' for either period of the gait cycle, nor was there any main effect for 'limb'.

There was, however, a statistically significant main effect for group during period 1 [$F(32,278)=2.63$, $p<0.001$, $\eta_p^2=0.23$; Wilks' Lambda=0.59]. When the results of the dependent variables were considered separately for period 1, knee angle (sagittal plane) at HS was significant [$F(5,154)=7.88$, $p=0.001$, $\eta_p^2=0.09$] in the between groups comparison. Post-hoc analyses revealed that 'CAI' participants had a more flexed knee angle at HS than controls ($p<0.001$).

There was also a statistically significant main effect for the group during period 2 [$F(32,278)=3.77$, $p<0.001$, $\eta_p^2=0.30$; Wilks' Lambda=0.49]. When the results of the dependent variables were considered separately for period 2, hip angle at TO in the sagittal plane [$F(5,154)=5.59$, $p=0.005$, $\eta_p^2=0.07$], total ankle joint displacement in the frontal plane [$F(5,154)=8.82$, $p<0.001$, $\eta_p^2=0.10$], maximum internal joint moment at the knee in the sagittal plane [$F(5,154)=5.48$, $p=0.005$, $\eta_p^2=0.07$] and maximum internal joint moment at the ankle in the frontal plane [$F(5,154)=4.90$, $p=0.009$, $\eta_p^2=0.06$] were significant in the between-groups comparison. Post-hoc analyses revealed that 'CAI' participants exhibited more hip extension at TO compared to 'copers' ($p=0.003$), more frontal plane ankle joint displacement compared to both 'copers' ($p=0.001$) and controls ($p=0.003$) and more maximum internal knee extension moment compared to controls ($p=0.008$).

Inspection of the mean scores for each limb implied that the above findings were bilateral in nature for both periods of the gait cycle.

Results of the between-group comparisons are presented for periods 1 and 2 in **Table 2**. Descriptive statistics for the dependent variables delineated by group, period and limb are presented in **Table 3**.

Discussion



This is the first analysis to 'pre-emptively' stratify a cohort of participants with a 6-month history of first-time LAS into 'CAI' and 'coper' subgroups. While the actual long-term injury outcome of these subgroups (CAI or coper) cannot be technically confirmed at this time, as the initial LAS was only 6 months ago

[12], this exploratory analysis has revealed several consistencies between the movement strategies exhibited by the 'CAI' subgroup, cohorts in the acute phase of LAS injury [8], and those with a confirmed diagnosis of CAI [2]. On this basis, it would seem that long-term injury outcome (CAI vs. coper) is potentially detectable 6 months after the acute LAS injury.

For instance, a characteristic feature of the entire LAS cohort when they were assessed in the acute phase of their injury was an increase in knee flexion at HS compared to controls [8]. With regards to the current findings, this was evident in the CAI subgroup bilaterally wherein they displayed $\approx 3^\circ$ more knee flexion than control participants. 'Coper' participants in this study also displayed more knee flexion ($\approx 2^\circ$ more) bilaterally at HS than controls; however, this was not statistically significant at the a-priori alpha ($p=0.011$). As was previously alluded to, the similarity between the 'CAI' and 'coper' subgroups can be linked with patterns displayed by the entire LAS cohort in the acute phase of injury, wherein they displayed increased knee flexion at HS on both their "involved" ($\approx 3.5^\circ$) and "uninvolved" ($\approx 3^\circ$) limbs [8]. Whether this strategy was adopted in the acute phase of injury and has since become redundant (because both LAS subgroups currently display it), or preceded the initial injury, is unknown. As this variable did not differentiate the 'CAI' and 'coper' subgroups in the current study, we do not consider it likely to predicate the extent of ankle-associated disability reported by the 'CAI' subgroup. Such characteristics would be unique to the 'CAI' subgroup compared to both the 'copers' and controls.

One such feature unique to 'CAI' participants was the bilateral greater total frontal plane displacement at the ankle joint ($\approx 11^\circ$ in the 'CAI' group) during period 2 compared to both 'copers' ($\approx 8.9^\circ$) and controls ($\approx 8.7^\circ$). Like the greater knee flexion at HS, this feature was comparable with the study of this cohort in the acute phase of their injury, wherein LAS participants displayed greater inversion at HS [8], and was also consistent with observations made of populations with a confirmed diagnosis of CAI [2,6,20]. A propensity for greater inversion at the ankle joint is considered to be a key contributor to the increased risk of re-spraining experienced by CAI populations [27]. That this feature was evident immediately following ankle sprain [8] and seems to persist into chronicity may indicate a number of underlying injury-affiliated events. For example, damage to the calcaneofibular ligament (CFL), which likely occurred at the initial LAS event [36], may have compromised static joint stability at the ankle in the frontal plane [28], thus increasing the potential for excessive inversion motion and the risk for re-sprain to occur [27]. Central mechanisms of sensorimotor control may also play a role: that the 'coper' subgroup (who have the same injury exposure as the 'CAI' subgroup) exhibited similar frontal plane displacement at their ankle to controls implicates sensorimotor control in the

Table 1 Participant demographics and questionnaire scores [mean with SD (standard deviation)] for the involved limb of the LAS subgroups and the control group.

	Age (yrs) [SD]	Body mass (kg) [SD]	Height (m) [SD]	CAIT (/30) [SD]	FAAMadl (%) [SD]	FAAMsport (%) [SD]
LAS (n=69; 43 males, 26 females)	22.88 [4.19]	73.69 [13.60]	1.73 [0.09]	22.81 [5.51]	95.73 [5.81]	84.46 [15.23]
'CAI' subgroup (n=33; 19 males, 14 females)	22.30 [1.96]	76.60 [15.12]	1.72 [0.09]	18.03 [3.90]	93.59 [7.11]	78.70 [17.53]
'Coper' subgroup (n=36; 24 males; 12 females)	23.42 [5.47]	70.87 [11.51]	1.73 [0.10]	27.08 [2.21]	97.88 [2.91]	90.40 [9.49]
Control (n=20; 15 males, 5 females)	22.60 [1.70]	71.4 [11.29]	1.73 [0.10]	30.00 [0]	100.00 [0]	100.00 [0]

LAS=lateral ankle sprain; CAI=chronic ankle instability FAAMadl=activities in daily life subscale of the Foot and Ankle Ability Measure; FAAMsport=sports subscale of the Foot and Ankle Ability Measure

Table 2 Results of the post-hoc analysis for the kinematic and kinetic dependent variables comparing the 'CAI' and 'coper'/control groups during periods 1 and 2 of the gait cycle.

		CAI		Coper		Control	
		Mean	SD	Mean	SD	Mean	SD
Period 1 (200ms pre-HS to 200ms post-HS)							
Kinematic	Max hip angle	17.46	8.74	19.61	7.57	17.47	4.82
	Hip position at HS	14.29	9.85	16.22	8.48	14.94	5.22
	Hip displacement	12.91	4.31	11.43	4.03	11.59	4.13
	Max knee angle	45.72	9.82	46.28	9.46	46.77	8.85
	Knee position at HS	3.27 ^b	4.61	2.21	5.07	-0.73	4.53
	Knee displacement	44.98	9.12	45.84	9.61	48.88	7.65
	Max ankle angle (frontal plane)	3.94	5.98	5.11	5.96	4.18	4.44
	Ankle angle at HS (frontal plane)	1.15	6.51	2.76	6.13	1.87	4.01
	Ankle displacement (frontal plane)	11.07	4.49	10.73	4.43	9.99	3.45
	Max ankle angle (sagittal plane)	2.12	2.86	2.55	2.34	1.45	2.81
	Ankle position (sagittal plane) at HS	-3.14	4.05	-2.33	3.49	-4.16	3.84
Ankle displacement (sagittal plane)	12.03	2.84	12.03	2.76	13.06	2.14	
Kinetic	Max hip moment	0.48	0.42	0.44	0.32	0.63	0.21
	Max knee moment	0.57	0.70	0.66	0.70	0.57	0.33
	Max ankle moment (frontal plane)	0.14	0.27	0.13	0.23	0.22	0.16
	Max ankle moment (sagittal plane)	0.31	0.59	0.43	0.79	0.29	0.37
Period 2 (200ms pre-TO to 200ms post-TO)							
Kinematic	Max hip angle	14.71	10.02	17.68	7.79	16.69	5.75
	Hip position at TO	-11.55 ^a	8.84	-7.19	6.02	-9.66	6.09
	Hip displacement	34.46	8.37	34.05	7.78	34.62	6.44
	Max knee angle	58.80	8.68	58.21	7.59	58.98	6.86
	Knee position at TO	40.90	6.90	42.07	6.01	40.33	6.03
	Knee displacement	53.21	9.22	52.29	8.55	54.70	6.26
	Max ankle angle (frontal plane)	4.54	5.45	5.28	5.50	2.89	4.11
	Ankle angle at TO (frontal plane)	0.93	6.29	1.87	6.08	-1.03	5.16
	Ankle displacement (frontal plane)	11.29 ^{a,b}	3.91	8.88	3.82	8.72	2.44
	Max ankle angle (sagittal plane)	8.25	3.27	8.86	3.58	6.97	5.50
	Ankle position (sagittal plane) at TO	-18.75	7.77	-17.02	5.90	-18.40	3.52
Kinetic	Ankle displacement (sagittal plane)	29.62	7.33	27.97	6.43	31.46	6.73
	Max hip moment	-0.46	0.36	-0.50	0.35	-0.47	0.34
	Max knee moment	0.26 ^b	0.55	0.25	0.48	-0.05	0.28
	Max ankle moment (frontal plane)	0.27	0.33	0.28	0.32	0.10	0.14
	Max ankle moment (sagittal plane)	0.90	0.84	0.93	0.83	0.48	0.63

CAI = chronic ankle instability; HS = heel strike; TO = toe-off. Flexion, dorsiflexion and inversion are positive for the kinematic variables; extension, plantar flexion and eversion are negative. Extension, plantar flexion and inversion moments are positive for the kinetic variables; flexion, dorsiflexion and eversion are negative. The kinetic variables were calculated only when the foot was in contact with the ground (200ms post-HS to HS and 200ms pre-TO to TO). Kinematic variables were measured in degrees (°); Kinetic variables were measured in Newton-meters per kilogram of body mass (Nm/Kg) ^aIndicates statistically significant difference compared to 'copers'; ^bIndicates statistically significant difference compared to controls

emergence of this strategy. Indeed, ankle sprain injury is likely to be associated with alterations in alpha motor neuron excitability [17] and previous research has shown that CAI participants activate their peroneus longus (PL) prior to HS earlier than non-injured controls [9]. This early activation may coincide with a decrease in the excitability of spinal reflexes of the lower extremity [18,19] and may prevent normal medial displacement of the center of pressure during the stance phase of gait, resulting in individuals with CAI bearing weight more laterally on their foot (and increasing total frontal plane displacement) [9]. Participants with a history of ankle sprain have previously been shown to apply greater loading through the lateral column of their foot during the latter part of stance [23,32]. Thus, it is plausible that the greater frontal plane displacement displayed by the 'CAI' subgroup was underpinned by these anomalous activation strategies [23,32]. This finding is in agreement with those of Brown et al., who also identified an increase in frontal plane ankle joint displacement during gait in participants with established CAI compared to copers [2].

In further comparing the results of the current analysis to those of its predecessor [8], it is interesting that the 'CAI' subgroup displayed significantly greater hip extension than the 'coper' subgroup. In the acute stage of injury, the LAS cohort as a whole displayed less hip extension in this period [8]. Due to the design of the present study, caution must be exercised in designating the importance of this, but the features that are unique to the 'CAI' subgroup may represent some of the anomalous strategies leading to a worse outcome. Because no significant differences were evident in the comparison between the 'coper' subgroup and the controls, it is possible that the coping strategies following a first-time LAS are 'successful' because they are similar to those of individuals with no LAS injury history. To our knowledge, only 3 previous studies have been published to date that have evaluated the walking gait of established copers following LAS [2,4,31], with only 2 of these making a comparison between copers and controls [4,31]. While differences in the acquisition methods [4], the prescribed gait task [31] and the definition of 'CAI' and 'coper' groups utilized differ between

Table 3 Descriptive statistics (mean and SD) for all the dependent variables delineated by group ('CAI', 'coper', control), period (1, 2) and limb ('involved', 'uninvolved').

	Limb	Period 1 (200 ms pre-HS to 200 ms post-HS)				Period 2 (200 ms pre-TO to 200 ms post-TO)			
		Involved		Uninvolved		Involved		Uninvolved	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Max hip angle	'CAI'	17.17	8.57	17.75	9.05	15.29	9.08	14.13	11
	'Coper'	19.47	7.05	19.76	8.17	17.61	7.99	17.74	7.71
	Control	17.82	3.37	17.12	6.02	16.62	4.79	16.77	6.72
Hip position at HS/TO	'CAI'	13.88	9.61	14.7	10.23	-11.34	8.31	-11.76	9.48
	'Coper'	16.08	8.35	16.36	8.75	-7.84	6.37	-6.55	5.67
	Control	15.23	3.72	14.66	6.49	-9.96	5.77	-9.36	6.54
Hip displacement	'CAI'	13.29	4.37	12.54	4.29	34.6	8.94	34.32	7.91
	'Coper'	11.08	3.26	11.77	4.7	34.36	8.27	33.73	7.36
	Control	12.54	3.87	10.65	4.26	35.17	7.01	34.06	5.97
Max knee angle	'CAI'	44.31	9.50	47.14	10.1	58.46	8.39	59.14	9.09
	'Coper'	46.48	9.30	46.08	9.77	58.29	7.42	58.13	7.87
	Control	47.74	9.15	45.81	8.68	59.4	7.66	58.55	6.16
Knee position at HS/TO	'CAI'	3.31	3.73	3.22	5.41	39.94	5.89	41.86	7.75
	'Coper'	2.06	4.90	2.36	5.31	41.14	6.63	43.01	5.25
	Control	-0.31	4.27	-1.15	4.86	40.37	6.63	40.28	5.56
Knee displacement	'CAI'	44.26	8.67	45.7	9.64	53.32	9.45	53.1	9.13
	'Coper'	46.48	8.56	45.2	10.65	52.31	9.01	52.27	8.22
	Control	49.41	8.17	48.35	7.29	54.76	7.08	54.64	5.53
Max ankle angle (frontal plane)	'CAI'	3.65	5.63	4.24	6.39	3.64	4.96	5.44	5.85
	'Coper'	4.84	5.10	5.37	6.79	5	5.08	5.56	5.96
	Control	3.03	3.13	5.33	5.3	2.55	4.79	3.23	3.42
Ankle angle at HS/TO (frontal plane)	'CAI'	0.80	6.22	1.49	6.88	0.16	5.99	1.71	6.59
	'Coper'	2.58	5.77	2.94	6.56	2.2	4.71	1.55	7.26
	Control	0.85	3.20	2.88	4.55	-1.88	5.6	-0.19	4.68
Ankle displacement (frontal plane)	'CAI'	12.05	5.02	10.09	3.74	10.92	3.65	11.67	4.17
	'Coper'	11.35	5.16	10.12	3.53	8.48	3.29	9.28	4.29
	Control	8.55	2.52	11.43	3.71	8.87	2.03	8.57	2.84
Max ankle angle (sagittal plane)	'CAI'	2.51	2.33	1.74	3.31	8.16	3.6	8.33	2.95
	'Coper'	2.59	2.35	2.51	2.37	8.03	3.37	9.69	3.64
	Control	1.76	3.32	1.13	2.24	7.05	6.11	6.9	5
Ankle position (sagittal plane) at HS/TO	'CAI'	-2.77	3.39	-3.52	4.64	-18.33	7.14	-19.17	8.45
	'Coper'	-2.27	3.01	-2.4	3.96	-16.15	4.96	-17.89	6.68
	Control	-4.26	4.13	-4.06	3.64	-18.74	3.99	-18.06	3.06
Ankle displacement (sagittal plane)	'CAI'	11.94	2.33	12.12	3.31	29.32	6.92	29.92	7.83
	'Coper'	11.87	2.59	12.19	2.95	26.83	5.76	29.1	6.94
	Control	13.26	2.19	12.86	2.14	31.96	7.07	30.97	6.54
Max hip moment	'CAI'	0.50	0.44	0.46	0.4	-0.52	0.43	-0.41	0.29
	'Coper'	0.42	0.31	0.45	0.34	-0.51	0.37	-0.49	0.32
	Control	0.64	0.19	0.61	0.23	-0.66	0.37	-0.27	0.13
Max knee moment	'CAI'	0.52	0.49	0.61	0.86	0.3	0.59	0.21	0.51
	'Coper'	0.61	0.65	0.7	0.75	0.23	0.54	0.26	0.42
	Control	0.45	0.37	0.7	0.22	-0.1	0.33	0	0.2
Max ankle moment (frontal plane)	'CAI'	0.12	0.23	0.17	0.31	0.32	0.39	0.23	0.25
	'Coper'	0.11	0.23	0.15	0.23	0.31	0.36	0.24	0.28
	Control	0.13	0.10	0.31	0.17	0.2	0.14	0.01	0.01
Max ankle moment (sagittal plane)	'CAI'	0.31	0.53	0.32	0.65	1.05	0.98	0.76	0.67
	'Coper'	0.42	0.89	0.44	0.69	1.05	0.88	0.81	0.78
	Control	0.37	0.32	0.2	0.4	0.97	0.57	-0.01	0.07

CAI = chronic ankle instability; HS = heel strike; TO = toe-off; SD = standard deviation. Flexion, dorsiflexion and inversion are positive for the kinematic variables; extension, plantar flexion and eversion are negative. Extension, plantar flexion and inversion moments are positive for the kinetic variables; flexion, dorsiflexion and eversion are negative. The kinetic variables were calculated only when the foot was in contact with the ground (200 ms post-HS to HS and 200 ms pre-TO to TO). Kinematic variables were measured in degrees (°); Kinetic variables were measured in Newton-meters per kilogram of body mass (Nm/Kg)

these studies and ours [2,4,31], our findings are in agreement with these papers in that no features were unique to the walking gait of LAS copers compared with CAI participants and non-injured controls [4,31].

Finally, the last feature which differentiated the 'CAI' subgroup from controls in this study was greater extensor dominance at

the knee in the pre-TO period. Like the increase in knee flexion at HS, this too was evident in the acute study [8], but has not previously been documented in CAI or coper populations. The underlying mechanisms contributing to this increase in knee extension can be explained in view of the coinciding kinematics. In the case of the 'CAI' subgroup, ligamentous damage and the

coinciding alterations in sensorimotor control [23,32] which manifested in the increase in frontal plane displacement at the ankle may have been one such cause of the increased knee extension moment; the increased frontal plane displacement at the ankle may have compromised this joint's ability to produce the 'push' force necessary for walking gait [21,22,33]. Excessive frontal plane motion of the ankle joint around TO could theoretically limit the normal medial displacement of the foot center of pressure and the subsequent utilization of the first ray of the foot to 'push off' the supporting surface [9]. A more rigid strategy at the knee may have been adopted in compensation. That all of the above between-groups differences were evident bilaterally is a particularly interesting finding from this study, and may represent further evidence of changes in central control mechanisms following ankle sprain injury [17–19]. The bilateral nature of the observed deficits is not a unique finding in the CAI literature during a gait task [14,29] and may be underpinned by the cyclical nature of walking gait, which necessitates some degree of symmetry between the lower extremities [10,22]. LAS disability may therefore have resulted in a bilateral manifestation of anomalous movement in the interest of maintaining inter-limb symmetry. Alternatively, the observed movement patterns may simply have preceded the injury. Herein lies one of the primary limitations of the current study: whether these movement and motor control patterns preceded the initial LAS and whether or not they actually cause CAI is yet to be elucidated. Furthermore, while our findings are important in the context of understanding how coping mechanisms may develop following LAS, their generalizability is low because these are ultimately temporary subgroups; it is possible that individuals in the 'coper' subgroup may yet develop CAI and vice versa at the 1-year time-point.

Conclusions

The most important implication of this study is that, even 6 months prior to the time when a diagnosis of CAI/coper can be confirmed [12], some individuals with a history of first-time LAS already display characteristics akin to their chronically impaired counterparts (i.e., the 'CAI' subgroup) while others do not (the 'coper' subgroup).

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