Title: Gait biomechanics in participants, six-months after first-time lateral ankle sprain.

Running title: Gait patterns after first-time ankle sprain

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. Sixty-nine participants with a 6-month history of acute first-time lateral ankle sprain were divided into sub-groups ('chronic ankle instability' and 'coper') based on their self-reported disability and compared to twenty non-injured participants during a gait task. Lower extremity kinematic and kinetic data were collected from 200ms pre- to 200ms post-heel strike (period 1) and from 200ms pre- to 200ms 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.

Key words: ankle joint [MeSH]; biomechanical phenomena [MeSH]; kinematics [MeSH]; kinetics [MeSH].

INTRODUCTION

Safe locomotion is dependent on the synchronous interaction of pre-programmed sensorimotor efferents and afferent feedback mechanisms [1]. Disturbance of this synchrony, as may occur on 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 theorised 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 reorganisation, 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 lend 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, 31].

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].

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 six-months later to complete the same protocol. In this way, we sought to evaluate the motor control and coinciding movement patterns that characterise these individuals, now six-months into their recovery/compensation and prior to the establishment of CAI or coper 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 sixty-nine participants who were recruited from a University affiliated hospital Emergency Department (ED) within two-weeks of sustaining an acute first-time LAS injury attended our laboratory six-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 Emergency Department: they were each encouraged to weight-bear and walk within the limits of pain. Activities of daily living were encouraged. Whether the patient sought additional medical treatment was not controlled in the current study. An additional convenience sample of twenty 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

Two assessment tools were used to quantify ankle function[15] and perceived instability[3]: the activities of daily living 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 two 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 1000 Hz. Joint moments at the hip, knee and ankle were calculated from 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. Five '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 familiarised with testing procedures prior to commencement.

Prior to data analysis all values of force were normalised with respect to each subject's body mass (BM).

Data analysis

A vertical component GRF threshold of 10N was used to identify foot contact with the force plate [24]. Kinematic and kinetic data relating to two periods for both limbs were analysed using the Codamotion software: period 1 extended from 200ms pre-heel strike (HS) to 200ms post-HS and period 2 extended from 200ms pre-toe off (TO) to 200ms 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, 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 400ms 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 200ms time windows following HS and prior to TO. This resulted in sixteen variables for each limb in each time period.

Statistical analyses

The LAS cohort was split into sub-groups 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 were 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 sixteen 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 sixty-nine 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 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' sub-groups. While the actual long-term injury outcome of these sub-groups (CAI or coper) cannot be technically be confirmed at this time as the initial LAS was only six-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 six-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 ≈ 3˚ more knee flexion than control participants. 'Coper' participants in this study also displayed more knee flexion (≈ 2˚ 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" (≈ 3.5˚) and "uninvolved" (≈ 3˚) limbs [8]. Whether this strategy was adopted in the acute phase of injury and has since become redundant (because both LAS sub-groups currently display it), or preceded the initial injury, is unknown. As this variable did not differentiate the 'CAI' and 'coper' sub-groups 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 (≈ 11˚ in the 'CAI' group) during period 2 compared to both 'copers' (≈ 8.9˚) and controls (≈ 8.7˚). 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 be under lied by a number of injury-affiliated events. For example, damage to the calcaneofibular ligament (CFL) which likely occurred at the initial LAS event[4], 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 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 centre 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

249 'CAI' subgroup was underpinned by these anomalous activation strategies [23, 32]. This
250 finding is in agreement with those of Brown et al., who also identified an increase in frontal
251 plane ankle joint displacement during gait in participants with established CAI compared to
252 copers [2].
253 In further comparing the results of the current analysis to those of its predecessor [8], it is
254 interesting that the 'CAI' sub group displayed significantly greater hip extension than the
255 'coper' subgroup. In the acute stage of injury, the LAS cohort as a whole displayed less hip
256 extension in this period [8]. Due to the design of the present study, caution must be exercised
257 in designating the importance of this, but the features that are unique to the 'CAI' subgroup
258 may represent some of the anomalous strategies leading to worse outcome. Because no
259 significant differences were evident in the comparison between the 'coper' subgroup and the
260 controls, it is possible that the coping strategies following a first-time LAS are 'successful'
261 because they are similar to those of individuals with no LAS injury history.
262 To our knowledge, only three previous studies have been published to date which have
263 evaluated the walking gait of established copers following LAS [2, 4, 31], with only two of
264 these making a comparison between copers and controls[4, 31]. While differences in the
265 acquisition methods [4], the prescribed gait task [31] and the definition of 'CAI' and 'coper'
266 groups utilised differ between these studies and ours [2, 4, 31], our findings are in agreement
267 with these papers in that no features were unique to the walking gait of LAS copers compared
268 with CAI participants and non-injured controls [4, 31].
269 Finally, the last feature which differentiated the 'CAI' subgroup from controls in this study
270 was greater extensor dominance at the knee in the pre-TO period. Like the increase in knee
271 flexion at HS, this too was evident in the acute study [8], but has not previously been
272 documented in CAI or coper populations. The underlying mechanisms contributing to this
273 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 centre of pressure and the subsequent utilisation 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 lays one of the primary limitations of the current study: whether these movement and motor control patterns preceded the initial LAS and whether they actually cause CAI or not is yet to be elucidated. Furthermore, while our findings are important in the context of understanding how coping mechanisms may develop following LAS, their generalisability 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 six-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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