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Impairment in Dynamic Postural Control Following an Acute Lateral Ankle Sprain

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IMPAIRMENTS IN DYNAMIC POSTURAL CONTROL FOLLOWING AN ACUTE LATERAL ANKLE SPRAIN

by

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(Under the direction of Thomas Buckley)

ABSTRACT

Lateral ankle sprains are the most common injury in sports, with an estimated 25,000 per day. Current research assesses postural control deficits following lateral ankle sprains; however most studies use static stances instead of dynamic stances. Most of the current research compares injured limb to non-injured limb, however bilateral impairments have been found to be present. Twenty Division I student athletes will be recruited to participate in this study, ten subjects will be NCAA Division I student athletes who have suffered a lateral ankle sprain. Control subjects will be healthy NCAA Division I student athletes, matched by height and gender with the injured subjects. Dynamic postural control will be evaluated by gait initiation, which will be assessed using the Vicon system and then compared to matched healthy control values. MANOVA revealed no significant difference in dynamic postural control following a lateral ankle sprain when compared to control group. Significant differences were found in range of motion assessment as well as perceived function assessment.

INDEX WORDS: Lateral ankle sprain, Postural Control, Gait initiation

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CHAPTER 1

INTRODUCTION

Lateral ankle sprains account for $2 billion in medical costs and are the most common injury in sports, with an estimated 25,000 per day and account for 16% of all sports injuries.[1,2] As many as one-sixth of all time loss injuries in sports are related to lateral ankle sprains.[2] Further, up to 75% of all ankle sprains occur in ankles that were previously sprained with potential contributing factors including: mechanical and functional instability, muscular weakness, limited mobility, improperly fitted footwear or old footwear, and damage to the proprioceptors in the ligaments of the ankle.[3-5] Following a lateral ankle sprain it has been suggested that the individual has reduced postural control, or the ability to maintain a desired postural orientation in response to perturbations generated from either internal or external sources.[6] These impairments, likely associated with proprioceptive deficits secondary to mechanoreceptor disruptions, not only predispose the individual to re-injury, but may be the potential underlying mechanism for chronic ankle instability.[4,7,8] Individuals who have experienced a lateral ankle sprain have an elevated risk of recurrent injury for 12 months after, a high risk for developing CAI, and are predisposed for developing osteoarthritis.[9-13]

During a lateral ankle sprain the ligaments that provide support to the lateral aspect of the joint, the anterior talofibular ligament (ATFL), the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL) are injured due to a hypersupination of the ankle. [7] The first to be injured is the ATFL, then the CFL, and lastly the PTFL.[7] When the foot is plantarflexed the ATFL becomes taut and parallel to the long axis of the leg.[14] As plantarflexion increases there is an increased tension on the ATFL.[15] Studies involving cadaveric-sectioning have found that after rupture of the ATFL, the amount of internal rotation of the rearfoot

increases substantially. St Pierre et al. studied the tensile strength to destruction of 36 ATFLs. Eighteen ligaments failed by bone avulsion from the talus, the other 18 had a midsubstance failure of the ligament.[14] In two studies of accidental lateral ankle sprains in the biomechanical lab setting, the ankle was in dorsiflexion and excessive inversion and internal rotation. [16,17] The ATFL was sprained in both cases; however unlike the hypothesized position of the ankle, dorsiflexion occurred rather than plantarflexion. In fact, kinematics revealed significantly lower plantarflexion values in the injury trial in comparison with the normal trials. In both cases there was a lateral shift of Center of Pressure (COP) which has been suggested to make the ankle vulnerable and may be considered a risk factor to sustain an ankle sprain.[16,17]

The lateral ankle ligaments are innervated by mechanoreceptors, which sense change in the joint position.[18] The decreased ability to sense changes in the ankle joint has a negative effect of postural control, thus after suffering a lateral ankle sprain, postural control impairments are present.[9,10,19] Postural control is the ability to maintain a desired postural orientation in response to perturbations generated from either internal or external sources.[6] Conversely, dynamic postural control is the ability to tolerate separation of center of mass (COM) and COP while transitioning from static to dynamic tasks.[20] When the distance between the COM and the COP increases, mechanical stability decreases, and postural control must act to return the COM to a stable position.[21] McKeon and Hertel suggest there are postural control deficits in individuals with an acute lateral sprain when compared to a healthy control group which have been identified through postural sway assessment.[10] Postural sway when balancing on the injured limb distributes forces across a larger area of the foot when compared to healthy individuals.[7,8] This suggests that the injured group uses a larger area of the foot to keep their balance. The modified star excursion balance test also detects deficits in postural control. The

farther the individual reaches out along the line, the more the separation of COM and COP is challenged, therefore making the individual less stable.[12] Time-to-boundary (TTB) measures have also been used to assess postural control. The lower the TTB measure, the greater postural instability, meaning the faster the COP reaches the boundary of the base of support leading to unsteadiness.[11]

In a systematic review by Wikstrom, they reported that most investigations used bilateral comparison to evaluate postural control deficits in the injured limb.[9,11,22,23] Often times when evaluating an injury the injured limb is compared to the contra lateral limb as "normal" for the individual. [9,11,22,23] However, recent findings have suggested both peripheral and centrally mediated bilateral impairments in postural control following an acute ankle sprain.[22] Because of this apparent bilateral impairment, caution must be taken when comparing the injured limb to the uninjured limb while assessing the injury and progress, and in making return to play decisions. These deficits may also indicate a larger motor control deficit.[9,24] Alterations in muscles proximal to the ankle joint have also been identified by Bullock-Saxton et al. who found alterations in hip extensor activity in both injured and uninjured limbs after a severe unilateral ankle sprain.[25] These central impairments and the resulting impaired postural control, may put the individual in greater risk for recurrent injury or other lower extremity injury and may be the cause of chronic ankle instability.[24] Most studies to assess postural control deficits use static tasks, however gait initiation has detected impairments in other populations.

Gait initiation (GI), the act of starting to walk from a stationary position, has been sensitive in determining postural control impairments for individuals suffering from Parkinson's disease, stroke, elderly, amputee, and chronic ankle instability.[21,26-28] The combined findings from these studies have identified two specific potential markers of impairments in postural

control; the displacement of the COP during the anticipatory postural adjustment phase and the resultant separation of the COP-COM at the conclusion of the initial step.[21,26,29] When a person takes a step, or begins to walk, the COP shifts towards the stepping limb initially to prepare for forward movement. This phase is known as the anticipatory postural adjustment (APA) phase, or segment 1 (S1). These anticipatory postural adjustments are likely controlled by the premotor cortex.[30] Movement initiation (MI) occurs when the COP and COM decouple by the activation of the tibialis anterior and gluteus medius, and inhibition of the triceps surae.[29] The COP then moves fully under the stance limb while the stepping limb is in the swing phase by the activation of the gluteus medius. This is known as the transitional phase, or segment 2 (S2). The movement of COP towards the toes of the stance limb for toe off by the triceps surae and inhibition of the tibialis anterior, marks segment 3 (S3) or the locomotor phase. The COM moves opposite the COP and is slightly in front of the individual. The larger the separation between COP and COM, the more unstable position the individual is in, and the more their postural control is challenged.[21]

Current investigations of impairments in postural control mostly focusses on static stances following a lateral ankle sprain and are limited to either cross-sectional design or comparing the injured to uninjured limb; however this design suffers from inherent limitations. Therefore, the purpose of this study was to conduct an investigation of impairments in postural control during the transitional movement task of GI following a lateral ankle sprain utilizing a between subjects design as well as comparing within the injured group between healthy and injured ankles. Specifically, we aim to compare postural control when the subject is cleared for full weight bearing after sustaining a lateral ankle sprain. We hypothesized that there would be

differences in COP displacement, COP-COM separation, and spatiotemporal measures between the lateral ankle sprain group and the control group. (Appendix A)

CHAPTER 2

METHODS

Subjects

Twenty Division I student athletes volunteered to participate in this study. Ten subjects were NCAA Division I student athletes who sustained a lateral ankle sprain who were recruited for participation in this study through referral from their athletic trainer. (Appendix C; Table 1) Control subjects were healthy NCAA Division I student-athletes who were matched by height ± 5 cm and gender with the injured subjects. (Appendix C; Table 1) The experimental subjects participated in a variety of collegiate sports including, baseball, cheer, diving, football, men's soccer, softball, women's basketball, women's tennis, and volleyball and were included in the study if the individual sustained a lateral ankle sprain during sport activity. Exclusion criteria for the control group included; history of chronic ankle instability as identified by using the foot and ankle ability measure (FAAM) and ankle instability instrument (AII) to evaluate self-perceived function and stability with a score no lower than 95% on either ankle and lower extremity injury within the previous 6 months. Exclusion for both groups included neurological conditions, including concussion, in the last 12 months. All subjects provided written informed consent prior to participating in the study as approved by the University's Institutional Review Board.

Instrumentation

Dynamic postural control was assessed with four force plates that were 400x600mm and embedded level with the floor (AMTI, model OR-6 and BP400600; Watertown, MA) that collected at 1000 Hz. Motion capture and analysis was done using the Vicon Motion System with eight cameras that collected at 100 Hz (Vicon; Lake Forest, CA). The subject's self-perceived ankle function was assessed by the valid and reliable foot and ankle ability measure

(FAAM) (Appendix C; Figure 3) and the ankle instability instrument (AII). (Appendix C; Figure 4) [7,9,31,32] Ankle joint swelling was assessed with a tape measure using the valid and reliable figure-of-eight method; passing over the navicular tuberosity, over the instep of the foot, across over the medial longitudinal arch, passing just proximal to the base of the fifth metatarsal, over the apex of medial malleoli, around the Achilles tendon, and passing over the apex of the lateral malleoli finishing at the starting position.[33] Range of motion was also assessed using an EZ Read Jamar goniometer (Patterson medical; Bolingbrook, IL) with normal ankle goniometer measures for dorsiflexion and plantarflexion.[34] The axis was placed over the lateral malleoli with the stationary arm in line with the fibula. The movement arm was parallel to the fifth metatarsal.[34]

Procedures

Subjects completed the FAAM and the AII on the first day of the study to confirm inclusion and exclusion criteria were met. The subjects anthropometric data including height, weight, swelling, and ROM was collected. The test day was on the day the athlete was cleared by their athletic trainer or physician to be full weight bearing.

Kinematic data was collected utiziling the Vicon motion capture system which used thirty-nine retro-reflective markers based on the plug-in gait system.[35] The 14mm retro-reflective markers were placed bilaterally on the 2nd MTP head, heel, ankle, lower leg, knee, thigh, anterior superior iliac crest, posterior superior iliac crest, shoulder, upper arm, elbow, forearm, distal radius and ulna at the wrist, 2nd MCP, forehead, and posterior head. Single markers were placed on the jugular notch, inferior sternum, C7, T10, and right scapula. Subjects were barefoot for all trials and had as many practice trials as needed to ensure comfort in the

task. The GI trial started with the subject standing with the right foot on force plate 1 and the left foot on force plate 3 (Appendix C; Figure 1). Subjects initiated gait with their non-injured limb and continued walking down a 4.9 m walkway. Subjects completed 5 trials of self-selected speed gait initiating with the non-injured limb and 5 trials of self-selected speed gait initiating with the injured limb. Controls determined which foot to initiate gait with first based off of their matched injured subject.

Data Analysis

Movement initiation during GI was identified by the first change in vertical ground reaction forces (GRF) (mean +/- 2 SD's).[27] GI was divided into 3 segments based on 4 landmarks (Figure 2). [36] Landmark 1 was identified when COP was the most lateral and posterior in the direction of the initial swing limb and marked the beginning of the unloading phase as the heel of the initial swing limb lifted. Landmark 2 was identified as the point when COP shifted from lateral to anterior motion and marked the point when swing leg toe-off occurred. Landmark 3 was identified as the end of the locomotion phase when toe-off of the stance limb occurred. The final landmark (HS^{-1}), was identified as the last vertical GRF at the end of single limb stance where the separation between COM and COP was the greatest. The spatiotemporal characteristics of the initial step including length, width, and velocity were also calculated. Step length (m) was determined by calculating the anterior displacement of the swing limb heel marker from MI to HS^{-1}. Stance width (m) was determined by measuring the distance between the left and right heel marker at MI. Step width (m) was determined by measuring the distance between left heel marker at HS^{-1} and right heel marker at MI. Step duration (s) was calculated by the time from MI to HS^{-1}.(Appendix C; Figure 2) Step velocity (m/s) was calculated as step length divided by the step duration.[36]

Statistical Analysis

Descriptive statistics were calculated for height, weight, age, and sport for each group. The mean individual values for the 5 trials of GI for each dependent variable with each limb as the initiator was calculated and used for analysis. Four dependent t-tests were run for all dependent variables (displacement of COP during S1, S2, and S3 of GI; COP-COM separation at MI, landmark 1, landmark 2, and HS^{-1}, and spatiotemporal characteristics for step length, step velocity and step width) based off if the injured limb was the initial stance limb or swing limb between the LAS and control group as well as within each group. Two 2-way ANOVAs were run to assess ROM and FAAM scores. Each test was run between LAS group and the control group, as well as within the LAS group and control group. The alpha level was set at $< .05$. All statistical testing was done using SPSS 17.0 (Chicago, Illinois).

CHAPTER 3

RESULTS

All participants were able to complete the 5 GI trials. There were no significant differences in demographic information between the LAS group and the control group (Table 1).

COP Displacement

Dependent t-test revealed that during GI the S1 A/P phase there was a trend towards significance between the LAS group and the control group when the injured limb was the initial stance limb and matched limb in the control group ($t(9) = -2.015$, $p = 0.075$) (Appendix C; Table 2B), with the control group having a larger shift. No significant difference was found between the LAS group and the control group when the injured limb was the initial swing limb and matched limb in the control group, or within the LAS group or control group (Appendix C; Table 2A&B). During the M/L component of the S1 phase was found to have a trend towards significance within the LAS group, with a greater shift when the healthy limb was the initial stance limb ($t(9) = -2.065$, $p = 0.069$), but was not significant within the control group. No significant difference was found between the LAS group and control group with the injured limb as the initial stance limb and matched limb in the control group or with the injured limb as the initial swing limb and matched limb in the control group for S1 M/L phase of GI (Appendix C; Table 2B).

During GI, the S2 A/P phase of GI was not found to be significantly different between the LAS group and the control group with the injured limb as the initial stance limb and matched limb in the control group, with the injured limb as the initial swing limb and matched limb in the control group, or within either group (Appendix C; Table 2A&B). There was a trend towards significant differences for the S2 M/L phase of GI between groups with the injured limb as the

initial stance limb and matched limb in the control group (t (9) = 2.214, p = 0.054), with a larger shift in the LAS group, as well as within the LAS group with a larger shift when the injured limb was the initial stance limb (t (9) = 2.152, p = 0.060) (Appendix C; Table 2A&B). No significance was found between the LAS group and the control group with the injured limb as the initial swing limb and matched limb in the control group or within the control group for the S2 M/L phase of GI (Appendix C; Table 2A&B).

No significant differences were found for the S3 A/P or M/L phase of GI between the LAS group and control group with the injured limb as the initial stance limb and matched limb in the control group or swing limb and matched limb in the control group, or within either group (Appendix C; Table 2A&B).

COP-COM Separation

Dependent t-tests revealed no significant difference between the LAS group and control group with the injured limb as the initial stance limb and matched limb in the control group or swing limb as initial stance limb and matched limb in the control group, or within groups for MI, L1, L2, or HS^{-1} (Appendix C; Table 3A&B).

Spatiotemporal Measures

A dependent t-test revealed significant differences between the LAS group and control group for step width with the injured limb having a greater step width than the matched limb in the control group (t (7) = 2.556, p =0.038) (Appendix C; Table 4A). No significant differences were found between groups with the injured limb and matched limb in the control group as the initial swing limb for step width. No significant differences were found for step width within the

LAS group or the control group (Appendix C; Table 4A). There were no significant differences within or between groups for the initial stance width (Appendix C; Table 4A&B).

Dependent t-test revealed significant differences within the LAS group for step velocity ($t(7) = -2.04$, $p = 0.047$). There was a faster step velocity observed when the subjects' initial stance limb was the injured limb (Appendix C; Table 4A). Step velocity was found to be approaching significance with the control group having a faster velocity when compared to the LAS group with the injured limb as the initial stance limb and matched limb in the control group ($t(7) = -2.274$, $p = 0.057$). No significant differences were found between the LAS group and the control group with the injured limb as the initial swing limb and matched limb in the control group for step velocity, or within the control group (Appendix C; Table 4B).

Step length was not found be significantly different between the LAS group and the control group with the injured limb as the initial stance limb and matched limb in the control group, or with the injured limb as the initial swing limb and matched limb in the control group, or within either the LAS group or the control group (Appendix C; Table 4A&B).

Range of Motion

Two-way ANOVA revealed a significant difference between groups for range of motion ($F(1, 1) = 15.09$, $p < 0.001$, $\eta^2 = .295$), with the control group having a larger range of motion than the LAS group. There was no significant difference within the LAS group when comparing the injured ankle to the healthy ankle, however, on average the injured ankle lacked 7° of range of motion ($F(1, 1) = 0.49$, $p = 0.489$, $\eta^2 = 0.01$) (Appendix C; Figure 5).

FAAM

Two way ANOVA revealed there was a significant main effect for group on FAAM scores, as well as between groups and within groups ($F(1, 1) = 56.87$, $p < 0.001$, $\eta^2 = 0.626$) (Appendix C; Figure 6). Within groups, the control group had a larger percentage, and within the LAS group, the healthy limb had a higher percentage. On average the injured population scored the injured limb 75% out of 100% and the healthy limb in the LAS group as well as both limbs in the control group were scored to be 100%.

CHAPTER 4

DISCUSSION

The purpose of this study was to conduct an investigation of impairments in postural control during the transitional movement task of GI following an acute LAS utilizing a between subjects design as well as comparing within the injured group between healthy and injured ankles. The primary finding of this study was that there was no significant difference in COP displacement or COP-COM separation during GI between subjects who sustained a LAS and the healthy matched controls. There was a significant difference observed between the LAS group and control group for step width, as well as a trend towards significance with step velocity. A trend towards significance was observed during the S1 A/P phase of GI as well. GI challenges the postural control system because it involves transitioning from a static stance to a dynamic stance. [18,24] In a healthy person, the momentum necessary for GI is developed during the APA phase, of GI.[30] Populations with impairments of dynamic postural control may have a decreased posterior displacement in the S1 phase due to adopting a more conservative approach to GI. This may in turn create a shorter step length and lower step velocity because of the inability to generate momentum forward.[26]

We hypothesized that a more conservative approach to GI would be adopted in the LAS group, however this was not seen in COP displacement. Specifically, we were interested in the COP during the APA phase, however both groups in this study presented with normal displacement.[26] Healthy young adults COP displacement in the A/P part of the APA phase is on average 4.7 ± 1.5 cm.[26] In this study a much lower average was observed (2.79 ± .92 cm) during the APA A/P phase of GI in this sample with the injured limb as the initial stance limb. This is lower than that of a healthy older adult (3.5 ± 1.4 cm) reported by Halliday et al, and even

lower than the displacement of an individual suffering from Parkinson's disease (2.94 ± 1.6 cm).[26] We did see a trend toward significance within the APA A/P phase, which can likely be attributed to the small sample size. Previous literature has found that healthy young adults displace COP 3.63±0.9 cm in the M/L direction of the APA phase.[26] In the current study M/L COP displacement was larger, displacing 4.37 ± 1.32 cm in the LAS group, but also larger in the control group, having a displacement of 4.56 ± 1.70 cm. Interestingly, when compairing the injured limb and healthy limb in the LAS group a trend towards significance was seen in the M/L portion of the APA phase. When the healthy limb was the original stance limb there was a greater displacement. This indicated that the COP was shifted more over the initial swing limb before shifting over the stance limb with the healthy limb as the stance limb. This may be a way of adopting a more conservative approach when the injured limb is the stance limb, keeping the COP closer to the COM at the beginning of GI.

A significant difference for step velocity within the LAS group between the injured ankle and the healthy ankle was found most likely because when the injured limb is the stance limb, the individual is stepping with the healthy limb, meaning they are balancing on the injured limb (Table 4). It is likely that the individual would step faster with the injured limb as stance limb, so that double limb stance is achieved sooner, and essentially a more stable state is achieved. There was a trend toward significant differences between the LAS group and the control group for step velocity with the injured limb as the initial stance limb and matched limb in the control group. We likely would see significant differences in step velocity with a larger sample size. Participants demonstrated normal step length as previously reported by Naugle for healthy young adults, averaging between 60-65 cm (Table 4).[37]

In this study no statistical differences were observed in COP-COM separation. Separation of COP-COM for the control group averaged to be 29.43 cm and the LAS group averaged 27.66 cm with the injured limb as the stance limb and matched limb in the control group, which is lower than previously reported for healthy individuals (36 cm), however not significantly different between groups.[38] The separation of COP-COM reflects the individual's ability to tolerate a dynamic unsteady state that accompanies forward momentum.[21] This is a previously validated tool in assessing individuals with a balance dysfunction when compared to healthy older adults.[39] We may not see differences between groups or within the LAS group between ankles because of small sample size as well as the possibility that changes over time may contribute to these impairments observed in other populations such as aging individuals and individuals with CAI.

There were significant differences for ROM and FAAM scores between the LAS group and the control group. On average, when comparing the LAS group injured ankle to the matched ankle of the control, the LAS group lacked 18° of range of motion. When comparing the healthy ankle in the LAS group to the matched ankle of the control, the LAS group lacked 12° of range of motion. Within the LAS group, the injured ankle lacked 7° of motion when compared to the healthy limb. This finding confirms that there are bilateral functional impairments observed following a lateral ankle sprain. These impairments may predispose the individual to recurrent injury, as well as putting him/her at risk for injury to the lower extremity of either limb. On average, the injured subjects rated their injured ankle to have a self-perceived function of 75% out of 100%, and a self-perceived function of 100% in reference to their non-injured ankle. Interestingly, while the healthy ankle in the injured group was lacking on average 12° of range of motion, they perceived their ankle function to be 100%. On the day the student athlete was

cleared to be FWB, they perceived their ankle to only be functioning at 75%. This is concerning because they feel the injured limb has a deficit of 25% compared to their healthy ankle and are walking without assistance. Wikstrom et al. suggested that self-perceived function is a potential indicator of the development of CAI.[40]

The results of this study revealed significant differences or a trend towards significant differences in dynamic postural control between individuals who had recently sustained a LAS and matched, healthy controls in step width, step velocity, ROM, and FAAM scores, however only trends towards significant differences in COP displacement were observed and no significant differences in COP-COM separation. A potential explanation for this may be that our sample size was too small to see significant differences. Another potential explanation is that while impairments occur, they are not central or affecting the premotor cortex which is likely responsible for cued GI,[30] rather there are functional and peripheral changes that occur in both ankles. Impairments observed in other populations during GI may be due to changes that occur over time from compensation or central disease, not from an acute injury. This may be seen in aging individuals as well as individuals who have developed CAI. The significance of both ROM and FAAM scores between groups bilaterally, suggests bilateral peripheral functional impairments occur, confirming the use of the non-injured limb as "healthy" is inaccurate. Therefore, investigation using pre injury, baseline data may be able to better identify the degree of impairments associated with an acute LAS.

Limitations

We attempted to control for factors that may have affected the outcome of this study, however a few limitations were still present. One limitation of this study is that because we used

an injured population we could not control for previous ankle or other lower extremity injury in the LAS group. Additionally, due to the use of an injured population, there is a small sample size that limits the power of this study. There were 13 possible participants with LAS occurring in the testing period, however two did not wish to participate and one participated, however had to be removed because of problematic data collection. That left 10 subjects who sustained a LAS to participate. Lastly, we could not control for the number of days between the occurrence of injury and the day the subject was cleared to be full weight bearing by their athletic trainer or physician.

Conclusion

This is the first study to our knowledge to use the task of GI to assess dynamic postural control following an acute LAS. The results of this study revealed that subjects who sustain an acute LAS have significant difference when compared to the control group for step width and within the LAS group for step velocity. There is a significant deficit in ROM and self-perceived function between the LAS group and the control group. Perceived function has been linked to the ability to cope with injury as well as predict the development of CAI. The LAS group had trends towards significant deficits in postural control during the APA phase of GI when compared to healthy, matched controls. Further research should be conducted using pre-injury data to have a greater understanding of the impairments a person is experiencing following a LAS because the use of the contralateral limb is unreliable due to bilateral impairments.

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Appendix A

Research Hypothesis

H_o1: LAS group will have no differences in separation of COP-COM compared to control group.
H_a1: LAS group will have a difference in separation of COP-COM compared to control group.

H_o2: LAS group will have no differences in COP displacement in the S1 phase of gait initiation compared to control group.
H_{a2}: LAS group will have a difference in COP displacement in the S1phase of gait initiation compared to control group.

H_{o3}: LAS group will have no difference in spatiotemporal measurements compared to control group.
H_a3: LAS group will have a difference in spatiotemporal measurements data compared to control group.

H_o4: LAS group FAAM scores will have no differences compared to the control group scores.
H_a4: LAS group FAAM scores will have differences compared to the control group scores.

H_o5: LAS ROM values will have no differences compared to the control group scores.
H_{a5}: LAS ROM values will have differences compared to the control group scores.

Limitations

- Cannot control for a history of ankle sprains/other injury
- Cannot control for the number of days between injury and day cleared to be FWB

Delimitations

- NCAA Division 1 student athletes from one university

Assumptions

- Gait initiation accurately identifies impairments in postural control
- 100% effort is being applied by participants

Appendix B

Literature Review

ANATOMY

The ankle complex comprises 3 articulations: the talocrural joint, the subtalar joint, and the distal tibiofibular syndesmosis. These 3 joints work together to allow coordinated movement of the rearfoot. Pronation, supination, internal and external rotation occur at the rearfoot. Rearfoot motion occurs simultaneously in the three cardinal planes, fontal-plane, sagittal-plane, and transverse-plane. These joints are not only supported by boney articulation, but also with ligaments and tendons.[13]

Articulations of the dome of the talus, the medial malleolus, the tibial plafond, the surface farthest from the midline, and the lateral malleolus make up the talocrual joint. The primary talocrual motions are in the sagittal plane, plantarflexion and dorsiflexion. The medial and lateral ligaments, including the anterior talofibular (ATFL) ligament, the calcaneofibular ligament (CFL), and the posterior talofibular ligament (PTFL) of the ankle provide static support. The ATFL runs from the lateral malleolus to the talus and prevents anterior translation of the talus and excessive inversion and internal rotation of the talus on the tibia. The ATFL is the weakest of the three lateral ankle ligaments, and has the least elastic transformation properties. The ATFL is approximately 6-10 mm in wide, 15-20 mm long, and 2 mm thick.[14] The CFL runs from the lateral malleolus to the lateral aspect of the calcaneus and prevents excessive inversion and internal rotation of the rearfoot.[13] The ATFL and the CFL act synergistically to control lateral stability of the ankle.[15] The CFL is a strong, flat oval ligament. The diameter of the ligament is 4-8 mm, it is 20 mm long, and 4-5.5 mm wide.[14] The PTFL, the

strongest ligament of the lateral ankle, runs from the lateral malleolus to the posterolateral aspect of the talus and prevents excessive inversion and dorsiflexion.[13,15] The PTFL is trapezoidal, approximately 30 mm long, 5 mm wide, and 5-8 mm in thickness.[14] The deltoid ligament is the primary static stabilizer of the medial aspect of the ankle and prevents excessive eversion and external rotation.[13] Not only is the talocrual joint an important in ankle movement and stability, the subtalar joint also provides stability to the ankle complex.

The talus and calcaneus articulate to form the subtalar joint which has two separate joint cavities, an anterior and posterior. This joint allows for rearfoot inversion and eversion. The two joint cavities share a common axis of rotation; however have two separate ligamentous joint capsules.[13] The posterior subtalar joint is formed between the inferior posterior facet of the talus and the superior posterior facet of the calcaneus.[41] The ligamentous support of the subtalar joint is divided into 3 groups: deep ligaments, peripheral ligaments, and retinacula. The deep ligaments are the cervical and interosseus ligaments.[13] The cervical ligament is located in the sinus tarsi and supports the anterior and posterior cavity of the subtalar joint. The cervical ligament is the strongest of the subtalar ligaments and helps prevent inversion of the ankle. The interosseus ligament lies posterior to the cervical ligament, and originates on the calcaneus and inserts on the talar neck. The interosseus ligament is also known as the ligament of the canalis tarsi. The peripheral ligaments are the CFL, the lateral talocalcaneal ligament (LTCL), and the fibulotalocalcaneal ligament (FTCL). The LTCL runs parallel and anterior to the CFL and also helps prevent ankle inversion. The FTCL originates on the posterior surface of the lateral malleolus and inserts on the posterolateral surface of the talus and calcaneus. The FTCL also assists in preventing excessive ankle inversion. The inferior extensor retinacula (IER) and the bifrucate ligament provide support to the lateral aspect of the subtalar joint as well. The

bifrucate ligament has two branches, the dorsal calcaneocuboid and the dorsal calcaneonavicular, and helps to resist inversion of the midfoot.[13] Another joint important to discuss in regards to stability of the ankle complex is the distal tibiofibular joint.

The distal tibiofibular joint is a syndesmotic joint that is formed by the articulation of the tibia and fibula. This joint is supported statically by the interosseous membrane, the anterior tibiofibular ligament, and the posterior tibiofibular ligament. Both the anterior and posterior tibiofibular ligaments originate on the distal lateral tibia and inserts on the lateral malleolus on the anterior and posterior aspect respectively. These structures are most often injured by excessive eversion, external rotation, and hyper dorsiflexion causing a syndesmotic sprain.[13]

MUSCULATURE

The muscles in the anterior compartment, the tibialis anterior, extensor digitorum longus, extensor digitorum brevis, and peroneous tertius, of the lower leg are thought to dynamically stabilize the ankle through eccentric contraction to slow plantar flexion motion[42]. The tibialis anterior, originating on the lateral condyle of the tibia and proximal tibia and inserting on the plantar surface of the 1st cuneiform and the base of the first metatarsal contributes to dorsiflexion of the ankle and assists in inversion of the foot. The tibialis anterior is innervated by the deep peroneal nerve. The extensor digitorum longus originates on the lateral condyle of the tibia and proximal, anterior fibula and inserts on digits 2-5 by four separate tendons. The extensor digitorum longus is innervated by the peroneal nerve. The extensor digitorum brevis originates on the distal, superior and lateral surface of the calcaneus and inserts on digits 1-4 and is innervated by the deep peroneal nerve. Both the extensor digitorum longus and brevis extend the metatarsophalangeal joints, and assists in extending the interphalangeal joints of the second

through fifth digits. The peroneus tertius originates on the distal, anterior fibula and inserts on the dorsal surface of the base of the fifth metatarsal. The peroneus tertius muscle dorsiflexes the ankle joint and everts the foot, and is innervated by the deep peroneal nerve. [42]

The lateral compartment is comprised of the peroneus longus and brevis which primarily stabilize the lateral ankle. Peroneus longus originates on the lateral aspect of the fibular head and inserts on the base of the first ray. Peroneus brevis originates on the distal third of the fibula and inserts on the base of the 5th metatarsal. Not only do the peroneals control inversion of the foot eccentricly, but concentricly perform eversion of the foot and are both innervated by the superficial peroneal nerve. [13,42]

The posterior lower leg is split into two compartments, superficial and deep. The superficial and deep posterior compartments are separated by the deep fascia. The superficial posterior compartment is made up of the gastrocnemius and the soleus. The gastrocnemius has a dual head origin, that originates on the proximal posterior aspect of the medial and lateral femoral condyles. The soleus originates on the posterior fibular head and proximal body of the fibula, as well as the medial border of the tibia. The gastrocnemius and the soleus share a common insertion onto the calcaneus via the Achilles tendon and share a common innervation, the tibial nerve. Both the gastrocnemius and soleus plantar flex the ankle joint, while the gastrocnemius assists in knee flexion. The deep compartment houses the tibialis posterior, flexor hallucis longus, flexor digitorum longus, and plantaris. The tibialis posterior originates on the interosseous membrane, the lateral posterior aspect of the tibia and the medial surface of the fibula. The insertion of the tibialis posterior spans the entire midfoot, specifically to the navicular tuberosity, three cuneiforms, cuboid, and base of the second through fourth metatarsal bones. The tibialis posterior inverts the foot and assists in plantar flexion of the ankle joint. The

flexor hallucis longus originates on the posterior surface of the distal 2/3 of the fibula, interosseous membrane, and adjacent intermuscular fascia and inserts on the plantar surface of the base of the distal phalanx of the great toe. The flexor hallucis longus flexes the interphalangeal joint of the great toe and assists in flexion of the metatarsophalangeal joint, plantar flexion of the ankle joint, and inversion of the foot. Flexor digitorum longus originates on the middle 3/5 of the posterior tibia and inserts on the base of the distal phalanges of the second through fifth digits. The flexor digitorum longus flexes the proximal and distal interphalangeal and metatarsophalangeal joints of the second through fifth digits and assists in plantar flexion of the ankle joint and inversion of the foot. The plantaris originates on the distal part of the lateral supracondylar line of the femur and inserts on the posterior calcaneus. The plantaris plantar flexes the ankle joint and assists in flexion of the knee joint. All the muscles of the deep compartment are innervated by the tibial nerve.

The lumbar and sacral plexes supply motor and sensory innervations to the ankle complex. The motor supply comes from the tibia, deep peroneal, and superficial peroneal nerves. The sensory supply comes from these nerves as well as the sural and saphenous nerves. The lateral ligaments and joint capsule of the talocrual joint have been shown to be innervated by mechanoreceptors, which contribute to proprioception. [18,43,44] The ankle joint has vascular supply from the dorsal pedis and posterior tibial arteries.

PATHOPHYSIOLOGY OF AN ANKLE SPRAIN

Injury to the ankle often occurs from extreme amounts of supination of the rearfoot.[45,46] Excessive inversion and internal rotation of the rearfoot coupled with external rotation of the lower leg results in strain to the lateral ligaments of the ankle, ATFL, CFL, and PTFL. If the

strain in any ligament exceeds the tensile strength of the tissues, ligament damage occurs, often referred to as a sprain.[47]

The most common ligament injured during a lateral ankle sprain is the ATFL, followed by the CF.[48] When the foot is plantarflexed the ATFL becomes taut and parallel to the foot. As plantarflexion increases there is an increase in the strain placed on the ATFL.[15] Studies involving cadaveric-sectioning have found that after rupture of the ATFL, the amount of internal rotation of the rearfoot increases substantially. St Pierre et al studied the tensile strength to destruction of 36 ATFLs. Eighteen ligaments failed by bone avulsion from the talus, the other 18 had a midsubstance failure of the ligament.[14] This increase in rearfoot movement puts excess stress on the remaining intact ligaments.[49] Isolated injury to the CFL occurs when the ankle is in neutral flexion. Injury to the PTFL is typical in severe ankle sprains and often accompanied by fracture or dislocation.[50] Conversely, in two studies of accidental lateral ankle sprains in the biomechanic lab setting, the ankle was in dorsiflexion and excessive inversion and internal rotation. The ATFL was sprained in both cases; however unlike the hypothesized position of the ankle, dorsiflexion occurred not plantarflexion. In fact, kinematics revealed significantly lower plantarflexion values in the injury trial in comparison with the normal trials. This suggests that the ATFL can be injured in both plantarflexion and dorsiflexion. In both cases there was a lateral shift of COP which has been suggested to make the ankle vulnerable and may be considered a risk factor to sustain an ankle sprain.[16,34]

Sprains can be classified as first-degree, second-degree, or third-degree. A first-degree sprain results in stretching of the ligament with little or no tearing of its fibers. No laxity is present in the joint, and a firm end-point is present. Local pain, point tendereness over the injured ligament(s), and slight swelling of the joint are present. A second-degree sprain results

in partial tearing of the ligament's fibers, resulting in joint laxity with a soft end-point present. Moderate pain and swelling is often present and loss the joint's function is noted. A third-degree sprain results in a complete rupture of the ligament, causing gross joint laxity, instability, and presents with an empty or absent end-point. Swelling and complete loss of function of the joint are noted. Pain may be limited secondary to tearing of the local nerves.[34] Depending on the severity of the injury, function usually returns over the course of a few days to a few months.[13] Proprioceptive deficits are present following injury to the ankle as well as neuromuscular recruitment impairments. This is most commonly assessed by looking at the reflexive response times of the peroneal muscles to inversion or supination perturbations. Sudden, forceful inversion of the ankle can lead to tearing of the lateral ligaments.[34] It is thought that the peroneal muscles are able to respond fast enough to protect the lateral ligaments from being injured once the ankle begins to rapidly invert. The estimated time frame for inversion motion to occur upon landing may be as short as 40 milliseconds.[13] Dynamic protection reaction of the peroneal muscles takes at least 126 milliseconds to occur after unexpected perturbations.[51] Fong et al suggest healthy male subjects peroneal muscle reaction time is between 55 to 80 milliseconds based off myoelectric investigation.

The lateral ligaments of the ankle are innervated by mechanoreceptors; receptors which sense the change in the joint position and are known to be impaired following a lateral ankle sprain.[18] . The mechanoreceptors are most active in the sensation of joint movements near the end ranges of motion. It is thought that after an injury to the lateral ankle, mechanoreceptors in the nervous tissue take much longer to heal than the ligament.[7] The decreased ability to sense changes in the ankle joint may have negative effect of postural control.[7] Mechanical instability of the ankle is a result of anatomical changes after the initial ankle injury. These changes include

pathologic laxity, impaired arthrokinematics, synovial changes, and the development of degenerative joint disease.[10] Pathologic laxity is often a result of ligamentous damage. The extent of pathologic laxity is dependent on the amount of damage caused to the lateral ligaments of the ankle. Arthrokinematic impairments is another insufficiency that may contribute to mechanical instability. An arthrokinematic restriction related to repetitive ankle sprains involves an anteriorly and inferiorly displaced distal fibula. This displacement may cause the ATFL to be slack in its resting position thus allowing a greater range of rearfoot supination before the ATFL becomes taut. Hypomobility from this change in fibular placement may also predispose the lateral ankle to injury. If the talocrual joint is not able to fully dorsiflex the joint will never reach its closed-pack position during stance which allows for greater movement. Mechanical instability may also result due to synovial hypertrophy and impingement or the development of degenerative joint lesions. Synovial inflammation often causes patients to report pain and instability due to impingement of hypertrophied synovial tissue between bones of the ankle complex. Repetitive bouts of ankle instability may also cause degenerative changes in the ankle complex. [13]

POSTURAL CONTROL

Postural control is the ability to maintain a desired postural orientation in response to perturbations generated from either internal or external sources.[6] Postural stability is the ability to maintain the body center of mass (COM) with respect to the base of support.[20] A person's COM is located around S1 or S2, and base of support, the feet, is small, which makes stability more difficult.[52] When postural control is stressed humans have the ability to correct the COM in order to maintain balance and not fall when their COM falls outside their base of support.[9]

Maintenance of postural control involves use of three somatosensory systems, visual, vestibular, and somatosensory systems.[53]

One of the most commonly utilized measures of sensorimotor outcome is single leg postural control.[9] Impaired postural control during single-leg stance has been demonstrated frequently in individuals after acute ankle sprain.[13] The Romberg test is a commonly used non-instrumental test of static postural control. Impaired postural control is likely due to a combination of both impaired proprioception and neuromuscular control. The foot pronates and supinates when balancing in a single leg stance in efforts to try and keep the COM above the base of support because of the impairments.[13,10] Impairments may be identified through a variety of tests. Single stance balancing reveals deficits in postural control and may be performed on force plates to collect COP data. The star excursion balance test also detects deficits in postural control. The star excursion balance test requires the subject to balance on the injured limb while reaching out as far as they can with the opposite limb along a line in the anterior, posteromedial, and posterolateral planes. The farther the individual reaches out along the line, the more the separation of COM and COP is challenged.

Dynamic postural control is defined as the ability to tolerate separation of COM and center of pressure (COP) while transitioning from static to dynamic tasks.[20] When the distance between the COM and the COP increases mechanical stability decreases and postural control must act to return the COM to a stable position.[21]

POSTURAL CONTROL DEFICITS FOLLOWING A LATERAL ANKLE SPRAIN

Following a lateral ankle sprain, multiple studies have identified impairments in postural control.[9,10,19] Gross and Marti reported more osteophytes and subchondral sclerosis in volleyball players with a history of recurrent ankle sprains in comparison to a healthy control group. [54]

Changes to the neuromuscular system that provides dynamic support to the ankle may occur following damage to the lateral ligaments which may be observed by balance deficits. It was initially reported that impaired postural control following a lateral ankle sprain occurred secondary to damaged mechanoreceptors in the lateral ligaments which resulted in proprioceptive deficits.[55] However, more recent findings have suggested that impaired proprioception does not fully account for why ligament damage predisposes athletes to functional instability. Impaired neuromuscular control results in deficits of the "dynamic defense mechanism" which protects the complex from hypersupination of the rearfoot.[56] Functional insufficiencies following ankle sprains have been demonstrated by deficits in ankle proprioception, cutaneous senstation, nerve-conduction velocity, neuromuscular response times, postural control, and strength.[57] It has been suggested that alteration in muscle-spindle activity in the peroneal muscles may be more important than altered mechanoreceptor activity in proprioceptive deficits at the ankle.[57] Peroneal nerve palsy has been reported following lateral ankle sprains resulting in impaired cutaneous sensation and slowed nerve-conduction velocity.[58] Impaired neuromuscular-recruitment patterns have been observed in individuals with a history of repetitive ankle sprains. This is commonly assessed by looking at reflexive response times of the peroneal muscles to inversion perturbations.[25] If peroneal response is impaired it may be due to impaired proprioception, slowed nerve-conduction velocity, or central impairments in neuromuscular-recruitment strategies.[13]

In a systematic review by Wikstrom, investigators used bilateral comparison to identify postural control deficits in the injured limb.[9] Often times when evaluating an injury the injured limb is compared to the contra lateral limb as "normal" for the individual. However, there has been a suggested bilateral impairment of postural control following an acute ankle sprain.[9]

Bullock-Saxton et al, found bilateral deficits of gluteus medius recruitment in subjects with a history of severe unilateral ankle sprain.[25] This bilateral impairment suggests that central changes may occur after lateral ankle sprain in addition to the peripheral changes, thus indicating central neural adaptations to peripheral joint condition.[9,13] The central impairments may put the individual in greater risk for recurrent injury or other lower extremity injury and may be the cause of chronic ankle instability.[24] Because of this bilateral impairment, caution must be taken when comparing the injured limb to the uninjured limb while making return to play decisions and when conducting research. Longitudinal assessment of postural control impairments may be much more useful in determining the significance of impairments. By comparing an assessment post-injury to a pre-injury assessment, a better determination of the degree of impairment is possible.

GAIT INITIATION

Dynamic balance requires the central nervous system to integrate multiple sensory and motor pathways so that the body can coordinate both postural intentional movement components. It has been suggested that COM-COP distance may be used as a variable sensitive to changes in postural stability.[21] The ability of the postural control system to handle the separation in COP-COM during GI is often used as a measure of dynamic stability.[20] .[39] GI begins with a separation of the COM and COP.[21] This is a transitional phase between static stance and the start of steady-state walking.[39] Muscles of the lower extremity activate to create movement during gait initiation. Initially, there is an inhibition of the soleus and an onset of the tibialis anterior of both the swing and stance limb. With this activation there is a backward shift of the COP. Swing limb hip abductors also aid in the shift of the COP towards the swing limb. Activation of muscles at the ankle and hip then shifts the COP forward towards the intended stance limb.[59]

Decoupling of the COM and COP completes the first phase of gait initiation according to Jian et al. called anticipatory phase.[60,61] This phase ends with toe-off of the swing limb. The second phase of gait initiation is the stepping motion from toe-off of the swing limb to heel-strike of the same limb, and toe-off of the stance limb, called the execution phase.[59,61] Breniere et al. found that the higher the intended gait velocity the longer the duration of the anticipatory phase of gait initiation and the longer the step length.[61]

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Appendix C

TABLES AND FIGURES

Table 1: Demographic Information

Group	Mean Age (yrs)	Mean Height (cm)	Mean Weight (kg)
LAS	20 ± 1.5	176.41 ± 12.15	82.94 ± 22.67
Control	19 ± 1.1	176.16 ± 10.44	78.10 ± 21.41

There were no significant differences between groups for age, height, or weight.

Table 2 A: COP Displacement Within Groups

	Injured Stance	Injured Swing	t	p
Mean LAS S1 A/P (cm)	2.79 ± 0.92	3.50 ± 1.55	-1.80	0.106
Mean CTRL S1 A/P (cm)	3.81 ± 1.09	4.41 ± 1.58	-1.46	0.177
Mean LAS S1 M/L (cm)	4.37 ± 1.32	5.22 ± 2.08	-2.07	0.069#
Mean CTRL S1 M/L (cm)	4.56 ± 1.70	5.06 ± 1.89	-1.56	0.154
Mean LAS S2 A/P (cm)	2.19 ± 1.10	1.86 ± 0.65	0.85	0.415
Mean CTRL S2 A/P (cm)	2.31 ± 2.14	1.48 ± 0.87	1.08	0.309
Mean LAS S2 M/L (cm)	19.62 ± 2.66	18.47 ± 3.72	2.15	0.060#
Mean CTRL S2 M/L (cm)	17.57 ± 4.62	17.97 ± 4.37	-0.56	0.590
Mean LAS S3 A/P (cm)	15.45 ± 1.70	17.00 ± 3.17	-1.61	0.142
Mean CTRL S3 A/P (cm)	17.87 ± 3.82	17.85 ± 2.74	0.03	0.981
Mean LAS S3 M/L (cm)	1.86 ± 1.41	1.49 ± 0.64	0.83	0.426
Mean CTRL S3 M/L (cm)	1.51 ± 1.03	1.91 ± 1.23	-1.35	0.208

There were no significant differences within either group for COP displacement in S1, S2, or S3.
= trend towards significance

Table 2 B: COP Displacement Between Groups

	LAS	CTRL	t	p
Mean Injured Stance S1 A/P (cm)	2.79 ± 0.92	3.81 ± 1.09	-2.015	0.075#
Mean Injured Swing S1 A/P (cm)	3.50 ± 1.55	4.41 ± 1.58	-1.320	0.219
Mean Injured Stance S1 M/L (cm)	4.37 ± 1.32	4.56 ± 1.70	-0.396	0.701
Mean Injured Swing S1 M/L (cm)	5.22 ± 2.08	5.06 ± 1.89	0.283	0.784
Mean Injured Stance S2 A/P (cm)	2.19 ± 1.10	2.31 ± 2.14	-0.142	0.890
Mean Injured Swing S2 A/P (cm)	1.86 ± 0.65	1.48 ± 0.87	1.346	0.211
Mean Injured Stance S2 M/L (cm)	19.62 ± 2.66	17.57 ± 4.62	2.214	0.054#
Mean Injured Swing S2 M/L (cm)	18.47 ± 3.72	17.97 ± 4.37	0.475	0.646
Mean Injured Stance S3 A/P (cm)	15.45 ± 1.70	17.87 ± 3.82	-1.919	0.087
Mean Injured Swing S3 A/P (cm)	17.00 ± 3.17	17.85 ± 2.74	-0.683	0.512
Mean Injured Stance S3 M/L (cm)	1.86 ± 1.41	1.51 ± 1.03	0.760	0.467
Mean Injured Swing S3 M/L (cm)	1.49 ± 0.64	1.91 ± 1.23	-1.118	0.293

There were no significant differences between groups for COP displacement in S1, S2, or S3.
= trend towards significance

Table 3 A: COP-COM Separation Within Groups

	Injured Stance	Injured Swing	t	p
Mean LAS MI (cm)	4.85 ± 0.87	4.40 ± 0.73	1.55	0.164
Mean CTRL MI (cm)	4.87 ± 1.43	4.42 ± 1.59	1.34	0.221
Mean LAS L1 (cm)	10.21 ± 1.87	11.07 ± 2.92	-1.48	0.184
Mean CTRL L1 (cm)	10.35 ± 2.70	10.95 ± 3.11	-1.08	0.317
Mean LAS L2 (cm)	15.83 ± 3.60	16.67 ± 2.56	-0.94	0.377
Mean CTRL L2 (cm)	15.16 ± 4.06	15.26 ± 3.46	-0.09	0.933
Mean LAS HS^{-1} (cm)	27.66 ± 3.95	29.02 ± 4.22	-0.92	0.388
Mean CTRL HS^{-1} (cm)	29.43 ± 3.08	30.56 ± 4.61	-1.66	0.140

There were no significant differences within groups for COP-COM separation for MI, L1, L2, or HS^{-1}.

Table 3 B: COP-COM Separation Between Groups

	LAS	CTRL	t	p
Mean Injured Stance MI (cm)	4.85 ± 0.87	4.87 ± 1.43	-0.05	0.960
Mean Injured Swing MI (cm)	4.40 ± 0.73	4.42 ± 1.59	-0.07	0.948
Mean Injured Stance L1 (cm)	10.21 ± 1.87	10.35 ± 2.70	-0.26	0.805
Mean Injured Swing L1 (cm)	11.07 ± 2.92	10.95 ± 3.11	0.28	0.791
Mean Injured Stance L2 (cm)	15.83 ± 3.60	15.16 ± 4.06	0.77	0.467
Mean Injured Swing L2 (cm)	16.67 ± 2.56	15.26 ± 3.46	1.74	0.126
Mean Injured Stance HS^{-1} (cm)	27.66 ± 3.95	29.02 ± 4.22	-1.59	0.157
Mean Injured Swing HS^{-1} (cm)	29.43 ± 3.08	30.56 ± 4.61	-0.71	0.499

There were no significant differences between groups for COP-COM separation for MI, L1, L2, or HS^{-1}.

Table 4 A: Spatiotemporal Measures Within Groups

	Injured Stance	Injured Swing	t	p
Mean LAS Step Length (m)	0.59 ± 0.05	0.62 ± 0.09	-1.53	0.170
Mean CTRL Step Length (m)	0.64 ± 0.06	0.65 ± .08	-1.22	0.263
Mean LAS Step Velocity (m/s)	0.56 ± 0.08	0.61 ± 0.09	-2.40	0.047*
Mean CTRL Step Velocity (m/s)	0.63 ± 0.09	0.61 ± 0.10	0.89	0.405
Mean LAS Stance Width (m)	0.24 ± 0.03	0.24 ± 0.03	-0.28	0.786
Mean CTRL Stance Width (m)	0.23 ± .06	0.22 ± 0.06	2.02	0.083
Mean LAS Step Width (m)	0.23 ± 0.04	0.23 ± .05	-0.16	0.877
Mean CTRL Step Width (m)	0.19 ± .05	0.24 ± 0.07	-1.98	0.089

There were significant differences within the LAS groups for step velocity. There were no significant differences within groups for step length, stance width, or step width.
* = significance

Table 4 B: Spatiotemporal Measures Between Groups

	LAS	CTRL	t	p
Mean Injured Stance Step Length (m)	0.59 ± .05	0.64 ± 0.06	-1.64	0.145
Mean Injured Swing Step Length (m)	0.62 ± 0.09	0.65 ± 0.08	-0.87	0.413
Mean Injured Stance Step Velocity (m/s)	0.56 ± 0.08	0.63 ± 0.09	-2.27	0.057#
Mean Injured Swing Step Velocity (m/s)	0.61 ± 0.09	0.61 ± 0.10	0.04	0.971
Mean Injured Stance Stance Width (m)	0.24 ±0.0 3	0.23 ± 0.06	0.74	0.486
Mean Injured Swing Stance Width (m)	0.24 ± 0.03	0.22 ± 0.06	1.41	0.202
Mean Injured Stance Step Width (m)	0.23 ± 0.03	0.19 ± 0.05	2.56	0.038*
Mean Injured Swing Step Width (m)	0.23 ± 0.05	0.24 ± 0.07	-0.29	0.779

There were significant differences between groups for step width and a trend towards significance for step velocity. There were no significant differences within groups for step length, or step width.
* = significance; # = trend towards significance

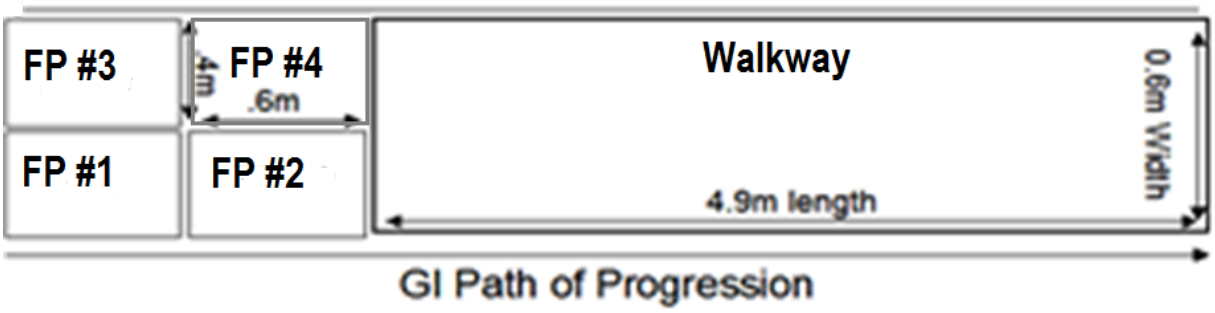

Figure 1: Force plate and walkway set up.

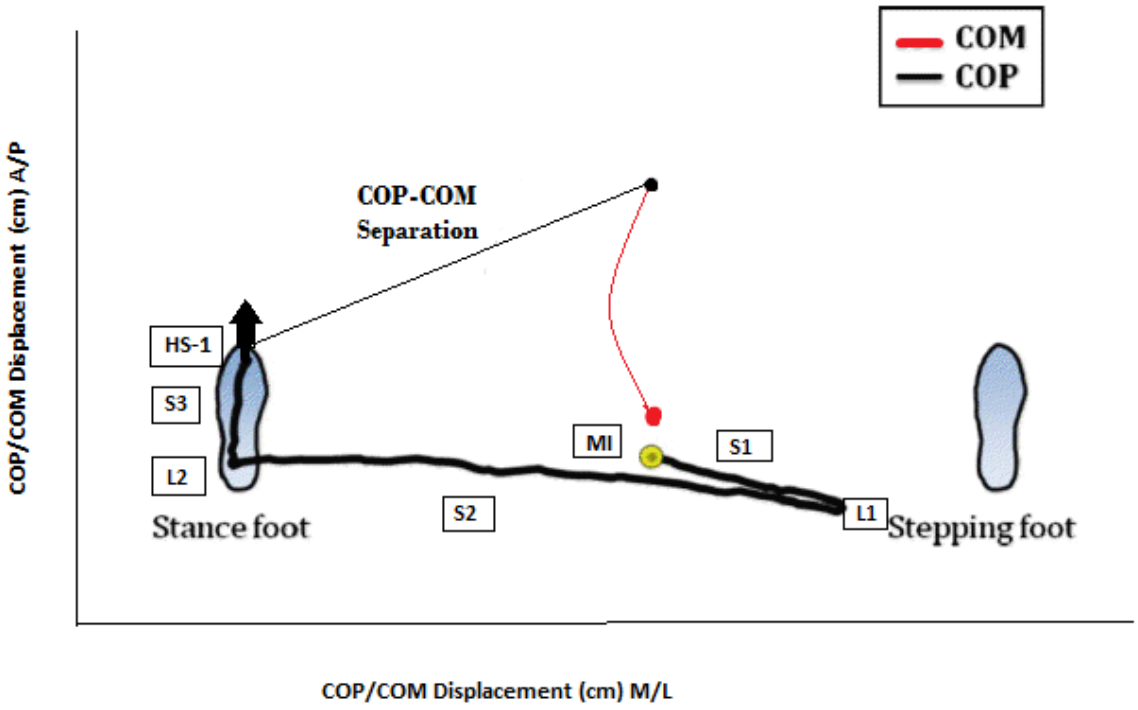

FIGURE 2: Overhead view of displacement of COP and COM during gait initiation with the right foot as the initial stepping foot.

Figure 3: FAAM Questionnaire

Revised Foot and Ankle Ability Measure

Subject Number: _____ **Date:**_____

Foot and Ankle Ability Measure (FAAM)

Please answer **every question** with one response that most closely describes to your condition within the past week.

If the activity in question is limited by something other than your foot or ankle mark not applicable (N/A).

	Side	No difficulty	Slight Difficulty	Moderate Difficulty	Extreme Difficulty	Unable to do	N/A
Standing	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking on Even Ground	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking on even ground without shoes	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking up hills	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking down hills	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Going up stairs	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Going down stairs	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking on uneven ground	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Stepping up and down curbs	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Squatting	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Coming up on your toes	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking initially	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking 5 minutes or less	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking approximately 10 minutes	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Walking 15 minutes or greater	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐

Because of your **foot and ankle** how much difficulty do you have with:

Standing	Side	No difficulty	Slight Difficulty	Moderate Difficulty	Extreme Difficulty	Unable to do	N/A
Home responsibilities	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Activities of daily living	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Personal Care	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Light to moderate work (standing, walking)	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Heavy work (push/pulling, climbing, carrying)	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Recreational activities	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐

How would you rate your current level of function during your usual activities of daily living from 0 to 100 with 100 being your level of function prior to your foot or ankle problem and 0 being the inability to perform any of your usual daily activities?

Right: ☐☐☐.0 %

Left: ☐☐☐.0 %

FAAM Sports Scale

Because of your **foot and ankle** how much difficulty do you have with:

	Side	No difficulty	Slight Difficulty	Moderate Difficulty	Extreme Difficulty	Unable to do	N/A
Running	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Jumping	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Landing	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Starting and stopping quickly	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Cutting/lateral movements	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Low impact activities	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Ability to perform activity with your normal technique	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐
Ability to participate in your desired sport as long as you would like	Right	☐	☐	☐	☐	☐	☐
	Left	☐	☐	☐	☐	☐	☐

How would you rate your current level of function during your sports related activities from 0 to 100 with 100 being your level of function prior to your foot or ankle problem and 0 being the inability to perform any of your usual daily activities?

Right: ☐☐☐.0 %

Left: ☐☐☐.0 %

Overall, how would you rate your current level of function?

Right:

☐ Normal ☐ Nearly normal ☐ Abnormal ☐ Severely abnormal

Left:

☐ Normal ☐ Nearly normal ☐ Abnormal ☐ Severely abnormal

Figure 4: Ankle Instability Index Questionnaire

Revised Ankle Instability Instrument

Subject Number: _____ Date: _____

Ankle Instability Instrument

Instructions

This form will be used to categorize your ankle instability. Please fill out the form completely. If you have any questions, please ask the administrator of the survey. Please mark the ○ completely. Thank you for your participation.

1. Have you ever sprained an ankle?	Right	○Yes	○ No
	Left	○ Yes	○ No
2. Have you ever seen a doctor for an ankle sprain?	Right	○ Yes	○ No
	Left	○ Yes	○ No
3. Did you ever use a device (such as crutches) because you could not bear weight due to an ankle sprain?	Right	○ Yes	○ No
	Left	○ Yes	○ No
If yes, 3a. In the most serious case, how long did you need the device? Right: ○ 1-3 days ○ 4-7 days ○ 1-2 weeks ○ 2-3 weeks ○ >3weeks Left: ○ 1-3 days ○ 4-7 days ○ 1-2 weeks ○ 2-3 weeks ○ >3weeks			
4. Have you ever experienced a sensation of your ankle "giving way"?	Right	○ Yes	○ No
If yes,	Left	○ Yes	○ No
4a. When was the last time your ankle "gave way"? Right: ○ <1 month ○ 1-6 months ago ○ 6-12 months ago ○ 1-2 years ago ○ >2 yrs Left: ○ <1 month ○ 1-6 months ago ○ 6-12 months ago ○ 1-2 years ago ○ >2 yrs			
5. Does your ankle ever feel unstable while walking on a flat surface?	Right	○ Yes	○ No
	Left	○ Yes	○ No
6. Does your ankle ever feel unstable while walking on uneven ground?	Right	○ Yes	○ No
	Left	○ Yes	○ No
7. Does your ankle ever feel unstable during recreational or sport activity?	Right	○ Yes	○ No ○ N/A
	Left	○ Yes	○ No ○ N/A
8. Does your ankle ever feel unstable going *up* stairs?	Right	○ Yes	○ No
	Left	○ Yes	○ No
9. Does your ankle ever feel unstable going *down* stairs?	Right	○ Yes	○ No
	Left	○ Yes	○ No

	Right	**Left**
How many times have you sprained your ankle in the past?	_____	_____
How long has it been since your last significant ankle sprain (in months)?	_____	_____
How many times in the past 3 months has your ankle felt like it "gives way"?	_____	_____

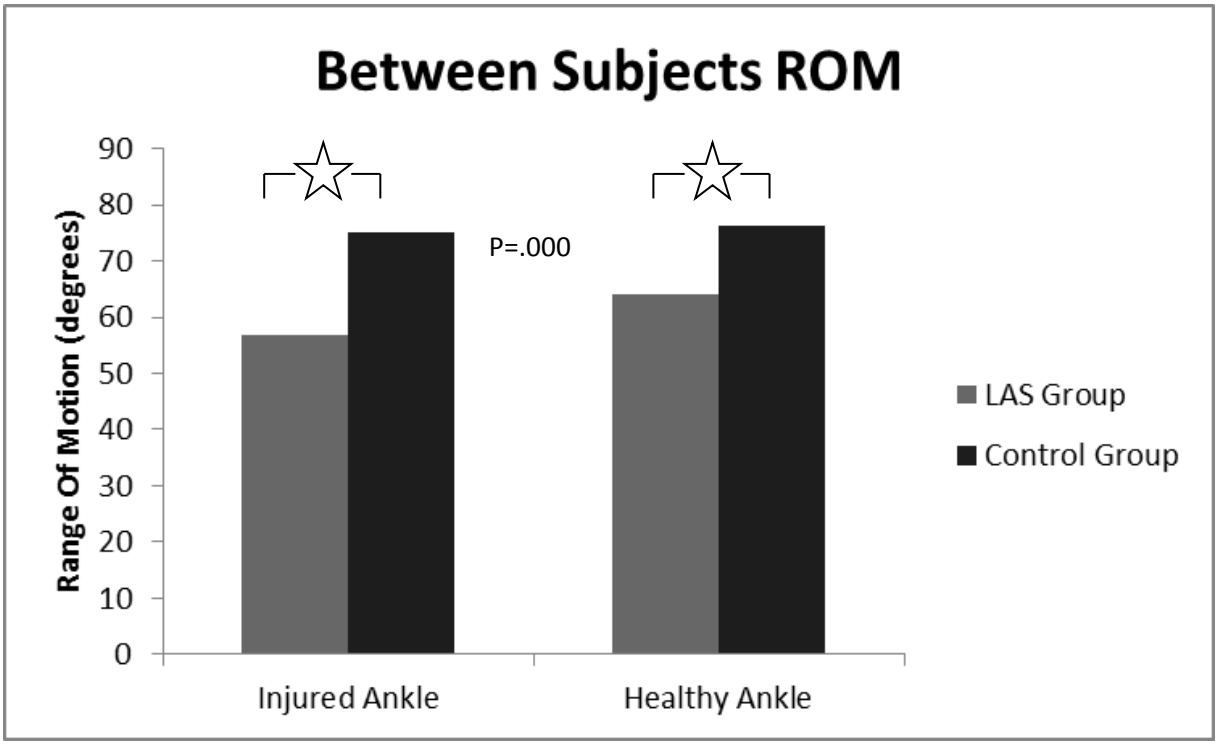

Figure 5: Mean values for ROM. Significant differences were found between groups. There was a significant difference between groups.
=☆ significance, p<.001

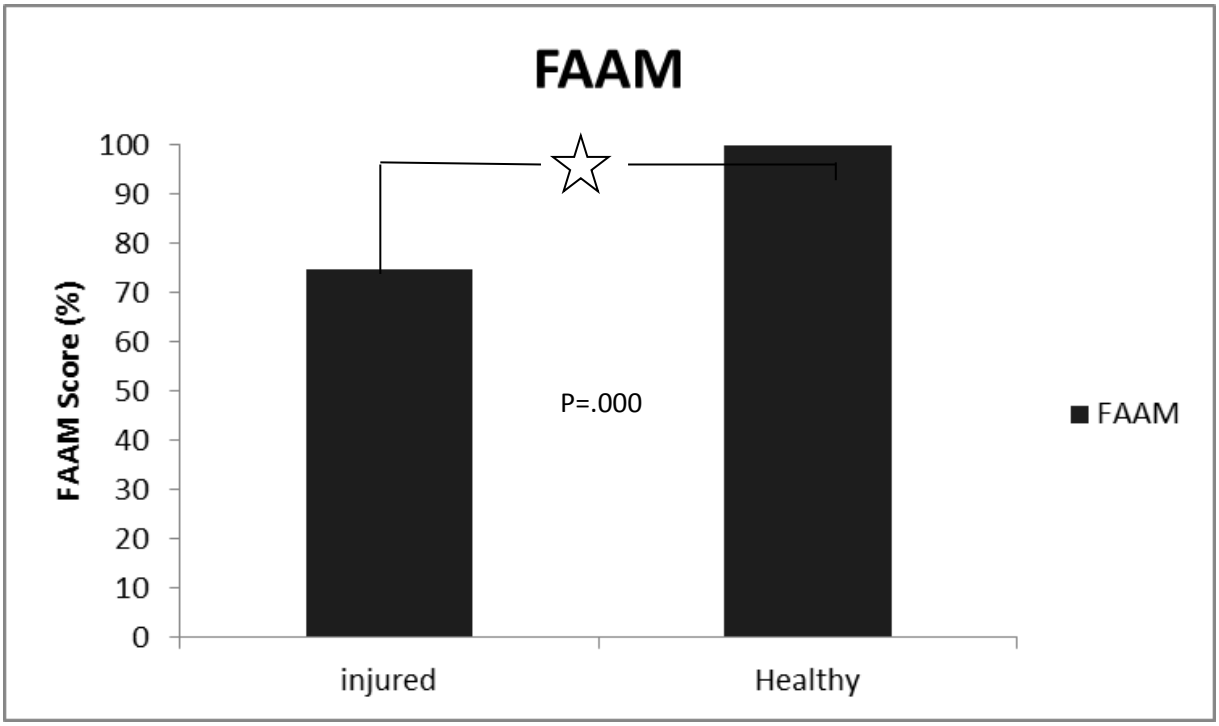

Figure 6: Mean FAAM scores within the LAS group. Significant difference was found between ankles within the LAS group.
☆ = significance, p<.001