EFFECTS OF INVERSION PERTURBATION AFTER DROP LANDING ON THE

LATENCY OF THE ANKLE MUSCULATURE

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EFFECTS OF INVERSION PERTURBATION AFTER DROP LANDING ON THE LATENCY OF THE ANKLE MUSCULATURE

Adam Charles Knight

A Dissertation

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VITA

Adam Charles Knight was born on December 30, 1981 in Hattiesburg, MS. He is the son of Chuck and Cindy Knight, and has two younger sisters, Lisa and Lori, and is married to the former Amy Wade. In 2000, he graduated with special honors from Oak Grove High School in Hattiesburg, MS. He then entered the Athletic Training program at the University of Southern Mississippi, serving as a student athletic trainer for various athletic teams, including the 2003 Conference USA football champions. He graduated in May of 2004 with highest honors with a Bachelor of Science Degree in Athletic Training. In the fall of 2004, the author began graduate school at Auburn University, serving as a graduate assistant athletic trainer for the football, cheerleading, and equestrian teams, including the 2004 undefeated and SEC Champion football team. In 2006, he completed a Master's of Education, and has since been pursuing a doctorate in Biomechanics. He married the former Amy Wade, daughter of Buddy and Janet Wade, on June 7, 2008.

DISSERTATION ABSTRACT

EFFECTS OF INVERSION PERTURBATION AFTER DROP LANDING ON THE LATENCY OF THE ANKLE MUSCULATURE

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Ankle sprains are a common injury affecting a large number of athletes and workers every year. Of specific concern is the latency of the peroneus longus and peroneus brevis which provide a dynamic defense mechanism against a lateral ankle sprain when the ankle is forced into excessive inversion. Previous research has used a tilt

platform or trap door mechanism to measure the latency of these two muscles with and without ankle support; however these studies lacked the dynamic nature usually encountered when a person lands on an obstacle that forces the foot into inversion. For the present study an outersole with fulcrum was developed to more accurately replicate the mechanism of a lateral ankle sprain when participants land from a jump.

A startle response, indicated by co-contraction of antagonist muscles has been noted for movements associated with whiplash injuries to the neck, however, the startle response has not previously been applied to the ankle. If present, this finding would have large implications for the future prevention of lateral ankle sprains. Therefore, the purpose of this study was to measure latencies of the peroneus longus, peroneus brevis, and tibialis anterior muscles during the performance of a single leg drop landing with the ankle forced into 25° of inversion by a fulcrum and outersole attached to the bottom of the participant's shoe. The latencies were measured across participants with no history of an ankle sprain, a history of a lateral ankle sprain, and a history of a high ankle sprain. Moreover, the influence of ankle taping on this response was also measured, as well as the ratio of evertor/invertor activity to determine if the startle response was present.

The results revealed that the latency of the peroneus longus and peroneus brevis was shorter using the outersole mechanism than those measured using a tilt platform/trap door, but there was no difference in the latency across the different injury groups. There was a significant interaction between the startle event and injury group, and the trend for an increasing ratio of evertor/invertor activity was present. In addition, there was a significant reduction in the latency of the peroneus longus when using ankle taping.

Future research should continue to use the outersole methodology to examine the latency of the ankle musculature. Participants with more recent ankle injuries should be examined; the startle response should be investigated further for the development of training protocols; and the affects of physical activity on the latency of the ankle musculature with ankle taping should also be examined.

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The author would like to thank Dr. Wendi Weimar for giving him the opportunity to further his academic career in the field of Biomechanics. Through countless hours of guidance, wisdom, and support, she has shaped my understanding of biomechanics and helped me develop into the educator and researcher that I am today.

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CHAPTER 1

INTRODUCTION

The lateral, or inversion ankle sprain, which damages the lateral ligaments of the ankle complex, is the most common injury in athletics (Beynnon et al., 2002; Hertel et al., 1999). The ankle supports the most weight per unit area of any joint in the human body, which contributes to the high rate of injury (Morrison & Kaminski, 2007). This injury is often seen in sports that involve repeated starting, stopping, cutting, jumping, and landing. These movements put the ankle at risk because of the frequent opportunity for the foot to be placed on another player's foot, or to catch the side of the foot and "roll the ankle" (Thacker et al., 1999). Many preventative measures have been taken to reduce the amount of lateral ankle sprains, including taping, bracing, footwear, and training. It has been proven that ankle support decreases the number of ankle sprains in high risk sports such as basketball and soccer (Eils et al., 2006) by reducing the amount of inversion at the subtalar joint. However, the effectiveness of these devices, specifically the athletic tape and the cloth wrap, are reduced as the amount of exercise time increases (Ashton-Miller et al., 1996). The ankle brace also loses its effectiveness as the amount of exercise time increases, but unlike athletic tape, the brace may be re-tightened to help limit the amount of inversion present at the subtalar joint (Thonnard et al., 1996).

The lateral ankle sprain is an injury that occurs when the ankle is forced into excessive inversion and plantarflexion (Moiler et al., 2007). Plantarflexion occurs at the

talocrural joint in the sagital plane, and inversion occurs primarily at the subtalar joint and transverse tarsal joint in the frontal plane (Kaminski & Hartsell, 2002). Fuller (1999) described a pathomechanical model that theorized the cause of a lateral ankle sprain as an increased supination moment at the subtalar joint. In an open kinetic chain, supination consists of plantar flexion, inversion, and internal rotation, while in a closed kinetic chain, supination consists of dorsiflexion, inversion, and internal rotation (Rocker, 1995). Fuller's hypothesis was that a foot with a center of pressure (COP) medial to the subtalar-joint axis will have a greater supination moment from the ground reaction force than a foot that has a more lateral relationship between the joint axis and the COP. In addition, excessive inversion and internal rotation of the rearfoot, due to a more medial COP, would also result in an increased supination moment and could lead to an injury of the lateral ligaments of the ankle (Fuller, 1999; Hertel, 2002).

When the ankle/foot complex is in this inverted/plantar flexed position the anterior talofibular ligament (ATFL) is stretched and as such is the primary ligament damaged by an inversion ankle sprain. The posterior talofibular ligament (PTFL) and the calcaneofibular ligament (CFL) also provide support to the lateral aspect of the ankle and may often be injured by the excessive inversion and plantarflexion motion that is associated with a lateral ankle sprain. Once the ATFL is damaged, a domino effect is seen, with the role of eliminating excess joint movement falling to the PTFL and CFL. Examination of cadaveric section ligaments has demonstrated that once the ATFL is ruptured, the amount of transverse plane motion of the rearfoot will increase substantially, which will cause further stress to the PTFL and the CFL (Hertel 2002). Other joint structures that may be at risk during a lateral ankle sprain include the joint

capsule of the talocrual joint and the ligaments of the subtalar joint. The peroneus longus and brevis may also be strained by the mechanism that causes a lateral ankle sprain (Hertel, 2002).

There are many factors that lead to a lateral ankle sprain. These factors may be classified as intrinsic or extrinsic factors. Intrinsic factors include deficits in strength in one or both ankles, anatomic variations (Barbaix et al., 2000), joint laxity, decreased range of motion, deficiency in proprioceptive feedback (Mohammadi, 2007), and a previous history of an ankle sprain(s) or instability (Hertel, 2002). Extrinsic factors include the type of sport, the condition of the field or court (environmental factors), errors in training, the skill of the competition, and the type of equipment and footwear (Eren et al., 2003). Of all of the listed intrinsic and extrinsic factors, the greatest predictor of a lateral ankle sprain is a previous ankle sprain(s). The re-injury rate among athletes has been reported as high as 70-80% (Hertel et al., 1999).

Ankle sprains were chosen for this project because the frequency of this injury demands attention. Second, the literature has indicated deficits in the understanding of the mechanisms of lateral ankle sprains and suggests that future research into ankle sprains should examine a dynamic task that more closely replicates a lateral ankle sprain than previously used methods and should include participants with a prior history of an ankle sprains (Cordova et al., 2000; Kernozek et al., 2008; Midgley et al., 2007; Shima et al., 2005). Specifically, the participant population for this project will include three groups, one with no previous history of an ankle sprain, one with only previous lateral ankle sprain history, and one with only high ankle sprain history.

High ankle sprains were included in the present study due to the relative lack of research on the injury. While there is limited research regarding the high ankle sprain, the mechanism of this injury is well understood. The high ankle sprain occurs at the distal tibiofibular joint, which is the articulation between the distal tibia and fibula (Williams et al., 2007). The ligaments of the distal tibiofibular joint include the anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, and the interosseous ligament (Norkus & Floyd, 2001). Stability of the distal tibiofibular joint is imperative for the ankle and lower extremity to function properly as the bones of this joint form the mortise that allows the foot to be able to move in the sagital plane and allows for the transmission of forces up the kinetic chain. The two most common mechanisms of injury for this joint are: (a) external rotation or (b) hyperdorsiflexion, or a combination of the two. External rotation and excessive dorsiflexion are two mechanisms that widen the mortise and thus contribute to high ankle sprains. Excessive external rotation widens the mortise between the tibia and fibula by forcing the talus into the fibula, and in excessive dorsiflexion, the talus is forced between the distal tibia and fibula, thus "wedging" them apart (Williams et al., 2007).

Compared to the lateral ankle sprain, the high ankle sprain is much less common and has been found to only account for 1-11% of all ankle injuries (Williams et al., 2007). The high ankle sprain is difficult to evaluate and diagnose, which has led to the limited research on evaluation, treatment, and long-term prognosis for this type of injury (Norkus & Floyd, 2001). However, as the recovery of this injury is much longer than for a lateral ankle sprain and due to the small amount of research on the long terms effects

of a high ankle sprain, it is beneficial to learn how a person with a history of a high ankle sprain will respond to an inversion perturbation.

Laboratory replication

Excessive inversion at the subtalar joint is the most common mechanism leading to a lateral ankle sprain (Hertel, 2002). Many laboratory experiments have replicated this mechanism of injury by using a tilt platform/trap door that randomly inverts the ankle to 30° while the latency of the peroneus longus and peroneus brevis is measured with and without ankle support (Ashton-Miller et al., 1996; Cordova et al., 2000; Cordova & Ingersoll, 2003; Hopkins et al., 2007; Karlsson & Andreasson, 1992; Kernozek et al., 2008; Konradsen et al., 1997; Lohrer et al., 1999; Lynch et al., 1996; Midgley et al., 2007; Mitchell et al., 2008; Shima et al., 2005). While these experiments provide a valuable step in our understanding of lateral ankle sprain mechanics, the process employed lacks construct validity in its ability to replicate a lateral ankle sprain. This is due to the fact that many ankle sprains occur during foot placement, and when using the tilting platform/trap door, there is often equal weight distribution and the proprioceptive cues from the hip and ankle are different for when the floor falls out from underneath the person than would occur when landing on the foot of another person (Shima et al., 2005).

The Startle Response

The startle response is defined as a quick acting muscular response caused by a rapidly applied, intense stimuli. It is believed to be a primitive response to protect people from external threats. An example of the startle response would be when a person is

struck from behind unexpectedly while driving a car. The human body has developed a pre-programmed response to help prevent injury during this type of unexpected perturbation. It may be triggered by acoustic, visual, or tactile stimuli (Rodriguez-Fornells et al., 1999). The startle response is elicited in humans whenever a transient perturbation is introduced. The initial reaction to this perturbation is exaggerated and is an example of the startle response. Research into the startle response evoked by rear end collisions has shown that the startle response involves co-activation of both the agonist and antagonist muscle groups (Siegmund et al., 2008). This co-activation serves as a "clamp down" strategy to protect the body. After repeated exposures to the same stimulus, this response shows habituation, which results in a decreased response of the antagonist muscle group and an increased response of the agonist muscle group. This causes attenuation in the muscle group that opposes restoration of the neutral posture and provides the agonist muscle an unencumbered avenue to provide the required/desired joint action (Siegmund et al., 2008).

When the ankle is forced into excessive inversion, one of three possible outcomes will occur. It is known that during normal physical activity (running and jumping) there is co-contraction of both the evertor muscle group and the invertor muscle group in order to keep the ankle complex stiff and protect it from injury. This joint stiffness maintains the ankle in a neutral position and prevents excessive range of motion (Hertel, 2002). Therefore, one possible outcome is co-contraction of the evertors and invertors of the ankle immediately after the ankle is forced into inversion. This type of action would not help prevent the excessive inversion moment, as the activity of the invertor muscle group would negate the protective mechanism of the evertor muscle group. A second action

that may occur after the ankle is forced into inversion is greater activity in the invertor muscle group than the evertor muscle group. This type of action would increase the magnitude of the inversion moment and could potentially increase the magnitude of the ankle sprain. The third action that may occur is greater activity in the evertor muscle group when compared to the invertor muscle group. This response would be beneficial as the evertor muscle group would help prevent, or at least attenuate, the excessive inversion moment produced by the external perturbation.

It is hypothesized by the author that when the startle response is applied to the ankle musculature, the first exposure to the inversion perturbation will cause co-activation of both the invertor and evertor muscle groups. The evertor muscle group, primarily the peroneus longus and brevis, provides dynamic protection against the lateral ankle sprain while the invertor muscle group, which primarily includes the tibialis anterior and tibialis posterior contribute to an inversion joint action. Ideally, when an ankle is forced into inversion, it would be desirable for only the evertors to be active, as these muscles would be in a position to stop or at least attenuate the inversion moment. However, if the startle response co-contraction occurs during initial exposure to the inversion perturbation, then both the evertors (protectors) and the invertors would be active and the invertors would act to attenuate the protective ability of the evertors. Therefore, the startle response would reduce the ability of the ankle musculature to prevent a lateral ankle sprain by negating the eversion moment provided by the peroneus longus and peroneus brevis. After repeated exposure to the inversion perturbation, there should be less activity in the invertor muscle group and the same level of activity in the evertor muscle group, based on previous work that found less activity in the antagonist

muscle group and the same level of activity in the agonist muscle group (Siegmund et al., 2008). This habituation response would help protect against lateral ankle sprains. This startle response mechanism is intriguing and has been included in the present study because: (1) it is not known if it exists and (2) if it does, there are tremendous applications for prevention training.

It is apparent that the ankle has received much attention in the literature; however there is still work to be conducted into the investigation of ankle sprains. Specifically, this project hopes to contribute to the findings of previous work by: (1) utilizing an inverting mechanism that more closely replicates the loading pattern of ankle sprains that occur outside the laboratory; (2) by investigating individuals who have sustained a previous lateral ankle sprain and individuals who have sustained a previous high ankle sprain; (3) by investigating the role of the startle response on muscle activation patterns during a loading pattern that more closely replicates a lateral ankle sprain; and (4) by investigating the role that ankle taping plays in the latency of the ankle musculature when exposed to an inversion perturbation during a drop landing.

Statement of Purpose

The purposes of this study are: (1) to determine the latency of the peroneus longus and peroneus brevis (separately) to an inversion perturbation during a single leg drop landing; (2) to determine how participants with healthy ankles, participants with a prior history of a lateral ankle sprain, and participants with a prior history of a high ankle sprain differ in the latency of the peroneus longus and peroneus brevis when exposed to an inversion perturbation during a single leg drop landing; (3) to determine if the startle

response is part of the initial exaggerated reaction to an inversion perturbation during a single leg drop landing; and (4) to determine the effect of athletic tape on the latency of the peroneus longus when the ankle is forced into inversion during a single leg drop landing.

Hypotheses

The study has the following hypotheses. The main hypothesis is as follows:

1) The latency of the peroneus longus and peroneus brevis will be statistically significantly greater in participants with a reported history of a lateral ankle sprain and a reported history of a high ankle sprain than participants with reportedly healthy ankles.

The secondary hypotheses are as follows:

2) The startle response will be part of the exaggerated first reaction to the ankle inversion perturbation. This will be evidenced by a co-contraction of the peroneus longus/peroneus brevis and tibialis anterior during the initial presentation of the ankle perturbation. Further exposure to the ankle perturbation will cause a decrease in the previously recorded magnitude of the tibialis anterior muscle.
3) The application of athletic tape will cause the participants have a significantly shorter latency in the peroneus longus when compared to no athletic tape condition.

Limitations

Although the proposed study will attempt to replicate the mechanism of a lateral ankle sprain, the exact mechanism of a lateral ankle sprain is difficult to recreate in any laboratory setting. Most ankle sprains occur when people meet an unexpected perturbation that forces the ankle into excessive inversion and plantar flexion. While this study will force the participants into inversion, excessive joint actions will be avoided to prevent actually injuring the participants. The amount of inversion produced will be approximately 25°, while the reported amount of inversion needed to sprain the ankle ligaments has been reported to be in excess of 40° (Ashton-Miller et al., 1996). In addition, the participants may anticipate the perturbation with activity in the ankle musculature before landing and encountering the perturbation. To avoid this anticipation, a flat sole will be used in the study to help prevent the participants from anticipating the perturbation and having excessive muscle activity prior to touchdown.

Delimitations

The study will only included participants between the ages of 19-30, as this is the age range of most college and professional athletes. Additionally, participants older than 30 years of age will be excluded due to changes in the neuromuscular system that occurs with aging.

Definition of Terms

Lateral Ankle Sprain

The lateral, or inversion ankle sprain, is a sprain of the lateral ligaments of the ankle caused by the mechanism of excessive inversion or excessive inversion and plantar flexion. This is the most common type of ankle sprain, and the ligament frequently sprained with a lateral ankle sprain is the anterior talofibular ligament. The calcaneofibular ligament and the posterior talofibular ligament may be sprained as well. This type of ankle sprain commonly occurs in sports that involve frequent stopping, starting and cutting, and in sports that require jumping where the athlete may land on the foot of another player (Hertel, 2002; Migdley et al., 2007).

High Ankle Sprain

This is also referred to as a syndesmosis ankle sprain. This sprain is much less common than the lateral ankle sprain; it involves the ligaments of the distal or inferior tibiofibular joint. The common mechanism for a high ankle sprain is hyperdorsiflexion or external rotation. Both of these mechanisms will force the talus upward between the tibia and fibula and sprain the anterior inferior talofibular ligament, the posterior inferior talofibular ligament, and/or the interosseous membrane. The rehabilitation from a high ankle sprain is much longer than that for a lateral ankle sprain, and the long term effects of a high ankle sprain are unknown (Norkus & Floyd, 2001; Williams et al., 2007).

Startle Response

The startle response is caused by a quick, rapid stimulus that results in a whole body muscular response (Rodriguez-Fornells, 1999). The first contact with an unexpected perturbation typically results in an exaggerated muscle response. This response likely contains a startle component and a postural component. Research into rapid accelerations of the trunk has found that a centrally generated postural response is initiated first in order to maintain head-trunk stabilization (Blouin et al., 2003). It is the startle response, when added to the normal postural response, which causes the exaggerated muscle response. This initial exposure involves co-activation of both the agonist and antagonist muscle groups of the body segment that experiences the greatest velocity. After repeated exposures to the same stimulus, this response shows habituation, with less activity in the muscles (antagonist muscle group) that oppose restoration of the original posture (Siegmund et al., 2008). This attenuation is explained by extinction of the startle response, leaving only the postural response.

Muscle Latency

For this study, latency will be defined as the time from contact of the fulcrum with the ground (beginning of inversion moment) to the time of first rise in muscle activity (5 standard deviations above muscle activity 200 ms before touchdown), as recorded by electromyography. Latency defines the time it takes for muscle activity to begin after contact with the ground. The peroneus longus and peroneus brevis help prevent lateral ankle sprains by providing an eversion moment through an eccentric contraction which will creates a dynamic defense mechanism. However, this is only

effective if the muscles contract quickly enough to prevent the excessive inversion range of motion.

CHAPTER II

REVIEW OF LITERATURE

The lateral ankle sprain, accounting for 85% of all ankle sprains (Shaw et al., 2008) is the most frequently occurring injury in athletics, resulting in a high cost of care and more lost time from competition than any other joint related injury (DiStefano et al., 2008; Fox et al., 2008). Each year in the United States, over two million people sustain an ankle sprain (Ubell et al., 2003) and it has been reported that ankle sprains account for up to 25% of all time lost from athletic competition (Ashton-Miller et al., 1996). The most common injury in volleyball is the ankle sprain (Midgley et. al, 2007), which accounts for 40% of all injuries sustained while playing the sport, and the ankle sprain accounts for 30% of all injuries during high school basketball (DiStefano et al., 2008). Once a person sustains an ankle sprain, he or she is at a much greater risk for re-injury or chronic ankle instability (Midgley et. al., 2007; DiStefano et al., 2008).

It is evident from the research that the ankle is the most commonly injured joint in athletics and is a common injury in non-athletic settings as well (Beynnon et al, 2002). It can result in time lost from practice and competition and time lost from work and everyday activities. In light of these statistics it is important to understand the mechanisms that cause a lateral ankle sprain and the structures that are injured during a lateral ankle sprain. It is also important to understand how the body responds to a perturbation that may cause an ankle sprain.

This chapter will be divided into five sections. Section one will describe the anatomy of the talocrural, subtalar, and distal tibiofibular joints. It will also describe the mechanism of injury for a lateral ankle sprain and a high ankle sprain, and briefly review chronic ankle instability and how it relates to this study. Section two will present research that forced the ankle into inversion as part of their methodology, which has helped develop the proposed methodology for this study. Section three will present research into ankle taping and bracing and their effects on the latency of the ankle musculature. Section four will describe the startle response and its importance in the prevention of ankle sprains. Section five will provide a brief summary of the reviewed research.

Section one: Ankle Anatomy and mechanism of injury

Talocrural Joint

The talocrural joint is formed by the articulation of the dome of the talus, the medial malleolus (distal tibia), and the lateral malleolus (distal fibula) (Figure 1). Due to the shape of talocrural joint, it is considered a hinge joint that primarily allows motion only in the sagital plane, i.e. plantar flexion and dorsiflexion. The stable and tightly packed arrangement of bones and ligamentous support allows force to be transmitted from the lower leg to the foot during weight bearing. Ligamentous support for the talocrual joint is provided by the anterior talofibular ligament (ATFL), the posterior talofibular ligament (PTFL), and the calcaneofibular ligament (CFL) on the lateral side (Figure 2), and the deltoid ligament on the medial side (Hertel, 2002).

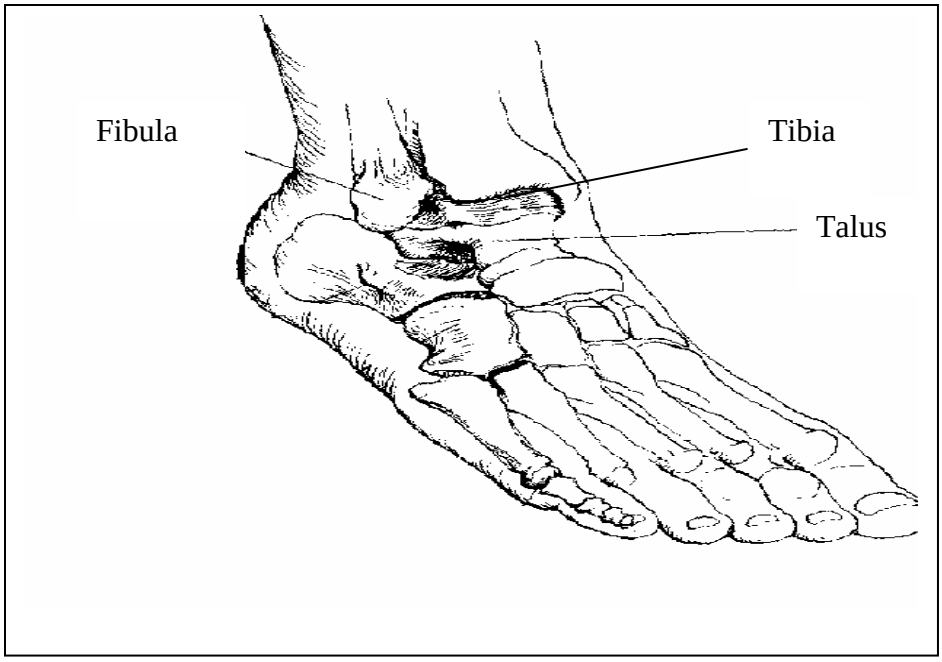

Figure 1- Bones of the talocrural joint (Hoppenfeld, 1976, pg. 203).

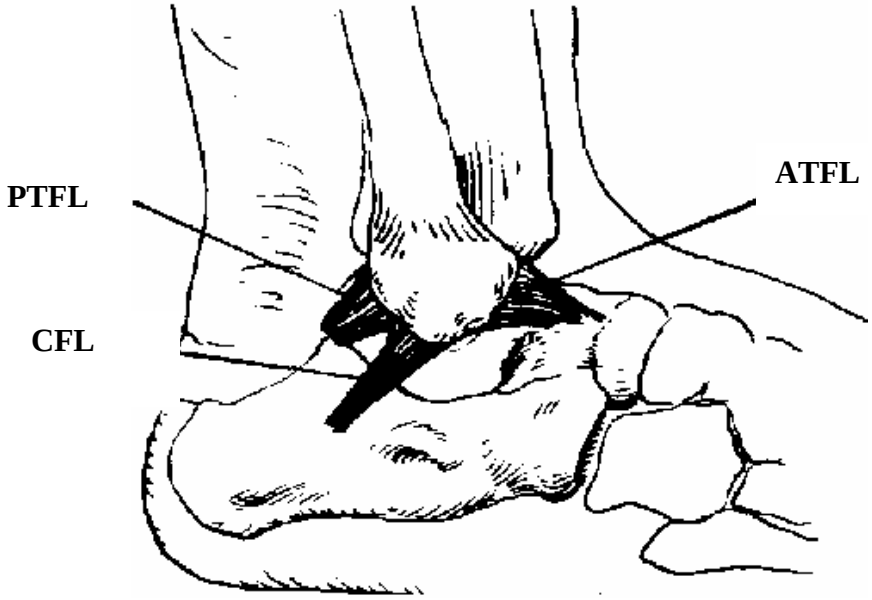

Figure 2- Ligaments of the lateral ankle (Hoppenfeld, 1976, pg. 217).

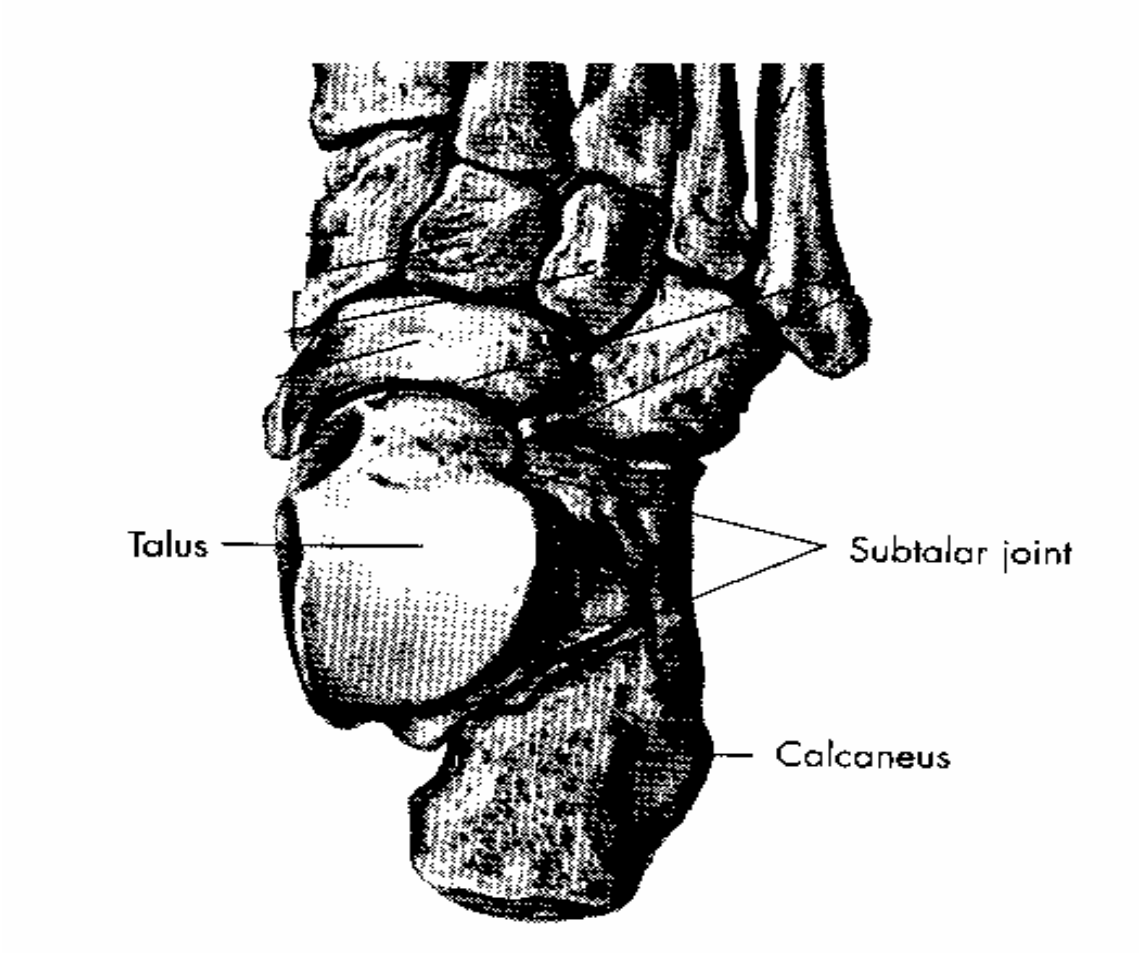

Figure 3- Subtalar joint (Floyd, 2007, pg. 281).

Subtalar Joint

The subtalar joint is located below the talocrual joint and allows for the joint actions of inversion and eversion, which primarily occur in the frontal plane (Figure 3). The subtalar joint is often divided into anterior and posterior joints that share the same axis of rotation but have separate joint cavities. The anterior subtalar joint is formed by the articulation of the talus, calcaneus, and navicular and the posterior joint is formed by the articulation of the talus and calcaneus. The subtalar joint receives ligamentous support from the cervical and interosseous ligaments, which act as cruciate ligaments for the subtalar joint. The subtalar joint also gains ligamentous stability from the CFL, lateral talocalcaneal ligament, and the fibulotalocalcaneal ligament. The ligaments of the subtalar joint may also be injured during a lateral ankle sprain (Hertel et al., 1999).

Distal Tibiofibular Joint

The distal tibiofibular joint is the syndesmotic articulation between the convex surface of the distal fibula and concave surface of the tibia (Figure 4). This joint is where the syndesmosis sprain, or high ankle sprain occurs. The ligaments of this joint are the anterior inferior tibiofibular ligament, the posterior inferior tibiofibular ligament, and the interosseous ligament. In order for the ankle and lower extremity to function properly, it is imperative that this joint be stable as it is the "receiving" portion of the force transmissions up the kinetic chain and provides the bracket of the hinge joint. Additionally, the fibula must be able to move in order to house the talus during gait, as the talus is wedged upward between the tibia and fibula during dorsiflexion. The anterior inferior tibiofibular ligament functions to hold the fibula tight against the tibia, prevent

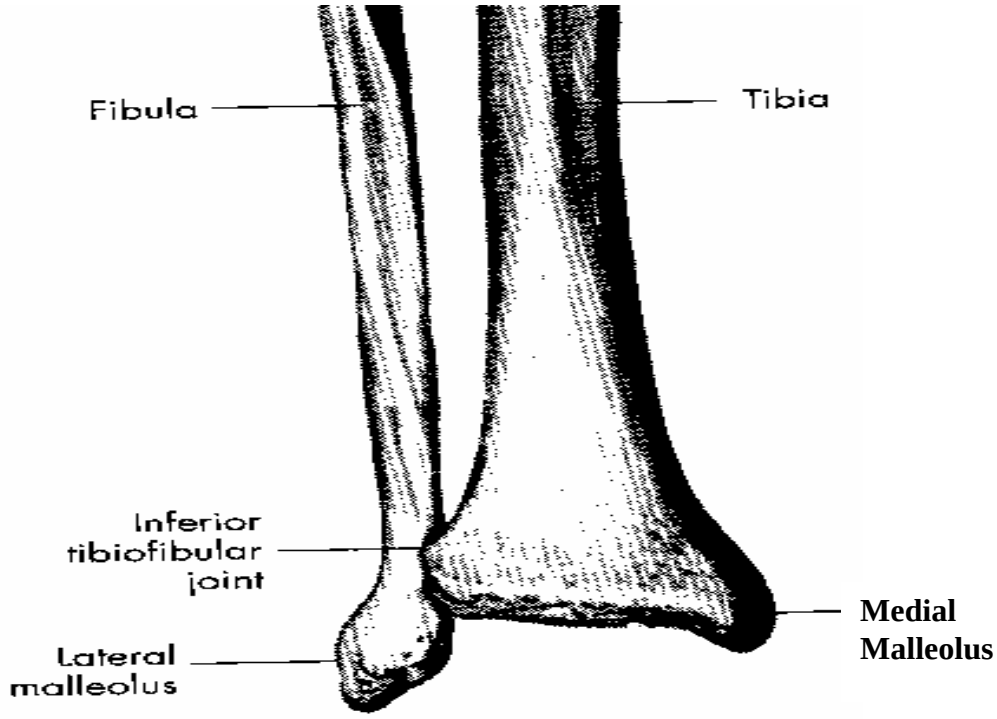

Figure 4- Inferior, or Distal tibiofibular joint (Floyd, 2007, pg. 280).

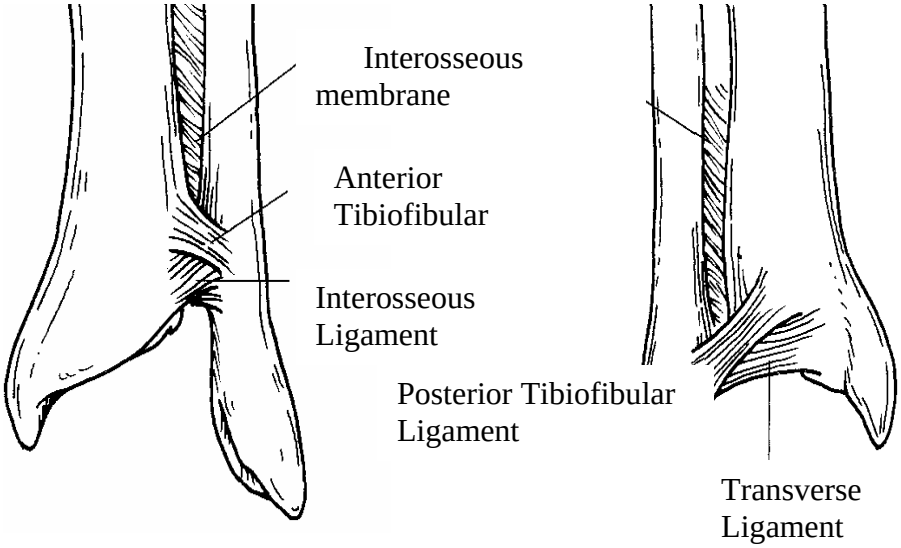

Figure 5- Ligaments of the Distal Tibiofibular Joint. Left-Anterior View; Right-Posterior View (Nordin & Frankel, 2001, pg. 246).

excessive fibular movement, and prevent external talar rotation. The ligament runs from the longitudinal tubercle on the anterior aspect of lateral malleolus to the anterolateral tubercle of the tibia. The fibers of the ligament increase in length as they move from the proximal end to the distal end, with the distal fibers being the longest. The posterior inferior tibiofibular ligament has both superficial and deep components. The superficial component runs from the posterior tubercle of the tibia and proceeds distally and laterally to the posterior lateral malleolus. The superficial ligament works in conjunction with the anterior inferior tibiofibular ligament to hold the fibula close to the tibia. The deep compartment of the posterior inferior tibiofibular ligament is the transverse tibiofibular ligament. It passes from the posterior tibial margin to the osteochondral junction on the posterior and medial aspects of the distal fibula. This ligament helps create a posterior labrum, deepening the articular surface of the distal tibia and preventing posterior tibial translation (Norkus & Floyd, 2001; Williams et al., 2007).

The slightly pliable interosseous membrane is very important in that it allows for slight separation between the medial and lateral malleolus during dorsiflexion at the talocrual joint in order to house the talus during dorsiflexion. The interosseus ligament can be considered as a thickening of the distal interosseous membrane. This ligament runs from the anteroinferior triangular segment of the medial aspect of the distal fibular shaft to the lateral surface of the distal tibia. Figure 4 illustrates the anatomy of the distal tibiofibular joint (Floyd & Norkus, 2001).

All three syndesmotic ligaments are important in providing stability to the distal tibiofibular joint (Figure 5). Testing of cadaver specimens revealed that the anterior

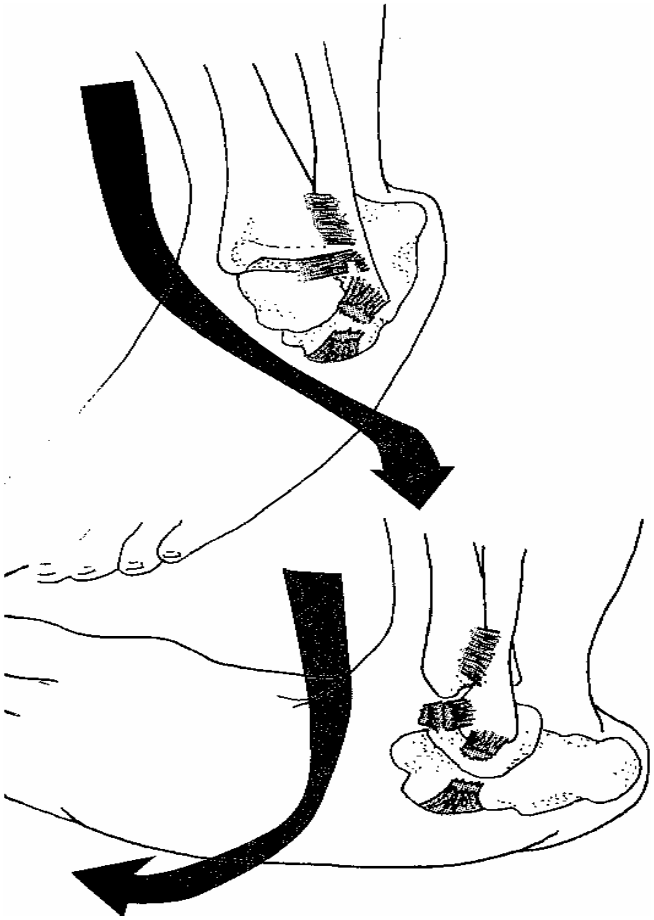

Figure 6- Mechanism of a lateral ankle sprain (Prentice, 2007, pg. 575).

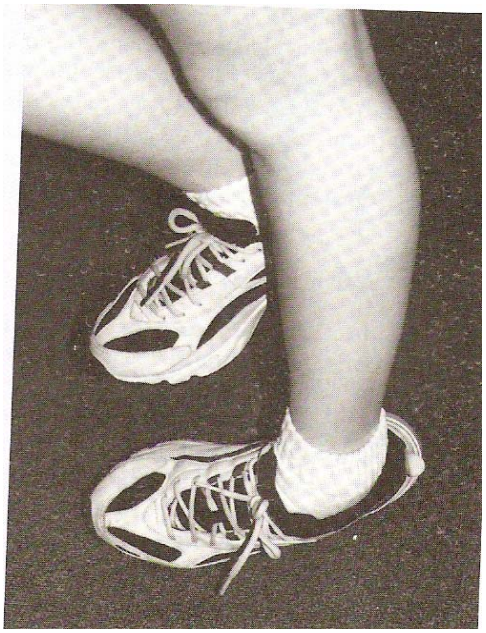

Figure 7- Replication of a lateral ankle sprain (Nordin & Frankel, 2001, pg. 246).

inferior tibiofibular ligament provides 35% of passive stability, the transverse ligament provides 33% of passive stability, and the interosseous ligament provides 22% of passive stability, and the superficial posterior inferior ligament provides 9% of passive stability against lateral fibular displacement (Norkus & Floyd, 2001). Thus, the ligaments of the inferior tibiofibular joint account for more than 90% of the total resistance to lateral fibular displacement by providing stability to the syndesmosis. Damage to any of the ligaments will disrupt the integrity of the joint and will make it more susceptible to future injury, provide poor force transmission, and altered plantar/dorsiflexion (Norkus & Floyd, 2001).

Mechanism of Injury: *Lateral Ankle Sprain*

The common mechanism of a lateral ankle sprain is excessive inversion at the subtalar joint and plantar flexion at the talocrural joint (Moiler et al., 2007) (Figures 6 and 7). Many lateral ankle sprains occur when a player jumps and lands on the foot of another player. This forces the ankle into supination, which is a combination of inversion (subtalar joint), plantar flexion (talocrural joint), and internal rotation (subtalar joint), and may result in a sprain of the lateral ankle ligaments (Ashton-Miller et al., 1996, Midgley et al., 2007). The lateral ankle sprain will primarily damage the ATFL, however, if the force exceeds the tensile strength of the ATFL, the CFL and the PTFL may also be sprained (Hertel 2002). To explain the kinetics of the mechanics of a lateral ankle sprain Fuller (1999) developed a pathological (pathomechanical) model that describes the position of the center of pressure and its relation to the subtalar joint and how this relationship may cause a lateral ankle sprain.

Center of Pressure

The center of pressure (COP) may be thought of as the average of all the forces acting on the foot when it is in contact with the ground. It is defined as the point where the applied forces do not cause a moment. Depending on the location of the vertical ground reaction force and its relationship to the subtalar joint axis, it may or may not cause a moment at the subtalar joint. If the vertical ground reaction force passes directly under the subtalar joint axis, no moment will occur. The location of the center of pressure must be known in order to combine the effects of the ground reaction force on the rearfoot and forefoot and determine the summative influences of the ground reaction force at the subtalar joint. The ground reaction force may not be the only force causing a moment at the subtalar joint. Moments from muscles, bones, and ligaments may need to be added to the moment from the ground reaction force. The ground reaction force may cause a supination moment and the muscles of the foot/ankle complex may cause an equal pronation moment. The addition of these two moments, because they are equal in magnitude but opposite in direction, causes no movement, even though an external moment is produced at the subtalar joint from the ground reaction force. There are three things that must be known to calculate the amount and direction of the moment at the subtalar joint from the ground reaction force. These three things are the magnitude of the force, its location, and the distance from the line of action of the force to the subtalar joint axis (Fuller 1999).

The position of the subtalar joint axis will vary greatly among different people. The foot with a more medially deviated subtalar joint axis is more likely to have a

pronation moment from the ground reaction force, and a supination moment will likely be caused by the ground reaction force in a foot that has its subtalar axis more laterally deviated (Fuller 1999). It is known that a lateral ankle sprain results from excessive supination of the subtalar joint, and that the ground reaction force will cause a larger supination moment at the subtalar joint in a person whose center of pressure is medial to the subtalar joint axis. Thus, the greater the magnitude of the ground reaction force that causes a supination moment at the subtalar joint, the greater the chance of the ankle complex having an excessive supination moment and suffering a lateral ankle sprain (Fuller 1999).

This pathomechanical model (Fuller 1999) has helped guide the development of the outersole and fulcrum for this proposed study. When a person lands from a jump onto the foot of another person, the center of pressure will normally be located medially to the subtalar joint axis. This will result in a supination moment at the subtalar joint and may cause a lateral ankle sprain. This proposed project will use a detachable outersole with a fulcrum located 20 mm from the medial side of the outersole to force the subtalar joint into inversion upon landing and replicating the mechanism of a lateral ankle sprain. This allows for the study of the mechanism of a lateral ankle sprain when a person lands from a jump onto the foot of another person.

Mechanism of Injury: High ankle sprain

In addition to lateral ankle sprains the ankle complex can be damaged through a high ankle or syndesmosis ankle sprain. The two most common mechanisms of a high ankle sprain are external rotation and hyperdorsiflexion, either in combination or

separately. Additional causes of injury to the syndesmosis include excessive eversion, inversion, plantar flexion, pronation, and internal rotation. External rotation typically causes damage only to the tibiofibular ligaments because external rotation widens the mortise by forcing a normally, rotationally stable talus into external rotation, which forces the fibula away from the tibia. This mechanism of injury, depending on the magnitude of the force applied, will tear the anterior tibiofibular ligament, the superficial tibiofibular ligament, the transverse tibiofibular ligament, or any combination of these three ligaments. This rotational injury may even tear the interosseous ligament or fracture the fibula (Clanton 2003; Norkus and Floyd, 2001; Pajaczkowski 2007).

The most common sporting activities where syndesmosis injuries occur are American football, soccer, and skiing. There are two common football mechanisms of this injury. The first involves a direct blow to the lateral leg of a tackled player whose foot is held in external rotation. The second mechanism also involves external rotation of the foot, this time caused by a blow to the lateral part of the knee while the planted foot is in external rotation, with the body spinning in the opposite direction. Both of these mechanisms result in significant forces directed at the ankle, lower leg, or both. In soccer, the player's foot may become fixed in the turf while the foot is forced into external rotation as the lower leg internally rotates. In skiing, this injury may occur when a ski becomes stuck in the snow, which causes internal rotation of the lower leg and rotation of the body in the opposite direction. The posterior tibiofibular ligament is commonly injured with this type of skiing injury (Clanton 2003; Norkus and Floyd, 2001).

Excessive dorsiflexion may also lead to a syndesmosis injury. When dorsiflexion occurs within the normal range of motion, the interosseous ligament becomes taut. As dorsiflexion continues past the physiological limit, the anterior aspect of the talus wedges the malleoli apart. This separation of the malleoli will sprain or rupture the anterior and posterior tibiofibular ligaments. This excessive dorsiflexion may occur in running and jumping sports when the foot is planted and the athlete falls or is pushed forward. This hyper-dorsiflexion can occur when an athlete stops and his or her inertia continues to carry their center of gravity over the ankle (Norkus & Floyd, 2001).

Ankle musculature

A key component in the prevention of a lateral ankle sprains are the muscles and tendons that cross the ankle complex and provide dynamic support through eccentric muscle actions. The most important muscles for providing dynamic stability against excessive inversion are the peroneus longus and peroneus brevis. These are the main evertors of the subtalar joint; by contracting eccentrically, these muscles help control supination of the rearfoot by negatively accelerating the subtalar joint in the presence of an inversion moment. The anterior muscles, which include the tibialis anterior, extensor digitorum longus, extensor digitorum brevis, and peroneus tertius, also add to the dynamic stability of the ankle complex, by eccentrically contracting to negatively accelerate the talocrual joint when it is forced into plantar flexion, thus helping prevent injury to the lateral ligaments of the ankle (Hertel 2002).

The peroneal muscles are located in the lateral compartment of the lower leg and receive innervation from the superficial peroneal nerve. The peroneus longus originates

on the head of the fibula and the lateral condyle of the tibia, and the peroneus brevis originates on the middle third of the fibula. Both muscles enter into one synovial sheath approximately 4 cm above the lateral malleolus. They pass behind the lateral malleolus through the *retromalleolar groove*, with the peroneus longus passing posterior to the peroneus brevis.

The main function of the peroneus brevis is abduction and eversion of the rearfoot and plantar flexion of the ankle. The function of the peroneus longus is to evert the hindfoot, plantar flex the first ray, and plantar flex the ankle. The peroneus longus and brevis are antagonists to the tibialis posterior, tibialis anterior, flexor hallicus longus, and flexor digitorum longus. The peroneus longus and brevis together contribute 63% of the total hindfoot eversion strength, with the peroneus longus contributing 35% and the peroneus brevis 28%, respectively. However, the peroneus longus and brevis only provide 4% of plantar flexion strength, with the gastrocnemius-soleus complex providing 87% of plantar flexion strength. The remaining plantar flexion strength is provided by the tibialis posterior, flexor hallicus longus, and flexor digitorum longus (Heckman et al., 2008).

The tibialis anterior muscle originates on the upper two-thirds of the lateral surface of the distal tibia and inserts on the inner surface of the medial cuneiform and the base of the first metatarsal. The main functions of the tibialis anterior are dorsiflexion of the talocrual joint and inversion of the subtalar joint. The tibialis posterior originates on the posterior surface of the proximal tibia and fibula and inserts on the lower inner surfaces of the navicular and cuneiform and the base of the second, third, fourth, and fifth metatarsals. The main functions of the tibialis posterior are inversion of the subtalar joint

and plantar flexion of the talocrual joint (Floyd 2007). The tibialis posterior provides a supination moment (Frigg et al., 2006), while the peroneus longus and peroneus brevis provide a pronation moment (Heckman et al., 2008).

The peroneus longus and peroneus brevis provide the primary defense against a lateral ankle sprain. Therefore, this proposed study will measure the latency of these muscles when a person is exposed to an inversion perturbation. This latency gives a measure of the amount of time it takes the ankle musculature to provide a dynamic defense against a lateral ankle sprain. Furthermore, once an ankle has sustained a sprain, it is reasonable to conclude that original anatomical arrangement has changed. For example, ligaments may be stretched leading to joint laxity, or perhaps scar tissue has made the ligaments less pliable, in either case, the function of the musculature of the ankle may be altered. In addition, since previous injury is the best predictor of future injury, how the musculature of a previously sprained ankle responds to an inversion perturbation may shed some light onto this increased propensity for injury (Hertel 2002).

Chronic Ankle Instability

One sample of participants in the proposed study will be people that have sustained at least one prior lateral ankle sprain. This study will not seek to recruit participants with chronic ankle instability or quantify people with chronic ankle instability (CAI). To understand how the ankle responds after a lateral ankle sprain and a high ankle sprain, this is best achieved by understanding the previous literature on chronic ankle instability, as it is reasonable to conclude that people with a previous history of an ankle sprain may perform similarly to people classified as having CAI.

Ankle sprains are a common injury, and although it is possible to rehabilitate quickly from an ankle sprain, the re-injury rate is very high and may lead to chronic ankle instability (Denegar and Miller, 2002). One study even reported that the greatest predictor of chronic ankle dysfunction is a syndesmosis ankle sprain (Williams et al., 2007).

People that have chronic ankle instability often have a deficit in sensorimotor function when compared to people with healthy ankles. The sensorimotor system organizes the interaction of different constraints, including the organism, environment, and task (dynamic systems theory) to produce movement. When a perturbation is applied to a system, these different components work together under the influence of the brain in order to maintain balance. Chronic ankle instability may be modeled as a constraint to the sensorimotor system that interacts with the other constraints and limits the ability of the body to develop patterns to maintain posture in the presence of an external perturbation (Brown & Mynark, 2007). One study found that patients with chronic ankle instability have a longer time to stabilization (TTS) when landing from a jump. This longer time may be due to the sensorimotor system reorganizing a stable pattern after the perturbation (Brown & Mynark, 2007). The proposed study will examine how participants that have sustained a lateral ankle sprain and high ankle sprain respond to an inversion perturbation upon landing from a drop landing. Based on this previous work, it would seem logical that participants with a history of lateral ankle sprains and participants with a history of a high ankle sprain would have a greater latency in the ankle musculature after an inversion perturbation than healthy participants. This work also seems to indicate that participants with a history of a lateral ankle sprain will have a

quicker time to failure (will complete the 25° of inversion faster) than the healthy participants. It is not known specifically how a high ankle sprain affects the sensorimotor system. This proposed study will help to answer these questions.

A study by Konradsen et al. (1998) examined the mechanical and functional parameters of the ankle and foot after an inversion ankle sprain and monitored these parameters during the healing process. The results of the study revealed that after an inversion ankle sprain, there is a significantly reduced ability to assess ankle inversion position, and that this deficit was still present 12 weeks after injury. There was also a reduction in the strength of the evertor muscles after an inversion ankle sprain, but the strength levels return to those of non-injured participants within 6 weeks of injury. Also, the reaction time of the peroneal muscles to a sudden inversion perturbation was not affected 3 weeks after injury (Konradsen et al., 1998).

While the Konradsen et al. (1998) study did not find a difference in the latency of injured ankles versus healthy ankles, that study did not use a dynamic landing task. Therefore, the proposed study will build upon this work by determining if there is a difference in the latency of the ankle musculature in people with a history of a lateral ankle sprain and a high ankle sprain on a dynamic landing task.

Section two: Inversion Mechanism

Most ankle sprains occur in sports that involve frequent jumping and running (Konradsen et al., 1998); for example, when a player jumps and lands on the foot of a teammate or opponent, which forces the ankle into excessive inversion (Midgley et al., 2007). Research has been conducted to replicate this mechanism of injury. The two

most often used methods utilized for replicating lateral ankle sprains are the tilt platform/trap door and the outersole with fulcrum.

Tilting platform/Trap door

The most common methodology used in the literature to replicate the mechanism of a lateral ankle sprain has been a tilting platform/trap door. With the tilting platform/trap door, participants stand with equal weight distribution on both feet on two separate trap doors. The researchers are able to randomly release the trap door, which causes approximately 30° of inversion at the subtalar joint and allows for study of the response of the ankle musculature using electromyography (Cordova et al., 2000; Cordova et al., 2003; Hopkins et al., 2007; Karlsson & Andreasson, 1992; Kernozek et al., 2008; Konradsen & Ravn, 1991; Konradsen et al., 1997; Lohrer et al., 1999; Lynch et al., 1996; Midgley et al., 2007; Mitchell et al., 2008; Shima et al., 2005). The tilt platform/trap door has also been integrated into a walkway in which a trap door falls away when a participant steps upon one of trap doors (Hopkins et al., 2007; Konradsen et al., 1997; Migdley et al., 2007).

Outersole with Fulcrum

Although seldom used in the literature, an outersole with a fulcrum placed along the medial side is another way to replicate the mechanism of a lateral ankle sprain. The outersole allows the researcher to force the ankle into inversion when landing from a jump, which is a frequent occurrence in athletics. A study conducted by Ubell et al. (2003) examined a dynamic landing task in which participants attempted to prevent a

forced inversion upon landing on one foot upon which the outersole and fulcrum were attached. The primary objective of the study was to measure the effectiveness of three different ankle braces at preventing this dynamic forced inversion. A detachable sole made of aquaplast was heat molded to fit a low-top basketball shoe. The outersole included a 6 mm wide fulcrum that was placed 20 mm medial to the midline along the length of the outersole. The fulcrum was 27 mm high and caused an inversion angle of 24° when the outer edge of the sole touched the ground. Three different ankle braces were tested and included the Swede-O Ankle Lok™, Aircast Sport Stirrup™, and the Bledsoe™ Ultimate ankle brace. Participants performed a 60 cm forward jump off the left leg from a take off platform onto the center of the force platform, landing on the right foot. A switch circuit was created between the force platform and lateral edge of the outersole to determine if participants could prevent 24° of dynamic forced inversion. If contact was made between the lateral edge of the outersole and the force platform, then the participant did not prevent the dynamic forced inversion. Trials included the outersole with a fulcrum and an outersole without a fulcrum (flat sole). The flat sole was included in the protocol in an attempt to prevent the participant from anticipating the dynamic forced inversion (Ubell et al., 2003). The results of the study revealed that for the unbraced trials, the success rate at preventing the 24° of inversion was 18% and half of the 14 participants were unsuccessful at preventing inversion on any of the trials, indicating the difficulty of the task. The Bledsoe™ ankle brace was the most successful, preventing 52% of the inversion perturbations, the AirCast™ prevented 46%, and the Swede-O™ prevented 34%. These numbers indicate the potential for ankle braces, and in

turn other ankle support methods, to prevent injury during a challenging dynamic task (Ubell et al., 2003).

There are several aspects of this previous research that have implications for the proposed study. First, the outersole with fulcrum used to cause the dynamic forced inversion is similar to the one that was developed for this study as well as the inclusion of a flat sole. Second, the results revealed that this activity was a challenging task with a low success rate when not wearing an ankle brace, indicating that the task should yield a stratified set of test scores. Third, the increased success rate while wearing ankle braces suggests that in the proposed study there should be a higher success rate when wearing ankle tape. Fourth, no measure of muscle activity was taken in the Ubell et al. (2003) study. The proposed study will measure the muscle latency during dynamic forced ankle inversion and determine the difference between participants with healthy ankles, participants with a history of one of more lateral ankle sprains, and participants with a history of a high ankle sprain.

Drop Landings

Drop landings have been used extensively to study lower leg kinematics and kinetics. Some of these studies examined the effects of ankle support on the kinetics and kinematics of a drop landing. The proposed study will use a similar drop landing with ankle taping as part of its methodology. A study by Riemann et al. (2002) found that ankle support decreased the amount of time to the first peak of the ground reaction force while DiStefano et al., (2008) found that ankle support had no effects on the ground reaction force, but did significantly change the joint angles at the knee and ankle. Other

research has found that external ankle support reduced the amount of ankle dorsiflexion during a drop landing (McCaw & Cerullo, 1999). While these studies found differences in the kinetics and kinematics during a drop landing while wearing ankle support, they did not measure the latency of the ankle musculature during this task nor did they include a forced inversion perturbation. The proposed study will address these deficits in the literature.

Summary

In summary, the tilting platform/trap door has been a valuable tool that has allowed researchers to approach replication of a lateral ankle sprain and study how the ankle musculature (with and without taping/bracing) responds to this inversion mechanism. While the tilting platform/trap door methodologies have helped shed light on the mechanism of a lateral ankle sprain, it lacks construct validity and does not truly replicate the mechanism of a lateral ankle sprain, which normally occurs during foot placement (Shima et al., 2005). The outersole with fulcrum, developed by Ubell et al. (2003), allows researches to more closely replicate the mechanism of a lateral ankle sprain during a dynamic task. However, this device has not been used to examine the response of the ankle musculature or the effects of ankle taping to this perturbation. Drop landings are also a dynamic task that has been frequently studied, but not in connection with a lateral ankle sprain. The proposed study will combine methodology from the tilting platform/trap door studies, the outersole and fulcrum projects, and the drop landing studies to more closely replicate the mechanism of a lateral ankle sprain and address this gap in the literature.

Section three: Inversion Platforms/Muscle Activation

Studies that have employed the tilt platform have added invaluable insight into the muscle response to unexpected ankle inversion, both with and without the participants being tape/braced (Cordova et al. 2000; Cordova & Ingersoll, 2003; Hopkins et al. 2007; Karlsson & Andreasson, 1992; Kernozek et al. 2008; Konradsen & Ravn, 1991; Konradsen et al. 1997; Lohrer et al., 1999; Lynch et al. 1996; Midgley et al. 2007; Mitchell et al., 2008; Shima et al., 2005). Within each study, electromyographic activity was recorded from the main evertors of the ankle (peroneus longus and peroneus brevis) and the main invertor (tibialis anterior), if the ankle was forced into a combination of inversion and plantar flexion in order to determine the latency of these muscles. Latency is defined as the time from platform release to the first rise in electromyographic activity.

Latency: *No Ankle Support*

Research has found that athletes with a functionally unstable ankle have an increased latency of the peroneus longus and brevis to sudden inversion as compared to athletes with stable ankles (Konradsen & Ravn, 1991). Lynch et al. (1996) found that the latency of the peroneus longus was significantly longer for the right leg (90.8 msec) than the left leg (87.6 msec). Konradsen et al. (1997) examined how the dynamic defense mechanism provided by the peroneus longus can help prevent lateral ankle sprains. Interestingly, data were only collected from the right leg, even though Lynch et al. (1996) found a difference in latency between the right and left peroneus longus. Konradsen et al. (1997) utilized four test conditions, three with the person standing and the ankle in 10° of inversion, subtalar neutral (0°), and 10° of eversion, and one with the person walking.

The results of the Konradsen et al. (1997) study revealed that when the peroneus longus was lengthened, the reflex latency was 51 ms, as compared to 54 ms when the peroneus longus was at resting length and 56 ms when the peroneus longus was shortened. The median peroneal reflex latency to a sudden inversion moment when walking was 48 ms, which was significantly less than the standing condition (Konradsen et al., 1997). A study by Hopkins et al. (2007) also examined the ankle musculature activation during standing and walking. The results of the study found that the latency of the muscles providing dynamic resistance to an inversion and plantar flexion moment are less (the musculature came on sooner) for a walking condition than a standing condition, which was in agreement with the findings from the Konradsen et al. (1997) study. A very recent study found that ankles with functional instability have a longer latency in the peroneus longus, peroneus brevis, and tibialis anterior when compared to healthy ankles (Mitchell et al., 2008).

Ankle Bracing and Latency

The next group of studies used the tilting platform/trap door to examine how ankle bracing affects the latency of the ankle musculature during an inversion moment (Cordova et al, 2000; Cordova & Ingersoll., 2003; Kernozek et al., 2008). A study by Cordova et al. (2000) evaluated the affects of two different ankle braces on the latency of the peroneus longus and how long term use of these braces effects this latency. The results revealed no significant interactions and no significant main effects, indicating that the use of an ankle brace over an eight week period did not cause a significant difference in latency of the peroneus longus (Cordova et al., 2000). Cordova and Ingersoll (2003)

expanded the work of Cordova et al. (2000) by investigating the amplitude of the peroneus longus stretch reflex after acute and long term use of two types of ankle braces (lace up and semi-rigid). The dependent variable was the amplitude of the peroneus longus stretch reflex (% of maximum amplitude), measured using electromyography (Cordova & Ingersoll, 2003). The results revealed that the lace up ankle brace group had a greater stretch reflex amplitude than the semi-rigid ankle brace group and control group immediately after application of the ankle brace. Also, the semi-rigid brace group had a higher stretch reflex amplitude than the control group or the lace up group after eight weeks of wear (Cordova et al., 2003). Kernozek et al. (2008) examined the combined affects of ankle bracing and various plantar-flexion angles on the latency of the ankle musculature and found no significant interaction between the bracing condition and the plantar flexion angle, no difference between the three plantar flexion angles and the latency in each of the three muscles, and no difference between the braced and unbraced conditions for each of the three muscle latencies (Kernozek et al., 2008). This work on ankle bracing and latency indicates that ankle support may reduce the latency of the peroneus longus, but this reduction was not found in all of the studies. Thus, the proposed study will add to the debate in this literature.

Ankle Taping and Muscle Latency

Ankle taping has been used for many years because of its ease of application and belief among athletic trainers, coaches, and athletes that it is an effective way to prevent ankle sprains. The utility of tape has been well documented however; there are drawbacks. It is costly to apply tape daily, repeated removal may cause skin irritation,

and tape may loose its effectiveness as the length of time of the activity increases. This decrement in effectiveness may occur in as little as 10 minutes (Ashton-Miller et al, 1996). Ankle taping helps restrict excessive range of motion by acting as an external ligament; however, researchers have produced opposing findings regarding the relationship between ankle taping and the latency of the peroneus longus to a sudden inversion perturbation (Lohrer et al., 1999; Karlsson & Andreasson, 1992; Midgley et al., 2007; Shima et al. 2005). Specifically, work by Karlsson and Andreasson (1992) indicated that ankle taping reduces the latency of the peroneus longus to a sudden inversion perturbation by increasing the sensory input, while Shima et al (2005) found an increase in the latency of the peroneus longus after tape application, and Lohrer et al. (1999) and Midgley et al. (2007) found no difference in the latency of the peroneus longus after the application of ankle tape. Additionally, Karlsson and Andreasson also found that healthy ankles had a shorter latency than ankles with a previous ankle injury, which is in agreement with previous work by Konradsen and Ravn (1991) and Mitchell et al. (2008); while Shima et al. (2005) found that injured ankles with hypermobility had a shorter latency than healthy ankles with normal mobility.

Due to the prevalence of ankle injuries in athletic events, ankle taping will continue to be used in order to provide mechanical stability to the ankle. There has been a large amount of research into the effectiveness of ankle taping in restricting range of motion in static conditions, but more research is needed into its effectiveness during dynamic tasks (Cordova et al., 2002).

This section has summarized some of the previous research using a tilting/trap door mechanism to replicate an inversion moment that causes a lateral ankle sprain in

order to measure latency of the ankle musculature (Cordova et al., 2000; Cordova and Ingersoll., 2003; Hopkins et al., 2007; Karlsson & Andreasson, 1992; Kernozek et al., 2008; Konradsen & Ravn, 1991; Konradssen et al., 1997; Lohrer et al., 1999; Lynch et al., 1996; Midgley et al., 2007; Shima et al., 2005). Some research examined the affects of ankle taping (Karlsson & Andreasson, 1992; Lohrer et al., 1999; Midgley et al., 2007; Shima et al., 2005) and bracing (Cordova et al., 2000; Cordova & Ingersoll, 2003; Kernozek et al., 2008; Midgley et al., 2007) during a forced inversion on latency of the ankle musculature. This research has brought attention to the response of the ankle musculature when it is forced into inversion and how taping and bracing affects this response. However, the literature has been inconsistent in its findings. The proposed study will build on this previous work by examining the response of the ankle musculature during a task that more closely replicates the mechanism of a lateral ankle sprain. Not only will healthy ankles be tested, but participants with a history of a lateral ankle sprain and a high ankle sprain will be tested as well. The effects of ankle taping on latency of the ankle musculature will also be measured across different samples of ankle injuries. The table below (Table 1) summarizes the methodology and main findings from the previously reviewed studies using the tilting platform/trap door and demonstrates the inconsistency in the results.

Table 1 Summary of tilt platform/trap door literature

Study	Participants	Ankle Support	Findings on latency
Cordova et al, 2000	Healthy	Braces	No effect on latency
Cordova & Ingersoll, 2003	Healthy	Braces	Braces increased stretch reflex amplitude
Hopkins et al., 2007	Healthy	No Support	Reduced latency when walking compared to standing
Karlsson & Andreasson, 1992	Healthy and Ankle Instability	Ankle Tape	Taping reduced latency Healthy ankles shorter latency than injured ankles
Kernozek et al., 2008	Healthy	Braces	No effect on latency
Konradsen & Ravn, 1991	Healthy and Functionally Unstable	No Support	Increased latency for in unstable ankles
Konradsen et al., 1997	Healthy	No Support	Reduced latency when walking compared to standing
Lohrer et al., 1999	Healthy	Ankle Tape	No effect on latency
Lynch et al., 1996	Healthy	No Support	Latency shorter on left ankle than right ankle
Midgley et al., 2007	Healthy	Ankle Bracing and Taping	No effects on latency
Mitchell et al., 2008	Healthy and Ankle Instability	No Support	Increased latency in injured ankles when compared to uninjured ankles
Shima et al., 2005	Healthy and Ankle Instability	Ankle Tape	Taping increased latency Injured ankles had shorter latency than un-injured ankles

Section four: Startle response

Humans encounter many different unexpected transient perturbations in everyday life. In athletic settings, this may include being struck from behind by another player or jumping in the air and landing on the foot of another player. These perturbations involve forces that move the body or disrupt its base of support. Humans have developed many different and varying responses to deal with these perturbations to restore posture, and prevent injury. These perturbations are normally applied very quickly and the response to the perturbation will vary according to the magnitude, knowledge, and anticipation of the perturbation. Exaggerated responses are normally evoked by large, unexpected perturbations where smaller or more familiar perturbations normally evoke smaller responses. An exaggerated response is typically evoked the first time a person is exposed to an unexpected perturbation. This exaggerated response consists of both a postural component that attempts to restore the initial posture and a startle response that causes the exaggeration in the initial response (Siegmund et al., 2008). The startle response is a short latency muscular response caused by a quick, intense stimulus (Rodriguez-Fornells, 1999).

When the body is first exposed to an unexpected perturbation, there is generally a muscular response that involves co-contraction of both the agonist and antagonist muscle groups (Siegmund et al., 2008). When this model is applied to a lateral ankle sprain, this co-contraction would involve simultaneous contraction of both the invertors and evertors of the subtalar and transverse tarsal joints. The evertor muscle group would counter the moment produced by the inversion perturbation, such as landing from a jump onto the foot of another player. If the invertor muscle group contract simultaneously with the

evertor muscle group after the ankle is forced into excessive inversion, the activity of the invertors would negate the protective moment produced by the evertor muscle group Therefore, this startle response to an unexpected perturbation would prevent the evertor muscle group from limiting the amount of inversion caused by the perturbation. This co-contraction likely occurs in order to stiffen the body and prevent excessive joint movement, however, in this case, is likely to negate the protective mechanism from preventing excessive inversion.

After the body is repeatedly exposed to the same stimulus, the muscle response attenuates. This process is also known as habituation. Habituation is a form of non-associative learning, in which the response decrement is dependent on the presentation of the response-eliciting stimulus (Pitz et al., 2004). When a person is repeatedly exposed to the same stimulus or perturbation, there will be attenuation in the muscle group that opposes restoring the original posture. This is evidenced by a reduction or even disappearance of activity in the antagonist muscle group and a fine tuning of the agonist muscle group to restore the original posture. Research has shown that during a first time exposure to a seated forward acceleration (similar to low speed rear end collision), there is a muscular response in both the anterior and posterior muscles of the neck and restoration of upright head posture. By the third exposure to this stimulus, activity in the posterior muscles diminishes, and the activity in the anterior muscles is attenuated (Siegmund et al., 2008).

If this habituation can be noted in the ankle, it may play an important role in the prevention of ankle sprains. The first time a person encounters an inversion perturbation, if the startle response is a component of the exaggerated first response to the ankle

perturbation, there will be activity in both the invertors (antagonist) and evertor (agonist) muscle groups. The evertor muscle group is attempting to restore the initial neutral posture of the ankle complex, while the invertor muscle group prevents this restoration. If the musculature at the ankle can be "trained" to elicit a smaller response in the invertor muscle group and an unchanged response in the evertor muscle group, it may be possible for the ankle musculature to prevent more ankle sprains or perhaps the severity of the sprains.

Previous work into rapid, whole body accelerations found that upon initial presentation of the stimulus, a centrally generated postural response is triggered in order to stabilize the head and trunk. The work also found that after repeated exposures to the same stimulus, the participants were able to modify their musculature response (Blouin et al., 2003). The authors of a recent study on whole body forward accelerations (Siegmund et al., 2008) argue that the first time a whole body acceleration is applied to a person, the elevated prehabituated muscle response is composed partly of a startle response. This theory is appealing because it explains that the first, exaggerated response to a whole body acceleration is caused by the startle response, which will vanish with habituation, and leave only the response evoked by the postural response system to correct for the perturbation. This theory, despite its simple approach, has been hard to express in experiments. The experiment by Siegmund et al. (2008) attempted to restore the initial response to a whole body acceleration by superimposing an acoustic startle response into an already habituated startle response.

For the experiment, a low speed rear end impact collision was simulated by exposing subjects to abrupt forward horizontal acceleration on a linear sled.

Electromyographic (EMG) activity was recorded from the sternocleidomastoid (SCM), scalene, and cervical PARA muscles. Accelerometers and motion tracking markers were used to record the kinematics of the crash. In order to create a stabilized habituated response the participants were first exposed to 11 identical whole body accelerations (peak 15.2 m/s^2), and then they were exposed to 5 additional whole body accelerations in which a superimposed loud acoustic stimulus was presented to generate a startle response. The peak EMG amplitude and peak kinematic responses across the 16 trials were compared.

The results revealed that the addition of the auditory tone restored EMG amplitudes in all three of the neck muscles and the peak amplitudes of four (out of five) of the habituation-effected kinematic variables to levels that were not significantly different than those measured during the first acceleration only trial; before habituation. This led the authors to conclude that the first exposure to a forward acceleration contains a combined startle response and a postural response (Siegmund et al., 2008).

The results of this previous work are important for shaping the present study and the inclusion of the startle response and how it may play a role in ankle injuries. These authors (Siegmund et al., 2008) found that the first response to a whole body acceleration is a combination of a postural response and a startle response. The next step in this process is to determine if the first response to an external perturbation that forces the ankle into inversion is a combination of the startle response and postural response. After making this determination, the next step would be to determine if this startle response can be attenuated in order to create less activity in the inverter muscle group and/or more

activity in the evertor muscle group, which would hopefully lead to a reduction in the number of or the severity of inversion ankle sprains.

A study examining habituation of the startle response in mice attempted to investigate where in the neural circuit this habituation occurs by using different behavioral methods. The startle response was elicited with two different sensory modalities, acoustic and tactile stimuli, to determine if habituation may be generalized between the two pathways. Specifically, this study sought to determine if habituation to either the acoustic or tactile startle response would result in generalization to the other startle response. For example, does attenuation of the acoustic startle response cause attenuation of the tactile startle response? The authors hypothesized that if habituation occurs in the sensory-specific pathway, before or in synapse with caudal pontine recticular nucleus, habituation in one of the two pathways should not be generalized to the other pathway. However, if the habituation occurs post-synaptically, the startle pathway would be shared by the acoustic and tactile modalities, and habituation would be generalized (Braff et al., 2001).

The authors completed four different experiments with the mice. The first experiment examined the acoustic affects of tactile stimuli, or how sound associated with a tactile stimulus affects the startle response. Rats with an intact tympanic membrane and rats with a ruptured tympanic membrane were exposed to a tactile airpuff (they were exposed to a tactile stimulus that also had an auditory component). The results revealed that the startle response caused by the tactile stimulus that also contained an auditory component causes an increased startle response. The rats with the ruptured tympanic membrane had a lesser startle response because they were not able to hear the auditory

stimulus associated with the tactile stimulus, which indicates that if the auditory stimulus associated with a tactile stimulus is eliminated there is a decrease in the startle response (Braff et al., 2001).

The second experiment investigated the habituation of the tactile startle response in two different strains of mice. The mice were tested in the same manner as experiment one, but with the tactile stimulus was applied to different parts of their bodies to evaluate the effect of the position of the tactile stimulus and its affects on the startle response. The results revealed no significant difference in the tactile startle response between the two strains of mice. The results also showed a decrease in the tactile startle response during repetitive stimulation due to habituation (Braff et al., 2001).

The third experiment investigated whether the short-term habituation of the startle response is sensory specific. Habituation to tactile and sensory stimuli was induced by using both modalities. The rats were exposed to either an acoustic stimuli or tactile stimuli on separate days. The results revealed that the rats did habituate their response to the auditory stimulus, but when the tactile stimulus was presented immediately after the auditory stimulus, no habituation was present. The same finding was present when the mice were first exposed to the tactile stimulus, and then to the auditory stimulus. This supports the hypothesis that habituation of the startle response is specific to the modality (Braff et al., 2001).

Experiment four replicated the third experiment with the exception that the background noise level of 100 dB was used in this experiment to help eliminate the affects of the acoustic artifact. The results were similar to the results in experiment three, with both strains of mice showing habituation to the acoustic or tactile stimuli before the

modality switch. There was no habituation to the acoustic stimuli in the tactile only mice group and no habituation to the tactile stimuli in the acoustic only mice group. Therefore, there was no generalization between the types of stimuli when a background noise was added to the experiments (Braff et al., 2001).

The findings of these four experiments indicate that there is habituation to acoustic stimuli after repeated exposures, and habituation is present to tactile stimuli after repeated exposures. However, there is no habituation to acoustic stimuli when repeatedly exposed to tactile stimuli and no habituation to tactile stimuli when exposed to acoustic stimuli. Therefore, there is no crossover habituation between the two types of stimuli and pathways. These findings add support to the theory that habituation of the startle response takes place in the afferent portion of the sensory pathway. This is important for the proposed study because it indicates that exposure to the acoustic startle response will not affect the tactile startle response. The proposed study will examine the tactile startle response, and since there is no crossover effect, the acoustic startle response does not need to be examined. The findings from this study (Braff et al., 2001) imply that after repeated exposures to the inversion perturbation, habituation should be present in the ankle musculature.

Joint Stiffness

The difference between the startle response and joint stiffness will now be reviewed. Stiffness, in general, is a mechanical property that determines how external forces acting on the skeletal system are transmitted or absorbed by the articular cartilage, while muscle stiffness is the stiffness properties of the muscles and tendons. Joint stiffness includes assistance from all structures located within and around a joint. This

includes muscles, tendons, cartilage, skin, fascia, ligaments, and the joint capsule (Riemann et al., 2001). Joint stiffness includes both passive and active contributions, with the active contributions provided by the muscles (Padua et al., 2006). Stiffness occurs at the ankle during walking, running, and jumping in order to adapt to different surfaces, and prevent excessive range of motion. Research has found co-activation of the dorsiflexors and plantar flexors of the ankle immediately after landing from a jump with no external perturbation (Bishop et al., 2006; Ferris et al., 2005; Santello & McDonagh, 1998).

Joint stiffness has been proposed to have both positive and negative effects. If the ankle is stiff when landing from a jump, its ability to absorb the energy enclosed by a destabilizing force will be increased. This may lead to a decrease in the number of subluxations or dislocations. However, an increase in stiffness may also increase the risk for an ankle injury. If the invertor musculature is stiff, it will cause an increase in the force production demands of the evertor muscle group to exceed the force produced invertor muscle group and limit the amount of inversion range of motion. (Riemann et al., 2001).

This author believes that if stiffness occurs at the ankle when it is forced into inversion by an external force, the protective mechanism supplied by the evertors will be cancelled by the invertors. Joint stiffness is not the same as the startle response. The startle response elicits co-contraction of both the agonist and antagonist muscle groups the first time the stimulus is presented. This initial exposure causes joint stiffness. After repeated exposures to the same stimulus, there is an increased or same amount of activity in the agonist muscle group; and less activity in the antagonist muscle group (Siegmund

et al., 2008). This attenuation of the activity of the antagonist muscle group, which for the ankle would be the invertors, is beneficial because a diminished response in the invertors will not negate the protective mechanism provided by the evertor muscle group. Therefore, stiffness will not occur after repeated exposures to the same inversion stimulus at the ankle.

Section five: Summary of Literature

The ankle sprain is the most common injury in athletics and a common injury among people in the general population (Hertel, 2002). Many ankle sprains occur when a person jumps and lands on the foot of another person (Midgely et al., 2007). Unfortunately, most of the research conducted on the response of the ankle musculature to an inversion perturbation has used a tilting platform/trap door mechanism that randomly inverts the ankle, often times while the participants were simply standing and awaiting the trap door to fall away. Specifically, in these previous studies the latency of the ankle musculature was measured using EMG to determine the amount of time from release of the trap door to first rise in EMG activity (Cordova et al., 2000; Cordova & Ingersoll,. 2003; Hopkins et al., 2007; Karlsson & Andreasson, 1992; Kernozek et al., 2008; Konradsen & Ravn, 1991; Konradsen et al., 1997; Lohrer et al., 1999; Lynch et al., 1996; Midgley et al., 2007; Mitchell et al., 2008; Shima et al., 2005). However, the trap door tool does not truly replicate the mechanism of a lateral ankle sprain, which frequently occurs during foot placement (Shima et al., 2005).

Several studies have utilized the drop landing, which more closely replicates the mechanism of a lateral ankle sprain, and some have even utilized the outersole with a

fulcrum, but these studies failed to collect muscle latency data (DiStefano et al., 2008; McCaw & Cerullo, 1999, Riemann et al., 2002; Ubell et al., 2003). The present study will advance the research in this area by utilizing a procedure that more closely replicates the lateral ankle sprain movement (drop landings, onto a fulcrum that will induce lateral ankle sprain posture), while measuring muscle latencies. In addition, the startle response has been noted in full body accelerations (Siegmund et al., 2008), but it has yet to be assessed in response to the perturbation of a single joint. The present study will attempt to determine if the startle response is present at the ankle during an unexpected perturbation. Furthermore, ankle taping and bracing has received much attention in the literature, but not in the context that will be employed in the present study. Previous literature has indicated that taping/bracing is helpful in reducing ROM and has been shown to generally decrease the latency of the ankle musculature, yet this study will advance this area by using a task that closely replicates one of the most common mechanisms of a lateral ankle sprain while taped and not taped. Last, no study has combined all of these factors while considering them across participants of varying ankle injury histories. It is these deficits within the literature that the proposed study will attempt to address.

CHAPTER III

METHODS

The primary purpose of this study was to examine the latency of the ankle musculature to an inversion perturbation during a drop landing among participants with healthy ankles, participants with a previous history of a lateral ankle sprain, and participants with a history of a high ankle sprain. The secondary purposes of this study were to 1) determine if the startle response is part of the first reaction to an unexpected ankle perturbation that can be attenuated and 2) to determine the effect of ankle taping on the latency of the ankle musculature to an inversion perturbation during a drop landing.

Participants

The participants in the present study included 19 to 30 year old students from Auburn University. Three groups of participants were used in the study. Ten to fifteen participants were included in each group, for a total of 40 participants. The first group included 15 participants that had no history of an ankle sprain or ankle injury. The second group included 15 participants that had a history of one or more lateral ankle sprains, previously diagnosed by a physician, but none within the past six months. The third group included 10 participants who had a history of a high ankle sprain, previously diagnosed by a physician, but none within the past six months. All participants were free of any lower extremity surgery or fracture. Any potential participants that had suffered

both a lateral ankle sprain and a high ankle sprain in the same ankle were not be allowed to participate in the study

Setting

All testing and data collection will occur in the Sport Biomechanics Laboratory at Auburn University. The Sport Biomechanics Laboratory is a large enclosed laboratory with the necessary equipment to carry out this research.

Materials

Outersole

Eight detachable outersoles (4 with fulcrum and 4 flat), made of orthoplast were developed for this project. A dummy and fulcrum outersole was attached to the bottom of the shoe. The fulcrum was designed to produce 25° of inversion upon landing. The fulcrum was placed 20 mm from the medial border of the outersole. It is 6 mm thick and

Figure 8- Outersole and fulcrum for creating 25° of inversion upon landing, Frontal view.

30 mm high (Figure 7 and Figure 8). Four flat soles were also created. The outersole was attached to the tennis shoe of the participants using Velcro™ straps. All participants were required to wear the same low top flat soled athletic shoe for testing

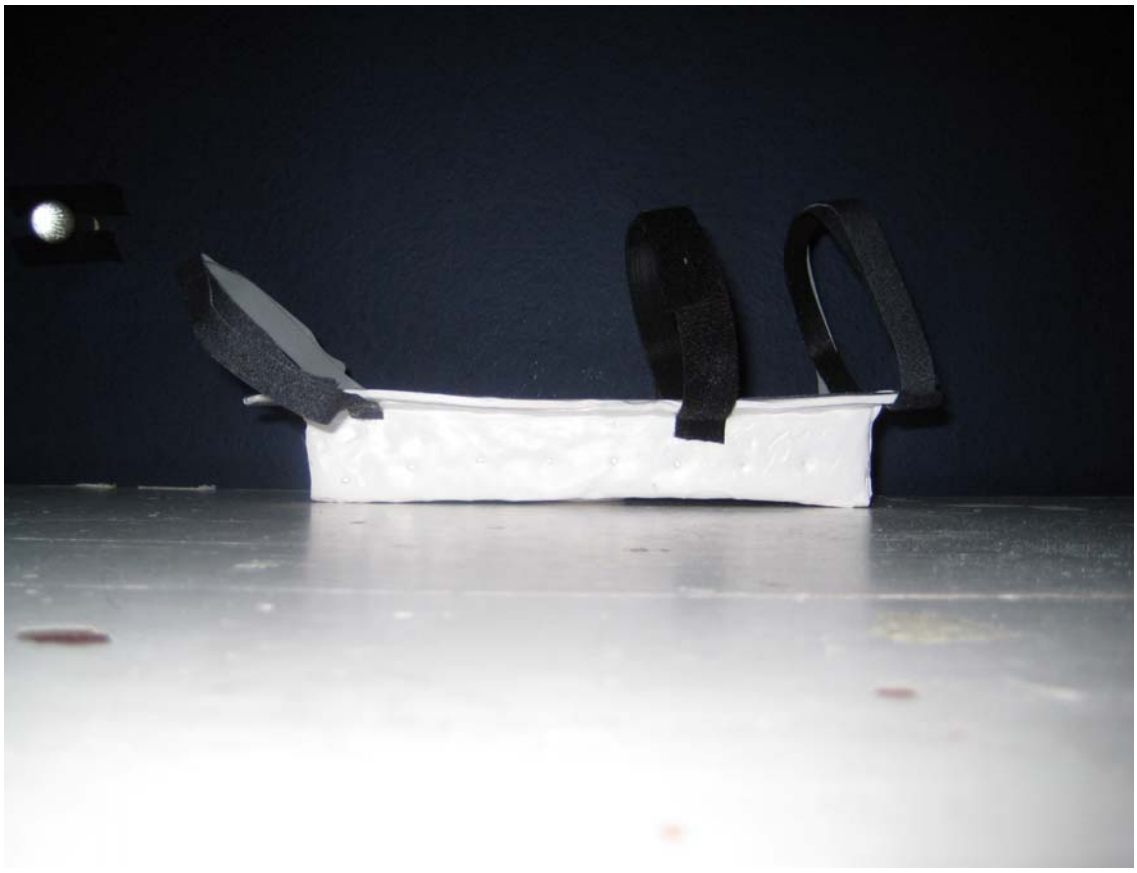

Figure 9- Outersole and fulcrum for creating 25° of inversion upon landing, side view.

Box

A 27 cm high wooden box was used from which the participants performed the single leg drop landing onto the landing area. The participants stood atop the box, on the non-testing leg and the outersole was placed on the testing leg. The participant then stepped down off the box and landed on the testing leg. The order of outersole assignment (fulcrum or flat) was randomized.

Figure 10- Box and outersole with fulcrum used for testing.

Athletic Tape

During the taped condition, the participants had both ankles taped with a closed basketweave procedure by the same Certified Athletic Trainer. Foam pre-wrap (Figure 10) was used as the underwrap followed by Coach™ Tape by Johnson and Johnson (Figure 11). The tape was anchored at the base of the gastrocnemius muscle on all participants.

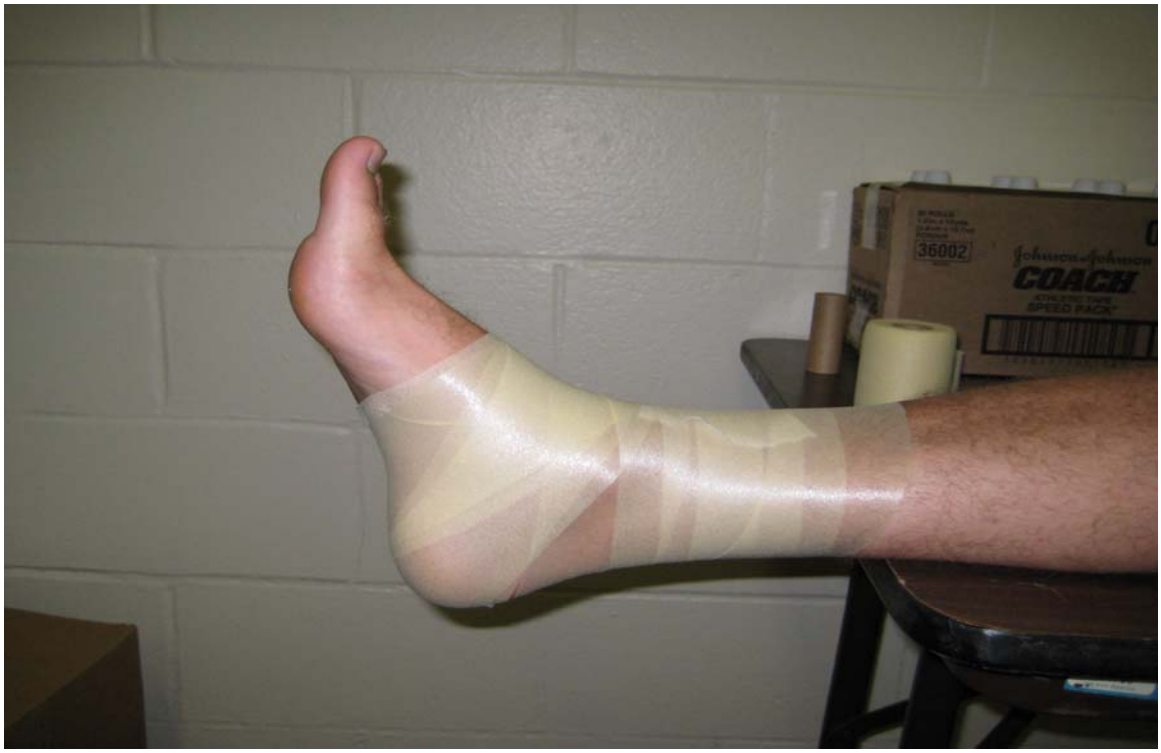

Figure 11- Foam pre-wrap applied over skin before Coach™ Tape.

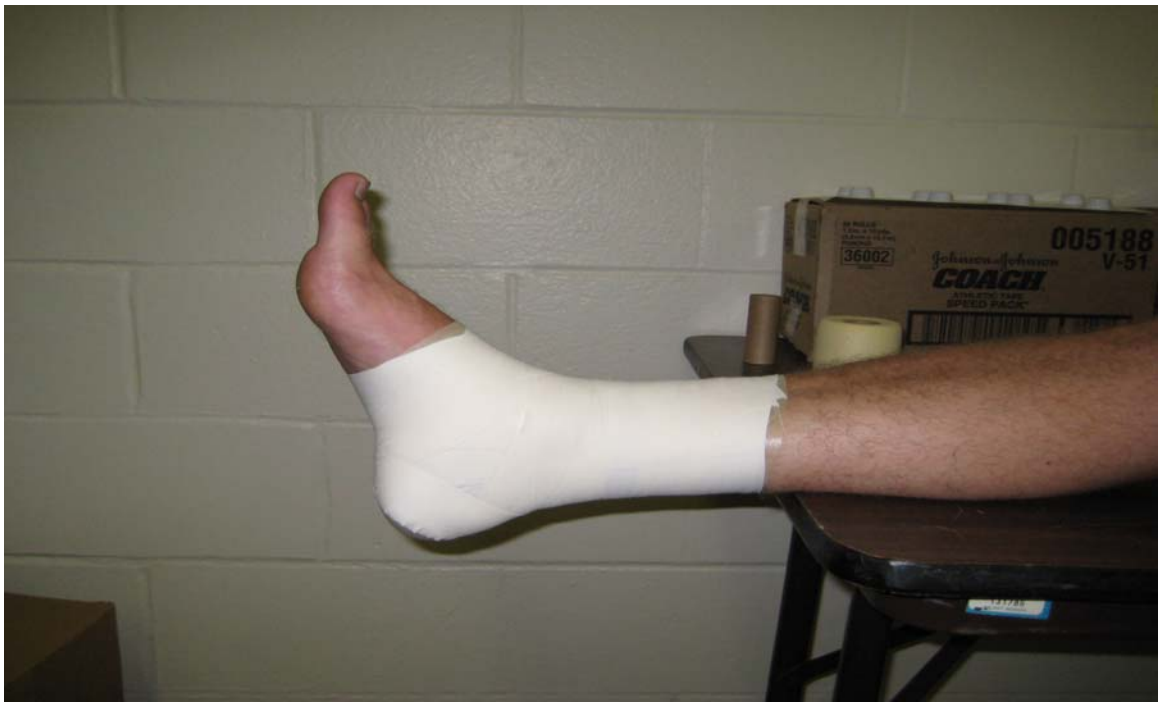

Figure 12- Closed Basketweave taping procedure.

Instrumentation

Muscle activity was recorded with a mutlichannel electromyography (EMG) amplifier/processor unit (MyoClinical, Noraxon USA INC; Scottsdale, AZ) using bipolar Ag/AgCl disc surface electrodes interfaced with a notebook computer (Figure 13). The EMG signal was sampled at 1000 Hz. The raw EMG signal was full wave rectified and passed through a 6^{th} order Butterworth filter.

A landing surface was developed so that a circuit was created between the fulcrum and the landing area. Two pieces of metal were placed on the landing area (Figure 14). Metal was also attached to the fulcrum and to the lateral border of the outersole (Figure 15). The electrical signal from the landing area and fulcrum was synchronized with the EMG signal and processed through the EMG amplifier. When the fulcrum made contact with the landing area, a spike was produced in one of the EMG channels, indicating ground contact. When the lateral border of the outersole made contact with the landing area, indicating failure, a second spike was produced in a different EMG channel. The time from the first spike to the second spike was calculated as the time to failure.

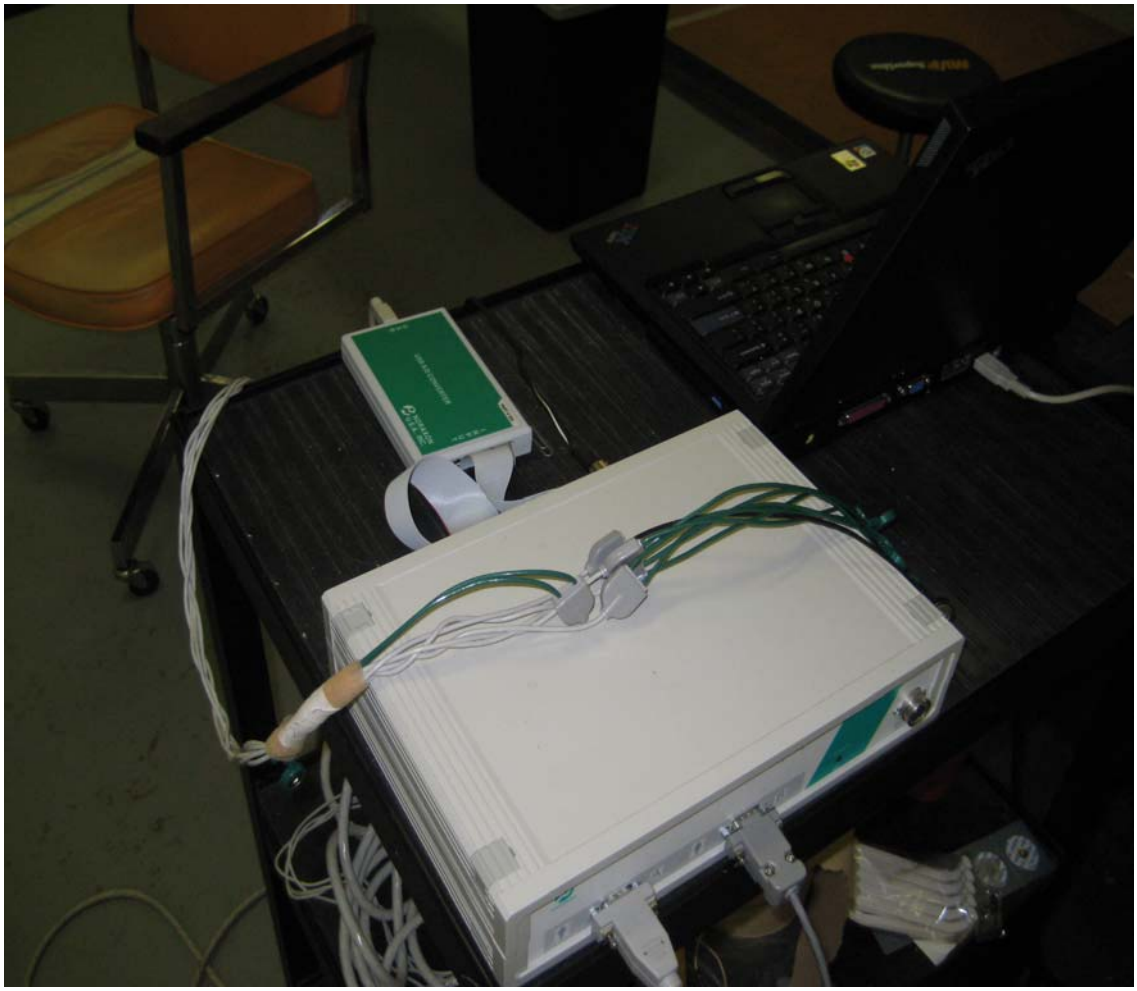

Figure 13- Noraxon Electromyography Processor and Amplifier.

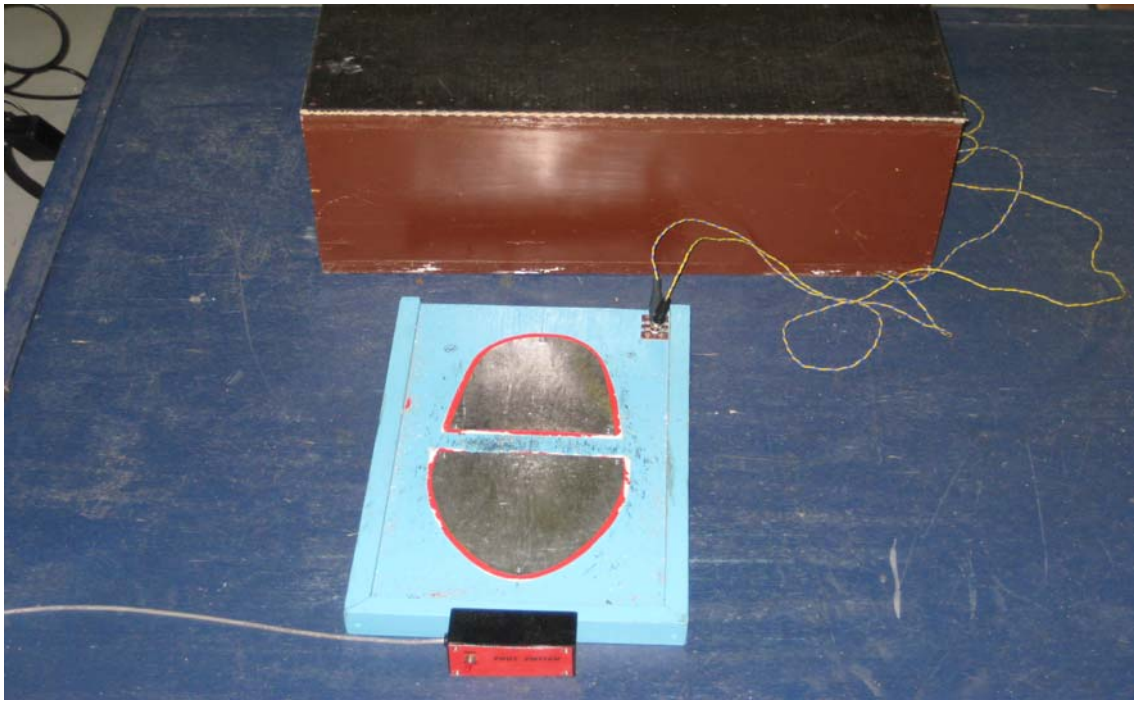

Figure 14- Landing Area with contact area for fulcrum and lateral border of outersole.

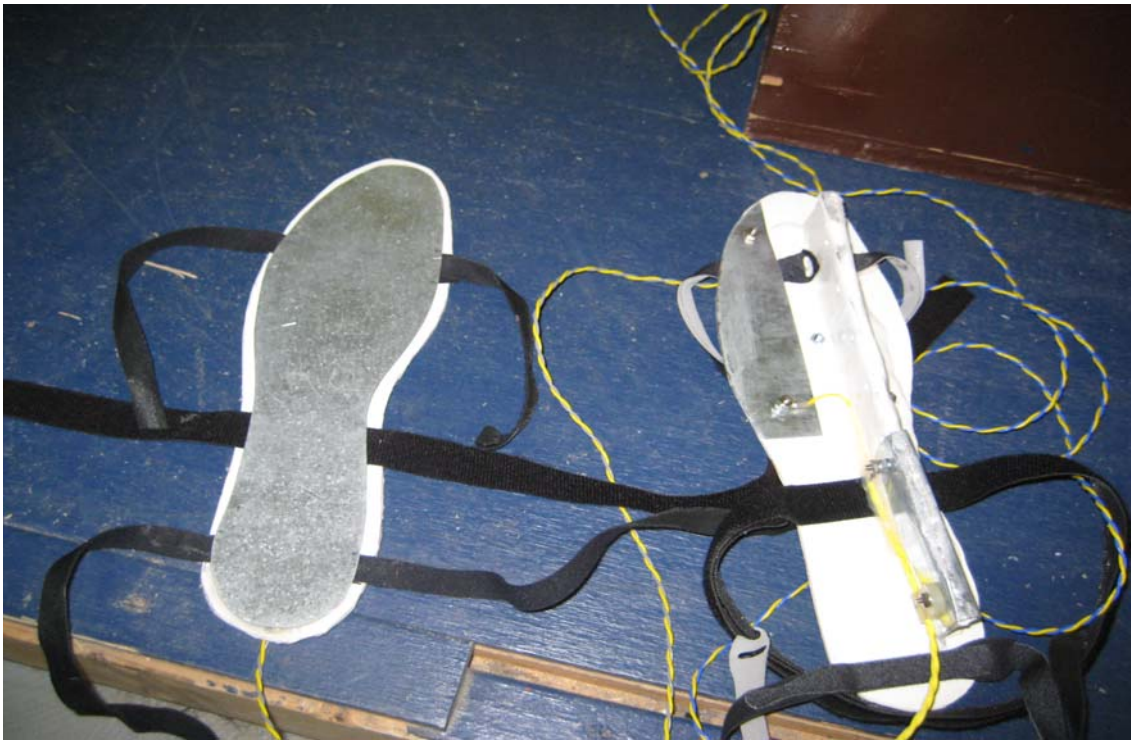

Figure 15- Flat outersole and outersole with fulcrum demonstrating the metal that made contact with the metal of the landing area to complete the circuit.

Design and Procedures

The participants reported to the Sport Biomechanics Laboratory for testing on three separate occasions. The first session included approximately ten minutes to read and fill out the informed consent and preliminary medical questionnaire. Participants indicated voluntary participation by signing the informed consent document approved by the Institutional Review Board of Auburn University. Furthermore, a medical questionnaire was used as a screening device that eliminated anyone who had (1) sustained a lower extremity injury within the last six months; (2) had ever had surgery or a fracture of the lower extremity; (3) had sustained both a lateral and high ankle sprain to the same ankle; (4) had an allergy to adhesives; and/or (5) currently had an inner ear disturbance or any other condition that would prevent them from completing the testing protocol successfully. The participants were then shown the testing protocol and allowed to become familiar with the procedures, without actually performing the test, in order to maintain the novelty of the movement for the assessment of the components of the startle response. The participants were allowed to practice stepping off the box but without either the fulcrum or flat sole. The participants then performed 2 blocks of testing. Each block consisted of 20 trials on one leg, 10 with the fulcrum and 10 without, for a total of 20 trials per leg. The integration of the flat outersole and fulcrum outersole into the testing rotation was randomized in an attempt to avoid anticipation by the participant. The first testing day was designed to determine if the startle response was part of the exaggerated first response to an inversion perturbation and how long this response persists. The first testing day was also designed to measure the latency of peroneus longus, peroneus brevis, and tibialis anterior across the three injury groups.

On the second or third testing days (depending on taping order), the participants had both ankles taped by a Certified Athletic Trainer. The order of taping assignment (day 2 or day 3) was random. Two blocks of trials (one on each leg) were performed on the second and third testing days. The order of testing (tape/no tape) was randomly assigned and alternated for each participant between the two testing days. The purpose of the second and third testing days were to determine the effects of ankle taping on the latency of the ankle musculature among the three groups during an inversion perturbation and to determine if there was a difference among the injury history groups and the ability to prevent the dynamic forced inversion.

Surface EMG electrodes were placed over the most prominent part of the muscle bellies of the peroneus longus, peroneus brevis, and tibialis anterior for the first testing day, and over the peroneus longus and tibialis anterior for the second and third testing days (Figure 16). The peroneus brevis was not measured the second and third testing days due to the application of athletic tape over the electrode placement site for this muscle. Electrode placement sites were shaved, abraded, and cleaned according to standard electromyographic procedures. The electrode placement was similar to that used by Lynch et al. (1996) and Kernozek et al. (2008). Proper placement was checked by using manual muscle testing. For the peroneus longus, the electrodes were placed at the junction of the proximal and middle thirds of the fibula over the palpable lateral compartment. For the peroneus brevis, the electrodes were placed one quarter of the distance between the lateral malleolus and fibular head, just anterior to the peroneus longus tendon. For the tibialis anterior, the electrodes were placed at the junction of the proximal and middle thirds of the tibia, over the largest palpable portion of the muscle

belly. Onset of muscle activity was defined as muscle activity after foot touchdown that exceeded 5 standard deviations from muscle activity 200 milliseconds before landing.

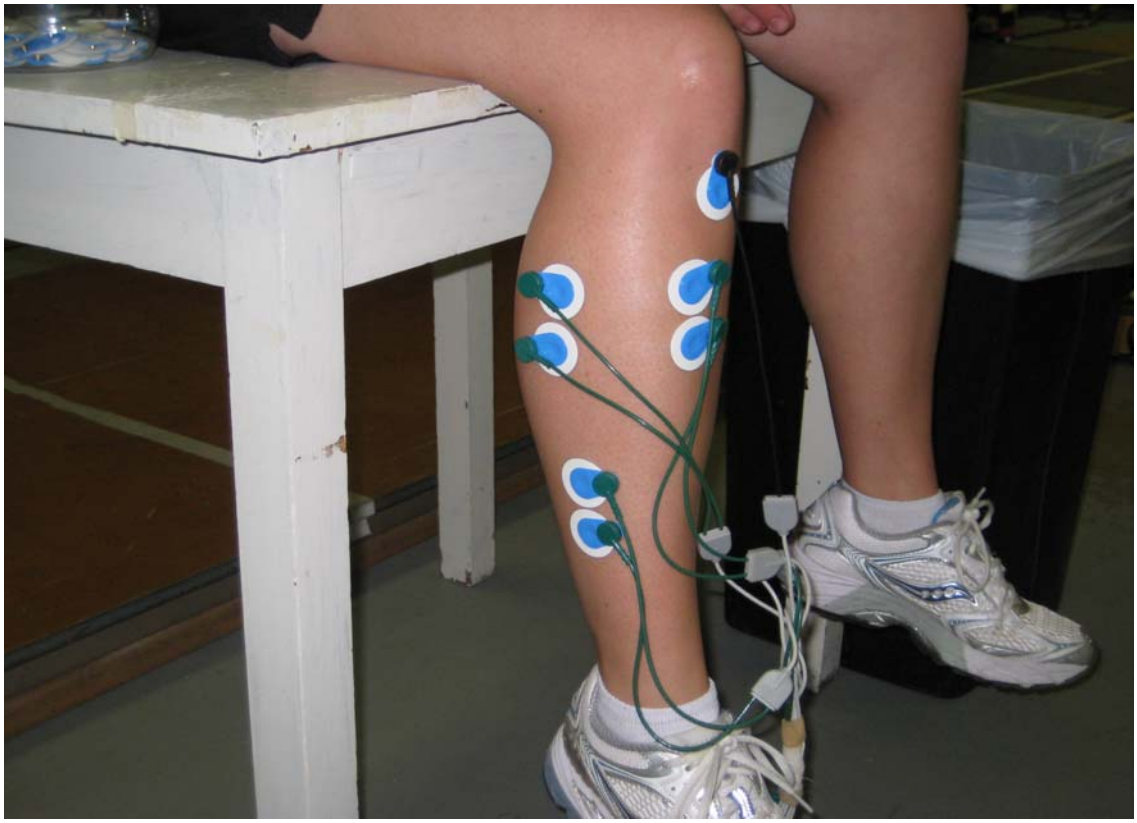

Figure 16- Electrode placement over the peroneus longus, peroneus brevis, and tibialis anterior.

Experimental Design

For the testing of the startle response, a mixed model 3 x 3 repeated measures design was employed. The first independent variable (between groups) was the injury groups, which contained three levels: no prior history, lateral ankle sprain, and high ankle sprain. The second independent variable was the between groups variable, and was the startle event, which had three levels (Startle event 1, event 2, and event 3). To determine the influence of ankle taping between the injury groups, a mixed model 3 x 2 repeated measures design was used for the second and third days of testing. The first independent

variable was between subjects and was the previous history of ankle injury of the participant. There were three levels to this variable, no prior ankle injury, one or more previous lateral ankle sprains, and one or more previous high ankle sprains. The second independent variable, which was within-subjects, was ankle taping, with two levels, no ankle tape and Coach™ Tape. The dependent variables were the latency of the peroneus longus and tibialis anterior, analyzed separately. Another dependent variable to be analyzed included the time to failure (time from touchdown of the fulcrum to time of contact of the lateral border of the shoe).

Data Analysis

To determine if the startle response was present, the ratio of activity of the evertors (peroneus longus and peroneus brevis) to invertors was calculated for each trial. The first trial (event one) was analyzed as a specific condition, because it is the only trial where the participants were completely unaware of the nature and mechanism of the inversion perturbation. To determine if the startle response is present and the response habituates, trials 2-5 were averaged to create one score (second event) and trials 6-10 (event three) were averaged to create one score (Blouin et al., 2003). The data were then analyzed with a 3 (injury group) x 3 (startle event) mixed model repeated measures ANOVA for each leg to determine if there is a statistically significant difference in the activity of the tibialis anterior, peroneus longus, and peroneus brevis among the different startle events and injury groups.

The muscle latency data from the first day were analyzed with a one way ANOVA with three levels (injury group) for the peroneus longus, peroneus brevis, and

tibialis anterior independently. These muscles were measured separately because:1) it is consistent with previous research and 2) these latencies are independent of each other. The muscle latency data from the second and third days of testing were analyzed with two separate 3 (injury history) x 2 (tape condition) mixed model repeated measures ANOVA to determine if there was a statistically significant difference in the latency of the peroneus longus among the different injury and taping groups. Another 3 (injury history) x 2 (tape condition) repeated measures ANOVA was conducted to determine if there is a statistically significant difference in the time to failure among the different injury groups and taping conditions.

CHAPTER 4

RESULTS

The results of the study will be presented in two separate sections. Section One will present the results from the first day of testing. The purpose of the first day was to determine if the startle response was part of the exaggerated first response to the inversion perturbation and to determine the latency of the peroneus longus, peroneus brevis, and tibialis anterior among the three ankle injury history groups. The purpose of the second and third testing days was to determine the affect of ankle taping on the latency of the peroneus longus across the three different ankle injury history groups. For all analyses, the dominant ankle of the healthy participants was compared to the injured ankle of the lateral ankle sprain and high ankle sprain group. The non-dominant ankle of the healthy participants was compared to the non-injured ankle of the lateral ankle sprain and high ankle sprain group. This was done because the dominant ankle is injured twice as frequently as the non-dominant ankle (Ashton-Miller et al., 1996) and to be consistent with previous research that compared latencies across injury groups (Karlsson & Anrdreasson, 1992; Konradsen et al., 1997; Konradssen & Ravn, 1991; Mitchell et. al, 2008; Shima et al., 2005).

Section One: Startle Response

Forty participants (19 female, 21 male) completed the testing protocol for the first day. This included 15 participants with no history of an ankle injury, 15 participants with a history of one or more lateral ankle sprains to the same ankle (7 left; 8 right), and 10 participants with a history of one or more high ankle sprains to the same ankle (4 left, 6 right). The descriptive statistics of the participants are presented in Table 2 below.

Table 2 Descriptive statistics for participants from the first day of testing (means and standard deviations)

Group	Age (years)	Height (m)	Mass (kg)
Total	21.48 ± 1.28	1.74 ± .094	73.29 ± 16.94
No Injury	21.07 ± 1.10	1.69 ± .095	63.46 ± 11.97
Lateral Ankle Sprain	21.20 ± 1.26	1.75 ± .079	74.37 ± 14.40
High Ankle Sprain	22.50 ± 1.08	1.81 ± .063	86.39 ± 18.56

All participants that completed the first day of testing were self-reported as being physically active, exercising a minimum of three days a week for at least 30 minutes a day.

To determine if the startle response was present, a ratio was calculated between the primary evertors of the ankle (peroneus longus and peroneus brevis) and the primary invertor of the ankle (tibialis anterior) for each trial. The average activity of each muscle was calculated 200 milliseconds before contact of the fulcrum with the ground for pre-activity, and the average activity of each muscle was calculated 200 milliseconds after contact of the fulcrum with the ground to determine post activity. The ratio of evertor activity to invertor activity was then created by dividing the muscle activity of the evertors by the muscular activity of the invertors. If the ratio was equal to 1.0, this

indicated equal activity in both the evertors and invertors. If the ratio was greater than 1.0, activity was greater in the evertor muscle group. If the ratio was less than 1.0, activity was greater in the invertor muscle group. The participant's performed 10 trials on both the dominant and non-dominant leg for the non-injured group and 10 trials on both the injured leg and non-injured leg for the lateral and high ankle sprain groups. The first trial was treated as a separate event, since it was the first time the participants were exposed to the inversion perturbation. Trials 2-5 were averaged and trials 6-10 were averaged to create the second and third events. If there was a significant difference across trials, there would be a significant change in the ratio of the evertor muscle group to invertor muscle group across the three events.

To analyze if the startle response was present, two separate analyzes were conducted. The first was conducted between the dominant ankle of the non-injured group and the injured ankle of the lateral and high ankle sprain groups. The second analysis was conducted between the non-dominant ankle of the non-injured group and the non-injured ankle of the lateral and high ankle sprain groups. A 3 (injury group) by 3 (startle event) ANOVA with repeated measures on the last factor was conducted. For the first ANOVA of the dominant and injured ankles, a significant interaction was found between the startle response and the injury group. This indicates that the different injury groups behaved differently across each startle event.

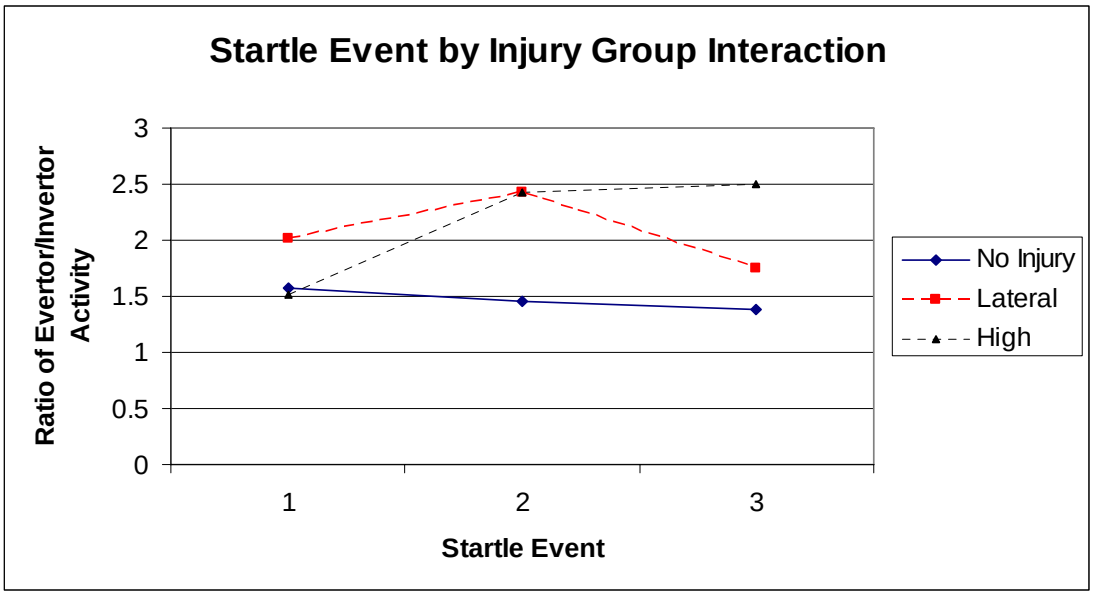

Figure 17- Startle Event by Injury Group Interaction for first startle analysis.

Figure 17 above demonstrates the significant interaction between the startle event and injury groups. The large effect size (η^2=.15) also indicates the relationship between the variables. However, the main effects of startle and injury were non-significant. The results of the analysis are presented in the ANOVA table in Appendix A. The means and standard deviations for the analysis are presented below in Table 3.

Table 3 Means and standard deviations for first startle analysis

Group	Injury	N	Mean % change EVR/INV	Standard Deviation
Startle 1	**No Injury**	15	1.58	.649
	Lateral	15	2.02	1.57
	High	10	1.52	.99
	Total	40	1.73	1.15
Startle 2	**No Injury**	15	1.45	.54
	Lateral	15	2.43	2.73
	High	10	2.43	1.56
	Total	40	2.06	1.89
Startle 3	**No Injury**	15	1.38	.46
	Lateral	15	1.75	.96

	High	10	2.50	1.75
	Total	40	1.80	1.15

While none of the main effects were significant, startle event 2 had the greatest percent change of any of the events. The high ankle sprain group showed the greatest change across the three events, going from 1.52 for event 1, 2.43 for event 2, and 2.50 for event 3, indicating that either the evertors increased or the invertors decreased across events. The effect size for injury group (η^2=.072) was moderate.

For the analysis of the non-dominant ankle and non-injured ankle of the lateral and high ankle sprain group, the ANOVA revealed no significant interaction between the startle event and injury group. There were also no significant main effects for the startle event or the injury group. The means and standard deviations for the second analysis are presented below in Table 4, while the results of the ANOVA are in Appendix A.

Table 4 Means and standard deviations for second startle analysis

Group	Injury	N	Mean % change EVR/INV	Standard Deviation
Startle 1	**No Injury**	15	1.21	.51
	Lateral	15	1.69	1.42
	High	10	1.66	.90
	Total	40	1.50	1.03
Startle 2	**No Injury**	15	1.80	.91
	Lateral	15	1.81	1.11
	High	10	1.97	1.06
	Total	40	1.85	1.00
Startle 3	**No Injury**	15	2.06	1.36
	Lateral	15	1.58	.81
	High	10	1.97	1.24
	Total	40	1.86	1.14

None of the main effects were significant, but startle event 3 had the greatest percent change at 1.86. The no injury group showed the greatest percent change across the three events, with a change of 1.21 for event 1, 1.80 for event 2, and 2.06 for event 3, indicating that either the evertors increased or the invertors decreased across events. The effect sizes for the injury group ($\eta^2 = .121$) and startle condition ($\eta^2 = .085$) were both moderate to large for this analysis.

The latencies of the peroneus longus, peroneus brevis, and tibialis anterior were also measured during the first day of testing. When muscular activity exceeded 5 standard deviations from muscular activity 200 milliseconds prior to contact with the ground, the muscle was considered to be on. There has been little consistency in past research examining muscle latency at the ankle in determining when the muscles are considered on. Some researches have used 2 standard deviations above resting levels to signal the onset of muscle activity (Hopkins et al., 2007; Konradsen et al, 1998; Midgley et al., 2007), while others have used 10 standard deviations (Kernozek et al., 2008; Lynch et al., 1996). Other studies have defined the onset of muscle activity as the first "rise" or "peak" in EMG activity after the ankle is tilted (Cordova et al., 2000; Karlsson & Andreasson, 1992; Konradsen et al., 1997; Mitchell et al., 2008; Shima et al., 2005). This study used 5 standard deviations because it is the opinion of the author that 2 standard deviations is not high enough a threshold (too sensitive), and 10 standard deviations is too high a threshold, and using the first "rise" in EMG activity is not a standardized, objective measure. Latency was measured in milliseconds (ms) as the time from contact of the fulcrum with the ground to when the muscle was considered on. A

separate three way (injury group) ANOVA was calculated for each muscle comparing the dominant leg of the non-injured group to the injured ankle of the lateral and high ankle sprain group and comparing the non-dominant ankle of the non-injured group to the non-injured ankle of the lateral and high ankle sprain group. The only ANOVA that was significant was for the peroneus longus of the non-dominant/non-injured ankles. The results of all the ANOVA's are presented in Appendix A. Since there was a significant difference between the injury groups for the peroneus longus non-dominant/non-injured ANOVA, Fisher's test of Least Significant Digits (LSD) was conducted to see where, among the three groups, the difference occurred. Fisher's LSD revealed a significant mean difference of 12.29 ms between the non-injury group (34.04 ms) and the lateral ankle sprain group (46.33 ms) indicating that the non-injury group had a more favorable latency than the lateral ankle sprain group. The means and standard deviations for each test are presented in the Tables 5-10 below.

Table 5 Means and standard deviations for the latency of the peroneus longus of the dominant/injured ankle

Injury Group	N	Mean Latency (ms)	Standard Deviation
No Injury	15	44.89	12.75
Lateral Ankle Sprain	15	45.43	10.92
High Ankle Sprain	10	44.84	12.46
Total	40	45.08	11.71

Table 5 demonstrates the consistency of the latency of the peroneus longus for the dominant/injured ankles across all three injury groups. The high ankle sprain group had the shortest latency at 44.84 ms.

Table 6 Means and standard deviations for the latency of the peroneus brevis of the dominant/injured ankle

Injury Group	N	Mean Latency (ms)	Standard Deviation
No Injury	15	57.07	19.41
Lateral Ankle Sprain	15	53.81	15.50
High Ankle Sprain	10	51.28	15.03
Total	40	54.40	16.70

Table 6 indicates that the high ankle sprain group had the shortest latency of the peroneus brevis among the dominant/injured ankle condition across all three injury groups at 51.28 ms however; there was no significant difference between the groups.

Table 7 Means and standard deviations for the latency of the tibialis anterior of the dominant/injured ankle

Injury Group	N	Mean Latency (ms)	Standard Deviation
No Injury	15	56.80	15.78
Lateral Ankle Sprain	15	64.05	14.51
High Ankle Sprain	10	64.99	21.86
Total	40	61.57	17.01

Table 7 reveals that although there was no significant difference between the injury groups, the no injury group had the shortest latency of the tibialis anterior of the dominant/injured ankle at 56.80 ms.

Table 8 Means and standard deviations for the latency of the peroneus longus of the non-dominant/non injured ankle

Injury Group	N	Mean Latency (ms)	Standard Deviation
No Injury	15	34.40*	6.91
Lateral Ankle Sprain	15	46.63*	13.76
High Ankle Sprain	10	41.26	11.49
Total	40	40.70	12.02

** Indicates a significant mean difference at the p < .01 level*

Table 8 shows that the no injury group had the shortest latency of the peroneus longus of the non-dominant/non-injured ankle among the three injury groups of 34.40 ms. This latency was significantly differently than that of the lateral ankle sprain group, which had a latency of 46.63 ms, indicating that the musculature of the non-injured participants was considered on sooner than that of the lateral ankle sprain participants. There was also a large effect size ($\eta^2 = .200$) for this test that indicates the relationship between the latency of the peroneus longus and the different injury groups.

Table 9 Means and standard deviations for the latency of the peroneus brevis of the non-dominant/non injured ankle

Injury Group	N	Mean Latency (ms)	Standard Deviation
No Injury	15	52.04	13.57
Lateral Ankle Sprain	15	51.30	18.47
High Ankle Sprain	10	55.77	16.18
Total	40	52.70	15.89

Table 9 demonstrates the latencies of the peroneus brevis of the non-dominant/non-injured ankles. Although not statistically significant, the lateral ankle sprain group had the shortest latency of 51.30 ms.

Table 10 Means and standard deviations for the latency of the tibialis anterior of the non-dominant/non injured ankle

Injury Group	N	Mean Latency (ms)	Standard Deviation
No Injury	15	57.22	15.82
Lateral Ankle Sprain	15	65.75	19.27
High Ankle Sprain	10	66.55	33.24
Total	40	62.75	22.29

Table 10 shows that the tibialis anterior of the no injury group had the shortest latency of 57.22 ms among the three injury groups of the non-dominant/no injury ankles at 57.22 ms, although these differences were not statistically significant.

The time to failure (TTF) was also measured for both ankles of each participant across all trials. The time to failure was the time from contact of the fulcrum with the landing area to contact of the lateral border of the outersole with the landing area. This represented the time it took the participants to go into full inversion. A 3 (injury group) by 2 (testing ankle: dominant/injured and non-dominant/non-injured) ANOVA with repeated measures on the last factor was employed to determine if there was a significant difference for the time to failure between the injury groups and dominant/injured ankles and non-dominant/non-injured ankles. The analysis revealed no significant interaction for injury and testing ankle and no significant main effects for the testing ankle and injury

group was noted either. The means and standard deviations for this analysis are presented in Table 11 below, while the results of the analysis are presented in Appendix A.

Table 11 Means and standard deviations for the time to failure of the dominant/injured ankle and the non-dominant/non injured ankle across the three injury groups

Ankle	Injury	N	Mean TTF (ms)	Standard Deviation
Dominant/Injured	No Injury	15	59.40	34.52
	Lateral Sprain	15	42.91	26.089
	High Sprain	10	45.82	22.81
	Total	40	49.82	29.15
Non-Dominant/ Non-Injured	No Injury	15	50.60	23.83
	Lateral Sprain	15	39.79	15.50
	High Sprain	10	40.39	11.67
	Total	40	43.99	18.66

Table 11 demonstrates that the dominant/ injured ankles, of the no injury group had the largest time to failure at 59.40 ms. For the non-dominant, non-injured analysis, the no injury group again had the longest time to failure at 50.60 ms. However, none of these differences were significant.

Of the 800 trials collected for each participants first day of testing, there were only 39 trials where there was no failure, indicating that the participants prevented the dynamic forced inversion by preventing contact of the lateral border of the outersole with the landing area only 4.88 % of time. Table 12 displays the prevention of the dynamic forced inversion by injury group and by dominant/injured ankle and non-dominant/non-injured ankle.

Table 12 Prevention of dynamic forced inversion by injury group and dominant/injured and non-dominant/non-injured leg for first testing day

Group	Dominant/Injured	Non-Dominant/Non-Injured
No Injury	16	9
Lateral Ankle Sprain	4	0
High Ankle Sprain	8	2
Total	28	11

Section Two: Influence of Ankle Taping

Thirty seven participants (17 female, 20 male) completed the testing protocol on the second and third testing days. This included 13 participants with no history of an ankle sprain, 14 participants with a history of one or more lateral ankle sprains of the same ankle (6 left; 8 right), and 10 participants with a history of one or more high ankle sprains of the same ankle (4 left; 6 right). The descriptive statistics for the participants are presented in Table 13 below.

Table 13 Descriptive statistics for participants from the second and third days of testing (means and standard deviations)

Group	Age (years)	Height (m)	Mass (kg)
Total	21.54 ± 1.28	1.75 ± .091	74.24 ± 17.03
No Injury	21.23 ± 1.09	1.70 ± .096	64.85 ± 12.31
Lateral Ankle Sprain	21.14 ± 1.29	1.75 ± .096	74.28 ± 14.94
High Ankle Sprain	22.50 ± 1.08	1.81 ± .063	86.39 ± 18.56

The participants were randomly assigned to ankle taping order. The latency of the peroneus longus and tibialis anterior were measured each day in milliseconds (ms), as well as the time to failure (TTF) in ms. The muscles were considered on when their activity level exceeded 5 standard deviations of muscle activity 200 ms before contact of the fulcrum with the ground, which is consistent with the methods from the first day of testing. To determine if there was a difference in the latency of the peroneus longus between the injury groups with and without ankle taping, a 3 (injury group) by 2 (ankle support) ANOVA with repeated measures on the last factor was conducted. A separate analysis was conducted comparing the dominant leg of the non-injured group to the injured ankle of the lateral and high ankle sprain groups and a separate analysis was also conducted comparing the non-dominant leg of the non-injured group to the non-injured leg of the lateral and high ankle sprain group. For the first analysis of the dominant/injured ankle, there was no significant interaction and no significant main effect for injury group. There was a significant main effect for ankle support. Fischer's

LSD found a significant mean difference of 10.41 ms ($p < .01$) in the latency of the peroneus longus without ankle taping (46.34 ms) when compared to the latency of the peroneus longus with ankle taping (36.35 ms). The results of the ANOVA are presented in Appendix B. The means and standard deviations are presented below in table 14, which shows that ankle taping significantly reduces the latency of the peroneus longus of the dominant/injured ankles. The large effect size (η^2=.359) for this test demonstrates the strong relationship between ankle taping and the latency of the peroneus longus. There was no significant difference in the latency of the peroneus longus among the three injury groups for no tape, but the lateral ankle sprain group had the shortest latency of 43.55 ms, and no significant difference in the latency of the peroneus longus among the three groups for ankle taping, but the no injury group had the shortest latency of 34.70 ms.

Table 14 Means and standard deviations for the latency of the peroneus longus of the dominant/injured ankle with and without ankle taping

Ankle Support	Injury	N	Mean Latency (ms)	Standard Deviation
No Tape	**No Injury**	13	47.30	12.03
	Lateral Sprain	14	43.55	11.65
	High Sprain	10	49.00	19.11
	Total	37	46.34*	13.93
Tape	**No Injury**	13	34.70	8.95
	Lateral Sprain	14	38.66	12.47
	High Sprain	10	35.25	17.89
	Total	37	36.35*	12.89

** Indicates a significant mean difference at the p < .01 level.*

For the second analysis of the latency of the peroneus longus of the non-dominant/non-injured ankle, there was no significant interaction and no significant main effect for

injury group. Again, there was a significant main effect for ankle support. There was a significant mean difference of 7.86 ms (p <.01) ms in the latency of the peroneus longus without ankle taping (41.21 ms) when compared to the latency of the peroneus longus with ankle taping (33.61 ms) indicating that the peroneus longus was activated quicker in the taped condition. The means and standard deviations are presented below in Table 15, and the results of the ANOVA are presented in Appendix B.

Table 15 Means and standard deviations for the latency of the peroneus longus of the non dominant/non injured ankle with and without ankle taping

Ankle Support	Injury	N	Mean Latency (ms)	Standard Deviation
No Tape	**No Injury**	13	37.68	16.16
	Lateral Sprain	14	40.46	12.17
	High Sprain	10	46.84	17.17
	Total	37	41.21*	15.09
Tape	**No Injury**	13	30.98	8.51
	Lateral Sprain	14	34.10	12.81
	High Sprain	10	36.34	11.25
	Total	37	33.61*	10.94

** Indicates a significant mean difference at the p < .01 level*

The table above demonstrates the significant difference between the latency of the peroneus longus for no tape and for ankle taping. There was also a large effect size (η^2=.204) for this test, which again shows the strength of the relationship between ankle taping and the latency of the peroneus longus. Although there was not a significant difference among injury groups for no tape, the no injury group had the shortest latency of 37.68 ms, and the no injury group had the shortest latency of 30.98 ms for the ankle taping condition.

To determine if there was a difference in the latency of the tibialis anterior between the injury groups with and without ankle taping, a 3 (injury group) by 2 (ankle support) ANOVA with repeated measures on the last factor was conducted. A separate analysis was conducted comparing the dominant leg of the non-injured group to the injured ankle of the lateral and high ankle sprain group and a separate analysis was conducted comparing the non-dominant leg of the non-injured group to the non-injured leg of the lateral and high ankle sprain group. For the first analysis of the dominant/injured ankle, there was no significant interaction between the injury group and ankle support and no significant main effect for injury group or significant main effect for ankle support. The results of the ANOVA are presented in Appendix B. The means and standard deviations are presented below in Table 16. The ankle taping condition had a shorter latency than the no ankle taping condition of 50.33 ms. The lateral ankle sprain group had the shortest latency for no ankle support at 47.56 ms and the shortest latency for ankle taping at 45.13 ms. However, none of these differences were significant.

Table 16 Means and standard deviations for the latency of the tibialis anterior of the dominant/injured ankle with and without ankle taping

Ankle Support	Injury	N	Mean Latency (ms)	Standard Deviation
No Tape	**No Injury**	13	55.73	17.15
	Lateral Sprain	14	47.56	16.55
	High Sprain	10	58.34	22.41
	Total	37	53.34	18.56
Tape	**No Injury**	13	47.49	14.65
	Lateral Sprain	14	45.13	15.86
	High Sprain	10	61.28	16.80
	Total	37	50.33	16.72

For the second analysis of the non-dominant/non-injured ankle, there was no significant interaction and no significant main effect for injury group or significant main effect for ankle support. The means and standard deviations are presented below in Table 17, and the results of the ANOVA are in Appendix B.

Table 17 Means and standard deviations for the latency of the tibialis anterior of the non-dominant/non-injured ankle with and without ankle taping

Ankle Support	Injury	N	Mean Latency (ms)	Standard Deviation
No Tape	**No Injury**	13	50.27	19.44
	Lateral Sprain	14	53.58	26.86
	High Sprain	10	61.21	16.16
	Total	37	54.48	21.70
Tape	**No Injury**	13	54.27	26.50
	Lateral Sprain	14	58.99	32.48
	High Sprain	10	47.07	11.89
	Total	37	54.11	25.95

The above table demonstrates that ankle taping condition had a shorter latency of the tibialis anterior of 54.11 ms when compared to the no ankle support condition. For the no tape condition, the no injury group had the shortest latency of 50.27 ms, while for the ankle taping condition; the high ankle sprain group had the shortest latency of 47.07 ms. None of these differences were significant.

To determine if there was a difference in the time to failure between the injury groups and across taping conditions, a 3 (injury group) by 2 (ankle support) ANOVA with repeated measures on the last factor was conducted. A separate analysis was conducted comparing the dominant leg of the non-injured group to the injured ankle of

the lateral and high ankle sprain group and a separate analysis was conducted comparing the non-dominant leg of the non-injured group to the non-injured leg of the lateral and high ankle sprain group. For the first analysis for the dominant/injured ankle, there was no significant interaction between testing ankle and injury group and no significant main effect for injury group or significant main effect for ankle support. The means and standard deviations are presented below in Table 18, and the results of the analysis are in Appendix B. The ankle taping condition had the longest time to failure of 59.29 ms. For no ankle support, the no injury group had the longest time to failure of 56.19 ms, while the ankle taping condition, no injury group also had the longest time to failure of 71.16 ms. These results were also not significant.

Table 18 Means and standard deviations for the time to failure of the dominant/injured ankle with and without ankle taping

Ankle Support	Injury	N	Mean Time to Failure (ms)	Standard Deviation
No Tape	**No Injury**	13	56.19	25.66
	Lateral Sprain	14	50.36	33.43
	High Sprain	10	46.78	21.99
	Total	37	51.44	27.54
Tape	**No Injury**	13	71.16	31.33
	Lateral Sprain	14	54.87	31.79
	High Sprain	10	50.05	22.12
	Total	37	59.29	29.94

The second analysis for the non-dominant/non-injured ankle yielded no significant interaction and no significant main effect for injury group nor significant main effect for ankle support. The results of the analysis are presented in the ANOVA table in Appendix B. The means and standard deviations are presented below in Table 19.

Table 19 Means and standard deviations for the time to failure of the non-dominant/non-injured ankle with and without ankle taping

Ankle Support	Injury	N	Mean Time to Failure (ms)	Standard Deviation
No Tape	**No Injury**	13	51.86	29.72
	Lateral Sprain	14	48.52	24.87
	High Sprain	10	49.24	31.65
	Total	37	49.89	27.76
Tape	**No Injury**	13	51.50	30.35
	Lateral Sprain	14	51.05	27.32
	High Sprain	10	48.13	25.57
	Total	37	50.42	27.24

The table above shows that the ankle taping condition had the longest time to failure at 50.42 ms. For the no ankle support condition, the no injury group had the longest time to failure at 51.86 ms, while for the ankle taping condition; no-injury group had the longest time to failure at 51.50 ms.

For the no ankle taping condition, there were 52 trials in which the participants did not fail (the lateral border of the outersole did not touch the landing area). This equals a success rate of 7.0 % at preventing the dynamic forced inversion. Table 20 below displays the number of successful preventions for each injury group for the no ankle taping condition.

Table 20 Prevention of the dynamic forced inversion for the no ankle taping condition

Group	Dominant/Injured	Non-Dominant/Non-Injured
No Injury	23	13
Lateral Ankle Sprain	6	6
High Ankle Sprain	0	4
Total	29	23

For the ankle taping condition, there were 29 trials in which the participants did not fail (the lateral border of the outersole did not touch the landing area). This equals a success rate of 3.9 % at preventing the dynamic forced inversion. Table 21 below displays the number of successful preventions for each injury group for the ankle taping condition.

Table 21 Prevention of the dynamic forced inversion for the ankle taping condition

Group	Dominant/Injured	Non-Dominant/Non-Injured
No Injury	6	6
Lateral Ankle Sprain	6	5
High Ankle Sprain	3	3
Total	15	14

Tibialis Anterior Activation

The data from the tibialis anterior revealed that this muscle did not reach the threshold (greater than 5 standard deviations above muscle activity 200 milliseconds before contact of the fulcrum with the landing area) for every trial. From the first day of testing, the tibialis anterior did not reach the threshold to be considered "on" 92 times out of 800 trials, or 11.5 %. From the second and third day of testing without ankle taping, the tibialis anterior did not turn on 216 times out of 740 trials, or 29.19 % of the time. From the second and third day of testing with ankle taping, the tibialis anterior did not turn on 216 times out of 740 trials, or 29.19 % of the time. The breakdown from each testing session is presented below in Tables 22-24 as well as the breakdown by injury group.

Table 22 Number and percentage of times the tibialis anterior failed to reach the threshold for activation for each injury group for the first day of testing

Group	Dominant/Injured Ankle	% of no activation	Non-Dominant/Non-Injured Ankle	% of no activation
No Injury	27	18.0 %	20	13.33 %
Lateral Ankle Sprain	12	8.0 %	10	6.67 %
High Ankle Sprain	14	14.0 %	11	11.0 %
Total	53	13.25 %	29	9.75 %

Table 23 Number and percentage of times the tibialis anterior failed to reach the threshold for activation for each injury group for the second and third day of testing without ankle taping

Group	Dominant/Injured Ankle	% of no activation	Non-Dominant/Non-Injured Ankle	% of no activation
No Injury	31	23.8 %	38	29.2 %
Lateral Ankle Sprain	40	28.6 %	38	27.1 %
High Ankle Sprain	34	34.0 %	35	35.0 %
Total	105	28.4 %	111	30.0 %

Table 24 Number and percentage of times the tibialis anterior failed to reach the threshold for activation for each injury group for the second and third day of testing with ankle taping

Group	Dominant/Injured Ankle	% of no activation	Non-Dominant/Non-Injured Ankle	% of no activation
No Injury	23	17.7 %	25	19.2 %
Lateral Ankle Sprain	41	29.3 %	38	27.1 %
High Ankle Sprain	43	43.0 %	36	36.0 %
Total	111	30.0 %	105	28.4 %

CHAPTER 5

DISCUSSION

The primary purpose of this investigation was to determine the latency of the peroneus longus and peroneus brevis to an inversion perturbation during a single leg drop landing. The secondary purposes were to: (a) determine the difference in the latency of the peroneus longus and peroneus brevis among participants with a history of no ankle sprain, participants with a history of a lateral ankle sprain, and participants with a history of a high ankle sprain, (b) to determine if the startle response is part of the initial exaggerated reaction to an inversion perturbation during a single leg drop landing, and (c) to determine the influence of ankle taping on the latency of the peroneus longus to an inversion perturbation during a single leg drop landing.

This section is divided into 6 sections. The first section discusses the latencies of the ankle musculature results from this study and how they compare to previous work. The second section discusses differences in the latencies of the ankle musculature among the different injury groups. Section 3 examines the results from the startle response data. The fourth section discusses the influence of ankle taping on the latency of the peroneus longus, and the fifth section examines the time to failure results. The final section includes conclusions and suggestions for future research.

Section One: Latency of the Ankle Musculature

Many previous studies have measured the latency of the ankle musculature of participants while stand on the tilt platform or walked across a runway with trap doors to force the ankle into inversion (Cordova et al. 2000; Cordova & Ingersoll, 2003; Hopkins et al. 2007; Karlsson & Andreasson, 1992; Kernozek et al. 2008; Konradsen & Ravn, 1991; Konradsen et al. 1997; Lohrer et al., 1999; Lynch et al. 1996; Midgley et al. 2007; Mitchell et al., 2008; Shima et al., 2005). All studied the latency of the peroneus longus (the main evertor of the ankle), and a few captured the latency of the peroneus brevis and tibialis anterior. However, to the author's knowledge, this was the first study that has used an outersole with fulcrum to cause an inversion moment at the subtalar joint when participants landed from a drop landing in order to replicate the mechanism of a lateral ankle sprain in an effort to measure the latency of the primary evertors of the ankle. The latency of the peroneus longus across all three injury groups for the dominant/injured ankles was 45.08 ms, and the latency of the peroneus longus for the non-dominant/non-injured ankles was 40.70 ms. This finding was shorter than the latency of the peroneus longus reported during standing conditions using the tilt platform or for the walking conditions that used a trap door. The range of the latency of the peroneus longus from previous research has been from 80.8 ms (Shima et al, 2005) to 46.4 ms (Cordova et al., 2000). The latencies from the tilt platform research for the peroneus longus, peroneus brevis, and tibialis anterior are presented below in Table 25.

Table 25 Latencies of the peroneus longus, peroneus brevis, and tibialis anterior reported from tilt platform research and runway with trap door

Study	Peroneus Longus Latency	Peroneus Brevis Latency	Tibialis Anterior Latency
Cordova et al, 2000 Standing on Tilt Platform	46.4 ms-Healthy	N/A	N/A
Cordova & Ingersoll, 2003; Standing on Tilt Platform	57.0 ms-Healthy	N/A	N/A
Hopkins et al., 2007 Standing on Tilt Platform	74.3 ms-Healthy	73.5 ms-Healthy	73.3 ms-Healthy
Hopkins et al., 2007 Runway with Trap Doors	59.5 ms-Healthy	60.1 ms- Healthy	65.0 ms-Healthy
Karlsson & Andreasson, 1992 Standing on Tilt Platform	68.8 ms-Healthy 84.5 ms-Injured	69.2 ms-Healthy 81.6 ms-Injured	N/A
Kernozek et al., 2008 Standing on Tilt Platform	50.3 ms-Healthy	51.2 ms-Healthy	N/A
Konradsen et al., 1997; Standing on Tilt Platform	54.5 ms-Healthy	N/A	N/A
Konradsen et al., 1997 Runway with Trap Doors	48 ms-Healthy	N/A	N/A
Lynch et al., 1996 Standing on Tilt Platform	76.6 ms-Healthy	83.0 ms-Healthy	88.2 ms-Healthy
Midgley et al., 2007 Standing on Tilt Platform	69.3 ms-Healthy	N/A	N/A
Mitchell et al., 2008 Standing on Tilt Platform	54.77 ms-Healthy 62.82 ms-Injured	56.86 ms-Healthy 65.46 ms-Injured	55.75 ms-Healthy 66.04-Injured
Shima et al., 2005 Standing on Tilt Platform	80.8 ms-Healthy	N/A	N/A

	78.1 ms- Injured

While the exact role of the peroneus longus in dynamically preventing a lateral ankle sprain is uncertain, it is reasonable to conclude that due to the mechanical function of the peroneus longus, the quicker it is activated the larger role it may play in preventing an inversion moment at the subtalar joint. There are two reasons why this author believes the latency of the peroneus longus was shorter in this study than in previous work. The first reason is that during a drop landing, there is muscular activity in the peroneus longus as it prepares for contact with the ground. Although a flat outersole was randomly interchanged with the fulcrum in the present study to prevent anticipation of the dynamic forced inversion, the participants knew they were going to make contact with the ground during every trial. It has been established that there is activity in the peroneus longus and peroneus brevis when going down stairs and landing from a jump (Hertel 2002), therefore it is reasonable to conclude that there would be activity in the peroneus longus and brevis prior to contact. The latencies of the peroneus longus have been shown to be shorter in studies that used the walking/trap door methodology vs. the standing on the tilt platform (Hopkins et al., 2007), and it is known that muscular activity is higher in a dynamic task as opposed to a static task (Nakazawa et al., 2004). Therefore, the shortened latency noted in this study is appropriate as the participants were involved in a more dynamic task than standing or walking.

The second reason the latencies of the peroneus longus were shorter was due to the angular velocity of the inversion perturbation. Previous work using the tilt platform and runway with trap door used an angular velocity between 200-400 deg/sec (Cordova

et al., 2000; Cordova et al., 2003; Hopkins et al., 2007; Karlsson & Andreasson, 1992; Kernozek et al., 2008; Konradsen & Ravn, 1991; Konradsen et al., 1997; Lohrer et al., 1999; Lynch et al., 1996; Midgley et al., 2007; Mitchell et al., 2008; Shima et al., 2005). Due to the nature of the inversion perturbation used in the present study, the angular inversion velocity was greater than in previous research. Based on the average time to failure of 50.60 ms for the dominant/injured ankles and 43.99 ms for the non-dominant/non-injured ankles, and the fact that there was 25° of inversion caused by the fulcrum, the average angular velocity was 494 deg/sec for the dominant/injured ankles and 568 deg/sec for the non-dominant/non-injured ankles. Previous research has shown that a higher rate of angular inversion velocity is associated with shorter muscle latencies (Lynch et al., 1996). In light of the differences in the methodologies and the reviews of previous literature, it is appropriate that the task utilized in this study would produce shorter latencies. Furthermore, it would appear that the shorter latencies are closer to those theoretically encountered during an actual lateral ankle sprain.

The latency of the peroneus brevis across all three injury groups for the dominant/injured ankles was 54.40 ms, and the latency of the peroneus brevis for the non-dominant/non-injured ankles was 52.70 ms. Although the latency of the peroneus brevis has not been investigated as frequently as the peroneus longus, the importance of this muscle's contribution as a dynamic stabilizer should not be overlooked and in fact, previous research has indicated that the peroneus brevis should be examined in any future work examining the latency of the ankle musculature (Lynch et al., 1996). These findings on the latency of the peroneus brevis are relevant because the peroneus brevis, after the peroneus longus, provides the second strongest dynamic eversion moment to

help prevent an inversion moment (Heckman et al., 2008). The reasons the latencies of the peroneus brevis were shorter in this study than in previous research is the same as those cited for the peroneus longus: (a) the dynamic nature of the task caused muscle activation prior to contact with the ground and (b) the higher angular inversion velocity used in the present study than in previous studies. Examination of Table 25 indicates that the latencies of the peroneus brevis found in the present study were shorter than all previous research with the exception of Kernozek et al. (2008). They may have found a shorter latency of the peroneus brevis because they used a combination of inversion and plantar flexion, so a direct comparison of their results to the current study cannot be accurately made

The findings of the shorter latencies of the peroneus longus and peroneus brevis call in to question some of the conclusions made from research using the tilt platform while standing and the trap door while walking. Based on the latencies of the peroneus longus and peroneus brevis measured using the tilt platform/trap door, several authors have concluded that the peroneus longus and peroneus brevis do not turn on quickly enough upon inversion to prevent a lateral ankle sprain (Hopkins et al., 2007; Kernozek et al., 2008; Konradsen et al., 1997). They base this estimate on 54 ms for the peroneal muscle group to turn on and 72 ms for force development to occur in the muscle due to electromechanical delay, and reasoned that this time is not fast enough to prevent the ankle from injury. However, the results from the present research found that the latencies of the peroneus longus and peroneus brevis were on average 10-15 ms less than the latencies reported previously by these researchers (Hopkins et al., 2007; Kernozek et al., 2008; Konradsen et al., 1997). Although the present study did not measure

electromechanical delay, the shorter latencies indicate that these muscles may be able to turn on quickly enough to prevent a lateral ankle sprain or at least help attenuate the rate at which the injury occurs. Since it is believed that the pre-activity in the ankle musculature contributed to the shorter latencies, the next questions to be answered are: (a) does the pre-activity in the ankle musculature during a landing task lead to a reduction in the electromechanical delay of the peroneus longus and peroneus brevis during a dynamic forced inversion, and (b) is there enough time for the peroneus longus and peroneus brevis to prevent a lateral ankle sprain based on the results using a more dynamic methodology?

Since the present study exposed the participants to an inversion moment only, comparisons of the latency of the tibialis anterior could only be made to other studies that forced the participants into inversion only. The latency of the tibialis anterior across all three injury groups for the dominant/injured ankles was 61.57 ms, and the latency of the tibialis anterior for the non-dominant/non-injured ankles was 62.75 ms. These findings fall into the range of previous work (Hopkins et al., 2007; Lynch et al., 1996; Mitchell et al., 2008). The latency of the tibialis anterior found in other studies may be found in Table 25.

The results from the current study indicate that the peroneus longus and peroneus brevis turn on faster using the current methodology, while the tibialis anterior has the same latency as that found using the tilt platform, which indicates that in the present study, the tibialis anterior turned on at an even later time after the peroneus longus and peroneus brevis turned on. This calls into question the role of the tibialis anterior during an isolated inversion moment at the ankle. The results of this study also found that on

day one of testing, the tibialis anterior failed to reach the threshold to be considered "active" 11.5 % of the time. This percentage increased even greater on days two and three of testing with and without ankle taping. The tibialis anterior failed to reach the threshold 29.19 % of the time during trials conducted on the second and third day. Previous research that used the tilt platform/trap door to cause an isolated inversion moment found that the tibialis anterior was not active 26 % of the time. This lead Lynch et al. (1996) to conclude that the tibialis anterior is a difficult muscle to analyze in this condition because of the high baseline activity before the inversion perturbation is applied; and due to the lack of a large external stimulus of the tibialis anterior during the inversion perturbation (Lynch et al., 1996).

It is true that the tibialis anterior is a difficult muscle to analyze. The present study used 5 standard deviations above muscle activity 200 milliseconds before touchdown as the threshold to consider the muscle "active" in an attempt to eliminate the influence of the high baseline activity that could obscure the latency. Therefore, this author believes that the reason the tibialis anterior failed to activate 23.3 % of the time over the course of the entire study is due to the lack of a strong external stimulus of the tibialis anterior during the inversion moment that would require a large activation of this muscle. When the ankle is forced into inversion, the peroneus longus and peroneus brevis are activated in order to prevent excessive inversion range of motion. Due to reciprocal inhibition (Latash 2008), the tibialis anterior muscle would be inhibited when the peroneus longus and peroneus brevis are excited. Since the tibialis anterior failed to activate at an even higher percentage on the second and third days of testing (29.19 %) compared to the first day of testing (11.5 %), it is possible that a training effect may have

been present where the tibialis anterior was activated less frequently after repeated exposures to the inversion mechanism. If this is the case, then the decrease in tibialis anterior activity would leave the protective mechanism of the peroneus longus and peroneus brevis to function without the negating activity of the tibialis anterior. This training effect will be further discussed in the startle response section.

Summary

The present study determined the latencies of the peroneus longus, peroneus brevis, and tibialis anterior upon a dynamic forced inversion after a drop landing. The latencies of the peroneus longus for both the dominant/injured ankles and non-dominant/non-injured ankles were shorter than those found by previous researchers. The latencies of the peroneus brevis for both the dominant/injured ankles and non-dominant/non-injured ankles were shorter than all previous studies with the exception of one, and the latencies of the tibialis anterior were very similar to those found by previous researchers. While the tilt platform/trap door is a valuable tool in evaluating muscle latency during an inversion moment, most ankle sprains do not occur a) when the person is standing still b) when the person is standing with equal weight distribution on both feet c) the floor falls away and d) at the rate of inversion angular velocity that occurs using the tilt platform. Many ankle sprains occur when landing from a jump or during foot placement, which is the mechanism the methodology of the present study attempted to replicate. This author believes that the finding of shorter latencies in the peroneus longus and brevis are meaningful since these two muscles are the ankle's primary active defense against a lateral ankle sprain, and the quicker these muscles are activated when the ankle

is forced into inversion, the more effective they will be at limiting the amount of inversion range of motion.

It is the belief of this author that the previous studies overestimated the latencies of the peroneus longus and brevis because these muscles did not develop pre-activity in preparation for a dynamic task and because of the slower rate of angular velocity in the previous work. As a result, tilt platform/trap door data must be read with some caution with regard to the values reported. Previous conclusions from tilt platform/trap door research indicating that the peroneus longus and peroneus brevis do not turn on quickly enough after the ankle is forced into inversion to prevent a lateral ankle sprain must now be reconsidered based on the results of this study.

Section Two: Latency of Ankle Musculature Among Different Injury Groups

The second purpose of the current study was to determine if there was a difference in the latency of the peroneus longus, peroneus brevis, and tibialis anterior among participants with no history of a lateral ankle sprain, participants with a history of one or more lateral ankle sprains, and participants with a history of one or more high ankle sprains on only one side of the body. Previous work has been inconsistent with regard to the latencies of these three muscles between healthy and unhealthy ankles. Several studies have found that healthy participants have a shorter latency in all three muscles than participants with functional ankle instability or chronic ankle instability (Karlsson & Anrdreasson, 1992; Konradssen & Ravn, 1991; Mitchell et. al, 2008), however other work has not found a significant difference in the latency of the peroneus longus between healthy and unhealthy ankles (Konradsen et al., 1997) and one study

even found that the injured sample had a shorter latency of the peroneus longus than the healthy sample (Shima et al., 2005).

In the present study, there was no significant difference in the latency of the peroneus longus of the dominant/injured ankles among the three injury groups, and all three groups had similar values (No injury = 44.89 ms; Lateral ankle sprain = 45.43 ms; High ankle sprain = 44.84 ms). There was a significant difference in the latency of the peroneus longus between the injury groups of the non-dominant/non-injured ankles, with the no injury group having a mean latency of 34.40 ms, which was significantly less than the latency of the lateral ankle sprain group (46.63 ms), and less (not significant) than the latency of the high ankle sprain group (41.26 ms).

There was not a significant difference in the latency of the peroneus brevis of the dominant/injured ankles among the three groups, but the high ankle sprain group (51.28 ms) and the lateral ankle sprain group (53.81 ms) both had shorter latencies than the no injury group (57.07 ms). For the non-dominant/non-injured ankles, there was not a significant difference in the latencies between the three groups. The lateral ankle sprain group (51.29 ms) and the no injury group (52.04 ms) had very similar latencies, while the high ankle sprain group had the longest latency of 55.77 ms.

These results are difficult to explain as the findings are not consistent with previous literature nor with expected outcomes. It was thought that the behavior of the peroneus longus across groups would be matched by the behavior of the peroneus brevis. As these muscles seem to have responded differently to the same stimuli, (that should have brought about analogous results), it would appear that the demands on these muscles

are not identical. Further research is required to determine the mechanical demands placed upon each of these muscles.

For the tibialis anterior, there was not a significant difference in latencies among the three injury groups for the dominant/injured ankles nor for the non-dominant/non-injured ankles. The no injury group had the shortest latency of the tibialis anterior of 56.80 ms for the dominant/injured ankles, while the lateral ankle sprain group (64.05 ms) and high ankle sprain group (64.99 ms) had latencies very similar to each other. For the non-dominant/non-injured ankles, the no injury group again had the shortest latency of 57.22 ms while the lateral ankle sprain group and high ankle sprain group again had similar latencies of 65.75 ms and 66.55 ms respectively. These results suggest that an ankle injury alters the performance of the tibialis anterior, although the difference on the non-dominant/non-injured side seems to suggest a more global pattern. More research is needed to determine if this pattern persists in inversion movements that include a plantar flexion moment as well.

One of the research hypotheses for the present study was that participants with a history of a lateral or high ankle sprain would have greater latency of the peroneus longus and peroneus brevis than participants with no history of an ankle sprain. The results of the present study do not support this hypothesis, as there was no difference between the injury groups. Two reasons may explain this lack of significant different between the no injury and injury groups in this study. The first is the criteria for inclusion in the study. In the present study, participants only had to have sustained a previous unilateral ankle sprain to be allowed in the study. Mechanical instability was not a requirement for inclusion, due to the nature of the inversion perturbation. Although residual symptoms

are common among people that have suffered an ankle sprain, it is not seen in all people. The previous research comparing a healthy sample to a sample that had a history of an ankle sprain required participants to have either chronic ankle instability or functional ankle instability. These participants had sustained more than one ankle sprain and had residual laxity of the ankle complex. Of the participants with a previous ankle sprain that were measured in the current study, only 3 out of the 25 in the lateral ankle sprain or high ankle sprain group had suffered more than one ankle sprain. The second potential reason why there was no difference between the injury groups was the pre-activity in the peroneus longus and peroneus brevis before contact of the fulcrum with the ground.. This pre-activity in the muscles due to stepping down and preparing for landing occurred in all three injury groups and may have eliminated differences between healthy and unhealthy participants that were found using the tilt platform. The differences found using the tilt platform may be attributed to the static nature of the task negating the necessity of pre-activity in the ankle musculature. This is an advantage of the tilt platform, because it has allowed researchers to demonstrate that participants that have suffered an ankle sprain have a longer latency in the injured ankles when compared to healthy participants. However, the present study would seem to suggest that this finding does not persist in more dynamic movements.

The only significant findings between the injury groups was the latency of the peroneus longus of the non-dominant/non-injured ankles, with the no injury group having a significantly shorter latency of 34.40 ms when compared to the latency of the lateral ankle sprain group of 46.63 ms. The shorter latency of the non-injured ankles may suggest that shorter latencies are beneficial in preventing lateral ankle sprains. Little

research has been conducted on the difference in the latency of the peroneus longus of the non-dominant ankle of non-injured participants when compared to the non-injured leg of participants that had sustained an ankle sprain (Mitchell et al., 2008), but what has been done noted no significant difference between the non-injured ankles of healthy vs. previously injured individuals. There are several potential explanations for this finding. First, the non-injured ankle of the lateral ankle sprain group may have a significantly longer latency than the no injury group due to the rehabilitation process. It is common after an ankle sprain to perform strength and proprioception exercises, but many times these exercises are only performed on the injured ankle, leading to possible deficits in the uninjured ankle. That is why it is important during the rehabilitation process to not neglect the non-injured ankle, or deficits in the latency of the ankle musculature may occur. Second, it is possible that there is a reason that the non-dominant leg is less injured, perhaps due to the different demands placed upon the non-dominant limb. Research has shown that the dominant leg produces a greater peak vertical force and higher vertical displacement during a single leg jump than the non-dominant leg (Stephens et al., 2007). More strength and power are produced by the dominant leg, and these demands may make it more susceptible to injury than the non-dominant leg.

Summary

There was no significant difference in the latency of the peroneus longus, peroneus brevis, and tibialis anterior among the dominant/non-injured ankles. This finding supports the work of Konradssen et al., 1998, but contradicts the work of Karlsson & Anrdreasson, 1992; Konradssen & Ravn, 1991; Mitchell et al. 2008 who did

find significantly different latencies between people with chronic or functional ankle instability and healthy participants. The inclusion criteria for the present study and/or the pre-activity developed in the ankle musculature before landing may have contributed to the non-significant finding in the present study. There was a significant difference in the latency of the peroneus longus of the non-dominant ankle of the no injury group compared to the non-injured ankle of the lateral ankle sprain group. Unfortunately, there is very limited research that has compared the non-dominant ankle of healthy participants to the uninjured ankle of participants that have sustained an ankle sprain, so the results are difficult to generalize. However, this increase in latency of the uninjured ankle of the lateral ankle sprain group may be a result of rehabilitation that only focused on the injured ankle or could be a reflection of the different demands required of the dominant and non-dominant legs. The relationship between the latency of the non-dominant ankle of healthy participants and the non-injured ankle that have sustained an ankle sprain should be examined further.

Section Three: Startle Response

The third purpose of the present study was to determine if the startle response was part of the initial response to an inversion perturbation, and if this response attenuates over time. Past research has found that the first time the body is exposed to an unexpected perturbation; there is co-activation of both the agonist and antagonist muscle groups. Over time, the response of the antagonist muscle group attenuates, and the activity of the agonist muscle group remains constant or increases (Siegmund et al., 2008). However, this response has not been applied to the ankle. If the startle response

is present, there would be co-activation of both the evertors of the ankle (peroneus longus and peroneus brevis) and invertors of the ankle (tibialis anterior) on the first trial (a ratio of evertor/invertor activity equal or near to 1.0). Over time (trials) this ratio could either increase, indicating more evertor activity or less invertor activity (which would be desirable), or decrease, indicating more invertor activity and/or less evertor activity (which would contribute to the inversion moment).

For the testing of the dominant/injured legs, there was a significant interaction between the startle event and injury condition. This finding indicates that the different injury groups acted differently across the startle conditions. There were no significant main effects for startle event or injury group. By examining the graph of the interaction (Figure 14), it is clear that there is little change in the ratio for the no injury group, from 1.58 for event 1, 1.45 for event 2, and 1.38 for event 3. The lateral ankle sprain group demonstrated an increase from event 1-2, but then a marked decrease from event 2-3 (2.02 for event 1, 2.43 for event 2, 1.75 for event 3) suggesting that in the injured ankle of participants that have sustained a lateral ankle sprain, the initial increase in evertor activity or decrease in invertor activity during trials 2-5 does not persist over the course of 10 trials. The reason for this is unclear and warrants further examination. Of particular interest is the high ankle sprain group which had an increase in the ratio across all three events, from 2.02 for event 1, 2.43 for event 2, and 2.50 for event 3. This result would seem to suggest that repeated exposure actually caused there to be either a decrease in the invertors or an increase in the evertors; in either case, this would be considered a desirable result. Interestingly, all three injury groups had a ratio greater than 1.50 for event 1, which demonstrates greater activity in the evertor muscle group than the

invertor muscle group during the first exposure to the inversion perturbation. This ratio increased to 2.06 for event 2, but decreased to 1.80 for event 3 when the ratio is collapsed across all groups. Although there was not a main effect for startle event, there is a promising trend in the data where the activity of the evertor muscle group was greater for event 2 and event 3 when compared to event 1. This trend is advantageous as an increase in the evertor activity and/or a concurrent decrease in the invertor activity would allow for a greater protective mechanism from the muscular support. As the foot falls into inversion, it is the role of the evertors to: (a) slow this fall; and/or (b) prevent the fall into inversion from happening at all. These results seem to indicate that this is indeed what is happening and has tremendous implications for future research regarding the training of the musculature to prevent ankle sprains or at least the severity of the ankle sprains.

For the testing of the non-dominant/non-injured legs, there was no significant interaction between startle event and injury group, and no significant main effect for injury group or startle event. Although the main effect for startle event was not significant, there was a large effect size for this variable ($\eta^2 = .085$), and the trend (increasing ratio across all three events) is present in the data. The mean for startle event 1 across all injury groups was 1.50, the mean for event 2 was 1.85, and the mean for event 3 was 1.86. This shows an increase in the ratio of evertor/invertor activity across all startle events and further supports the theory that the ankle musculature can be trained to provide greater evertor muscle activity and/or less invertor activity. Due to the stringent inclusion criteria, the sample size for this study was limited. With a greater number of participants, the main effect for startle, if the data were to follow the current trend, would be significant. For the no injury group and the high ankle sprain group,

there was an increase in the ratio across all three startle events and although not included in the results section, an independent analysis of the no-injury group revealed a significant main effect for startle event, where there was a significant increase in the ratio of evertor/invertor activity across all 3 startle events.

The results of this study signify that stiffness did not occur at the ankle when it was forced into inversion by an external force using the current methodology. There are three findings to support this conclusion. First, during the startle response trials, the ratio of evertor to invertor activity was always greater than 1.0, indicating greater evertor activity than invertor activity. For joint stiffness to have been present, the ratio would have been equal or close to 1.0. When the ratio was collapsed across all three injury groups for both startle analysis, it was always greater than 1.50. Therefore, there was always at least 50% greater activity in the evertor muscle group than the invertor muscle group. Second, the present study found that the latency of the tibialis anterior was consistent with latencies reported by other researchers, but the latency of the peroneus longus was shorter than those reported by other researchers. This indicates that in the present study, there was an even greater amount of time between the activation of the evertors of the ankle and the invertors of the ankle. Third, the tibialis anterior failed to reach the threshold for activation 23.13 % of the trials across all testing days. These findings are positive because the greater activity in the evertor muscle group, the less likely a lateral ankle sprain is to occur.

Summary

One of the purposes for examining the startle response at the ankle was to determine if the activity of the invertor muscle group may be negating the moment produced by the evertor muscle group, and to see if this response attenuates over time. Previous research has examined the eversion to inversion strength ratio and postural control between healthy ankles and ankles with functional ankle instability (Bernier et al., 1997; Kaminski et al., 1999; Mitchell et al., 2008; Ross & Guskiewicz, 2004), but has failed to examine this ratio of evertor to invertor activity during a dynamic forced inversion and how repeated exposures to the same mechanism changes this ratio The trends in the data from the current study indicate that after repeated exposures to an inversion perturbation, the ratio between the evertors and invertors of the ankle increases. The peroneus longus and peroneus brevis (evertors) may help prevent a lateral ankle sprain by contracting eccentrically when the ankle is forced into inversion, but if the tibialis anterior (primary invertor) is active as well, it may cancel the eversion moment produced by these muscles. The ratio increasing may be due to increased activity in the evertor muscle group or decreased activity in the invertor muscle group, either condition is favorable for the attenuation of the inversion moment. This trend in the data supports future research that could lead to the development of a training program that exposes participants to an inversion perturbation in order to train the musculature to produce a larger ratio of evertor to invertor activity when the ankle is forced into inversion, unexpectedly.

Section Four: Influence of Ankle Taping

The fourth purpose of this study was to determine the influence of ankle taping on the latency of the ankle musculature. The participants had both ankles taped and tested on days two or three (depending on random assignment to taping order) using the same methodology as the first day. Each participant had his or her ankles taped by a Certified Athletic Trainer in a closed basketweave manner before performing the testing. Previous research, which has used a variety of methodologies, has found differing results with regard to the influence of ankle taping on the latency of the peroneus longus. It was hoped that the use of a more dynamic testing procedure would shed more light on the conflicting results from previous research.

Consistent with the results from the first day of testing, there was no significant difference in the latency of the peroneus longus among the different injury groups for the no tape and ankle taping condition, for both the dominant/injured ankles and non-dominant/non-injured ankles. There was a significant difference in the latency of the no tape condition compared to the latency of the ankle taping condition for both the dominant/injured leg and the non-dominant/non-injured leg. These differences and their implications will be discussed further.

For the dominant/injured ankles, the no tape condition had a mean latency of 46.34 ms, while the taping condition had a mean latency of 36.35 ms. Not only is this difference significant, but it is also meaningful due to the large effect size (η^2=.359). For the non-dominant/non-injured ankles, the no tape condition had a mean latency of 41.21 ms while the taping condition had a mean latency of 33.61 ms. This difference is also

significant and has a large effect size (η^2=.204). These results indicate that immediately after the ankle is taped in a closed basketweave manner, there is a significant reduction in the latency of the peroneus longus of 9.90 ms for the dominant/injured ankles and 7.60 ms for the non-dominant/non-injured ankles. The potential application of this finding is that ankle taping significantly reduces the amount of time it takes the peroneus longus to turn on as the ankle is forced into inversion. This finding strongly supports the use of athletic tape in the prevention of ankle sprains, as the peroneus longus is the primary defense against a lateral ankle sprain, and the sooner it turns on, the more likely it is to prevent a lateral ankle sprain.

Summary

The research hypothesis for tape suggested that ankle taping would significantly reduce the latency of the peroneus longus when compared to the same test conducted in the absence of athletic tape. The results of this study support that hypothesis. These results further support the use of ankle taping as a prophylactic measure to prevent ankle sprains among participants that have never suffered an ankle sprain and participants that have a history of ankle injury, as the greatest predictor of a future ankle sprain is a history of one or more ankle sprains (Midgley et. al., 2007; DiStefano et al., 2008). These results revealed that the application of athletic tape will cause the peroneus longus, which is the primary active defense against a lateral ankle sprain, to activate significantly quicker than without the application of athletic tape. Past research has found that ankle taping will reduce the latency of the peroneus longus (Karlsson & Andreasson, 1992) because the athletic tape increases sensory input by stimulating mechanoreceptors in the skin, muscle

and joint capsule. This increased stimulation likely increases the sensitivity of the muscle spindles in the peroneus longus to a sudden inversion perturbation (Ashton-Miller et al., 1996, Midgley et al, 2007).

Section 5: Time to Failure

An additional set of data that were collected during this study was the time to failure. This was the time from contact of the fulcrum with the landing area to contact of the lateral border of the outersole with the landing area. The time to failure represents the amount of time it took the participants to go through the full 25° of inversion, so a longer time is desirable. This was measured for each participant for every trial. The time to failure from day one was analyzed separately from the data from days two and three. The time to failure results will now be discussed.

From the first day of testing, the data revealed that there was no significant difference in the time to failure between the different injury groups nor between the dominant/injured ankles and the non-dominant/non-injured ankles. Although there were no statistically significant differences, the trends in the data indicate that the dominant/injured ankles had a longer time to failure of 49.82 ms than the non-dominant/non-injured ankles, which had a mean time to failure of 43.99 ms, suggesting that the dominant/injured ankles were able to resist failure longer than the non-dominant/non-injured legs. The no injury group had the longest time to failure among the dominant/injured ankles of 59.40 ms, while the lateral ankle sprain group had the shortest time to failure of 42.91 ms (high ankle sprain group = 45.82 ms). This finding suggests that once a person has sustained an ankle sprain, their ability to resist the

inversion perturbation is diminished, and supports the theory that the greatest predictor of a future ankle sprain is a past ankle sprain (Midgley et. al., 2007; DiStefano et al., 2008). Closer inspection of the results also suggests that people who have never sustained an ankle sprain are better equipped for resisting an inversion moment which may cause a lateral ankle sprain. For the non-dominant/non-injured ankles, the no-injury group again had the longest time to failure of 50.60 ms, while the lateral ankle sprain group and high ankle sprain group had mean time to failures of 39.79 ms and 40.39 ms respectively. This finding is of particular importance as it compares non-injured to non-injured ankles. The finding of a longer time to failure for the non-injured population may suggest that there is a difference between people making some more susceptible to lateral ankle sprains than others.

Of the 800 trials performed by the 40 participants on day one of testing, there were 39 successful preventions of the dynamic forced inversion, meaning the lateral border of the outersole never made contact with the landing area. This equals a success rate of 4.88 %. The non-injured group was the most successful at preventing the dynamic forced inversion with 25 successful preventions (16 dominant, 9 non-dominant), followed by the high ankle sprain group with 10 successful preventions (8 injured, 2 non-injured) and the lateral ankle sprain group with 4 successful preventions (4 injured). To the author's knowledge, this was the first study that has utilized the metric of time to failure during a dynamic forced inversion. One previous study (Ubell et al., 2003) examined the effectiveness of different ankle braces at preventing a dynamic forced inversion of 24° upon landing from a 60 cm forward single leg jump. That study found a success rate of 18% at preventing the dynamic forced inversion with no ankle support, however, the

study only used participants with healthy ankles and only looked at success or failure, not the time to failure (Ubell et al., 2003). Another study found that participants with chronic ankle instability had a longer time to stabilization when landing from a jump than participants with no history of an ankle injury, however this study only included a landing task, there was no inversion perturbation (Brown & Mynark, 2007). From the second and third day of testing, the dominant/injured ankles and non-dominant/non-injured ankles were analyzed separately. For the dominant/injured ankles, there was no significant difference in the time to failure among the different injury groups or ankle support condition, however, as expected the ankle taping condition (59.29 ms) did have a greater time to failure than the no ankle tape condition (51.44 ms). The no-injury group had the longest time to failure among the different injury groups for no ankle support with a mean time to failure of 56.19 ms and for ankle taping with a mean time to failure of 71.16 ms. This finding continues to support the notion that (a) there may be a reason why the non-injured have avoided this injury or (b) that the injury has a global effect on both the injured and non-injured limbs of the non-healthy participants (non-healthy meaning that they had previously sustained an ankle sprain). For the non-dominant/non-injured ankles, there was no significant difference in the time to failure among neither the different injury groups nor the ankle support conditions. The ankle taping condition again had a greater time to failure of 50.42 ms than the no ankle taping condition, which had a mean time to failure of 49.89 ms. The no-injury group had the longest time to failure for the no ankle taping condition of 51.86 ms and the taping condition with a mean time to failure of 51.50 ms. These results are consistent with the first day of testing and also indicate that ankle taping did not make the lateral ankle sprain group or high

ankle sprain group more successful at preventing the dynamic forced inversion than the no injury group, and further supports the theory that the longer time to failure has aided the no-injury group in preventing an ankle sprain. Further, these results may indicate that once you sustain an ankle sprain, the other ankle may be at risk as well. A future question to be investigated should focus on the notion that there is a certain threshold time to failure for which if a person is below that value, then they may be classified as pre-disposed to ankle sprains (a predictor of injury).

For the no ankle taping condition across all injury groups, the success rate at preventing the dynamic forced inversion was 7.0%, which was higher than the overall success rate across all injury groups for day one. The no-injury group had the highest number of no failures with 36 (23 dominant ankle, 13 non-dominant ankle), while the lateral ankle sprain group had 12 no failures (6 injured ankle, 6 non-injured ankle), and the high ankle sprain group had 4 no failures (4 non-injured ankle). For the ankle taping condition, the success rate decreased to 3.9%. The no-injury group had the highest success rate with 12 no failures (6 injured ankle, 6 non-injured ankle), while the lateral ankle sprain group had 11 no failures (6 injured ankle, 5 non-injured ankle), and the high ankle sprain group had 6 no failures (3 injured ankle, 3 non-injured ankle). The decrease in success rate for the taping condition was surprising. The application of athletic tape caused a significant reduction in the latency of the peroneus longus and also increased the time to failure, but not the success rate at preventing the dynamic forced inversion. This may be due to the fact that the amount of inversion produced in the current study was only 25°. If a greater amount of inversion was caused by the fulcrum, the ankle taping condition may be more successful at preventing the dynamic forced inversion than the no

taping condition because a greater amount of range of motion would give the participant an increased amount of time to prevent the dynamic forced inversion.

The results of Ubell et al. (2003) discovered that ankle bracing increased the success rate of preventing the dynamic forced inversion between 34% to 52 %, while in the present study the application of athletic tape did not increase the success rate at preventing the dynamic forced inversion. While both studies used an outersole with fulcrum to create a dynamic forced inversion, the previous study (Ubell et al., 2003) used a forward jump, while the current study used a single leg drop landing. It is difficult to make a comparison between the two studies because in the current study, a large component of the ground reaction force was in the vertical direction, while in the Ubell et al. (2003) study there was a greater anterior-posterior component of the ground reaction force.

Summary

In the present study, although not statistically significant, the no-injury group had the longest time to failure for each of the testing conditions and was the most successful at preventing the forced dynamic inversion. This finding indicates that the non-injured participants were able to prevent the ankle from going into the full 25° of inversion longer than participants that have had an ankle sprain. There are several explanations for the decrease in time to failure for the lateral and high ankle sprain groups. An increase in laxity among the injured participants, which occurs in 32-40 % of people that sustain an ankle sprain (Mitchell et al., 2008) would make the subtalar joint hypermobile and therefore decrease the ability of the joint structure (capsule and ligaments) to resist a

dynamic forced inversion. Also, the electromechanical delay may have been greater among the injured participants than the non-injured participants, also as a result of the increased laxity. This would lead to a longer time for the peroneus longus and peroneus brevis to develop enough tension to prevent the inversion moment. Again, electromechanical delay was not measured in the current study, but future research should examine the electromechanical delay between different injury groups. The fact that some of the participants were able to prevent the 25° of dynamic forced inversion indicates that the peroneus longus and peroneus brevis may be able to prevent a lateral ankle sprain when the ankle is forced into inversion. The author believes the success rate would have been greater with a larger amount of inversion range of motion and future research should use a fulcrum that causes a greater amount of inversion range of motion.

Section 6: Conclusions and directions for future research

The final section will discuss the conclusions that can be drawn from this study and the directions for future research. This section will be broken down into four sections: 1) The latencies of the ankle musculature and the different injury groups; 2) the startle response; 3) influence of ankle taping; and 4) additional areas for research.

Latencies of the Ankle Musculature

It can be concluded from the results of the study that the peroneus longus and peroneus brevis have shorter latencies using the current methodology than when using the tilt platform. The author believes that the current methodology resembles the mechanism of a lateral ankle sprain more closely than the tilt platform/trap door because 1) the

current study used a dynamic task in which the participants were moving; 2) many ankle sprains occur when landing from a jump; and 3) the inversion angular velocity in the current study was greater than that used in the tilt platform/trap door studies and closer to actual values during a lateral ankle sprain. Future research should continue to use this methodology to investigate the mechanisms and outcome of ankle sprain mechanics. The electromechanical delay of the peroneus longus and peroneus brevis should also be measured using the outersole with fulcrum to determine how this dynamic activity affects the amount of time it takes to develop force in the main evertors of the ankle.

While this author believes that there are some clear advantages to this methodology over the tilt platform/trap door methods, some improvement should be considered. First, the height of the box should be increased. In the present study, 27 cm was chosen for the safety of the participants. Yet, when a person lands from a jump, it is typically from a height greater than 27 cm. Since no participants sustained an injury from this height, future research should increase this height by 5-10 cm. In addition, the fulcrum attached to the bottom of the shoe should yield an inversion angle greater than 25^{o}. Previous work on the tilt platform/trap door commonly caused 30-35° of inversion, so increasing the inversion angle should not pose any increased risk to the participants. While the current study did not find a difference in the latencies of the peroneus longus and brevis of the dominant/injured ankles between the different injury groups, other research has found a difference. Future research should use the above mentioned adaptations to more closely replicate a lateral ankle sprain, and participants that have recently suffered a lateral or high ankle sprain should be included in the participant population. Participants that have sustained an ankle sprain within the past 6 months but

are functionally able to withstand the testing should be investigated to determine if there is an acute difference in the latency of the peroneus longus and peroneus brevis soon after injury.

There has been limited research comparing the latencies of the peroneus longus and peroneus brevis of the non-dominant leg of healthy participants to the non-injured leg of participants that have sustained an ankle sprain. The current study found that the non-dominant leg of healthy participants had a significantly shorter latency of the peroneus longus than the non-injured leg of participants that had sustained a lateral ankle sprain. Although not included in the results of this manuscript, the non-dominant leg of the healthy participants had a significantly shorter latency of the peroneus longus than the dominant leg. Since more ankle sprains occur to the dominant leg (Ashton-Miller et al., 1996), this may be a predictor of a lateral ankle sprain. Future research should focus on a set of athletes over the course of a season and track the number of ankle injuries that occur. The latency of the peroneus longus and brevis of both the dominant and non-dominant leg should be measured before the start of the preseason and at the conclusion of the season to determine: 1) if there is a difference in latency between the two legs in the preseason, (2) if this latency exists, does it pre-dispose the athlete to an ankle sprain? 3) does the latency change over the course of an athletic season? And 4) is there a threshold value for the latency of the peroneus longus and peroneus brevis above which a person is pre-disposes to an ankle sprain?

Startle Response

The present study found an interaction between the startle event and injury group for the analysis of the dominant/injured ankles and although non-significant, a trend in the data with a large effect size for the non-dominant/non-injured ankles for the startle event. For both the dominant/injured ankles and the non-dominant/non-injured ankles, the ratio of evertor/invertor activity was greater than 1.0, indicating more activity in the evertor muscles group or decreased activity in the invertor group, which helps protect against a lateral ankle sprain. Unfortunately, this finding does not support the theory of equal activity in both the agonist and antagonist muscle groups the first time the participants are exposed to an inversion perturbation, suggesting that there is not a true startle response as defined by the whiplash research (Siegmund et al., 2008) at the ankle. For the non-dominant/non-injured ankles, this ratio increased across all three time events, while for the dominant/injured ankles, this ratio increased for the second startle event but decreased for the third startle event, although it was still greater than the first startle event. These results support the theory that the ankle musculature may be trained to provide a larger response by the evertor muscle group and a smaller response by the invertor muscle group when the ankle is forced into inversion. Also, the fact that in the current study, the tibialis anterior failed to reach the threshold for activation 11.5 % of the trials the first testing day and this percentage increased to 29.19 % of the trials the second and third testing days indicates that repeated exposure to an inversion perturbation may actually train the invertor muscle group to not activate when the ankle is forced into inversion. Future research should examine this relationship with more participants and across multiple days. A larger, healthy sample with no previous history of an ankle

sprain should be tested, to further determine how this ratio changes in healthy participants before it is applied to people that have sustained an ankle injury. In addition, research should also examine how long this increase in the ratio persists and if this training mechanism may be applied to athletes at high risk for a lateral ankle sprain, people in industrial settings and/or the general population who may have risk factors for a lateral ankle sprain.

Influence of Ankle Taping

It may be concluded from the results of this study that immediately after the application of ankle taping, the latency of the peroneus longus is significantly reduced. There was no difference across the injury groups, so this reduction in latency occurs for injured and non-injured people. These results support the use of ankle taping as a prophylactic mechanism to help prevent lateral ankle sprains. Future research should investigate if this reduction in latency persists after 30-45 minutes of physical activity, for it is known that tape loses its ability to prevent extreme ranges of motion at the ankle, over time (Ashton-Miller et al., 1996; Wilkerson 2002). For example, participants should be tested in the same manner as in the present study, and should then perform 30-45 minutes of standardized activity (taking the same number steps using a pedometer, or jogging on a treadmill) and have both ankles tested again to compare the results of pre-activity to post-activity. Research should also examine how ankle braces affect the latency of the peroneus longus and peroneus brevis using the same methodology and a comparison of the latencies to the values obtained while wearing athletic tape, should be made.

Additional Areas of Future Research

Excessive inversion is the primary mechanism that causes a lateral ankle sprain. However, many times the lateral ankle sprain occurs with a combination of plantar flexion at the talocrural joint and inversion at the subtalar joint. As the amount of plantar flexion increases, the amount of tension on the anterior talofibular ligament (the primary ligament damaged by a lateral ankle sprain) increases as well (Kernozek et al., 2008). First, an outersole with fulcrum should be designed that forces the participants into plantar flexion only to determine how this joint action in isolation affects the latency of the primary dorsiflexors of the ankle (tibialis anterior, extensor digitorum longus, and extensor hallicus longus). After these latencies have been measured, a fulcrum should be designed that causes inversion and plantar flexion (in combination) at the ankle to determine how this mechanism affects the latency of the ankle musculature and how the latencies obtained using this methodology compare to research using the tilt platform/trap door methodology that also cause a combination of inversion and plantar flexion.

The extensor digitorum longus (EDL) is a muscle that has received very little attention in previous research. This muscle is an evertor of the subtalar joint and dorsiflexor of the talocrural joint, in addition to its role of extension of the second through fifth toes (Floyd, 2007). If the ankle is forced into inversion or a combination of inversion and plantar flexion, the EDL is an additional muscle that could help prevent the excessive range of motion that may lead to a lateral ankle sprain. Only one previous study has examined the latency of the EDL. Using a tilt platform, it was found that there was a latency of 59.81 ms in healthy participants and 61.53 ms in participants that had

previously suffered an ankle injury (Mitchell et al., 2008). These values are similar to the latencies of the peroneus longus and peroneus brevis measured in previous research using the tilt platform/trap door. However, based on the results of the current study, the author believes the latency of the EDL would be shorter when using a dynamic task. Future research should examine the electromechanical delay, and the latency of the EDL using a more dynamic task such as the one used in the present study and measure this latency among different injury groups.

Increasing the strength and proprioception of the EDL may be vital to preventing lateral ankle sprains. While the peroneus longus and peroneus brevis are the main evertors of the ankle, and thus provide the dynamic defense against a lateral ankle sprain (Konradsen et al., 1997), they are also plantar flexors of the ankle. If these muscles are activated to prevent excessive inversion at the subtalar joint, they will also contribute to a plantar flexion moment at the talocrural joint. If the ankle is forced into inversion and plantar flexion, the peroneus longus and brevis would help protect against excessive inversion but would actually increase the plantar flexion moment. This same theory can be applied to the tibialis anterior. If the ankle is forced into excessive plantar flexion and inversion, then the tibialis anterior would provide an eccentric muscle action to provide a dorsiflexion moment and help prevent excessive plantar flexion at the talocrural joint. However, since the tibialis anterior is also an invertor of the subtalar joint, it would increase the inversion moment if it is activated to help control the excessive plantar flexion at the talocrural joint. The EDL would dynamically protect the ankle against both excessive inversion and plantar flexion. Therefore, by increasing the activity of the EDL, the dynamic defense mechanism against a lateral ankle sprain would be increased. Future

research should examine the activity of the EDL during a dynamic forced inversion and plantar flexion moment. Rehabilitation after a lateral ankle sprain should be applied to both the injured and non-injured ankles and also focus on both concentric and eccentric strengthening of the ankle musculature, with a specific emphasis placed on eccentric strengthening of the peroneus longs, peroneus brevis, and EDL, as these are the primary muscles whose eccentric muscle actions may prevent a lateral ankle sprain.

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APPENDIX A- Pictures of Testing Protocol

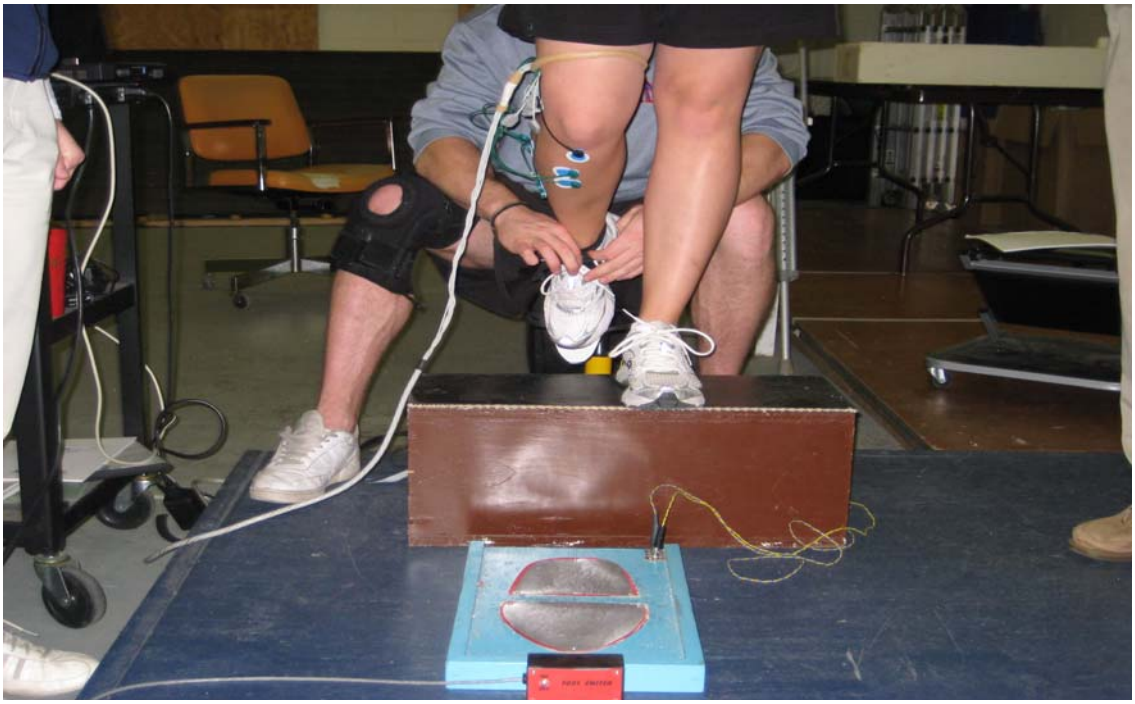


Figure 18- Placement of Outersole onto shoe, frontal view.

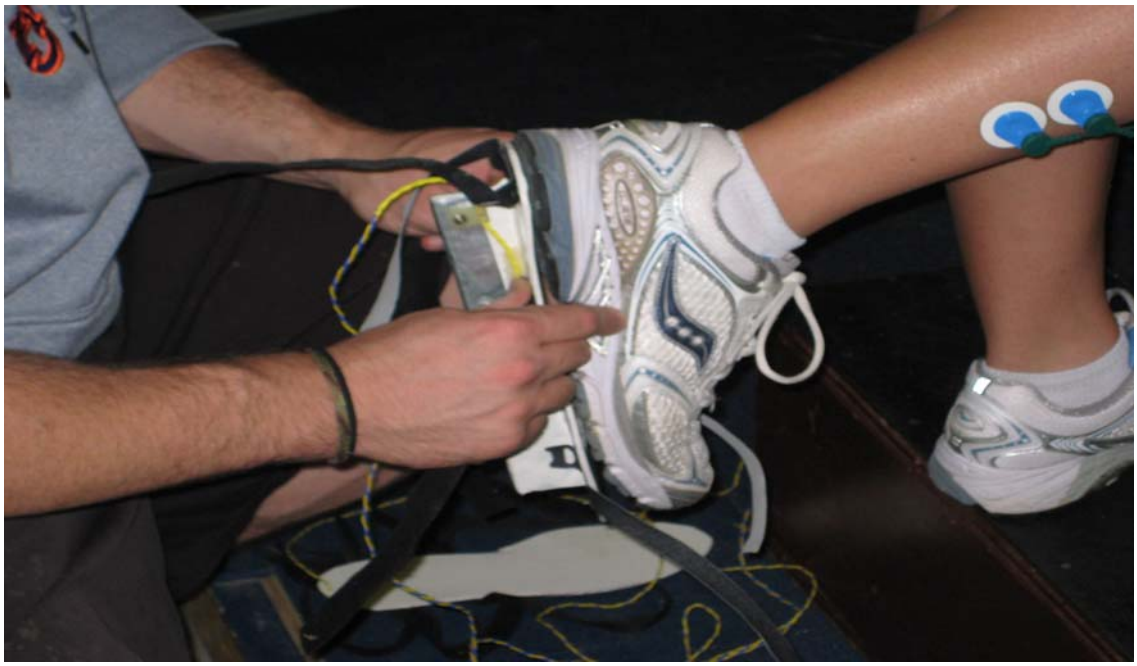


Figure 19- Placement of outersole onto shoe, side view

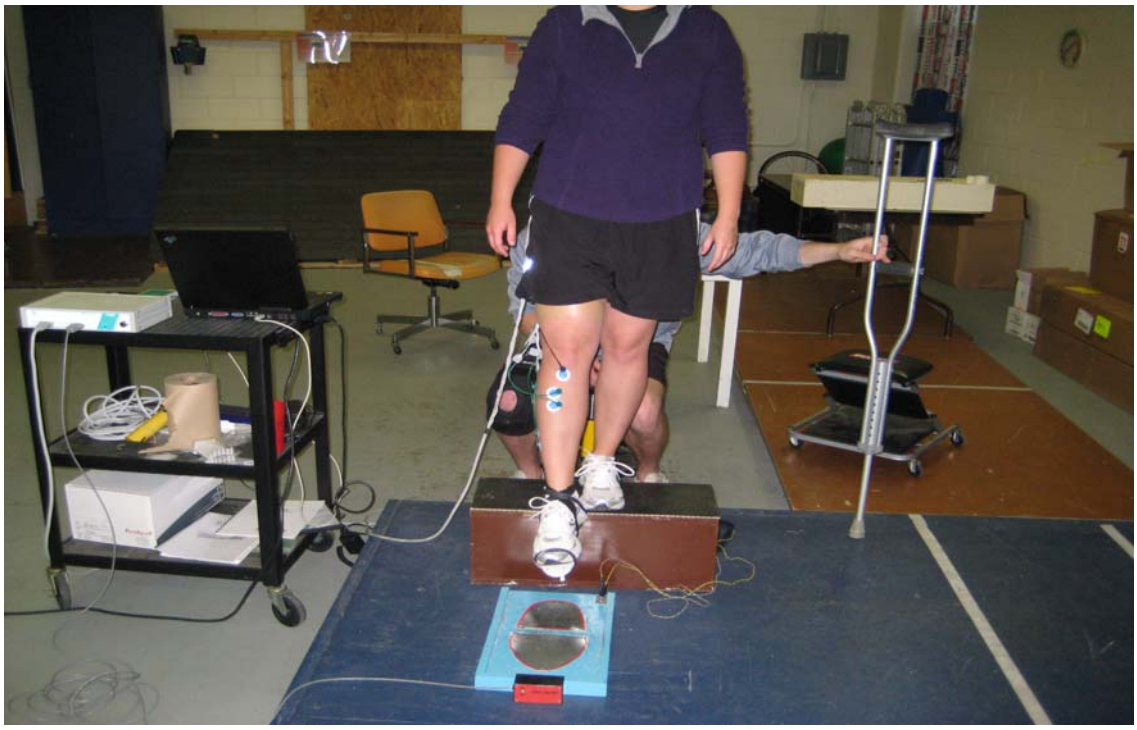


Figure 20- Participant waiting to perform drop landing with outersole and fulcrum, front view

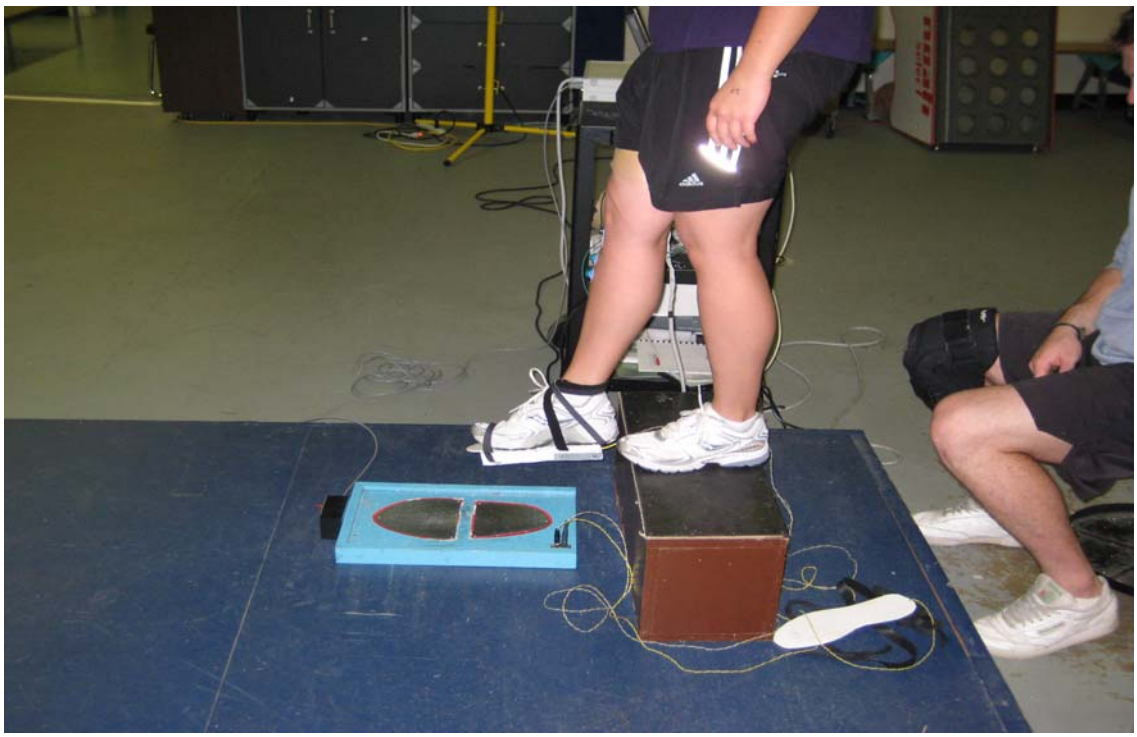


Figure 21- Participant waiting to perform drop landing with outersole and fulcrum, side view.

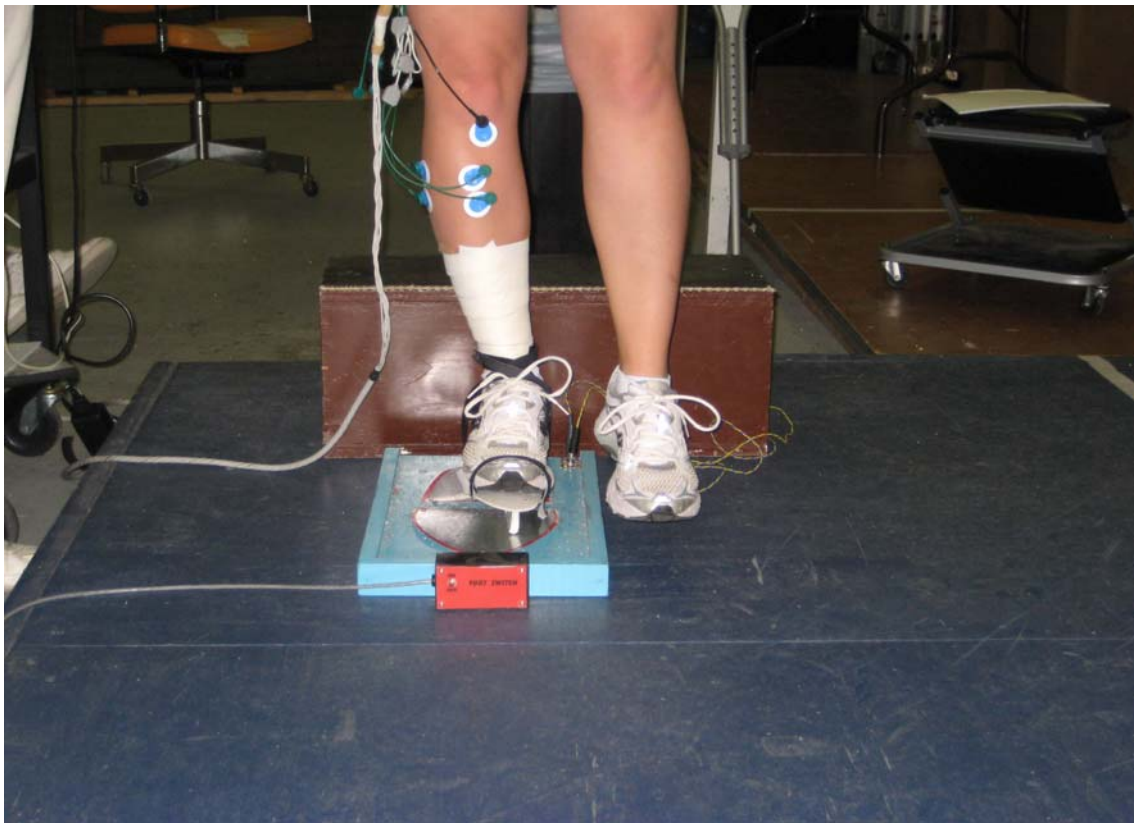


Figure 22- Participant landing on the fulcrum.

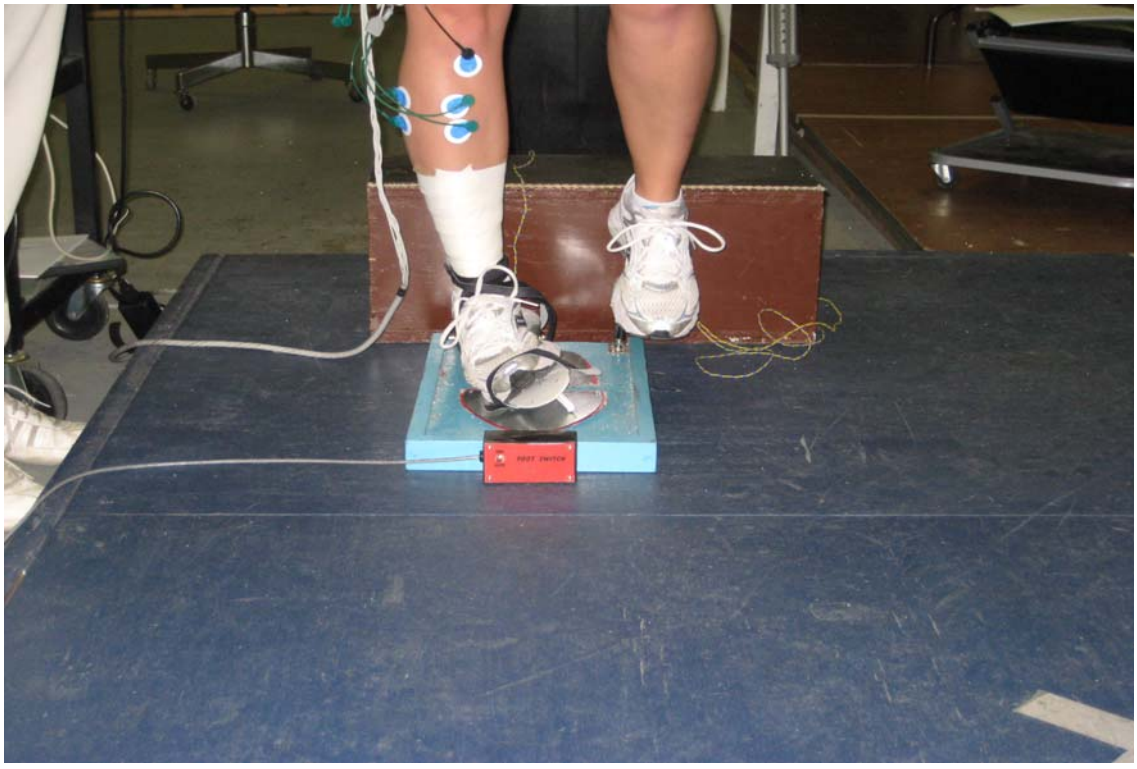


Figure 23- Participant going through full inversion ROM (failure).

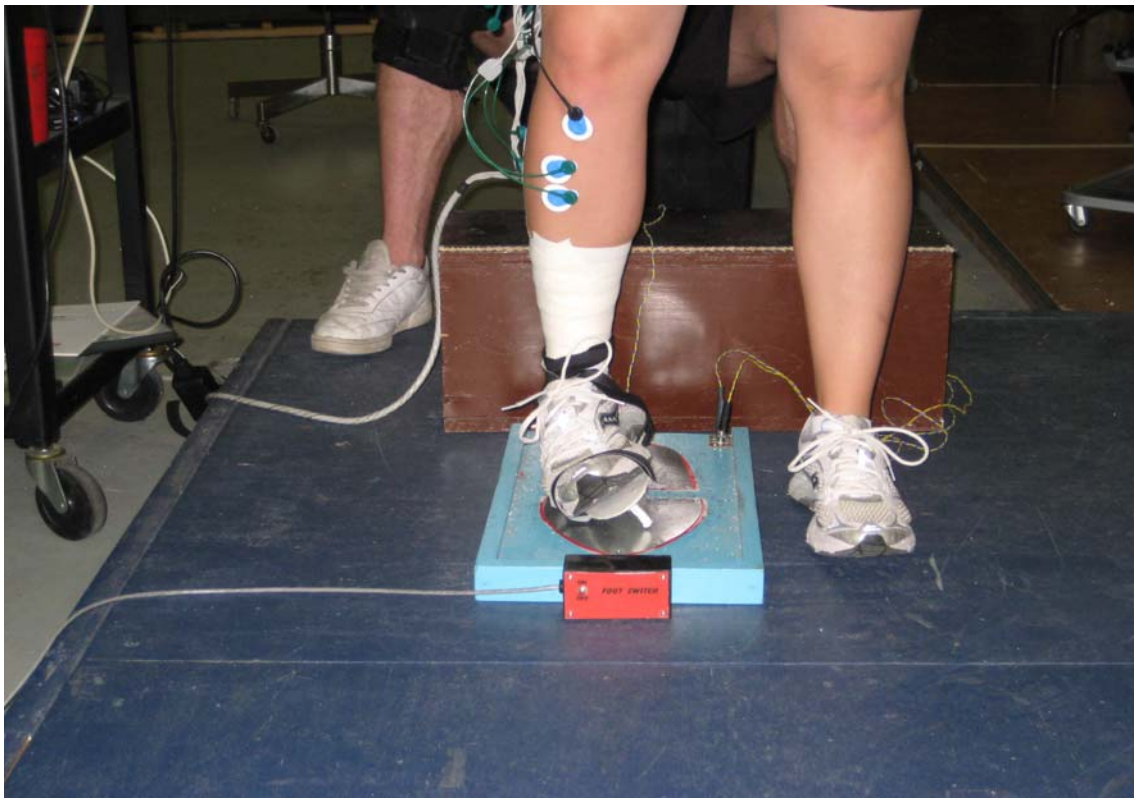


Figure 24- Participant completing the testing protocol with fulcrum, both feet are now on the ground

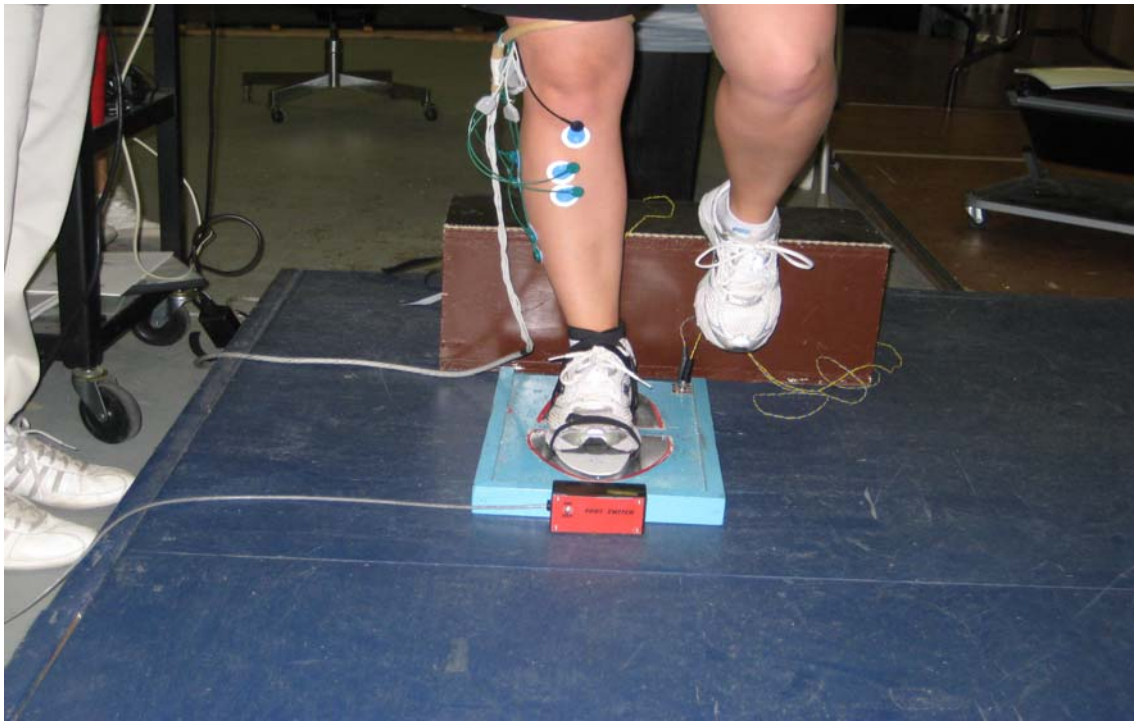


Figure 25- Participant landing on the flat outersole.

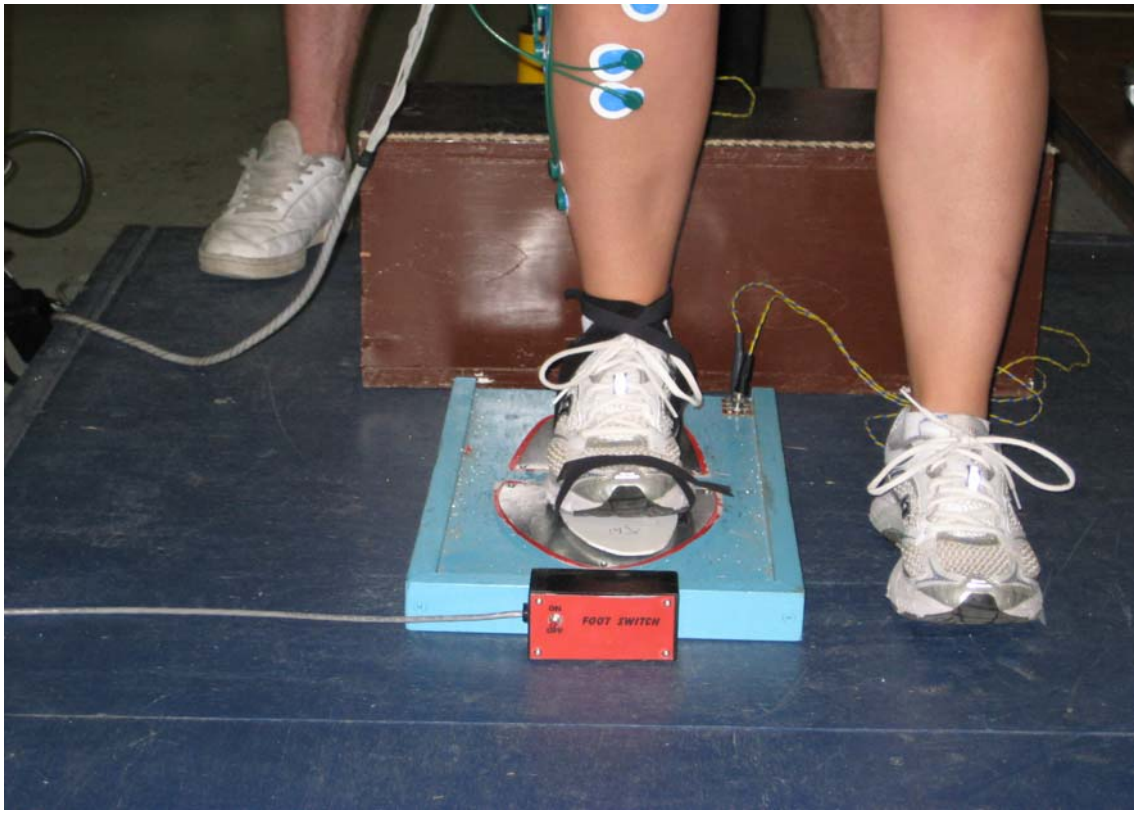


Figure 26- Participant completing the protocol with flat outersole, both feet are now on the ground.

APPENDIX B

Electromyography Signals from Testing with Outersole and Fulcrum

KEY

Channel 1- Peroneus Longus
Channel 2- Peroneus Brevis
Channel 3- Tibialis Anterior
Channel 13- Contact of Fulcrum with Landing Area
Channel 14- Contact of Lateral Border of Outersole with Landing Area

APPENDIX C

Electromyography Signals from Testing with Flat Outersole

KEY

Channel 1- Peroneus Longus
Channel 2- Peroneus Brevis
Channel 3- Tibialis Anterior
Channel 13- Contact of Flat Outersole with Landing Area

APPENDIX D

ANOVA TABLES FOR RESULTS OF DAY ONE TESTING

Analysis	Source	df	SS	MS	*F*	*p*	η^2	Power
Startle Dominant/ Injured	Startle*Injury	2	5.52	2.76	3.28	.049	.150	.587
	Startle	1	.577	.577	.685	.413	.018	.127
	Injury	2	11.25	5.62	1.44	.249	.072	.289
	Error (startle)	37	31.17	.842				
Startle Non-Dominant/Non-Injured	Startle* Injury	2	3.47	1.73	2.56	.091	.121	.479
	Startle	1	2.34	2.34	3.45	.071	.085	.440
	Injury	2	.694	.347	.144	.867	.008	.071
	Error (startle)	37	25.09	.678				
PL Latency Dominant/Injured	Injury	2	2.95	1.47	.010	.990	.001	.051
	Error (injury)	37	5344.14	144.44				
PB Latency Dominant/Injured	Injury	2	209.28	104.64	.363	.698	.019	.104
	Error (injury)	37	10668.14	288.33				
TA Latency Dominant/Injured	Injury	2	549.92	274.96	.948	.397	.049	.202
	Error (injury)	37	10732.71	290.07				
PL Latency Non-Dominant/Non-Injured Injured	Injury	2	1125.78	562.89	4.62	.016	.200	.746
	Error (injury)	37	4508.99	121.87				
PB Latency/Non-Dominant/Non-Injured	Injury	2	130.49	65.24	.249	.781	.013	.086
	Error (injury)	37	9711.76	262.48				
TA Latency/Non-Dominant/Non-Injured	Injury	2	737.60	368.00	.732	.488	.038	.165
	Error (injury)	37	18644.44	503.90				
TTF Dominant/Injured versus Non-Dominant/Non Injured	Ankle*Injury	2	122.16	61.08	.238	.789	.013	.085
	Ankle	1	645.57	645.57	2.52	.121	.064	.340
	Injury	2	3178.25	1589.17	1.73	.191	.086	.340
	Error (ankle)	37	9477.14	256.14				

APPENDIX E

ANOVA TABLES FOR RESULTS OF DAYS TWO AND THREE OF TESTING

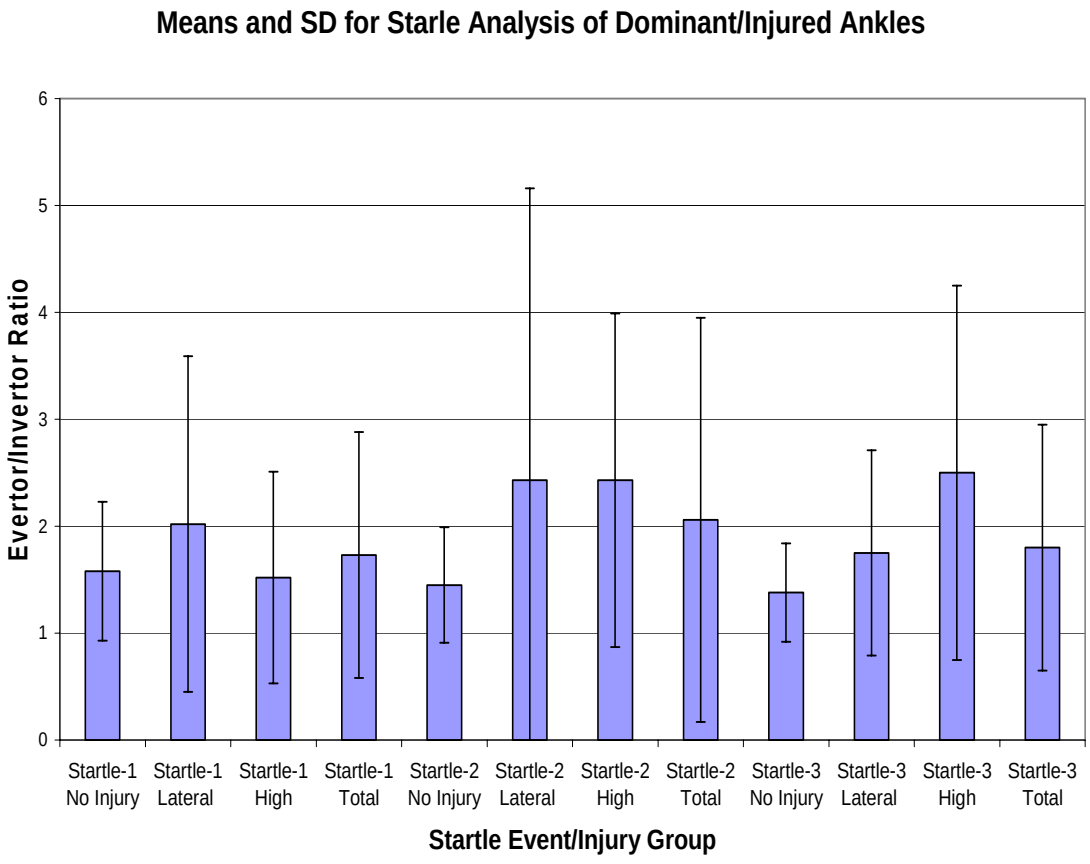
Analysis	Source	df	SS	MS	*F*	*p*	η^2	Power
PL Latency Ankle Support Dominant/ Injured	Support*Injury	2	296.87	148.44	1.25	.299	.068	.253
	Support	1	1965.18	1965.18	16.54	<.001	.327	.977
	Injury	2	16.84	8.42	.033	.967	.002	.055
	Error (support)	34	4038.07	118.77				
PL Latency Ankle Support Non-Dominant/Non-Injured	Support* Injury	2	57.97	28.93	.226	.799	.013	.082
	Support	1	1118.82	1118.82	8.72	.006	.204	.818
	Injury	2	596.43	298.22	1.36	.272	.074	.272
	Error (support)	34	4364.53	128.37				
TA Latency Ankle Support Dominant/Injured	Support*Injury	2	356.77	178.34	1.07	.355	.059	.222
	Support	1	119.90	119.90	.718	.403	.021	.131
	Injury	2	2115.54	1057.77	2.51	.096	.129	.469
	Error (support)	34	5675.32	166.92				
TA Latency Ankle Support Non-Dominant/Non-Injured	Support*Injury	2	1307.26	653.63	1.50	.238	.081	.297
	Support	1	45.14	45.14	.103	.750	.003	.061
	Injury	2	218.21	109.10	.149	.862	.009	.071
	Error (support)	34	14829.01	436.14				
TTF Ankle Support Dominant/Injured	Support*Injury	2	512.63	256.31	.958	.394	.053	.202
	Support	1	1042.43	1042.43	3.90	.057	.103	.483
	Injury	2	2973.14	1486.57	1.075	.353	.059	.223
	Error (support)	34	9093.68	267.46				
TTF Ankle Support Non-Dominant/Non-Injured	Support*Injury	2	46.42	23.21	.098	.907	.006	.064
	Support	1	2.26	2.26	.010	.923	.000	.051
	Injury	2	107.30	53.65	.039	.961	.002	.055
	Error (support)	34	8067.55	237.28				

APPENDIX F

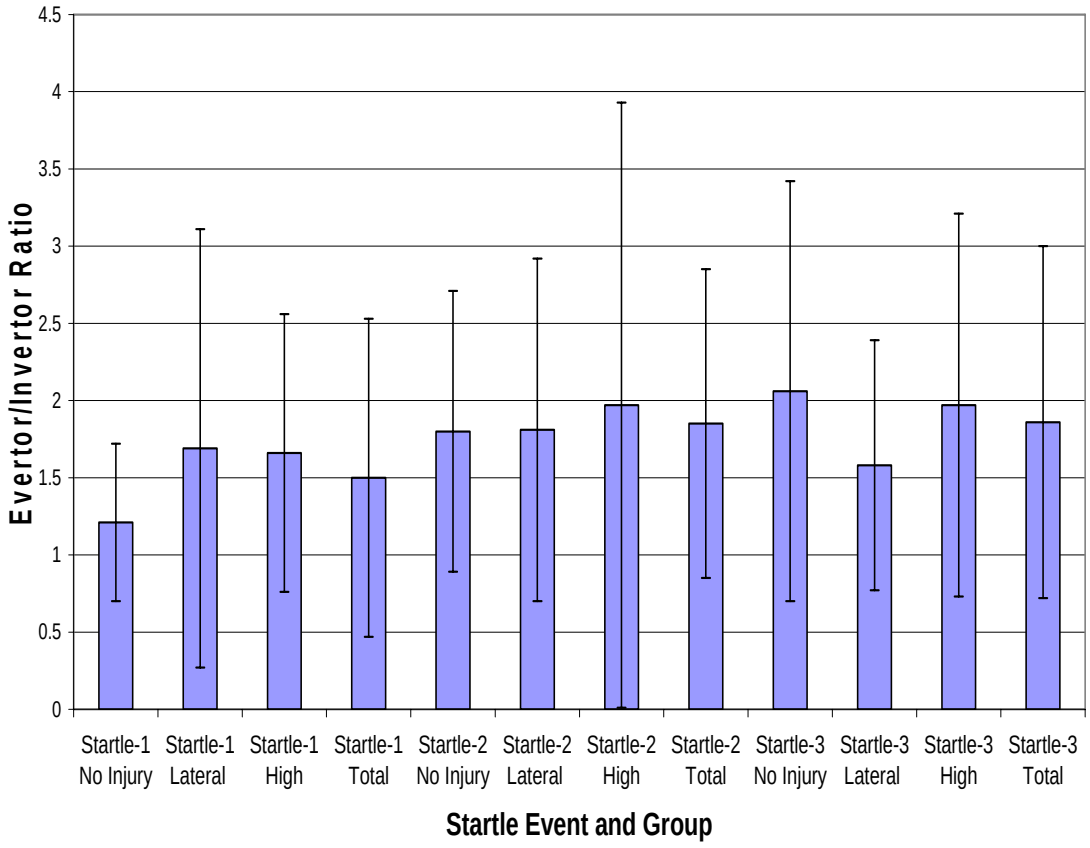
Graphs from Day One of Testing

KEY FOR GRAPHS

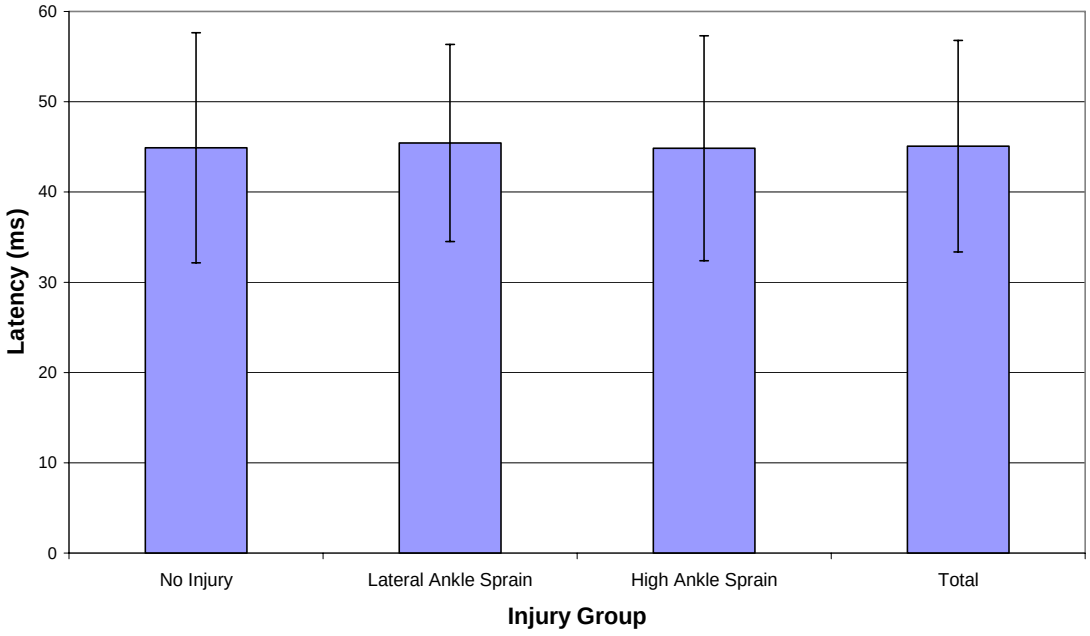
BLUE- Mean
Error Bar-Standard Deviation



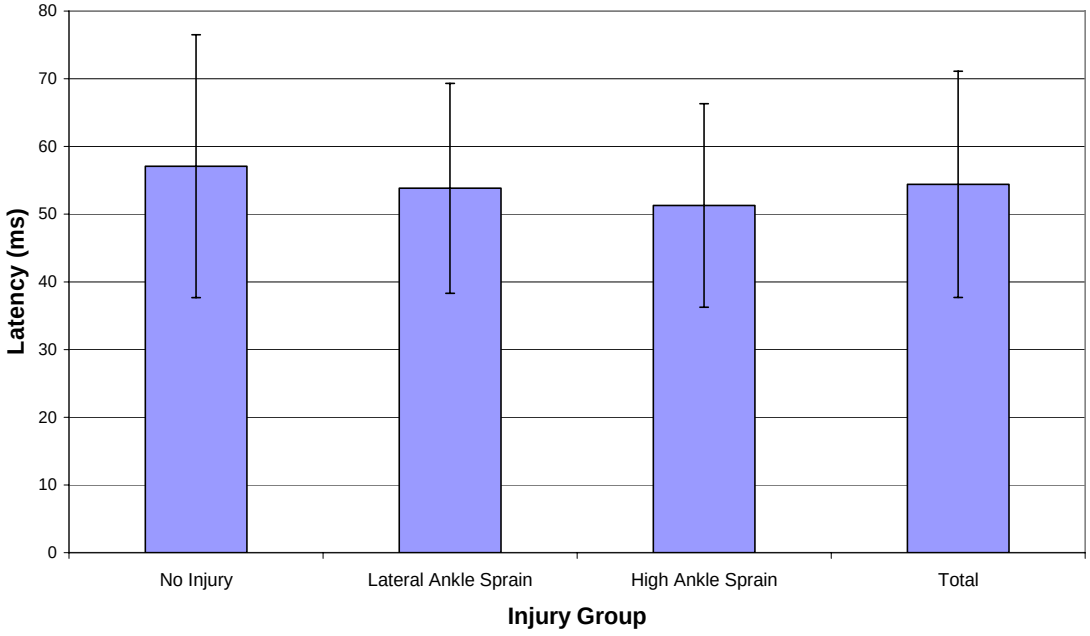
Means and SD for Startle Analysis of Non-Dominant/Non-Injured Ankles



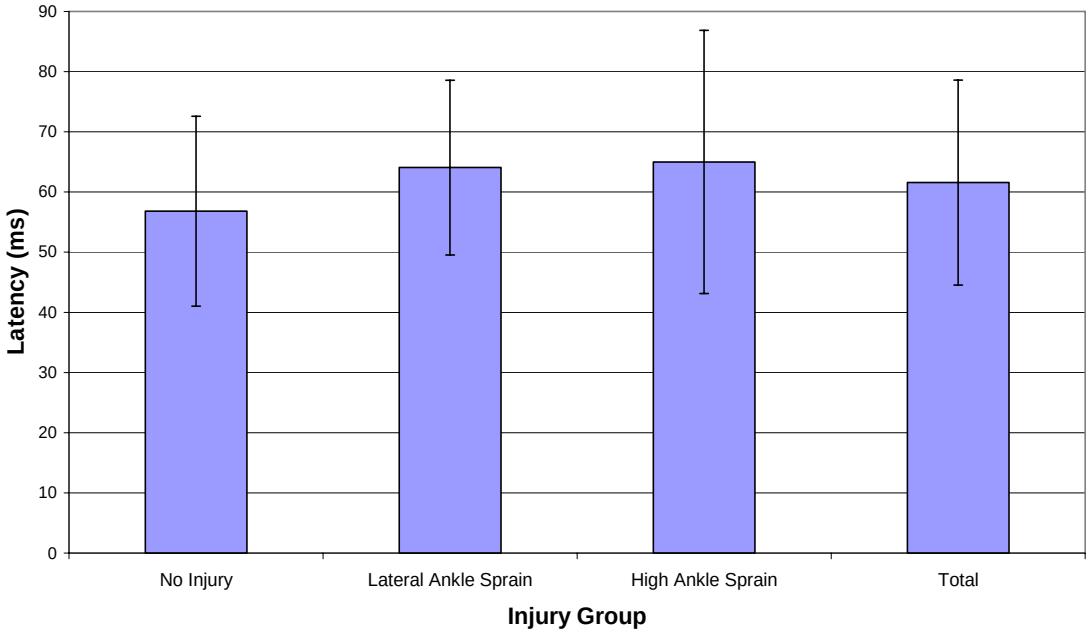
Means and SD for Peroneus Longus Latency of the Dominant/Injured Ankles



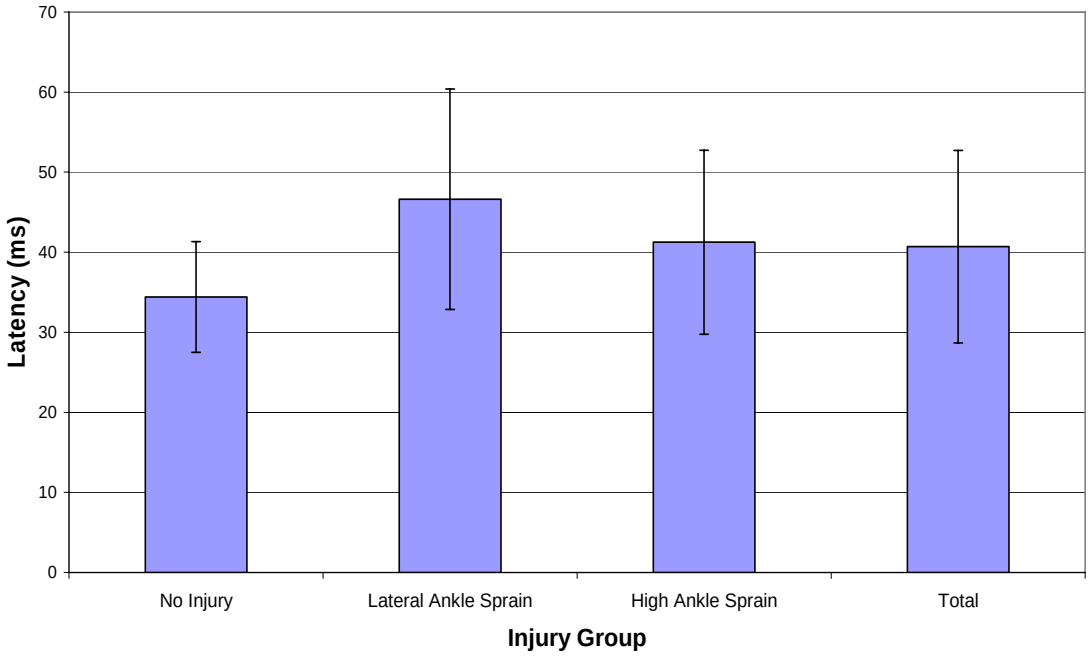
Means and SD for Peroneus Brevis Latency of the Dominant/Injured Ankles



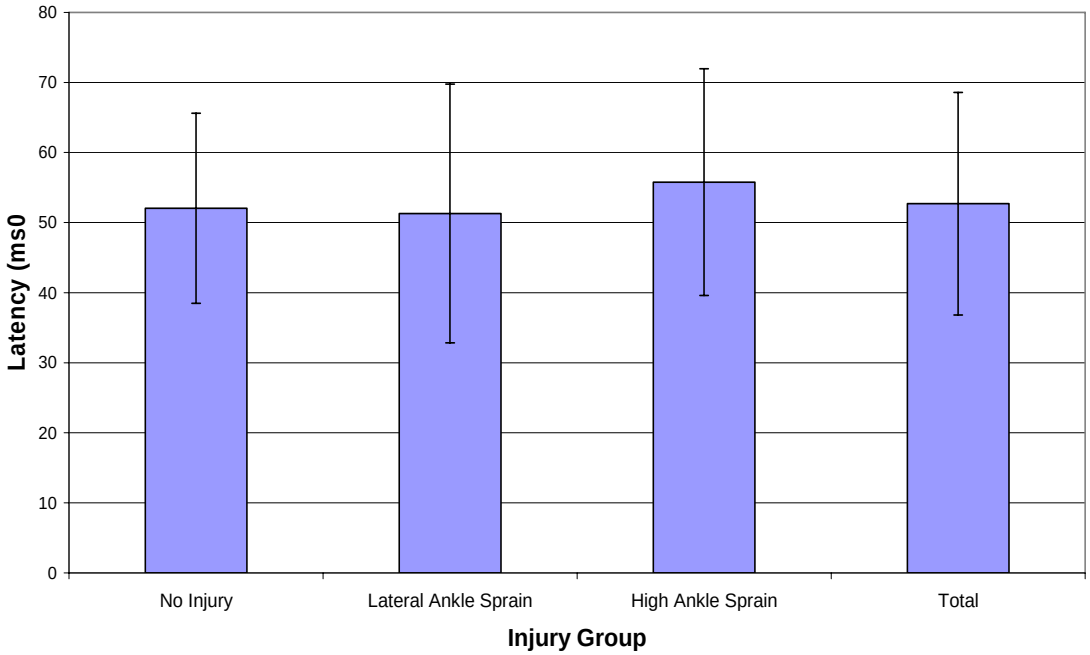
Means and SD for Tibialis Anterior Latency of the Dominant/Injured Ankles



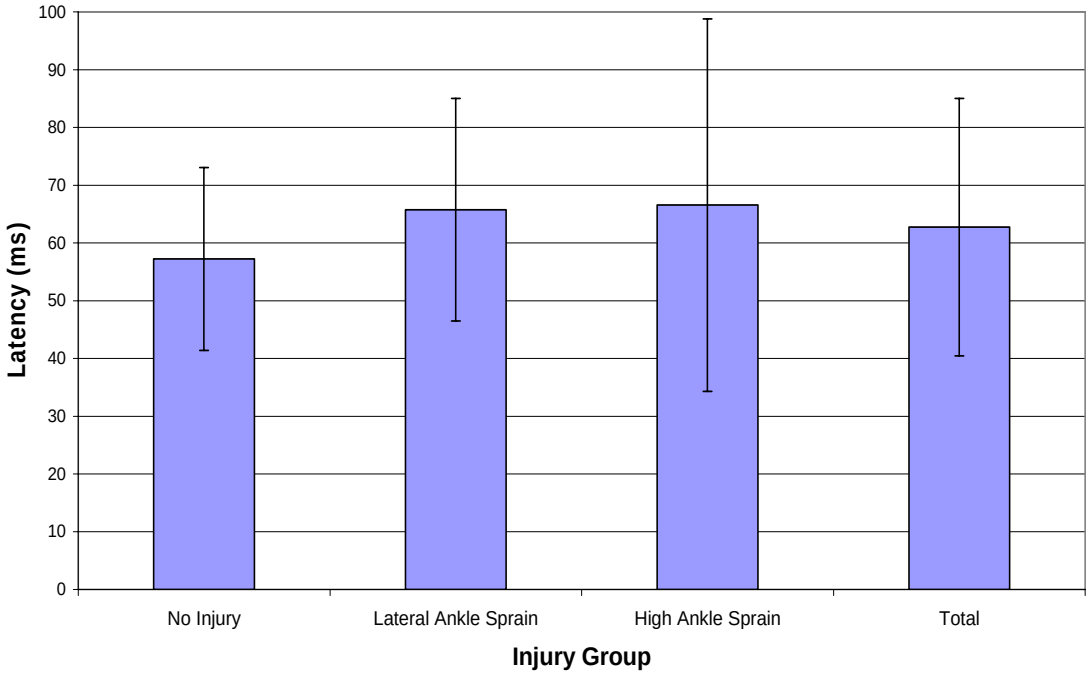
Means and SD for Peroneus Longus Latency of the Non-Dominant/Non-Injured Ankles



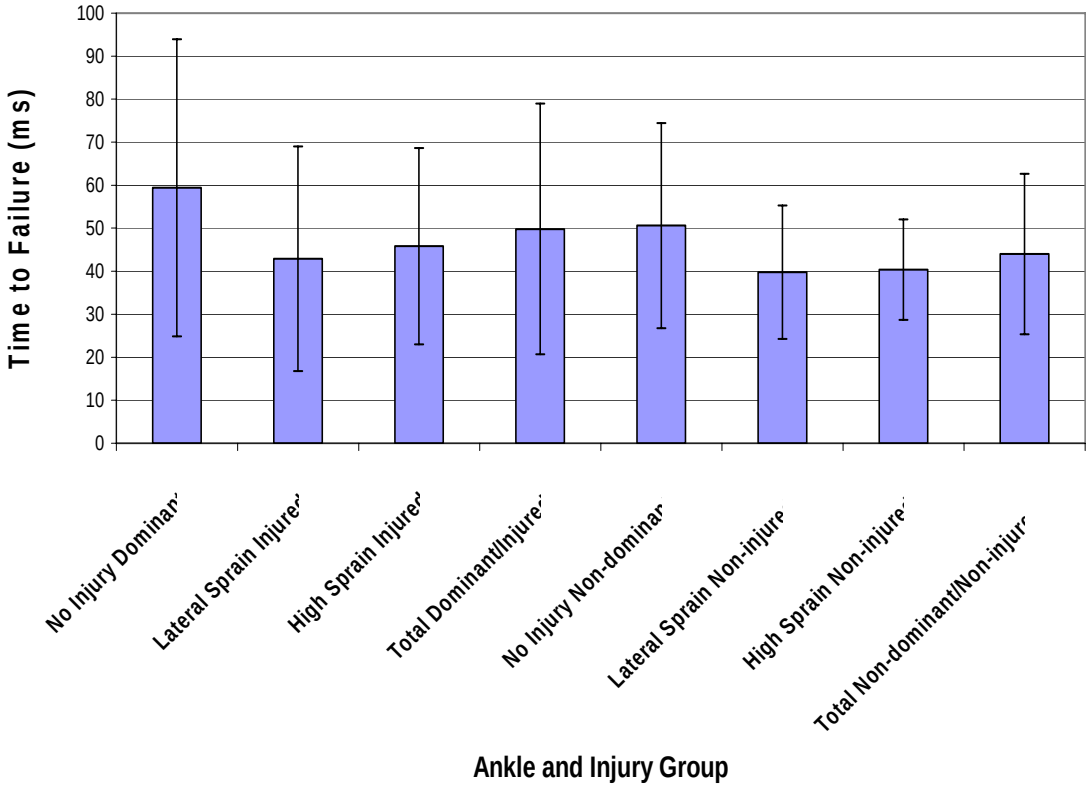
Means and SD for Peroneus Brevis Latency of the Non- Dominant/Non-Injured Ankles



Means and SD for Tibialis Anterior Latency of the Non- Dominant/Non-Injured Ankles



Means and SD for Time to Failure of Dominant/Injured Ankles and Non-Dominant/Non-Injured Ankles

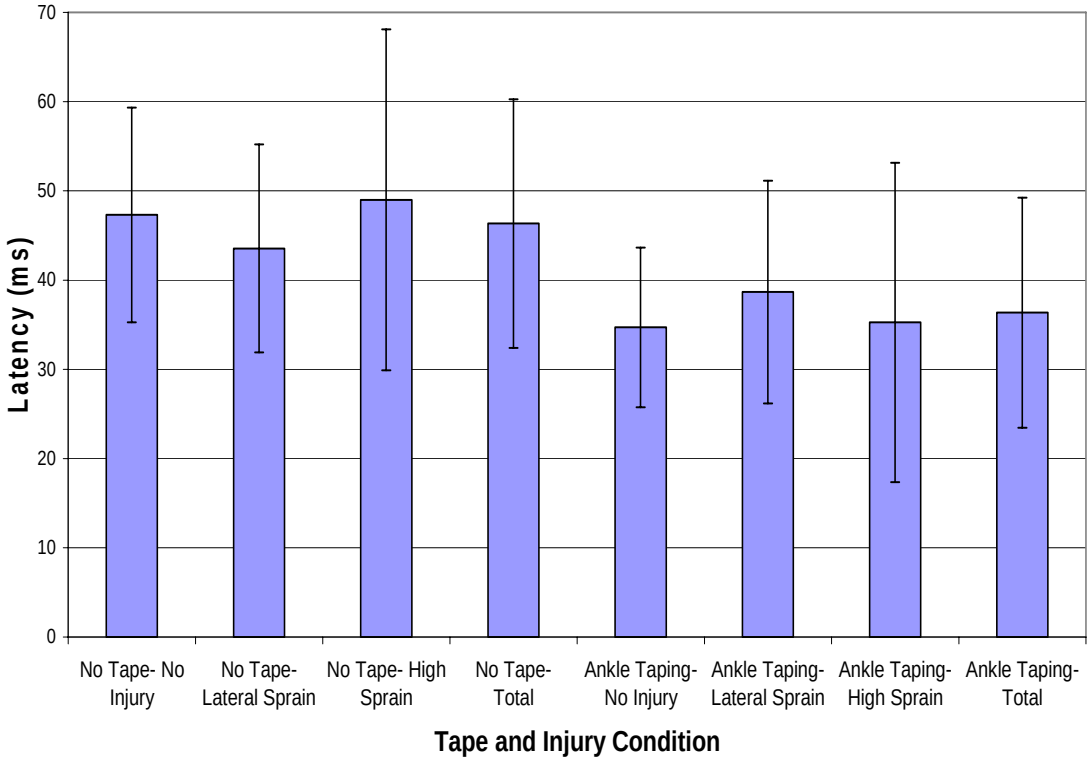


APPENDIX G- Graphs From Days Two and Three of Testing

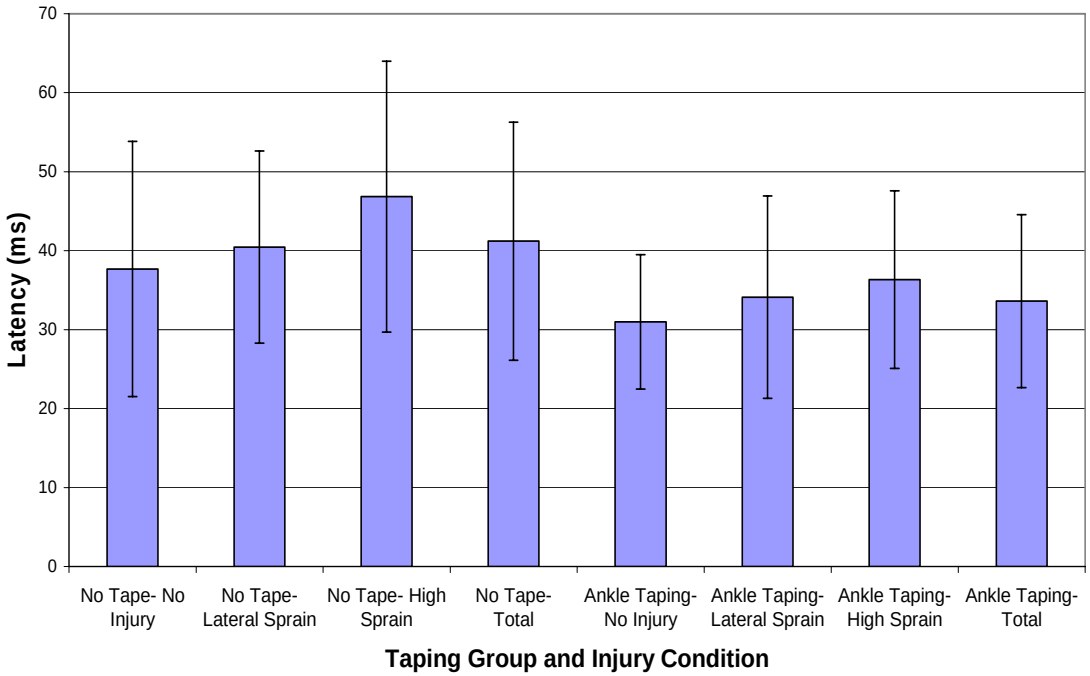
KEY FOR GRAPHS

BLUE- Mean
Error Bar- Standard Deviation

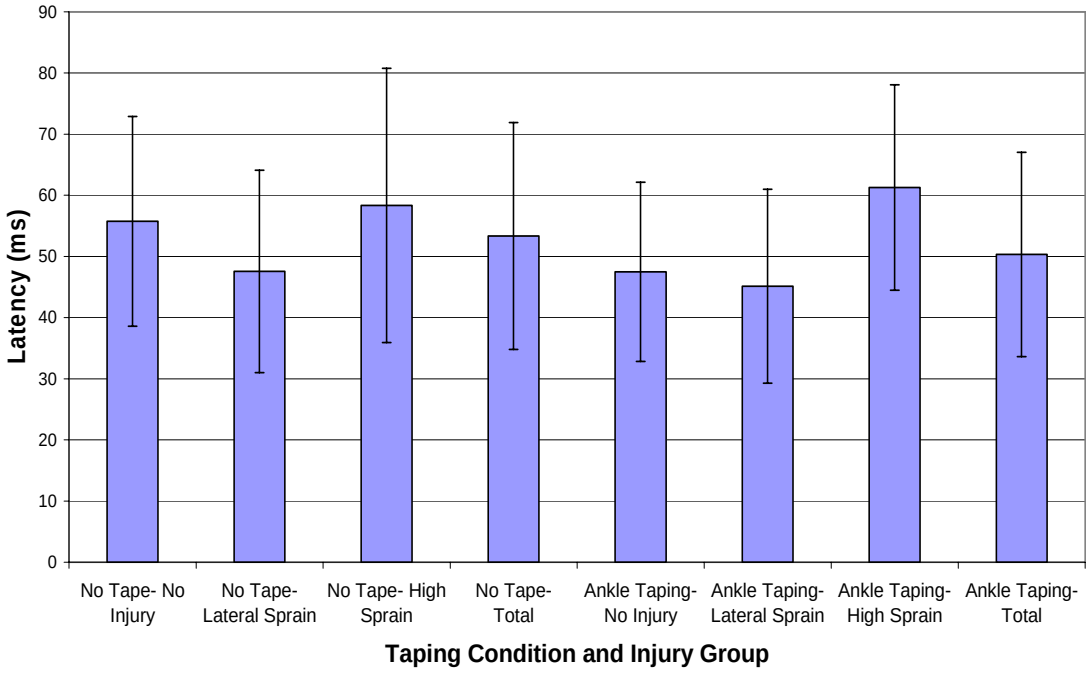
Means and SD for Peroneus Longus Latency of the Dominant/Injured Ankles with and without Ankle Taping



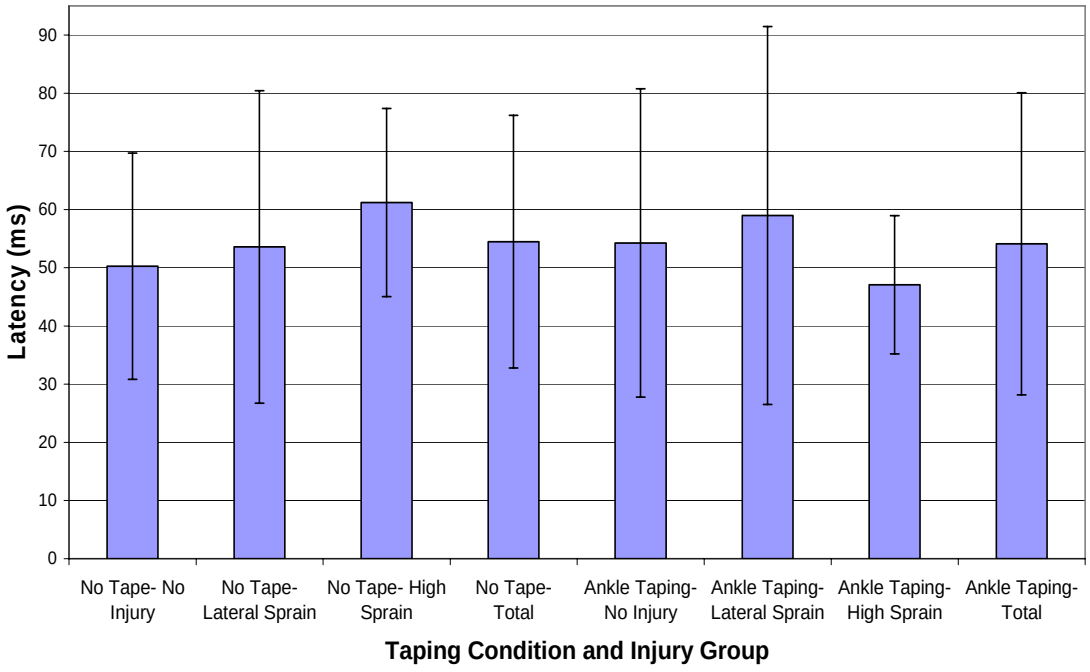
Means and SD for Peroneus Longus Latency of the Non-Dominant/Non-Injured Ankles with and without Ankle Taping



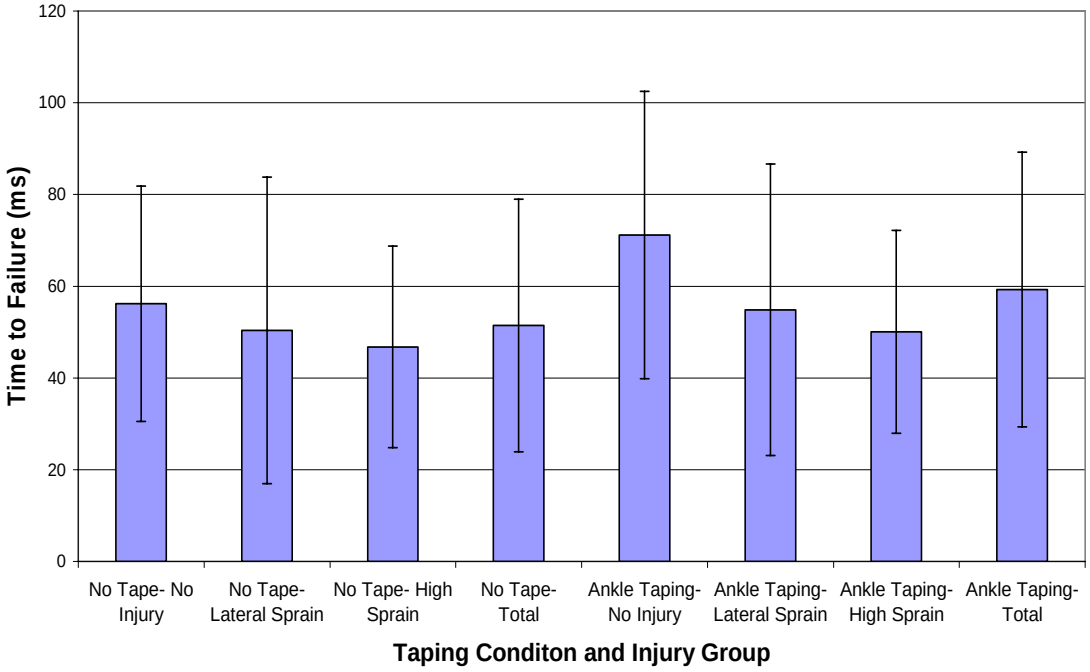
Means and SD for Tibialis Anterior Latency of the Dominant/Injured Ankles with and without Ankle Taping

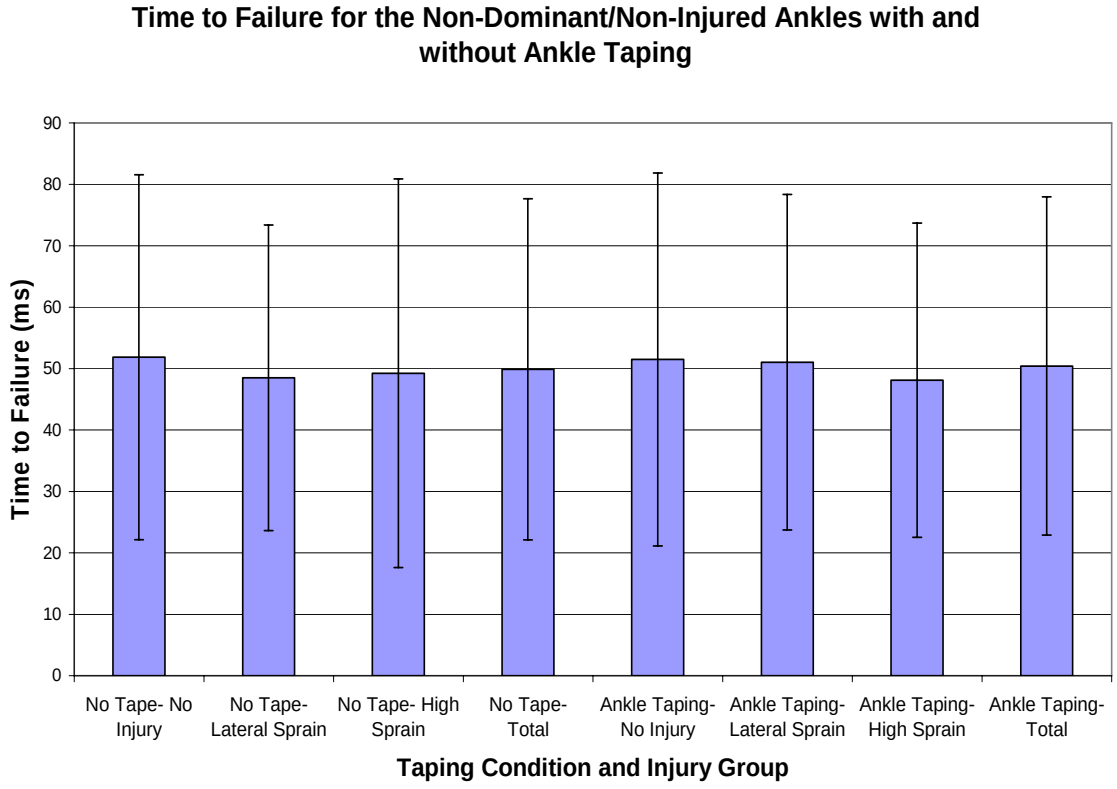


Means and SD for Tibialis Anterior Latency of the Non-Dominant/Non-Injured Ankles with and without Ankle Taping



Time to Failure for the Dominant/Injured Ankles with and without Ankle Taping




Time to Failure for the Non-Dominant/Non-Injured Ankles with and without Ankle Taping
Time to Failure (ms)
90
80
70
60
50
40
30
20
10
0
No Tape- No Injury
No Tape- Lateral Sprain
No Tape- High Sprain
No Tape- Total
Ankle Taping- No Injury
Ankle Taping- Lateral Sprain
Ankle Taping- High Sprain
Ankle Taping- Total
Taping Condition and Injury Group

APPENENDIX H

Institutionally Approved Informed Consent Document

INFORMED CONSENT
for a Research Study entitled
Effects of Inversion Perturbation After Drop Landing on the Latency of the Ankle Musculature

You are invited to participate in a research study to determine the effects of an inversion perturbation after a drop landing on the latency of the ankle musculature. This study is being conducted by Adam Knight, under the supervision of Dr. Wendi Weimar, Associate Professor of Biomechanics, in the Auburn University Department of Kinesiology. You were selected as a potential participant because your health condition and mobility might, through the pre-screening health questionnaire to follow, permit you to perform the test safely and successfully, you are age 19 and older, and you are a student in the Department of Kinesiology. The results of the study will be used to investigate how long it takes the ankle muscles to turn on when the ankle rolls after a drop landing, how ankle taping effects this response, and if the startle response is a part of the exaggerated first response to the inversion or plantar flexion perturbation. The purpose of this study is to investigate how long it takes the ankle muscles to turn on when someone rolls his or her ankle, and how ankle taping affects the time it takes the muscles to turn on.

What will be involved if you participate? If you decide to participate in this research study, you will be asked to report for testing on three separate days. The first day will not involve testing with ankle taping, but the second and third day will involve testing with ankle taping. The testing protocol will require you to perform a single leg drop landing off of a 27 cm box. You will have an outersole placed on the bottom of the shoe that will cause you to either invert (roll) or plantar flex (point toes downward) your ankle, but not enough to cause an injury. Testing will be conducted on both legs. Electrodes will be placed over the muscles on your lower leg to measure electrical activity. The second and third testing days, you will also have your ankles taped by a certified athletic trainer and perform the testing protocol. Your total time commitment will be approximately 80 minutes, or 25 minutes each day, with a slightly longer time commitment the first day to fill out the forms.

Are there any risks of discomforts? The risks associated with participating in this study include the possibility that you may sustain an ankle injury by performing the

_____
Participant's Initials

drop landing. However, the range of motion for this project is well below the amount required to sustain an injury to the ankle ligaments. Furthermore, spotters will be present during all testing in the case that there is a problem while performing the drop landing. Also, the possibility exists that you may have an allergic reaction to the athletic tape or the electrodes used to record muscle activity. In addition, you will be encouraged to end the test if you feel that you are about to unsure about any procedure or you feel that you may injure your ankle. Last, a Certified Athletic Trainer will be present at all testing sessions. If medical treatment is necessary due to an injury, you are responsible for any and all medical costs.

Are there any benefits to yourself or others? If you participate in this study, you can expect to learn a quantitative measure of the latency of the ankle musculature during an inversion or plantar flexion perturbation during a drop landing, and if the startle response if part of this first exaggerated response to an ankle perturbation. I can not promise you will receive any of the benefits described.

Will you receive compensation for participating? To thank you for your time, you will be given an extra credit voucher worth 10% on a quiz given by participating instructors in the Department of Kinesiology or Physical Education classes.

If you change your mind about participating, you can withdraw at any time during the study. Your participation is completely voluntary. If you choose to withdraw, your data can be withdrawn as long as it is identifiable. You will be given extra credit based on the amount of time that you put into the study. That is, your extra credit will be prorated. Your decision about whether or not to participate or to stop participating will not jeopardize your future relations with Auburn University, the Department of Kinesiology, or the Sport Biomechanics Laboratory.

Your privacy will be protected. Any information obtained in connection with this study will remain confidential. Information obtained through your participation may be published in a professional journal or presented a professional meeting.

If you have questions about this study, *please ask them now or* contact Adam Knight at knighad@auburn.edu, or Dr. Wendi Weimar at weimawh@auburn.edu. Furthermore, you may reach any of these two individuals by calling the Sport Biomechanics Laboratory at 844-1468. A copy of this document will be given to you to keep.

If you have questions about your rights as a research participant, you may contact the Auburn University Office of Human Subjects Research or the Institutional Review Board by phone (334)-844-5966 or e-mail at hsubjec@auburn.edu or IRBChair@auburn.edu.

HAVING READ THE INFORMATION PROVIDED, YOU MUST DECIDE WHETHER OR NOT YOU WISH TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE INDICATES YOUR WILLINGNESS TO PARTICIPATE.

_____	_____
Participant's signature Date	Investigator obtaining consent Date
_____	_____
Printed Name	Printed Name

APPENDIX I

Preliminary Medical Questionnaire*

Please read each question carefully and answer honestly. If you do not understand the question, please ask the investigator for clarification. Check the appropriate answer.

Participant Number: _____

YES	NO	
_____	_____	1) Are you under the age of 19?
_____	_____	2) Have you ever been told you have an inner ear disorder?
_____	_____	3) Have you ever had lower extremity surgery?
_____	_____	4) Have you ever had an ankle sprain?
_____	_____	5) Do you presently have any lower extremity disorders?
_____	_____	6) Have you ever had an allergic reaction to adhesives?
_____	_____	7) Has your doctor ever said that you have heart trouble?
_____	_____	8) Have you ever had a heart murmur, rheumatic fever or respiratory problems?
_____	_____	9) Has your doctor ever told you that you have a muscle, bone or joint problem such as arthritis that had been aggravated by exercise, or might be made worse by exercise?
_____	_____	10) Have you ever felt faint, dizzy or passed out during or after exercise?
_____	_____	11) Have you ever felt pain, pressure, heaviness or tightness in the chest, neck shoulders or jaws as a result of exercise?
_____	_____	12) Do you have any reason to believe that your participation in this investigative effort may put your health or well being at risk?
_____	_____	13) Are you able to stand on one leg without the use of aids?

_____ _____ 14) Are you currently taking any medication that you think might influence your ability to participant in this study?

15) If you answered yes to question number 4 or 5, please give an approximate date of the injury and brief description of the injury. Do you currently have any problems resulting from that injury?

*Adapted from British Columbia Department of Health and Michigan Heart Association

Signature of subject _____ Date _____