EFFECTS OF FATIGUE & GENDER ON PERONEAL REFLEXES AFTER ANKLE INVERSION

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EFFECTS OF FATIGUE & GENDER ON PERONEAL REFLEXES FOLLOWING SUDDEN ANKLE INVERSION

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(Abstract)

An estimated 23,000 ankle injuries occur every day in the U.S. Ankle sprains account for 85% of all ankle injuries and inversion ankle sprains account for 85% of all ankle sprains. There is growing evidence that suggests gender and fatigue may increase the risk for inversion ankle sprains. Investigating the effects of fatigue and gender on peroneal reflex response after ankle inversion may help explain the differences in sprain rates with fatigue and gender. Therefore, the purpose of this study was to investigate the effects of fatigue and gender on peroneus brevis and peroneus longus reflexes after ankle inversion. A “trap-door” platform was used to elicit peroneal reflexes from sixteen males and fifteen females by suddenly inverting the ankle to 20°. Five unfatigued peroneal reflex measurements were performed before and after a fatigue protocol that attempted to fatigue the ankle evertors over 12 minutes to 75% of the unfatigued MVC torque. Results showed that reflex delay was not affected by fatigue, gender, or their interaction. PL reflex amplitude was not affected by fatigue or gender but was affected by their interaction. Results showed that PL reflex amplitude decreased by 11.3% in males and increased 22.1% in females with fatigue. A secondary analysis attempted to rule out extraneous factors that could have contributed to the differences in reflex response, but no experimental explanations were found. The differences in PL reflex amplitude were attributed to biomechanical, physiological, and anatomical differences between males and females.
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ALL my friends: Thank you for the distractions from my work.

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DEDICATION

TO MOM & DAD
YOU ARE THE REASON FOR ALL MY SUCCESSES

TO LAUREN
YOU ARE THE BEST TWIN A SISTER COULD EVER HAVE
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ANKLE INVERSION

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CHAPTER 1

INTRODUCTION
1.1 Background

In collegiate volleyball and basketball, ankle injuries have accounted for 21%-33% of all injuries over the past 21 years [1]. Numerous other epidemiological studies of volleyball [2, 3], basketball, soccer, and running have reported similar ankle injury rates [4]. According to NCAA reports of specific injuries, the ankle is among the top three most injured joints for almost every sport played at the collegiate level [1]. For the general U.S. population including collegiate athletics, it has been estimated that 23,000 ankle injuries occur every day in the U.S. [5] amounting to over 8 million ankle injuries a year. Of these ankle injuries, approximately 75% to 85% are ankle sprains [6, 7]. Studies have shown that the amount of time missed because of ankle sprains averages less than one month suggesting that it is not the severity of ankle sprains but the incidence that is the real problem [8]. According to statistics from 1984, the diagnosis and rehabilitation of an ankle sprain is estimated to cost anywhere from $300-$900 [9], which comes to over $2 billion annually [9, 10]. This is roughly the money spent on coronary bypass surgical grafting in the U.S. [9].

1.2 Types and Grades of Ankle Sprains

Clinicians use three different grades to diagnosis the severity of an ankle sprain. A grade I ankle sprain involves the stretching of ligaments and in the extreme cases some mild tearing of the ligaments. A grade II ankle sprain involves moderate tearing of the ligaments while a grade III is the complete rupture of a ligament [6]. Of the number of ankle sprains, approximately 85% are lateral or inversion ankle sprains [7, 11], 10% are syndesmotic ankle sprains, and 5% are medial or eversion ankle sprains [11]. Inversion ankle sprains occur when the foot becomes hyper-inverted while eversion ankle sprains occur when the foot becomes hyper-everted. Syndesmotic ankle sprains involve either a forced dorsiflexion or a forced external rotation of the foot. The focus of the present paper will be on inversion ankle sprains because they are the most prevalent.
1.3 Characteristics of the Ankle

1.3.1 Ankle Anatomy

There are two main groups of ligaments that can be injured by an ankle sprain. The lateral stabilizing ligaments are the most often injured ligaments in the ankle [6] and include the anterior talofibular ligament (ATF), the calcaneofibular ligament, and the posterior talofibular ligament (PTF). Inversion ankle sprains are responsible for injuries to the lateral ligaments. Due to its orientation across the joint, the ATF is the most easily injured ligament while the PTF is very difficult to injure [6]. The medial stabilizing ligaments include the deltoid ligament and the anterior tibiofibular ligament; these ligaments are injured during eversion ankle sprains [6].

In addition to the ligaments that cross the ankle, there are thirteen muscles that cross the ankle. These muscles are organized into three main compartments. The anterior compartment is comprised of the tibialis anterior, the extensor digitorum longus, the extensor hallucis longus, and the peroneus tertius. The lateral compartment is comprised of the peroneus longus muscle (PL) and the peroneus brevis muscle (PB). In the posterior compartment, the three superficial muscles are the gastrocnemius, the soleus, and the plantaris. The deep muscles contained in the posterior compartment include the popliteus, the flexor hallucis longus, the flexor digitorum longus, and the tibialis posterior. The tibialis anterior and tibialis posterior are responsible for ankle inversion. The two muscles contained in the lateral compartment, the PB and PL muscles, are two muscles responsible for ankle eversion [12]. During an inversion ankle sprain, the peroneal muscles are stretched and a reflex is activated which acts to counter the inversion movement of the ankle. As a result, these muscles may have a role in the prevention of ankle sprains.

1.3.2 Joint Stability

From a functional point of view, an ankle sprain can be considered a loss of joint stability. Joint stability describes the ability of a joint to maintain or regain its position following an external disturbance [13]. All the structures in a joint – ligaments, tendons, muscles, bones – contribute to joint stability; however, during functional activities the
predominate mechanisms for maintaining joint stability are active muscle stiffness and active muscle reflexes [13-15].

1.3.3 Joint Stability - Joint Stiffness

One contributor to joint stability is joint stiffness and is defined as the resistance of a joint to stretch or deformation in response to external forces [16]. Joint stiffness promotes stability by helping to prevent joints from moving outside the normal range of motion [16]. The active muscle tissue provides the largest contribution to overall joint stiffness and is therefore the dominant contributor to functional joint stability. Contraction of the agonist muscles is important in controlling joint stiffness; however, the simultaneous contraction of both agonist and antagonist muscles is also important. Increases in this coincident contraction, termed co-contraction or co-activation, can increase joint stiffness [17] and in turn increase joint stability.

1.3.4 Joint Stability – Muscular Stretch Reflex

The second important mechanism that contributes to joint stability is active muscle reflex. Muscle spindles are sensory receptors within the muscle that detect muscle length and/or rate of change of muscle length [18]. If the spindles detect a change, a signal is sent to the nervous system which signals the muscle to activate. The initial muscle response is called the stretch reflex and is due to sudden changes in muscle length. Common parameters used to describe the stretch reflex are reflex delay and reflex amplitude.

In the ankle, sudden inversion causes the PB and PL muscles to stretch causing the activation of the peroneal stretch reflex. This activation of the peroneal reflexes in response to sudden inversion was shown by Grunebeg et al. [19] who found that peroneal reflex amplitude increased when landing on a tilted surface that caused inversion compared with a similar landing on a flat surface. Because of this increased reflex activation, it is possible that peroneal reflexes have a role in controlling ankle stability with inversion stress and thus have a role in stabilizing and protecting the ankle from inversion ankle sprains. This possible connection between peroneal reflexes and ankle sprains has led many researchers to investigate peroneal reflex response after sudden
ankle inversion [19-27]. Some of these studies have found increased peroneal reflex delay in individuals who exhibit functional instability from recurrent ankle sprains [22, 23]. Although it is difficult to determine if this increase in delay was present before and potentially contributed to the recurrent sprains, or if it resulted from the sprains, there remains an association between reflex delay and ankle sprains.

1.4 Risk Factors for Inversion Ankle Injury

1.4.1 Intrinsic Risk Factors

There are a number of factors that have been shown to increase the risk of an inversion ankle sprain. Perhaps the most frequently studied intrinsic risk factor is a previous history of ankle sprains [28, 29]. As stated previously, joint stability is closely linked to the occurrence of injury. With a previous ankle sprain, there is the possibility of ligament damage (increased joint laxity) and nerve damage, which could lead to decreased ankle stability and thus an increased risk of an ankle sprain [28]. Next are anthropometric factors like height and weight which may predispose an individual to a sprain [30, 31]. The greater an individual’s height or weight, the greater the inversion torque that the ankle must endure, which may increase the risk for an ankle sprain [28]. Joint laxity (e.g. mechanical instability), another intrinsic risk factor, refers to the looseness of the ankle due to structural ligament or soft tissue damage. Joint laxity has long been associated with an increased risk for ankle sprains by medical personnel due to decreased ankle stability [28]. However, researchers who have investigated joint laxity and the increased risk of an ankle sprain have come to different conclusions depending on the joint laxity assessment technique used [28]. Barrett et al [32] found that joint laxity did not predict ankle sprains when using both anterior drawer and talar tilt measurements of joint laxity. Baumhauer et al [33] found that using the anterior drawer test showed a connection to the occurrence of ankle sprains but that the talar tilt test did not. In a later study, results from the talar tilt were associated with the occurrence of ankle sprains in men but not women [31]. A fourth intrinsic risk factor is functional instability. Functional instability unlike joint laxity is associated with a disruption of the sensory receptors at the ankle and could lead to decreases in ankle proprioception [34]. A decrease in ankle proprioception could lead to a decrease in ankle stability and thus an increased risk for
ankle sprains. Lastly, increased postural sway has been linked to an increased risk for ankle sprains by several studies [35-37]. A possible explanation is the increase in functional instability that has been found with increased postural sway [38].

1.4.2 Extrinsic Risk Factors

Unlike the intrinsic factors described above, extrinsic factors like bracing and taping have been associated with a decreased risk for ankle sprains [30, 39-41]. Most of the risk factors for inversion ankle sprains are connected to either a loss of ankle stability or a decrease in ankle proprioception. Ankle braces have been suggested to improve both of these conditions. Several studies hypothesized that the reason for the decrease in occurrence of ankle sprains was due to an increase in mechanical stability of the ankle [40] and/or an increase in ankle proprioception [39]. The decrease in ankle sprain risk with bracing, however, does not suggest that sprains do not occur when bracing or taping is used. According to a study of English soccer players, 32% of all injuries occurred while players were wearing some form of external support [8]. So although there may be a decreased risk, there is still some risk of incurring an ankle sprain.

1.4.3 Fatigue as a Risk Factor

Many recent epidemiological studies have suggested neuromuscular fatigue as a potential risk factor for athletic injuries [8, 42-45]. Reports have found that 71% of rugby injuries occurred in the second half of matches [42], 48% of soccer injuries occurred in the last third of the first and second halves of matches [8], and 47% of ice hockey injuries occurred in the last 5 min of a period [44]. Moreover, a study of telemark skiers found that the most likely time for an injury was in the afternoon between 2 p.m. and 4 p.m. [46]. These findings have lead researchers to hypothesize that neuromuscular fatigue may contribute to the occurrence of injury [42, 47].

1.4.4 Gender as a Risk Factor

There have also been reports that suggest gender as a risk factor for some sports injuries. It is well documented that females have an increased risk of anterior cruciate ligament (ACL) injury when compared with males [48-50]. Per hour of play, females
have been estimated to have a four to eight times greater risk for ACL injury than males [48-50]. With respect to ankle injuries, there is evidence of increased ankle sprains in females compared to males. The most compelling study by Hosea et al. [51] found that females were 25% more likely than males to sustain a grade I ankle sprain during basketball. The mechanism behind this increased risk is unknown.

1.5 Effects of Fatigue and Gender on Joint Stability

Ankle stability is an important factor in determining whether an ankle sprain will occur. Many of the risk factors associated with inversion ankle sprains have been tied to decreases in joint stability; however, the mechanisms behind fatigue and gender as risk factors are relatively unknown. The importance of joint stability to injury occurrence has led researchers to investigate fatigue and gender with respect to joint stiffness and muscular reflex.

1.5.1 Effects of Fatigue

Several studies have investigated the effects of fatigue on joint stiffness and found very different results. One study found that fatigue had no effect on ankle stiffness [52] but this study used only four subjects. Another study found a decrease in stiffness at the knee and a similar trend at the ankle due to stretch-shortening cycle exercises [53]. Increases in co-contraction, and coincidently joint stiffness, have been found with fatigue of the trunk muscles [54] and the soleus and tibialis anterior muscles [55]. To our knowledge, there have been no studies specifically looking at the effects of fatigue on ankle co-contraction.

Studies of the muscular stretch reflex, the second main component of joint stability, have considered fatigue with respect to both reflex delay and reflex amplitude. Many studies have confirmed that neuromuscular fatigue has no effect on reflex delay for the vastus lateralis muscle [56], the knee extensors [57, 58], and the lateral gastrocnemius and soleus [16]. Conversely for reflex amplitude, studies have found both increases and decreases in reflex amplitude with fatigue. Reflex amplitude has been shown to increase in the knee extensors [57, 58] and elbow flexors [59] and decrease in the soleus muscle [60], finger flexors [61], and the first dorsal interosseus muscle [62] with fatigue.
1.5.2 Effects of Gender

The effect of gender on joint stiffness for multiple joints including the ankle has been investigated, although the results have been inconsistent. Several studies have found that females have a decreased stiffness when compared to males in the knee flexors [63, 64], the knee extensors [64], the ankle invertors and evertors [16], and in the lower extremity [65] An increased stiffness in females compared to males was also found for the plantar flexion/dorsiflexion (PD) stiffness of the ankle [16]. Moreover, larger amounts of co-contraction were found in females compared to males in the leg [65], which supports the idea that females have greater stiffness in the lower extremity. Some of the differences in stiffness with gender were attributed to anthropometric differences between males and females [63, 65].

Investigations of reflex with gender have found no differences with respect to reflex delay of the vastus lateralis muscle [56]. To our knowledge, no studies have investigated reflex amplitude with gender or peroneal reflex delay and amplitude with gender.

1.5.3 Effects of Fatigue and Gender

We were able to find only one study considering the effects of both fatigue and gender on ankle stiffness. Massimini et al. [16] showed that with fatigue, PD ankle stiffness increases in males and decreases in females even after accounting for size differences in joint structure between genders. The reason for the contradicting changes in stiffness with fatigue was not discussed. Our literature search revealed no studies looking at co-contraction at the ankle with regards to fatigue and gender.

The effects of fatigue and gender have been investigated with respect to both EMG amplitude of voluntary contractions and reflex delay and amplitude. Several studies looking at voluntary contractions with fatigue and gender have found increases in EMG amplitude for the trunk muscles [66] and decreases for the plantar flexor muscles [67] following fatigue, but no differences were found with gender and reflexes were not investigated. The behavior of voluntary contractions, however, may be different than the muscular reflex response. There has been one study considering the effects of both
fatigue and gender on muscular reflex. Moore et al. investigated the vastus lateralis muscle and found no change in reflex delay for both males and females or a change in reflex amplitude for females [56]. An increase in reflex amplitude was found for males. The gender difference in reflex amplitude changes with fatigue were attributed to differences in how males and females respond to isokinetic fatigue. These suggested differences in how males and females fatigue have been investigated extensively [68-71]. Results show that males and females respond differently to fatigue with respect to parameters like metabolic response [70], endurance time [68, 71], and median frequency shifts [68]. Many physiological and anatomical explanations have been offered to explain these fatigue differences including muscle mass, metabolic byproducts, and muscle type distribution.

1.6 Goals of the Present Study

Fatigue and gender have recently been realized as potential risk factors for ankle sprains because of the growing number of supporting epidemiological studies. Considering the prevalence and impact that inversion ankle sprains have on athletes, there is a growing need to investigate fatigue and gender as risk factors and to determine how they affect ankle sprains. The occurrence of ankle sprains is closely tied to changes in joint stability; however, studies looking at the effects of fatigue and gender on joint stability at the ankle are limited especially in the inversion/eversion (IE) direction. To our knowledge, there are no studies investigating the effects of fatigue and gender on IE ankle stiffness, co-contraction, or peroneal muscle reflexes. Therefore, to begin the investigation of how ankle stability in the IE direction is affected by fatigue and gender, a study was performed on the peroneal stretch reflexes. The goal of the present research was to investigate the effects of fatigue and gender on peroneal reflexes following sudden ankle inversion.

1.7 References


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CHAPTER 2

FATIGUE & GENDER EFFECTS ON PERONEAL REFLEXES FOLLOWING SUDDEN ANKLE INVERSION
2.1 Introduction

An estimated 23,000 ankle injuries occur every day in the U.S. [1] amounting to over 8 million ankle injuries a year. The cost of these injuries is over $2 billion annually [2, 3]. According to statistics from 1984, this is roughly equivalent to the amount of money spent on coronary bypass surgical grafting in the U.S. [2]. The NCAA’s Injury Surveillance System 2003-2004 Report indicated that the ankle has been the most injured joint in the body in collegiate volleyball and basketball over the last 21 years, accounting for 21%-33% of all injuries [4]. Numerous other studies of volleyball [5-7], basketball, soccer, and running have reported similar ankle injury rates [6].

Ankle sprains account for 75% to 85% of all ankle injuries [8, 9]. Inversion ankle sprains account for 85% of these ankle sprains [8, 10]. A number of proposed risk factors for inversion ankle sprains include a previous history of ankle sprains, height and weight, joint laxity, functional instability, and postural sway [11, 12]. Many of these risk factors are thought to either decrease the stability of the ankle or decrease the proprioceptive acuity at the ankle [11]. Functionally speaking, an ankle sprain can be considered a loss of joint stability, so by decreasing ankle stability, these risk factors are increasing the risk of a sprain. Conversely, bracing has been shown to decrease the occurrence of ankle sprains by increasing the mechanical stability of the joint [13] or increasing joint proprioception [14]. In addition to these risk factors, there is growing evidence that suggests gender and fatigue are also risk factors for inversion ankle sprains. In support of gender as a risk factor, females have been shown to be 25% more likely than males to sustain a grade I ankle sprain during basketball [15]. In support of fatigue as a risk factor, overall injury rates have been shown to increase toward the end of matches [16-20]. Specifically, epidemiological studies of several sports found that 71% of rugby injuries occurred in the second half of matches [17], 48% of soccer injuries occurred in the last third of the first and second halves of matches [20], and 47% of ice hockey injuries occurred in the last 5 min of a period [18].

Stretch reflexes in the peroneus brevis muscle (PB) and peroneus longus muscle (PL) may be important in preventing inversion ankle sprains. These muscles contribute to ankle eversion, and may help arrest sudden ankle inversion before tissue strain becomes excessive. Evidence for the role of peroneal reflexes in controlling ankle inversion was
shown by Gruneberg et al. [21] who found increases in peroneal reflex amplitude when landing on a titled surface that caused inversion versus a flat surface. Moreover, increased peroneal reflex delay has been associated with individuals who exhibit functional instability from recurrent ankle sprains [22, 23]. Although it is difficult to determine if this increase in delay was present before and potentially contributed to the recurrent sprains, or if it resulted from the sprains, the association between reflex delay and ankle sprains indicates some complicity between the two.

The connection between peroneal reflexes and ankle sprains have led many researchers to investigate peroneal reflex response after sudden ankle inversion [21-29]. Given the growing evidence supporting fatigue and gender as risk factors for inversion ankle sprains, investigating the effects of these factors on peroneal reflexes after sudden ankle inversion may help explain the differences in sprain rates with fatigue and gender. To our knowledge, no such studies exist. Therefore, the purpose of the present study was to investigate the effects of fatigue and gender on PB and PL reflexes following sudden ankle inversion.

2.2 Methods

Sixteen males (18-32 years of age) and fifteen females (18-26 years of age) participated in the experiment. Mean (SD) height for males and females was 180.7(7.2) cm and 167.4(7.8) cm, respectively; mean mass for males and females was 75.1(8.8) kg and 59.5(7.6) kg, respectively. None of the participants reported any history of ankle injury within the last twelve months, and all provided informed consent in accordance with the Virginia Tech Institutional Review Board before participation.

The experimental protocol involved four main stages: warm-up, unfatigued reflex measurements, fatigue protocol, and fatigued reflex measurements. Prior to the start of testing, participants were positioned on a Biodex System 3 isokinetic dynamometer (Biodex Medical Systems Inc.; Shirley, NY). The participant’s dominant foot (defined as the foot used to kick a soccer ball) was placed in the manufacturer’s ankle attachment and positioned in the anatomical position with the knee flexed at 45° and the hip flexed at 70° (Figure 2.1). In this position, the axis of rotation of the dynamometer passed through body of the talus at approximately 35° superiorly to the transverse plane and
approximately 15° medially to the sagittal plane (which follows the manufacturer’s recommended setup for ankle inversion/eversion testing). Two velcro straps secured the foot to the ankle attachment while a thigh support and strap were used to minimize movement of the leg. Maximum ankle inversion/eversion (IE) range of motion was measured and the dynamometer IE movement restricted to 90% of the maximum inversion movement and 90% of the maximum eversion movement. Testing began with a 2-minute isotonic warm-up with torque set at 0.678 Nm (0.5 ft-lbs) for both inversion and eversion. During warm-up, one repetition was performed every 4 seconds with eversion and inversion each lasting 1 sec followed by a 2-second pause. A metronome set to 60 beats/min was used to maintain a consistent repetition rate during these and all other IE movements.

Figure 2.1: Participants were positioned in a Biodex System 3 isokinetic dynamometer.
Following the warm-up, multiple maximum voluntary contractions (MVCs) and one reference contraction (RC) were performed. Three 60°/sec isokinetic eversion MVCs were performed to determine participants’ 20% isokinetic MVC torque that would be used in the upcoming fatigue protocol. After two minutes of rest, participants performed three isometric eversion MVCs to determine their 50% isometric MVC torque needed for the upcoming RCs (see below). These isometric MVCs were performed with the ankle in the anatomical position. After one minute of rest, participants performed an isometric RC for 5 sec at their 50% MVC torque. To perform this RC, participants were provided with a real-time display of the dynamometer torque and a visual marker indicating 50% of their isometric MVC torque. Once instructed, participants attempted to evert their ankle until the dynamometer torque reached the 50% MVC torque marker. Participants practiced the unfatigued RC several times prior to collection.

To elicit peroneal reflexes, a “trap-door” platform was used to provide sudden ankle inversions (Figure 2.2). Participants were positioned with the lateral border of their dominant foot aligned with the edge of the platform, feet shoulder width apart, weight equally distributed between both feet, and head facing directly forward. The platform was released by manually removing a support arm under the lateral border of the platform. After release, the ankle was inverted from the anatomical position to approximately 20° inversion in about 60 ms. A potentiometer, placed on the axis of rotation of the platform, was used to measure angular displacement of the platform. A metallic contact on the support arm, which lost contact when the platform was released, was used to identify the start time of inversion. Three practice reflex trials were performed to familiarize participants with the inversion motion. Five unfatigued peroneal reflex measurements were then performed at random intervals over a period of 45 seconds. In an attempt to distract participants during the reflex measurements, participants were instructed to count backward throughout all reflex measurements.
Reflexes were elicited using an inverting “trap door” platform. A block underneath the platform limited maximum inversion to 20° while a ledge prevented foot slippage upon platform inversion. A potentiometer and metallic contact circuit were integrated into the platform to measure angular displacement and the start of ankle inversion, respectively.
The fatigue protocol attempted to fatigue the ankle evertors at a linear rate over 12 minutes to 75% of the unfatigued isokinetic evertor MVC torque. Eversion movement was performed with an isotonic load starting at 20% of the maximum isokinetic MVC torque. Inversion movement was performed concentrically using a low isotonic load of 0.678 Nm (0.5 ft-lbs) to minimize invertor fatigue. One 60°/sec isokinetic eversion MVC was performed every 2 minutes to track the progress of fatigue. The eversion load was systematically adjusted every two minutes based on a comparison of the isokinetic MVC torque with the target MVC torque. The target MVC was determined using a linear decrease in torque from 100% to 75% of the MVC in 12 minutes. If the participant was not at or below the 75% target level at the end of 12 min, two additional minutes were added. As in the warm-up, one repetition was performed every 4 seconds with eversion and inversion each lasting 1 sec followed by a 2-second pause. Immediately following the fatigue protocol, a fatigued RC and five fatigued peroneal reflex measurements were performed in an identical manner as described for unfatigued measurements. These measurements were completed within 3 minutes of the completion of the fatigue protocol to minimize recovery from fatigue.

Muscle activity of the PB and PL were collected during the RCs and reflex measurements using Ag/AgCl electrodes. PB and PL electrode placement (Figure 2.1) was ¾ and ¼ of the distance from the lateral malleolus to the lateral epicondyle, respectively from the knee, and along the line connecting these anatomical landmarks [21]. Electrodes were placed longitudinally along the presumed direction of the muscle fibers with a 2 cm inter-electrode distance. A reference electrode was placed on the head of the fibula. Prior to electrode placement, the skin was shaved and abraded with an alcohol pad. Muscle activity was sampled at 1000 Hz and subsequently full-wave rectified and filtered with a 25 Hz low pass filter (4th order zero phase lag Butterworth). During each reflex trial, data from the platform’s potentiometer and metallic contact (which indicated platform release time) were collected and sampled at 1000 Hz.

Reference contractions were included in the protocol in an effort to compensate, if necessary, for the effects of fatigue on electromyogram (EMG) amplitude. EMG amplitude during sub-maximal isometric contractions increases with fatigue [30, 31]. The reason for this is two-fold: (1) the build-up of metabolic byproducts during fatigue
decreases the conduction velocity of action potentials leading to decreases in myoelectric frequency, and (2) the skin is a low-pass filter, so a greater amount of myoelectric activity can pass through the skin when the frequency is reduced during fatigue [32]. For these reasons, the same level of myoelectric activity within the muscle would appear larger in the fatigued condition. Because of this, it could be argued that any increase in reflex amplitude with fatigue was due to the effect of fatigue on EMG amplitude rather than the effect of fatigue on reflex amplitude per se. RCs were collected to provide a way to measure the effects of fatigue on EMG amplitude. Unfatigued reflex amplitudes were normalized by unfatigued RC amplitude. If RC amplitude increased with fatigue, then fatigued reflex amplitude would be normalized by the fatigued RC amplitude to provide compensation for the effect of fatigue on EMG amplitude. If RC amplitude did not increase with fatigue, then fatigued reflex amplitude would be normalized by unfatigued RC amplitude (just as the unfatigued reflex amplitude was normalized). Our analysis revealed no significant difference in RC amplitude with fatigue for the PB (p=0.258) or PL (p=0.305). Therefore, all the EMG reflex amplitude data was normalized using the unfatigued RC amplitude.

The primary analysis involved two dependent measures which were calculated to quantify PB and PL reflexes: reflex delay and reflex amplitude. Reflex delay was defined as the time difference between the start of platform inversion and EMG activity. The start of EMG activity was visually identified as the first point, 20 ms after the start of platform inversion, that exceeded mean baseline activity by two standard deviations. A cutoff of 20 ms was used because any activity prior to this point was assumed to be artifact. Other studies have used a similar approach to identifying reflexes [25, 27, 29, 33]. Reflex amplitude was systematically chosen as the highest point in the EMG signal within 150 ms of the start of inversion. Maximum platform inversion angle and time to maximum platform inversion angle were also analyzed to ensure consistent inversion parameters between fatigue conditions and across gender. In a secondary analysis, additional measures were considered to elucidate possible differences in peroneal reflexes with gender and fatigue. End fatigue level, defined by the final MVC taken during the fatigue protocol, was the first parameter tested to ensure that any differences in PB and PL reflex across gender were not due to differences in the level of fatigue of the participants.
Isometric evertor MVC torque was analyzed to discern strength differences between genders. Variations in muscle strength have been linked to differences in fatigue because of differences in muscle mass and blood perfusion [34]. Even though participant end fatigue level was controlled, if males demonstrated greater strength, their eversion repetitions would have been at a greater absolute torque than the females, leading to differences in blood perfusion of the leg. Lastly, EMG median frequency of the PB and PL during the RCs were analyzed to determine any potential difference in PB fatigue and PL fatigue.

Prior to analysis, visual inspection of all the dependent variables showed some to be positively skewed. After a log_{10} transformation, these variables conformed well to the normal distribution. The data were inversely transformed after parametric statistics were applied to the transformed data. The effects of fatigue and gender on each of the dependent measures were determined using a two-way repeated measure analysis of variance (ANOVA). Following a significant interaction, pairwise comparisons were evaluated within each level of fatigue and gender using paired t-tests and Holm’s sequential Bonferroni procedure to control for familywise error rate [35]. Significance was determined using p-values less than 0.05.

2.3 Results

Reflex delay (Figure 2.3) was not affected by fatigue (PB: p=0.441, PL: p=0.404), gender (PB: p=0.636, PL: p=0.305), or their interaction (PB: p=0.778, PL: p=0.116). The mean reflex delay for the PB and PL were 56.7(8.6) ms and 60.4(12.0) ms, respectively.

PB reflex amplitude (Figure 2.4) was not affected by fatigue (p=0.601), gender (p=0.799), or their interaction (p=0.099). PL reflex amplitude (Figure 2.4) was not affected by fatigue (p=0.484) or gender (p=0.858) but was affected by their interaction (p<0.001). Pairwise comparisons revealed a 11.3(15.9)% decrease in PL reflex amplitude for males (p=0.008) and a 22.1(25.2)% increase for females (p=0.003) with fatigue. There was no effect of gender on the unfatigued (p=0.736) or fatigued (p=0.494) PL reflex amplitude.
Figure 2.3: Reflex delay of the PB and PL was not affected by fatigue or gender.
The sudden inversion movement of the platform did vary slightly across experimental conditions. The maximum platform inversion angle averaged 22.2(1.0)°, and was not affected by fatigue (p=0.427), gender (p=0.200), or their interaction (p=0.448). However, time to maximum platform inversion angle decreased an average 1.2(2.1) ms with fatigue (p=0.003) and was 5.0 ms longer for females compared to males (p=0.030) when collapsed across fatigue conditions. A significant interaction between fatigue × gender (p=0.030) revealed that with fatigue, time to maximum platform inversion decreased by 2.0 ms in females but was unaffected by fatigue in males. The ≤ 2.0 ms difference in timing of the inversion movements with fatigue was 3.2% of the
unfatigued time to maximum platform inversion and was therefore thought to be practically insignificant.

Our secondary analysis included several additional parameters which were tested to rule out factors that could have potentially confounded the observed effects of fatigue and gender on peroneal reflexes. End fatigue level averaged 65.5(11.1) % and did not vary across gender (p=0.599). Thus, the interaction between fatigue and gender for PL reflex amplitude was not due to a gender difference in fatigue level. An analysis of isometric evertor MVC torque revealed that males were stronger then females (p<0.001) with an average isometric evertor MVC torque of 16.9(3.4) Nm for males and 11.5(3.3) Nm for females. EMG median frequencies of the RCs were not affected by fatigue (PB: p=0.703, PL: p=0.164), gender (PB: p=0.272, PL: p=0.593), or their interaction (PB: p=0.579, PL: p=0.372). These results reveal that if there were differences in PB and PL fatigue, the differences were not apparent in an EMG frequency analysis.

2.3 Discussion

The purpose of the present study was to investigate the effects of fatigue and gender on PB and PL reflexes following sudden ankle inversion. Our results showed no fatigue or gender effects on PB and PL reflex delay. However, PL reflex amplitude was found to increase by 22% in females and decrease by 11% in males following fatigue. The same trend was seen in PB reflex amplitude, although the changes were not statistically significant. The opposite effect of fatigue in males and females was unexpected, so our secondary analysis attempted to rule out any extraneous factors that could have contributed to the differences in reflex response. Other than minor differences in platform movement that were deemed practically insignificant, no experimental explanations were found. Thus, the differences in PL reflex amplitude were attributed to biomechanical, anatomical, and/or physiological differences between males and females.

The lack of a fatigue and gender effect on peroneal reflex delay is consistent with a similar investigation of the vastus lateralis muscle [33], while the lack of a fatigue effect on peroneal reflex delay is consistent for multiple knee extensor muscles [36, 37]. Reports of the effect of fatigue on reflex amplitude have been contradictory. Reflex amplitude has been shown to increase with fatigue in the knee extensors [36, 37] and
elbow flexors [38], and decrease with fatigue in the soleus muscle [39], finger flexors [40], and the first dorsal interosseus muscle [41]. However, none of these studies investigated gender. Moore et al. [33] reported a significant increase in vastus lateralis reflex amplitude in males with fatigue, but no change in vastus lateralis reflex amplitude in females with fatigue. These results are different than those presented here where PL reflex increased in females with fatigue and decreased in males with fatigue. The contradictory results could potentially be explained by differences between the muscle groups used or differences in data collection. There are two main data collection differences that could potentially have affected the vastus lateralis reflex results. First, the tendon tap used to elicit reflexes by Moore et al. [33] controlled force while the “trap-door” platform in the present study controlled position. Since females weighed less than males they potentially had a smaller muscle mass. Consequently, the controlled tap force would have had a greater effect on females because the force to muscle mass ratio was greater than in males. This proportionally greater force could have led to a greater reflex response in females which could have confounded the reflex amplitude results of the study. A second difference in data collection was that potential changes in EMG amplitude with fatigue were not addressed [30, 31] which could have confounded the reported trends in reflex amplitude. The incorporation of unfatigued and fatigued RCs in the present study allowed the effects of fatigue on EMG amplitude to be ruled out as a potential confounding factor.

There are several potential biomechanical, anatomical, and physiological explanations as to why changes in PL reflex amplitude with fatigue are different in males and females. Both reflexes and joint stiffness contribute to joint stability [42, 43] and may help to prevent inversion ankle sprains. It is possible that as a result of fatigue, or in an effort to compensate for fatigue, males and females use stiffness and reflexes differently. Massimini et al. [44] found that with fatigue, ankle plantar flexion/dorsiflexion stiffness decreased in females and increased in males. If similar changes in IE ankle stiffness occur with fatigue, a decrease in ankle stiffness in females could necessitate a simultaneous increase in reflex amplitude to offset the loss of joint stability. Conversely, an increase in stiffness in males could result in a simultaneous decrease in reflex amplitude to compensate for the changes in joint stability. If true, these hypothesized
trends in reflex amplitude would be consistent with those reported here. Changes in co-contraction have been linked to changes in ankle stiffness [42, 43, 45]. So, a second yet related potential explanation for the gender-specific changes in PL reflex amplitude with fatigue is a difference in the effect of fatigue on co-contraction with gender. Several researchers have found that the amount of agonist/antagonist co-contraction increases with fatigue [46, 47]. Given that the effects of fatigue on ankle stiffness have been shown to vary with gender, it is possible that changes in co-contraction with fatigue are different in males and females and could affect peroneal reflex amplitude. Unfortunately, the effects of fatigue and gender on co-contraction could not be evaluated in the present study because EMG was not collected from antagonistic muscles. A third potential explanation for the gender-specific changes in PL reflex amplitude with fatigue is the inherent anatomical and physiological differences between males and females and how they fatigue [34, 48, 49]. Some of these differences include muscle mass, metabolic byproducts, and muscle-fiber type distribution. For example, our results show that evertor muscle strength was significantly different between males and females suggesting that males had a greater muscle mass. Although all participants were fatigued to similar percentages of their MVC, males were performing the fatigue protocol using higher absolute torques which could lead to a greater loss of blood perfusion in the PB and PL during the fatigue protocol. Future studies investigating the effects of fatigue and gender on reflexes should include these factors to help understand how they may contribute to these observed trends.

Three potential limitations of this study warrant discussion. First, the peroneal muscles are relatively small and in close proximity to other muscles. Although we followed established guidelines for EMG electrode placement [21], our EMG signals could include crosstalk from nearby muscles. Second, since static and dynamic exercises are suspected to involved different aspects of the neuromuscular system [50], a fatigue protocol involving dynamic contractions was selected as more representative of typical athletic activities. External validity of dynamic versus static fatiguing is greater; however, there is a loss of experimental control and the possibility of fatigue in muscles other than the peroneal muscles with a dynamic fatigue protocol. Third, we did not actually measure ankle inversion, we measured platform inversion. Subtle differences could have existed.
between inversion of the platform and inversion of the foot that were undetectable by the potentiometer.

In conclusion, the results of this study showed that reflex delay in the peroneal muscles does not change with fatigue, but PL reflex amplitude increases in females and decreases in males with fatigue. The opposite effect in males and females may be due to biomechanical, anatomical, and/or physiological differences in how males and females fatigue and/or compensate for fatigue. The clinical relevance of this gender difference is unclear at this point, but these gender differences could be related to the gender differences in ankle injury rate. Future studies should consider IE ankle stiffness and co-contraction with fatigue and how these parameters and reflex amplitude may influence ankle sprains in males and females differently.

2.5 References


Erin L. Wilson       Chapter 2. Peroneal Reflexes       32


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2.6 Supplemental Materials I

Data Collection Sheet

____________________________________

____________________________________

ANKLE FATIGUE REFLEX STUDY

Setup Checklist

- Turn on:
  - EMG amplifier (30 min to warm-up)
  - Biodex (button in back and two greens ones on front)
- Biodex set-up on cart:
  - remove attachment
  - push start
  - re-attach ankle attachment
  - push ‘Attachment Select’ and cycle through until it says Ankle
  - push ‘Isokinetic’
- Biodex LabVIEW set-up:
  - open System Biodex Trq Pos Col.vi
  - change file name to C:\Erin Raw Data\subj_#
  - push reset button
  - open metronome.vi
- Check Battery Voltage, Plug two batteries in on Trap Door
- Potentiometer Calibration – use multimeter; once/day
  -19.0° (L): _____ V  0°: _____ V  11.2° (R): _____ V
- Get out supplies:
  - EMG electrodes (5)
  - Co-flex
  - Alcohol
  - athletic tape
  - razor
  - bathroom scale
  - timer
  - metronome
- Ensure all connections into A/D card are correct
- Check bolt on Biodex and ensure it is tight
1. Give summary of protocol and demonstrate
2. Obtain informed consent
3. Collect anthropometric measurements:
   age: ______ weight: ______ lbs height: ______ cm
5. Demonstrate EV and INV
6. Apply EMG electrodes (shave, alcohol):
   - reference electrode – head of fibula
   - peroneus longus – quarter of the way distal between head of fibula and lateral malleolus
   - peroneus brevis – three-quarters of the way distal between head of fibula and lateral malleolus; have subject move toes independently to feel extensor digitorum longus;
     peroneus brevis does not move with toes
7. Perform passive motion of ankle to ensure comfort
8. Set ROM; **Isokinetic** mode, adjust ROM to 80%: INV to 90%  EV to 90%
   Total ROM: ______  80% ROM: ______
9. Start metronome program, explain timing of repetitions: 1 sec, 1 sec, 2 sec pause
10. Do 2-min **Isotonic** warm-up session at 0.5 ft lb for inversion and eversion
11. Check for EMG signal using LabVIEW
12. Perform **Isometric** MVCs using Biodex:
   EMG 1: peroneus brevis gain setting: ___
   EMG 2: peroneus longus gain setting: ___
   Max Torque: ______ Nm ______ Nm ______ Nm
   filename: col ___ col ___ col ___
13. Two minute rest
14. **Isokinetic** MVCs with inversion at 500 deg/sec and eversion at 60 deg/sec
   Max Torque: ______ Nm comments: _______________________
15. One minute rest
16. Practice 50% Hold
17. **Isometric 50% MVC** (______) Trial for EMG Adjustment w/ foot in neutral position (5 sec)
18. Demonstrate mounting and inversion of platform; position subject on Zinderizer; align edge of foot with edge of platform; ensure feet are shoulder width apart

19. Demonstrate twice the release of the platform and the sudden inversion

20. Collect unfatigued reflex data (5 times with at least 10 sec rest in-between)

| filename: col | comments: __________________________ |
| Collect unfatigued reflex data | comments: __________________________ |
| Collect unfatigued reflex data | comments: __________________________ |
| Collect unfatigued reflex data | comments: __________________________ |
| Collect unfatigued reflex data | comments: __________________________ |

21. Ask subject to return to Biodex Chair; strap subject in

22. Make sure ROM is set to 90% for INV and EV

23. Check filename in LabVIEW and push reset button: C:\Erin Raw Data\subj_#

24. Turn on metronome; Begin Fatiguing Protocol

<table>
<thead>
<tr>
<th>Isokinetic for MVCs: INV: 500 deg/sec EV: 60 deg/sec</th>
<th>Isotonic for Reps: INV: 0.5 ft-lb EV: 50% MVC</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Isokinetic MVC</strong> MVC (ftlb):</td>
<td><strong>Time</strong></td>
</tr>
<tr>
<td>Isokinetic MVC 0:00</td>
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<tr>
<td>Isokinetic MVC 2:</td>
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<td>Isokinetic MVC 4:</td>
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<td>Isokinetic MVC 12:</td>
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<td>Isokinetic MVC 14:</td>
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<tr>
<td>Isokinetic MVC 16:</td>
<td></td>
</tr>
</tbody>
</table>

*** ASK IF TINGLY WITH SCALE FROM 1 TO 5 (5 is max tingly): _____ *****

**Special Instructions:**
- For MVCs, instruct subject to contract as hard and as fast as possible
- Allow one minute rest following warm-up and before 2<sup>nd</sup> set of MVCs
- Begin subject in full inversion for all sections
- Evert, Invert for one-second each, two second pause in full inversion
- Focus on completing the full range of motion in the one second allotted

25. Turn off metronome
26. **Isometric 50% MVC** Trial for EMG Adjustment with foot in neutral position (5 sec)

   filename: *col__* comments: ________________________________

27. Instruct subject to dismount biodex chair and balance on the zinderizer

28. Collect fatigued reflex data (5 times with at least 10 sec rest in-between)

   filename: *col__* comments: ________________________________

   Collect fatigued reflex data comments: ________________________________
   Collect fatigued reflex data comments: ________________________________
   Collect fatigued reflex data comments: ________________________________
   Collect fatigued reflex data comments: ________________________________

29. Offer subject a chair to rest

30. Remove EMG from subject.
    
    Give them stipend and have them sign payment sheet.
    
    Thank them, and see them to the door.

31. Turn off equipment

   - EMG amplifier
   - Biodex
   - Potentiometer
   - LabVIEW
   - Multimeter
   - Unplug batteries (2)

32. Clean up the laboratory

33. Move data to Y: Drive at end of day

**General Comments:**

________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________
EFFECTS OF FATIGUE & GENDER ON PERONEAL REFLEXES AFTER ANKLE INVERSION

ERIN LAWALL WILSON

MAIN APPENDIX

POSTURAL STRATEGY CHANGES WITH FATIGUE OF THE LUMBAR EXTENSOR MUSCLES
A.1 Introduction

Movements used to maintain standing balance in the sagittal plane following a postural perturbation have been described as a hip strategy, ankle strategy, or a combination of the two [1;2]. Numerous extrinsic and intrinsic factors affect which of these postural strategies are typically employed. Extrinsic factors include support surface size [3], perturbation magnitude [4], perturbation direction [5], and body position prior to the perturbation [6]. These studies suggest that postural strategy is affected by the biomechanical demands associated with maintaining balance. Intrinsic factors include fear of falling [7], expectation of perturbation characteristics [8], quality of somatosensory and vestibular feedback [9], Parkinson’s disease [10], aging [11], low back pain [12], and respiration [13].

Neuromuscular fatigue is another intrinsic factor that may affect postural strategy, but to our knowledge no studies have investigated fatigue in this regard. Fatigue has been shown to increase postural sway during quiet standing [14-20]. Increases in postural sway have, in turn, been associated with changes in postural strategy [21;22]. For example, Kuo et al. (1998) administered the Sensory Organization Test to healthy subjects and observed an increase in postural sway along with an increase in hip strategy when somatosensory information was disrupted. Although this study did not focus on fatigue, it demonstrates potential effects of a change in postural strategy on postural sway. Similarly, the documented increase in postural sway during quiet standing with fatigue may result from a change in postural strategy. If fatigue induces changes in postural strategy during quiet standing, it seems logical that similar changes in postural strategy will occur in response to a postural perturbation.

The purpose of this study was to investigate the effect of lumbar extensor fatigue on postural strategy in response to a balance perturbation. The effect of lumbar extensor fatigue level was also investigated to determine if increasing levels of fatigue have increasing effects on postural strategy. Understanding any changes in postural strategy with fatigue will further our basic understanding of postural control, and potentially contribute to the development of interventions aimed at mitigating the effect of fatigue on balance. One application of such an intervention would be in the construction industry where fatigue-related loss of balance may contribute to over 300 deaths/year from falls.
from heights [23]. Lumbar extensor fatigue was investigated because it is common during many occupational lifting tasks [24], and muscle fatigue in general has been shown to increase the risk of falling from heights [25].

A.2 Methods

Twelve physically active males (20-22 years of age) participated in the experiment. Mean (SD) participant height and mass were 173.7 (6.4) cm and 70.2 (6.6) kg, respectively. None of the participants reported any history of low back pain or injury, and all provided informed consent in accordance with the Virginia Tech Institutional Review Board before participation.

Participants completed three experimental sessions with 1-2 weeks between sessions. Each session consisted of four stages: warm-up, unfatigued balance perturbations, fatiguing protocol, and a fatigued balance perturbation. For warm-up, participants completed two cycles of the following activities: jogging at 2.0 km/hr on a treadmill for 2½ min, stretching the lumbar extensors, and performing five back extensions on a 45º Roman chair (New York Barbell, Elmira, NY). Following warm-up, the unfatigued isometric maximum voluntary contraction (MVC) moment of the lumbar extensors was measured at the level of L3. Participants were positioned in the Roman chair, attached to a load cell (Cooper Instruments and Systems, Warrenton, VA) at the mid-sternum via a modified construction harness, and instructed to extend at the back as hard as possible. In this position the lumbar flexion angle was approximately 45º.

Participants performed three isometric MVCs separated by one minute of rest; the largest of the three was recorded as the unfatigued MVC value.

Three unfatigued balance perturbations were performed following the warm-up. Prior to the perturbation, participants were instructed to stand quietly on a force platform without shoes, with their eyes closed, feet together, and arms resting at their side (Figure A.1). Participants were also told that if they stepped to regain their balance, another collection would be taken. (Two perturbations were repeated due to stepping.) Perturbations consisted of an anteriorly-directed force applied in the mid-sagittal plane at the level of the inferior margin of the scapulae, and were applied using a padded pendulum that was pulled a fixed distance away from the subject and released. These
perturbations were designed to mimic a bump to the upper back and challenge the postural control system without eliciting a stepping response. Using this method, the perturbation peak force was 177(21) N, and the perturbation impulse was 13.2(1.3) Ns. Perturbations were applied following a random delay (0-15 sec) from the beginning of each data collection to reduce participant anticipation.

In each of the three experimental sessions, the lumbar extensor muscles were fatigued to either 86%, 73%, or 60% of their unfatigued MVC. A balanced Latin square was used to determine the order of presentation of the three fatigue levels. The fatiguing protocol consisted of multiple sets of back extensions and intermittent MVCs performed on the Roman chair (see [15] for more details). Investigators attempted to fatigue participants such that their MVC moment decreased linearly over the duration of the fatiguing protocol and achieved the desired fatigue level over 14 minutes. The fatigue levels actually achieved were 82(5)%, 70(5)%, and 62(6)% MVC, which were significantly different from each other (p<0.001). Prior to performing the back extensions, participants were instructed to keep their feet flat on the base of the Roman chair and move through approximately 45° range of motion from 0° lumbar flexion to maximum flexion. Participants performed one set of back extensions every minute of the fatiguing protocol and an isometric back extension MVC every two minutes. MVCs were performed 10 seconds after completing the prior set of back extensions. A digital metronome set at 30 beats/min was used to ensure all participants performed the extensions at a similar rate. The number of repetitions in each set was systematically adjusted based upon a comparison of each measured MVC moment to the target moment [15]. If participants were not at or below the desired fatigue level at the end of the fatiguing protocol, two minutes were added with additional repetitions according to the same method. This process was continued until the MVC was below the desired fatigue level.
Figure A.1: Experimental set-up for applying postural perturbations. Subjects were perturbed by releasing the pendulum after pulling it back a fixed distance from the subject. Ground reaction force and body position data were recorded using the force platform and position markers shown.

Within 20 seconds after completing the fatiguing protocol, one fatigued balance perturbation was performed in an identical manner as the unfatigued perturbations. Only one fatigued perturbation was performed to minimize recovery from fatigue.

During each trial, the perturbation force was sampled at 1000 Hz from a load cell mounted inline with the pendulum and subsequently low-pass filtered at 25 Hz (4th order zero-phase-lag Butterworth filter). Triaxial ground reaction forces and moments were low-pass filtered with a 500 Hz anti-aliasing hardware filter, amplified, and sampled at 1000 Hz with a Bertec K20102 type 9090-15 force platform (Bertec Corp., Columbus, OH). These data were subsequently low-pass filtered at 10 Hz (4th order zero-phase-lag
Butterworth filter) and transformed to obtain ground reaction forces (GRF) and center of pressure (COP) data [26]. Body position was sampled at 50 Hz using a Vicon 460 motion analysis system (Vicon Motion Systems Inc., Lake Forest, CA). Markers were placed on both sides of the body at the acromion, iliac crest, greater trochanter, lateral femoral epicondyle, lateral malleolus, calcaneus, and head of the 5th metatarsal (Figure A.1). Marker position data was low pass filtered at 7 Hz (4th order zero-phase-lag Butterworth filter) and averaged across both sides of the body to create a sagittal plane representation of the body with five segments including the feet, shanks, thighs, pelvis, and trunk (defined by the iliac crest and shoulder). Using these five segments, joint angles were calculated for the ankle, knee, hip, and “low back” joints (the low back joint is defined as the angle between the trunk and pelvis segments). Joint torques were estimated using an inverse dynamics analysis with a model of the body consisting of five rigid segments connected by frictionless pin joints. The mass and inertial characteristics of the segments were defined using an anthropometric model [27].

Three dependent measures were calculated to quantify preparatory activity prior to the perturbation including initial joint angles, initial joint torques, and initial COP position relative to the ankle. These initial values were calculated as the mean over the time interval from 540 ms to 40 ms prior to the perturbation. Four dependent measures were calculated to quantify postural strategy during recovery from perturbations including peak joint angles, time to peak joint angles, peak joint torques, and time to peak joint torques. Because initial joint torques prior to the perturbation were affected by fatigue (see Results), peak joint torques relative to their initial values were used rather than absolute torques. Only data from the third unfatigued balance perturbation was used in the analysis because of possible learning effects within the three unfatigued perturbations [28].

To determine the effect of fatigue and fatigue level on postural strategy, two separate two-way repeated measures multivariate analysis of variance (MANOVA) were used. The first MANOVA used dependent measures quantifying preparatory activity prior to the perturbation, and the second used dependent measures quantifying strategy during recovery from the perturbation. Data from all four joints were included in each MANOVA with the independent variables being fatigue (two levels: unfatigued and
fatigued) and fatigue level (three levels: 86% MVC, 73% MVC, and 60% MVC). A significant main effect was followed by a two-way repeated measures analysis of variance (ANOVA) for each dependent variable. Correlation analysis was performed to determine the strength of any relationship between measures quantifying preparatory activity prior to the perturbation and measures quantifying strategy during recovery from the perturbation. A significance level of $p \leq 0.05$ was used for all tests.

To determine if perturbations were consistent across fatigue states, a two-way repeated measures MANOVA was also used to determine if perturbation peak force and impulse were affected by fatigue and fatigue level. Perturbation peak force decreased 4.3(6.4)% with fatigue ($p=0.013$), and there was no effect of fatigue level or fatigue × fatigue level interaction for either parameter.

A.3 Results

Although a fair amount of inter-subject variability was observed, a general pattern of joint angles and torques emerged across all subjects during unfatigued trials (Figure A.2). Prior to the perturbation, initial joint angles were approximately zero (i.e. anatomical position). Initial ankle and knee torques were plantar flexor and flexor dominant, respectively, while the initial hip and low back torques were more variable. Following the perturbation, the initial response was a flexor torque at the knee and extensor torques at the hip and low back. These torques were followed 10-30 ms later by a plantar flexor torque at the ankle and flexion at the knee, hip, and low back. Ankle plantar flexion began 20-80 ms after the ankle torque. The entire recovery duration was approximately 2 sec. The timing of joint angles and torques were consistent within the unfatigued trials and were not affected by fatigue, fatigue level, or their interaction.
Figure A.2: Representative joint angles and torques during recovery from an unfatigued postural perturbation. Positive values indicate extension movement (relative to anatomical position) and extensor torque. The perturbation time is indicated with a dotted line. The plots show the general patterns observed from the subjects.
Fatigued recoveries exhibited the same general pattern of joint angles and torques as in the unfatigued recoveries, but some specific differences existed. Prior to the perturbation, initial joint angles did not change with fatigue (p=0.441), but the COP position was 1.1(1.7) cm more anterior from the ankle compared to unfatigued trials (p=0.012). The anterior displacement of the COP during static stance suggests increased plantar flexor activity and necessitates a forward lean (the magnitude of which was evidently too small to be detected in joint angle measurements). As a result of this slight forward lean when fatigued, initial joint torques at all four joints changed with fatigue; offsets were in the direction of the maximum torque for each joint. More specifically, initial ankle torque shifted 7.1(1.1) Nm in the plantar flexor direction (p=0.006), initial knee torque shifted 4.8(3.8) Nm in the flexion direction (p=0.010), initial hip torque shifted 6.5(1.7) Nm in the extensor direction (p<0.001), and initial low back torque shifted 6.1(1.4) Nm in the extensor direction (p=0.001). There were no fatigue level effects or fatigue×fatigue level interactions for initial COP position, initial joint angles, or initial joint torques.

Both peak joint angles and torques after the perturbation were affected by fatigue (Figure A.3) and fatigue level. Fatigue increased peak ankle plantar flexion 2.5(0.5)° (p=0.002), did not affect peak knee (p=0.170) or hip (p=0.062) angles, and increased peak low back flexion 6.3(1.6)° (p=0.021). In addition, fatigue decreased peak ankle plantar flexion torque 3.2(2.1) Nm (p=0.002), did not affect peak knee torque (p=0.310), increased peak hip extensor torque 10.2(1.2) Nm (p=0.044), and increased peak low back extension torque 9.4(0.8) Nm (p=0.013). Fatigue level also had an effect on several dependent measures. Increasing fatigue level from 86% MVC to 60% MVC increased peak low back flexion 4.6(7.0)° (p=0.049), decreased peak ankle plantar flexor torque 5.8(6.4) Nm (p=0.030), decreased peak knee flexor torque 6.8(7.0) Nm (p=0.005), and decreased hip extensor torque 3.8(8.3) Nm (p=0.034). Fatigue level had no effect on peak low back torque (p=0.376), and there were no fatigue×fatigue level interactions for peak joint angles or torques.

Correlations between initial COP position and peak joint angles/torques revealed minimal covariance between these variables except for one peak torque value. Peak ankle torque exhibited a small but statistically significant correlation with initial COP.
position ($r^2 = 0.19; \ p<0.05$). The coefficient of determination for all other correlations were $r^2 = 0.01(0.01)$, and none were statistically significant.

Although all subjects exhibited the same general recovery patterns that are best described as a mixed strategy, subjects could be classified into two groups based on the amount of movement at the hip and ankle during unfatigued recoveries (Figure A.4). Through visual inspection of the unfatigued data, eight of the twelve subjects were found to move predominantly at the hip and low back and were classified as using more of a hip strategy. The remaining four subjects showed similar amounts of movement at the ankle, hip, and low back, and were classified as using a true mixed strategy. The effects of fatigue on postural strategy were more pronounced in the hip strategy group. For the hip strategy group, fatigue increased peak ankle plantar flexion 2.4(3.4)$^\circ$, increased hip flexion 5.2(8.0)$^\circ$, and increased low back flexion 8.4(11.0)$^\circ$. For the mixed strategy group, fatigue increased peak ankle plantar flexion 2.9(2.8)$^\circ$, increased hip flexion 1.9(2.1)$^\circ$, and increased low back flexion 3.5(3.9)$^\circ$, respectively.
Figure A.3: Representative joint angle and joint torque data during unfatigued and fatigued postural recoveries. Positive values indicate extension movement (relative to anatomical position) and extensor torque. Joint angle plots show increases in peak ankle plantar flexion and peak low back flexion with fatigue. Joint torque plots show initial torque offsets and increases in ankle, hip, and low back extensor torques with fatigue. Figures in the middle illustrate differences in unfatigued and fatigue recoveries at the instant of maximum hip flexion.
Figure A.4: Differences in peak joint angles and torques between the hip and mixed group. Both line plots show a greater affect of fatigue on the hip group as compared to the mixed group. The stick figures show the general patterns of movement for both groups. The dotted figure is the initial (anatomical) position while the solid figure is the maximum displacement from the initial position following the perturbation. Standard deviation bars were omitted for clarity, but these data are available from the authors.

A.4 Discussion

Neuromuscular fatigue has been shown to increase postural sway during quiet standing [14-20]. The increase in sway may, in part, be the result of a change in postural control strategy. The purpose of the present study was to investigate the effects of lumbar extensor fatigue and fatigue level on postural strategy in response to a balance perturbation. Our results showed both proactive and reactive changes in postural strategy with fatigue. For the purposes of this report, proactive changes are operationally defined to be changes occurring prior to the perturbation, and reactive changes are the changes occurring after the perturbation. Proactive changes involved adopting a slight forward lean prior to the perturbation as evidenced by an anterior shift in the COP, while reactive...
changes indicate a shift toward hip strategy. Increasing fatigue level exaggerated some, but not all, of these changes. Interestingly, the changes were not limited to the fatigue location.

The proactive change in postural strategy involved a 1.1(1.7) cm anterior displacement of the COP prior to the perturbation. Although a post hoc analysis of the angle between the ankle and whole-body center-of-mass (COM) prior to the perturbation did not show a significant increase with fatigue, mechanics necessitates at least a slight forward lean due to the COP shift.

In both unfatigued and fatigued recoveries, subjects generally used a mixed strategy; however, reactive changes with fatigue showed a shift toward more of a hip strategy. Changes in peak torques indicated a clear shift toward a hip strategy, as evidenced by a 3.2 Nm decrease in plantar flexor torque and a 10.2 Nm increase in hip extensor torque. The 2.5° increase in peak ankle plantar flexion is consistent with this shift, since the hip strategy involves simultaneous hip flexion and ankle plantar flexion. Although the increase in peak hip flexion was only a trend (p=0.062), peak low back flexion increased 6.3°, which provides a similar benefit to recovery as hip flexion by attempting to reduce the body moment of inertia about the ankle [4]. Taken together, we feel that these results indicate a shift toward more of a hip strategy with lumbar extensor fatigue. It was also interesting to note that peak low back extension torque increased 9.4 Nm despite subjects being fatigued.

According to past studies [5], the change in reactive postural strategy could be partially attributed to the forward lean. However, three arguments suggest otherwise. First, low correlations between initial COP position and peak joint angles/torques indicate a weak relationship between these measures. Second, a follow-up analysis was performed by repeating our statistical analysis on a subset of participants whose change in initial COP with fatigue was less than the overall mean of 1.1 cm (7 participants). This subset of participants showed no change in initial COP position with fatigue (p = 0.286), and significant changes in reactive measures with fatigue that were similar to the overall analysis: peak ankle angle increased (p = 0.046), peak hip extensor torque increased (p = 0.031), and peak low back extensor torque increased (p = 0.012). Third, comparing lean angles in the present study with those of another study suggest that the leans here were
too small to elicit a change in recovery strategy. Horak and Moore [6] reported that a hip strategy is favored when pre-leaning in the same direction as an induced sway. However, this shift in strategy was only found when subjects assumed a maximum forward lean (an average 6.09° from normal standing posture). No change in strategy was found when subjects assumed a half-maximum forward lean (an average 3.01°). The average forward lean in our study was 0.25°, which is much smaller than either of the leans reported. For these reasons, we do not attribute the reactive changes in postural strategy to the forward lean prior to the perturbation.

Reasons why a shift toward more of a hip strategy and an increase in forward lean were observed with lumbar extensor fatigue remain to be explained. In regards to the forward lean, we feel it is unlikely that lumbar extensor fatigue directly elicits an increase in plantar flexor activity without any other biomechanical or physiological explanation. One reason for the lean, independent of fatigue, may be that subjects unintentionally leaned forward with repeated exposure to the balance perturbation. Such a habituation has been reported [8]. This habituation, however, seems to occur mostly in the first few perturbations [28], and we allowed 3 unfatigued perturbations to allow for habituation prior to fatiguing subjects. Also, there was no difference in COP initial position between the 2nd and 3rd unfatigued perturbations (p=0.907), suggesting that habituation had already occurred. Another potential reason is related to motor control. Lumbar extensor fatigue has been associated with an increase in antagonist muscle activity in the rectus abdominus muscle (RA) [29]. We analyzed muscle activity data from 5 subjects that was collected for a separate study and found baseline RA activity increased an average of 54% with lumbar extensor fatigue prior to the perturbation (p=0.033). Forward leaning has also been associated with an increase in RA activity [6]. The increase in RA activity prior to the perturbation may be a part of a subtle adaptation in postural control that displaced the COP forward. Interestingly, Runge et al. [4] reported higher hip torques (i.e. more of a hip strategy) during perturbation recoveries that involved elevated RA activity (although their perturbation method involved a moving platform rather than a trunk impulse). Thus, an increase in RA activity from lumbar extensor fatigue has the potential to contribute to both the forward lean prior to the perturbation and the shift toward more of a hip strategy in response to the perturbation. The last potential
explanation is attractive because it links the effects of lumbar extensor fatigue with both the proactive and reactive changes in postural strategy observed.

It seems reasonable that increasing levels of fatigue would lead to larger influences on postural strategy. Such a dose-response relationship implies that increasing fatigue level would exaggerate the effect of fatigue. This was the case with peak low back flexion, peak ankle plantar flexor torque, and peak knee flexor torque. The decrease in ankle movement and increase in low back movement is consistent with more of a hip strategy. This was not the case, however, with peak hip extensor torque. Fatigue increased peak hip extensor torque, but increasing fatigue level from 86% MVC to 60% MVC decreased peak hip extensor torque an average of 3.85 Nm. In other words, increasing fatigue level decreased the effect of fatigue on the hip. This may reflect a continuous adaptation of postural strategy with increasing level of fatigue. It is interesting to note that the only joint that did not exhibit a change in peak torque with increasing level of fatigue was the joint that was fatigued.

It appears that even in the absence of any influencing factors, differences in preferred postural strategies exist between individuals. Subtle differences in peak joint angles during unfatigued perturbations allowed subjects to be categorized into two strategy groups (Figure A.4). Horak and Nashner [30] similarly reported that some subjects employed pure ankle or hip strategies while others used a mixed strategy even after 20-40 trials. In the present study, there was a tendency for subjects using more of a hip strategy when unfatigued to be more heavily influenced by fatigue compared to subjects using a mixed strategy when unfatigued. These data suggest that an individual’s preferred postural strategy may dictate how much they are affected by certain influencing factors. Postural strategy may be related to postural “performance” and the ability to prevent a fall after a perturbation, but future work is needed to establish this relationship.

Several methodological issues warrant discussion. First, one may question whether the measured changes in postural strategy were due to fatigue or the 4.3% decrease in perturbation amplitude. Runge et al. [4] reported greater shifts toward hip strategy with increasing perturbation amplitude (i.e. increases in platform velocity). Our perturbation amplitude decreased with fatigue by an average of 8 N (decrease in perturbation force). Because of this, we feel that if perturbation amplitude did not
decrease with fatigue, the measured shift toward hip strategy would have been larger. The reason for this change in perturbation amplitude with fatigue is beyond the scope of this study, but may be related to a decrease in trunk stiffness with lumbar extensor fatigue. Second, one may question whether the measured changes in reactive strategy were due to changes in initial COP position. While we cannot rule this out, we feel the low correlations between the initial COP position and reactive measures, along with the result from our follow-up analysis (individuals who had no change in initial COP position still exhibited a shift toward hip strategy with fatigue), provide strong evidence supporting the fact that almost all of the reactive changes were not due to changes in initial COP position. Third, all learning affects were assumed to have happened during the unfatigued balance perturbations. Fourth, we acknowledge that our relatively low marker sampling rate (50 Hz) may have limited our ability to identify subtle changes in postural strategy temporal characteristics.

In conclusion, lumbar extensor fatigue elicited a change in postural strategy in response to a perturbation. Both proactive and reactive postural compensations were identified and show a general shift toward hip strategy with fatigue. These findings illustrate that neuromuscular fatigue can influence postural strategy similar to previously reported intrinsic factors. Future studies are needed to determine how these strategies affect the ability to maintain balance following a postural perturbation.

A.5 References


A.6 Supplemental Material II

Data Collection Sheet

__________________________

__________________________

FATIGUE LEVEL STUDY

Setup Checklist

☐ 1. Turn on: (allow 30 minutes to warm up)
   - Vicon datastation
   - force platform amplifier
   - load cell amplifiers
   - EMG amplifier
   - DAQPad-6020E

☐ 2. Open Excel Spreadsheet (MVC Regression.xls) for fatigue protocol and save as
    subjectname.xls in subject folder.

☐ 3. Open load cell–emg.vi for EMG collection and real-time load cell viewing

☐ 4. Open workstation software and create Vicon directory with subject name and session number

☐ 5. Get out supplies:
   - EMG electrodes
   - Co-flex
   - alcohol
   - athletic tape
   - razor
   - bathroom scale
   - reflective markers (17)

☐ 6. Calibrate Vicon Cameras
   Make sure calibration is adequate:
   System > Calibrate cameras
   wand visibility > 70 %
   static reproducibility < 1.0%

☐ 7. Check Vicon data collection setting
   System > Video Setup
   video sampling rate: 100Hz
   System > Analog Setup
   analog sampling rate: 1000 Hz
   collection channels 1-7 (apply to current session)

☐ 8. Make sure that forceplate is not in contact with platform

Erin L. Wilson

Main Appendix: Postural Strategy

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FATIGUE LEVEL STUDY
Data Collection Sheet

Subject name: ________________________________ Date: _______________
Session # _____ Fatigue Level: _________ Start time: _________

1. Give subject proper clothing in which to change (shorts, tank top) and ask if they need to use the restroom
2. Give summary of protocol and demonstrate
3. Obtain informed consent
4. Ask subject to remove shoes
5. Collect anthropometric measurements:
   - age: _______ weight: _______ lbs height: _______ cm
6. Position subject on forceplate leaving a 10 cm gap from back to resting pendulum. Mark toe line and edges of base of support with tape and record dimensions position of front left corner of base of support for Time-to-Boundary calculations:
   - width: _______ cm length: _______ cm
   - _______ cm from posterior edge _______ cm from left edge
7. Adjust pendulum height Record height: _______
8. Adjust height of roman chair Record height: _______
9. Demonstrate balance measurement with pendulum
10. Ask subject to replace shoes
11. Place harness loosely onto subject
12. Apply EMG electrodes:
   - reference electrode (head of fibula)
   - erector spinae (4 cm lateral to L3 – even with iliac crest)
   - internal oblique
13. Apply reflective markers:
   - right shoulder right iliac crest
   - right back
   - right elbow (radial) right greater trochanter
   - right wrist (ulnar) right lateral epicondile
   - left shoulder left iliac crest
   - left elbow (radial) left greater trochanter
   - left wrist (ulnar) left lateral epicondile
14. Warm-up exercise (2 sets)
   - 2 ½ minutes jogging at 4.5 mph
   - back stretching
   - 5 back extensions
15. Ask subject to remove shoes and socks
16. Apply remaining reflective markers
   - right temple right maleolus
   - left temple left maleolus
   - forehead right 5th metatarsal
   - back of head left 5th metatarsal
17. Connect subject to EMG preamplifier
18. Check for EMG signal using LabView
19. Tighten harness
20. Perform MVCs: (LabView collection)
   - erector spinae
     - load cell max: ______ volts ______ volts ______ volts
       - filename: EMG ___ gain setting: ___
   - internal oblique
     - filename: EMG ___ gain setting: ___
   - erector abdominus
     - filename: EMG ___ gain setting: ___
   - external oblique
     - filename: EMG ___ gain setting: ___
21. Apply heel markers
22. Enter erector spinae MVC voltage into Excel spreadsheet
    record weight to be used for box lift (20% of MVC load cell force): ______ lbs
23. Put weight into box
24. Zero forceplate amplifier
25. Collect static marker calibration
    (trial type: subject calibration)
    - filename: collection __ comments: _________________________
26. Label subject markers in Vicon (trial > create auto label calibration, save trial)
27. Collect unfatigued balance (dynamic)
    (trial type: repeated capture)
    - filename: collection __ comments: _________________________
    - filename: EMG __ comments: _________________________

One minute rest
Collect unfatigued balance (dynamic)
    - filename: collection __ comments: _________________________
    - filename: EMG __ comments: _________________________

One minute rest
Collect unfatigued balance (dynamic)
    - filename: collection __ comments: _________________________
    - filename: EMG __ comments: _________________________

One minute rest
Collect unfatigued box lift time: 5 sec
    - filename: EMG __ comments: _________________________
28. Start metronome (30 beats per minute)
29. Begin fatiguing protocol
Fatiguuing Protocol

<table>
<thead>
<tr>
<th>Time</th>
<th>% MVC</th>
<th>Reps per minute</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 : 00</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 :</td>
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<td>13 :</td>
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<tr>
<td>15 :</td>
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</tbody>
</table>

**Linear fatigue protocol:**

- Extensions will be done through full 45° range of motion at a rate of approximately 30 reps/min
- Begin with number of repetitions every minute (rem) specified below
- Final MVC defined as two consecutive MVC falling below desired %
- MVC will be measured throughout protocol at exactly 10 seconds after completing every other set using load cell and compared to linear regression of target MVC calculated in Excel Spreadsheet `MVC regression.xlsx`

<table>
<thead>
<tr>
<th>Goal MVC</th>
<th>Beginning # of Reps</th>
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<tbody>
<tr>
<td>86.6 %</td>
<td>5</td>
</tr>
<tr>
<td>73.3 %</td>
<td>9</td>
</tr>
<tr>
<td>60 %</td>
<td>11</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in repetitions</th>
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</thead>
<tbody>
<tr>
<td>Above 5 %</td>
</tr>
<tr>
<td>+3</td>
</tr>
</tbody>
</table>

****Remind subject to keep feet flat and move through full range of motion.****
Collect fatigued balance (dynamic)
filename: collection __
filename: EMG __

Collect fatigued box lift time: 5 sec
filename: EMG __

Collect balance and lifting data every 3 minutes for 30 minutes
note: subject should remain standing at all times

3 min:
Collect fatigued balance (static only)
filename: collection __
filename: EMG __

6 min:
Collect fatigued balance (dynamic)
filename: collection __
filename: EMG __
Collect fatigued box lift time: 5 sec
filename: EMG __

9 min:
Collect fatigued balance (static only)
filename: collection __
filename: EMG __

12 min:
Collect fatigued balance (dynamic)
filename: collection __
filename: EMG __
Collect fatigued box lift time: 5 sec
filename: EMG __

15 min:
Collect fatigued balance (static only)
filename: collection __
filename: EMG __

18 min:
Collect fatigued balance (dynamic)
filename: collection __
filename: EMG __
Collect fatigued box lift time: 5 sec
filename: EMG __

21 min:
Collect fatigued balance (static only)
filename: collection __
filename: EMG __

24 min:
Collect fatigued balance (dynamic)
filename: collection __
filename: EMG __
Collect fatigued box lift time: 5 sec
filename: EMG __

27 min:
Collect fatigued balance (static only)
filename: collection __
filename: EMG __
30 min:
Collect fatigued balance (dynamic)
filename: collection __ comments: _________________________
filename: EMG __ comments: ____________________________
Collect fatigued box lift time: 5 sec
filename: EMG __ comments: ____________________________

☐ 33. Offer subject a chair to rest
☐ 34. Record end time: ______
☐ 35. Remove EMG, harness, and reflective markers from subject.
   Give them stipend and have them sign payment sheet.
   Thank them, and see them to the door.
☐ 36. Mark Vicon files for batch process and export to ASCII format through ‘pipeline’
☐ 37. Transfer Vicon files to network drive
☐ 38. Turn off equipment:
   - Vicon system
   - force platform amplifier
   - load cell amplifier
   - EMG amplifier
   - DAQPad-6020E
☐ 39. Reapply adhesive to reflective markers
☐ 40. Clean up the laboratory

General Comments:
______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________
______________________________________________________________________________

Erin L. Wilson
Main Appendix. Postural Strategy
ERIN L. WILSON

Erin L. Wilson was born in Newark, Delaware on May 8, 1982. She attended Rising Sun High School in North East, Maryland and graduated in 2000. She attended Virginia Polytechnic and State University where she received a Bachelor of Science in Engineering Science and Mechanics in 2004. She also completed a minor in mathematics and an option in biomechanics. Continuing in a 5 year BS/MS program, she completed her Master of Science at Virginia Polytechnic and State University in Biomedical Engineering. The focus of her research was on the biomechanical analysis of balance and sports injuries. Her main projects investigated the effects of fatigue and gender on ankle reflexes as well as how low back fatigue affected recovery from a perturbation. Erin has two loving parents, Richard and Nicolette, as well as a twin sister Lauren who unfortunately attended the University of Maryland. In her free time she enjoys reading, hanging out with all her Hokies, attending football games, grilling, hiking, and soaking up as much sun as possible.