Comparison of Muscle Latencies for Diabetic Neuropathy Patients Versus Healthy Controls During a Perturbed Balance Task

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COMPARISON OF MUSCLE LATENCIES FOR DIABETIC NEUROPATHY PATIENTS VERSUS HEALTHY CONTROLS DURING A PERTURBED BALANCE TASK

By

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Bachelors of Science in Kinesiology
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ABSTRACT

COMPARISON OF MUSCLE LATENCIES FOR DIABETIC NEUROPATHY PATIENTS VERSUS HEALTHY CONTROLS DURING A PERTURBED BALANCE TASK

by

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The purpose of this study was to compare muscle latencies for patients diagnosed with diabetic neuropathy versus healthy controls during a perturbed balance task, with a secondary purpose to distinguish postural control strategies the groups used based on the muscle latencies. Five participants diagnosed with diabetic neuropathy (DN; 4 male, 99.7 ± 7.95 kg, 176 ± 9.58 cm, 46.6 ± 16.55 years) and 5 healthy control (HC; 4 male, 100.36 ± 12.61 kg, 173.76 ± 9.66 cm, 47 ± 13.42 years) participants were recruited. Participants granted institutionally approved written consent before participating. Delsys Trigino Wireless EMG sensors were placed on the following muscles on each subject’s right side of their body: tibialis anterior, gastrocnemius medialis, rectus femoris, biceps femoris, rectus abdominis, and lumbar paraspinals at the iliac crest. Subjects performed three trials of a perturbed balance task (SOT VI). EMG data were analyzed in MatLab using custom written script. Latency was determined as the time from the perturbation to the time when the smoothed EMG data exceeded two standard deviations above the baseline for at least 50 ms. Dependent variables (latency for each muscle) were evaluated between groups using a paired t-test (SPSS Statistics 20; IBM; Armonk, NY). Single subject (SS) analysis between matched participants was performed using Microsoft Excel. No statistically
significant differences ($p > 0.05$) were observed between the groups for any muscle latency. Single subject analysis identified differences ($p < 0.05$) between some matched pairs with no distinguishable trend or pattern observed. Similar balance strategies based on muscle latency were observed between groups. The results of this study may be explained with current theory that has challenged the pathophysiology of DN especially regarding whether DN affects the motor system, and if DN may be attenuated by exercise. The results of this study continue to shed light on the complexity of DN.
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Lastly, to my future wife Chrisabelle Cempron I thank you for your love and patience. I would be a different man if not for you. I am guided by your light and will always be there for you. Your love is only trumped by one. *Soli Deo Gloria pro eo Fide Et Ratio.*

“All we have to decide is what to do with the time that is given us.”

— J.R.R. Tolkien, The Fellowship of the Ring
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INTRODUCTION

According to the Center for Disease Control and Prevention (CDC) in 2011 11.3% of adults age 20 or older have diabetes. Diabetes is also the leading cause of amputation and the seventh leading cause of death in the United States (National Diabetes Fact Sheet, 2011). Naturally, it would appear the number of Americans diagnosed each year with diabetes is increasing and many studies have been performed to combat the disease both in the areas of disease prevention and in injury prevention for those already diagnosed (Diabetes Public Health Resource, 2012).

Diabetic neuropathy (DN) is the medical term for damage caused by diabetes. Having high blood glucose over an extended period of time can damage blood vessels that bring oxygenated blood to some nerves (Diabetes Public Health Resource, 2012). These damaged nerves may not fire in synchronization with other nerves, may not fire at all, or may fire too slowly. People that suffer from DN typically have symptoms that include numbness, pain, and weakness in the hands, arms, feet, and legs with the most common being peripheral neuropathy affecting the arms and legs. Such symptoms in the legs can lead to postural instability (Diener et al., 1984; Boucher et al., 1995; Hurvitz & Richardson, 1995; Simmons et al., 1997; Yamamoto et al., 2001).

In many studies where the postural sway was analyzed in reference to a healthy control group or a diabetic group not diagnosed with DN, participants diagnosed with DN were found to have greater postural sway (Bonnet et al. 2009). Participants diagnosed with DN are therefore thought to have greater postural instability. Boucher et al. (1995)
demonstrated that DN patients showed wider ranges of sway, a faster sway speed, and a greater distribution of sway than did healthy control subjects under all conditions. Participants diagnosed with DN also showed similar or less stable postural performance with vision than healthy subjects without vision. A strong correlation between the severity of the neuropathy and the postural stability was demonstrated and this study showed that even with vision, the postural stability of neuropathic patients is impaired and may put them at higher risk of falling. It has been suggested that diabetic patients with neuropathy incur the risk of potential morbidity caused by falls, irrespective of age (Yamamoto et al. 2001).

Cavanagh et al. (1992) reported DN patients have an increased risk of injuries and falls by a factor 15. With older adults naturally having a greater risk of falling, and a greater risk of injury from a fall, it would seem pertinent to understand what balance strategies the DN population utilizes in order to stabilize themselves, relative to fall prevention. Simmons et al. (1997) utilized the Sensory Organization Test (SOT) using a Neurocom apparatus in an attempt at distinguishing whether DN patients use hip or ankle balance strategies. The results indicated hip strategies were used more during the two most difficult conditions of the SOT however the mechanism of control was not specified. Therefore, further investigation into balance strategies employed by DN patients, focusing on muscle function, is warranted.

**Purpose of the Study**

The purpose of this study was to compare muscle latencies for patients diagnosed with diabetic neuropathy versus healthy controls during a perturbed balance task. A
secondary purpose for this study was to distinguish postural control strategies the groups used based on the muscle latencies.

**Research Questions**

Is there a difference between groups in right leg and trunk muscle latencies of the tibialis anterior (TA), gastrocnemius medialis (GAS), rectus femoris (QUA), biceps femoris (HAM), rectus abdominis (ABS), and lumbar paraspinals at the iliac crest (PAR) muscles during condition VI of the sensory organization test (SOT)? The following muscles were chosen for their known function in balance strategies about the hip and ankle (Runge et al., 1999).

The following is a brief description of the SOT VI condition used. (SOT VI): subjects will have their eyes open and stand on a sway referenced surface while the visual surround moves.

**Significance of the Study**

Little is known about the postural strategies, whether hip or ankle, are invoked by persons with diabetes. Understanding how the DN population balances themselves in general can lead to greater fall prevention understanding. Therefore, this study aims to investigate the differences in muscle latencies. Investigating muscle latencies may contribute to knowledge of balance strategies used between diabetic neuropathy patients versus healthy equivalents.
Statistical Hypothesis

Null hypothesis I

- There will be no difference between groups for right leg and trunk muscle latencies (TA, GAS, QUA, HAM, ABS, or PAR) during the condition VI of SOT.

Alternate hypothesis I

- There will be a difference in muscle latencies between the groups during condition VI of SOT.

Null hypothesis II

- There will be no difference between groups in chosen postural control strategies.

Alternate hypothesis II

- There will be a difference between groups in chosen postural control strategies.

Variables

1. Independent Variable:

- Two levels: (1) Diabetic neuropathy group and (2) healthy control group

2. Dependent Variables:

- Muscle latency of the TA, GAS, QUA, HAM, ABS, or PAR muscles during SOT condition VI
**Definitions and Terms**

**Ankle Strategy**: Horak and Nashner (1986) characterized ankle strategy by early activation of posterior ankle muscles followed by activation of posterior thigh and trunk muscles.

**Hip Strategy**: Hip strategy was characterized by early activation of anterior trunk and thigh muscles associated with a relative increase of shear forces at the support surface (Horak and Nashner, 1986).

**Sensory Organization Test (SOT)**: Sensory organization (sensory integration; multisensory organization) is the ability of an individual to effectively process individual sensory system (somatosensory & vestibular) input cues to maintain balance control.

**Limits of Sway (LOS)**: It has been proposed that static balance is maintained if the COG is positioned within an area described as an inverted cone, with the apex of the cone centered under the feet and the open end forming an ellipsoid shape of 12.5° in the A–P direction. The maximum extent of the ellipsoid boundary has been termed the limits of sway (LOS). See Figure 1.

**Center of Pressure (COP)**: The point where the resultant of all ground reaction forces act.

**Center of Gravity (COG)**: Geometric center of the body.
Figure 1: Limits of Stability: The maximum extent of the ellipsoid boundary has been termed the limits of stability (LOS), an inverted cone, with the apex of the cone centered under the feet and the open end forming an ellipsoid shape of $12.5^\circ$ in the A–P direction.
CHAPTER 2

REVIEW OF RELATED LITERATURE

Diabetic Neuropathy and How it Affects The General Populace

Peripheral diabetic neuropathy (DN) is the most insidious chronic complication of diabetes. It usually leads patients to a progressive loss of their somatosensory sensitivity, proprioception, and muscular function (Sacco & Amadio 2003). DN is the medical term for damage caused by diabetes. An individual with high blood glucose, over an extended period of time, can damage blood vessels that bring oxygenated blood to some nerves (Diabetes Public Health Resource, 2012). These damaged nerves may not fire in synchronization with other nerves, may not fire at all, or may fire too slowly. People that suffer from DN typically have symptoms that include numbness, pain, and weakness in the hands, arms, feet, and legs (also known as “glove in stocking distribution”) with the most common being DN affecting the arms and legs.

DN is known to cause muscle weakness and loss of reflexes, especially at the ankle, leading to an altered gait (Diabetic Neuropathies: The Nerve Damage of Diabetes, 2012). Because of the lack of sensation due to nerve damage, unnoticed blisters and sores may appear on the foot because pressure or injury is not felt by the individual. If these injuries are not treated quickly infection may spread to the bone resulting in possible amputation of the foot.

According to the Centers for Disease Control and Prevention (CDC) approximately 50% of people diagnosed with diabetes have some form of neuropathy although some may not experience symptoms. At any point in time people with diabetes
may undergo nerve damage. The longer a person has diabetes the more at risk they are to develop some kind of neuropathy. Some people are at greater risk than others for developing DN including: those who have had diabetes for more than 25 years, people who have problems controlling their blood glucose levels, people with high blood fat content or hypertension, people who are overweight, and those over the age of 40 (Diabetes Public Health Resource, 2012).

The number of Americans diagnosed with diabetes has increased every year from 1958 to 2011. In 2011, 25.8 million (8.3%) people were diagnosed with diabetes and approximately 7 million undiagnosed. Number and percent of the U.S. population with diagnosed diabetes were obtained from the National Health Interview Survey of the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention (CDC) for years. With the augmenting diabetic populace may come a variety of problems for this population – one such complication may include a tendency to fall relative to the healthy population (Simmons et al., 1997; Richardson, Ching & Hurvitz, 1992; Cavanaugh et al., 1992; Yamamoto et al., 2001).

**Diabetic Neuropathy and Postural Stability: What We Know**

A general observation for people with diabetes is that they have greater postural sway, especially if they have DN (Simmons et al., 1997; Richardson, Ching & Hurvitz, 1992; Cavanaugh et al., 1992; Yamamoto et al., 2001). In particular, the static balance and dynamic balance have been studied in the diabetic population. A great number of these studies yielded results indicating participants diagnosed with DN had increased postural sway and instability.
Postural instability was found to be significantly associated within participants diagnosed with DN. However, diabetes in itself does not appear to have an effect on postural stability (Simoneau et al. 1994), but diabetic patients diagnosed with DN were reported to demonstrate more instability than healthy control subjects or diabetic patients alone (Uccioli et al. 1995). A strong correlation of instability in type 1 diabetic patients with peripheral neuropathy by posturography has been demonstrated (Uccioli et al., 1995; Giacomini et al., 1996; Uccioli et al., 1997). Simoneau et al. (1994) showed that the most significant correlation of instability is with the quantitative sensory measures of DN and age. Yamamoto et al. (2001) concluded that type 2 diabetic patients with DN also show significantly greater body instability than healthy patients without neuropathy. In diabetic patients with a long history of severe DN, the degree of instability is expected to be greater than in non-diabetic subjects (Yamamoto et al. 2001).

It has been suggested that static balance is maintained if the Center of Gravity (COG) is positioned within an area described as an inverted cone, with the apex of the cone centered under the feet and the open end forming an ellipsoid shape of 12.5° in the A–P direction (Nashner, 1993 from The of Balance Function Testing). The maximum range of the ellipsoid boundary has been labeled the limits of sway (LOS). An increase in body sway under static and dynamic conditions puts the DN patient at increased risk of exceeding the LOS and possibly at an increased risk of falling (Simmons et al., 1997).

Participants Diagnosed with DN and Falling

Diener et al. (1984) noted an increase in postural sway at low frequencies of perturbation in young healthy subjects with ischemically induced loss of ankle
proprioception but preserved strength. Thus, there is evidence that DN (whether inherited, acquired, or experimentally induced) significantly affects balance with and especially without vision.

Richardson, Ching & Hurvitz (1992) examined 25 pairs of age and sex matched subjects. They were interviewed to gather information about falling during the previous year. The results indicated that the DN group was 23 times more likely to fall than the controls. Other factors that have been associated with falls were not significantly different between the two groups. In a similar study by Cavanaugh et al. (1992) two groups of Type I diabetics with and without diabetic neuropathy were compared. Cavanaugh’s group found that the DN group was 15 times more likely to report an injury as the result of a fall and felt significantly less safe during standing and walking than healthy matched non-neuropathic subjects adjusted for gender, duration of diabetes, and retinopathy. These facts are strongly suggestive of the effect of neuropathy on posture and balance.

Uccioli et al. (1995) found the role of the peripheral nerve system is important in controlling body sway while analyzing static posture. Here, young DN patients were compared to healthy controls (HC) and diabetic patients without DN using posturography. A trace of the subject’s sway was found to be significantly large in the DN group versus HC and the diabetic patients without DN. Posturography was shown to be a valid tool in measuring postural control.

Boucher et al. (1995) demonstrated that patients with DN showed wider ranges of sway, a faster sway speed, and a greater distribution of sway than did healthy control subjects under all conditions (balancing on a force platform). They also exhibited similar
or less stable postural control with vision than that of healthy control subjects without vision. There was a strong correlation between the severity of the DN and the postural stability. This study shows that even with vision, the postural stability of neuropathic patients is impaired and may put them at higher risk of falling. Diabetic patients with neuropathy seem to have greater risk of potential injury caused by falls regardless of age.

Hurvitz & Richardson (1995) found DN to be significantly associated with self-reported of falls (11/20, 55% DN group vs. control group 2/20, 10%) and postural instability (7/9, 77% DN group vs. control group 0/0, 0%) over the previous year. In this study, a fall was defined as some portion of the subject’s body unintentionally coming into contact with the ground. Similar results can be seen in an analysis by Simmons et al. (1997) who indicated that sixteen DN patients recorded one or more falls during their sensory organization test (SOT) for a total of 29 trials with a loss of balance (a fall was recorded when the patient lost balance or moved their feet). This figure was significantly different from a total of five falls observed for the healthy control group. Diabetic patients with cutaneous sensory deficit in the foot exhibited significantly poorer equilibrium in comparison to control subjects (Simmons et al. 1997). These results indicate that significant balance loss associated with cutaneous deficit in the foot places the patient at increased risk for falling.

Oppenheim et al. (1999) performed a study with results indicating for positions with eyes closed, diabetic patients with severe and moderate neuropathy were significantly less stable than normal subjects and diabetic patients without neuropathy. Diabetic patients with severe and moderate neuropathy turned out to be as equally unstable as clinical control trials; clinical control group in this study consisting of 52
patients (14 with stage II Parkinson’s disease, 13 with brain damage, 7 with whiplash, and 19 with peripheral vestibular pathology). Moreover, Oppenheim et al. (1999) showed that patients with DN had significantly less stability during quiet stance and while their head was turned to the right or left compared to the clinical control group subjects.

Yamamoto et al. (2001) suggested results indicating there is strong evidence that DN patients demonstrate a less than normal ability to maintain posture. Their data revealed that DN plays an essential role in the instability in type 2 diabetic patients. Educational programs in order to avoid dangerous situations such as falls or injuries related to postural instability are necessary, especially for older DN patients, who should take precautions against the increased risk of falls, and perhaps require better understanding of the strategies DN patients use during instability.

**Diabetic Neuropathy and Balance Strategies**

According to Simmons et al. (1997) a decline in the LOS would also explain the observed shift in strategy usage from a largely ankle based adjustment in balance to a hip correction as the difficulty of maintaining balance increases, specifically during the SOT conditions V, where the patient cannot see and must rely on their vestibular system to balance, and IV, where the patient relies on the preference of their visual or vestibular system to balance. Typically the healthy controls combined high strategy scores, indicating predominantly ankle strategy usage, with high equilibrium scores. The DN subjects had significantly lower equilibrium scores in comparison to control subjects, indicating the ankle strategy was insufficient or restricted in controlling sway. Similar results were found by Muritz & Dietz (1980) and Giacomini et al. (1996) where subjects
had an induced cutaneous sensory deficit similar to that found in DN patients using ischemic techniques and for young DN patients. With a strong causality between DN and postural instability, how do participants diagnosed with DN correct balance or what strategies might they take?

Nashner and McCollum (1985) hypothesized the possibility of two discrete strategies that could either be used independently, or combined, by the nervous system to produce adaptable control of the horizontal position of the center of mass (COM) in the sagittal plane. The ankle strategy repositioned the COM by moving the whole body as a single-segment inverted pendulum by production of torque at the ankle. The hip strategy, in contrast, moved the body as a double-segment inverted pendulum with counterphase (an equal force in the opposite direction) motion at the ankle and hip. They also suggested in situations that limit the effectiveness of ankle torque at producing whole-body motion, hip strategy should be observed. Observations in a study by Horak and Nashner (1986) were consistent with this, showing that an ankle strategy was used to respond to translations during stance on a flat support surface (this was large enough for the participants diagnosed with DN feet to fit appropriately on) that moved backward, while hip strategy was witnessed during responses to backward translations during stance on a narrow 10 cm beam. Nashner and colleagues (1986) also predicted that mixed hip and ankle strategies would be observed in response to fast translations of a flat support surface.

Horak et al. (1990) showed subjects with insufficient information about the characteristics of their support surface due to somatosensory loss induced by ischemia, similar to that of Muritz & Dietz, 1980, responded to postural displacements with
increased shear forces at the surface, excessive hip movements, and increased proximal hip muscle activation. In addition, findings suggest that DN, which also affects large proprioceptive afferent fibers as well as smaller somatosensory afferent fibers, can result in large delays in EMG responses to surface displacements (Horak et al., 1990). These results suggest that proprioceptive inputs from the ankle joint muscles are primarily responsible for stimulating the initial postural responses to surface displacements while standing on a large, firm surface (Horak et al., 1990). Also, these results suggest that sensory information from the vestibular system and from somatosensory afferent fibers in the feet and ankles plays an important role in determining the availability of postural strategies.

In particular experimental conditions Horak & Nashner (1986) and Moore et al., (1986), associated each movement strategy with “stereotypical” muscle activation patterns. The ankle strategy was associated with a distal-to-proximal pattern of ankle-knee-hip muscle activation posteriorly while the hip strategy was associated largely with proximal, hip muscle activation anteriorly, although it should be mentioned that complex combinations of these two patterns have been seen (McCollum et al., 1984). Horak and Nashner (1986) concluded for a given environmental context, such as length of support base, there is a mechanical boundary within which the most optimal postural movement pattern is an ankle strategy and beyond which is a hip strategy. They suggest that in situations in which both the ankle and hip strategy are insufficient, subjects will use a "stepping or stumbling strategy" in which the base of support moves under a falling center of mass (Horak et al., 1990).
To further investigate this a model will be explored; developed by Kuo & Zajac (1993) who stated when a subject’s main goal is maintaining upright body alignment, the model predicted the use of ankle strategy, defined by Kuo as movement at the ankle joint without significant movement at the hip joint, to control posture. However, when either the goal of stability was optimized or the perturbation was large, thus requiring a fast, high-amplitude response, the model predicted the use of hip strategy, defined as the combined use of ankle and hip accelerations, to respond to postural perturbations on flat support surfaces. Based on the expectations of Kuo’s (1993) optimization model the hip strategy appears to require less muscle activity than ankle strategy to effect the same COM movement on a flat surface. The model suggests therefore that the choice of postural strategy depends both on the postural goal and on the environmental constraints (Kuo & Zajac, 1993). It should be noted this model is limited by the constraints of keeping the foot in contact with the floor and the keeping knee straight.

Similar to that of Horak and Nashner (1986), Kuo and Zajac (1993) found indications that biomechanical and control constraints play a role in forcing selection of strategies. Not only does the surface play a role but as perturbations increase in size, subjects place greater reliance on the hip strategy, which also appears to be more effective in stabilizing the COM than the ankle strategy.

To further explore the role of postural balance strategies, the effects of DN on the ankle’s nerves and muscles should be explored. As it’s becoming clear participants diagnosed with DN have greater sway and perhaps a change in balance strategies, it could be possible that the ankle is affected to a degree by the disease forcing the participants diagnosed with DN to possibly rely on an abnormal strategy.
Diabetic Neuropath and The Possible Causes for Postural Instability

Reduced Ankle Plantar Flexion and Moments

One long-term complication associated with DN is bilateral reduction or loss of somatosensory information in the hands and feet commonly referred to as the stocking and glove distribution. Since somatosensory information (with visual and vestibular information) is used in maintaining balance, a somatosensory deficit in the feet might compromise functional postural stability and gait (Simmons et al., 1997).

Mueller et al. (1994) reported that participants diagnosed with DN and a history of plantar ulcers had less ankle mobility, peak ankle plantar moment and power, and considering the impairment of the distal extremities observed in participants diagnosed with DN, they proposed that these patients change the ankle strategy to the hip strategy during gait in order to compensate for the smaller ankle moments of force due to the peripheral degeneration. Kwon et al. (1994) had similar results and stated there are several possible reasons why participants diagnosed with DN had a lower peak ankle plantar flexion moment than subjects in the control group. A compensatory adaptive strategy may be employed to maintain balance during walking. Another possible reason for reduced ankle moments during walking is reduced plantar flexor muscle strength. Salsich et al. (2000) report that participants diagnosed with DN had approximately 36% less concentric plantar peak torque compared with subjects in the control group.

Sacco & Amadio (2003) showed the EMG activity of the tibialis anterior was smaller and delayed compared to a healthy control group – this could represent an alteration in the contribution of the ankle during not only gait, but perhaps balance in
general. Furthermore, Sacco et al. (1999) performed a study whose main results demonstrated that participants diagnosed with DN have reduced active ankle ROM and dynamic ankle flexion at heel–strike as well as reduced amplitude (flexion–extension) when compared to non-diabetic subjects. Considering DN patients have an increased sway, reduced plantar flexion flexibility the LOS in the anterior-posterior direction could be reduced compounding the instability problem (Simmons et al., 1997).

Reduced Nerve Function

DN modifies the amount and the quality of the sensorial information necessary for proper motor control. Consequently, there is an increase in instabilities during gait and static posture (Richardson et al., 1992), which were formerly considered to be due largely to muscular weakness (Courtemanche et al., 1996). Considering that the peripheral sensorial information diminishes due to the injured peripheral nerves, that this loss starts in the lower extremities, and that the muscle spindles of these extremities are also damaged in diabetic neuropathy; the amount of information that comes from the ankle will be drastically reduced resulting in changes of gait and balance strategies (Van Deursen, 1997). As a consequence of the DN, participants diagnosed with DN may try to compensate for the small ankle activity and sensorial information by increasing the muscular activity of the hip (Mueller et al., 1994). Other studies (Delbridge et al., 1988; Mueller et al., 1989; Van Deursen, 1997) have also observed lesser ankle flexibility during specific ankle movements as well as in gait in DN patients.

With the progression of the DN, motor nerves are damage and dysfunctions and atrophy are the results of the motor component of DN. The fibular nerve, with the n. sural
and the n. plantar medial nerves are the nerves which present more abnormalities in electrophysiological tests in DN patients (Dick et al., 1985). According to Simmoneu et al. (1996), it can be expected that the fibular muscles—tibialis anterior and gastrocnemius—will also have their functions damaged. In particular, the vastus lateralis, tibialis anterior, and gastrocnemius have been the most affected by the neuropathy progression (Sacco et al. 2010). It is necessary to emphasize the importance of the sensorial and kinesthetic information (muscle spindles) of the lower extremities, especially of the ankle, which is crucial for a better control of gait and posture.

Patients with DN are generally unstable when standing quietly with eyes closed. However, not all patients are impaired to the same extent. Severity of unsteadiness depends not only on the degree of DN, but also on the type of afferent fibers involved (Nardone et al., 2007). DN may disrupt both afferent and efferent pathways of the lower extremity necessary for the maintenance of posture and normal gait (Mueller et al., 1994). DN affects and involves medium-sized afferent fibers typically resulting in instability, particularly with eyes closed (Nardone & Schieppati, 2004). A likely reason for the instability in patients with DN is that spindle group II afferent fibers are affected in addition to group Ia fibers. The group II fibers innervate the spindle secondary terminations, sensitive to changes in muscle length, and represent a more important source of sensory input for stance control than Ia fibers (Schieppati & Nardone, 1999).

For participants diagnosed with DN, balance may be unexpectedly better under dynamic conditions (standing and balancing on a platform constantly translating in the anterior-posterior direction) than under quiet stance, since other inputs and pathways (vestibular and visual) can play a role and provide crucial information (Nardone et al., 2007).
However, it is well established that proprioceptive information from the lower extremities is one of the main input sources (besides the visual and labyrinthine senses) that ensure and regulate postural control. As DN is intimately linked with considerable restriction in this sensory modality, disturbances of postural control are a frequent clinically well-known symptom of the diabetic patient (Oppenhein et al. 1999).

**Diabetic Neuropathy and EMG**

In earlier experimental studies, postural control strategies were characterized primarily by muscle activation patterns and body kinematics Horak & Nashner (1986). Ankle strategy was characterized by early activation of posterior ankle muscles followed by activation of posterior thigh and trunk muscles – for responses to backward translations on a beam. These muscle activations were associated with the production of torque at the support surface, and kinematic analyses showed body movement predominantly at the ankle joint, although some small movement at the hip was also observed. The hip strategy, observed in response to translations of a narrow beam, was characterized by early activation of anterior trunk and thigh muscles associated with a relative increase of shear forces at the support surface and little phasic activation of ankle muscles. Kinematic analyses showed trunk flexion paired with ankle extension.

The role of somatosensory information was examined by Horak et al. (1990) by comparing postural responses of healthy control subjects prior to and following somatosensory loss due to hypoxic anesthesia of the feet and ankles. Postural responses were quantified by measuring spatial and temporal features of leg and trunk EMG activation, ankle, knee, and hip joint kinematics, and surface forces in response to
anterior–posterior surface translations under different visual and surface conditions. In a similar study by Runge et al. (1999), where the strategies used for faster translation velocities by a normal healthy population, muscle activations were larger and some muscles that were silent at the slower velocities became active. Consistent with the distal-to-proximal EMG activity observed in past studies for ankle strategy responses to backward translations (Horak & Nashner (1986); Horak et al. (1990)), slow translations of approximately 5–20 cm/s induced corrective responses characterized by muscle activity on the posterior aspect of the body beginning with gastrocnemius.

Although non-diabetic patients were used in the Runge et al. (1990) study, the methodology may suit well to the present study. During the faster translation velocities of the Runge et al. (1990) protocol, hip strategy was added to the response, as demonstrated not only by rectus abdominis activity and increased hip flexion, but more importantly by an early hip flexor torque, which established active initiation of the hip flexion. The addition of a hip flexor torque to the postural response at faster translation velocities demonstrates a change in the control of balance to active generation of upper body flexion.

When hip flexor torque is not used to stabilize balance, the destabilizing force of gravity is countered by using plantarflexion torque generated about the ankle joints to halt the forward body rotation and COM movement. However, while plantarflexion on a fixed surface rotates the lower leg backward, the same torques (if unopposed) flexes the trunk forward. Keeping in mind this was a healthy population (Runge et al., 1990). It should be noted, as stated earlier, DN patients appear to have less ROM in the ankle joint during plantarflexion, and also have a possibility of increased ankle muscle weakness.
The multi-segmented human body can be controlled as a flexible, single-segment inverted pendulum only because passive anatomical structures and activations in proximal muscles (e.g. biceps femoris, lumbar paraspinals) limit the relative movement between body segments to the small deviations typically observed in the ankle strategy. The postural control strategy is the same: muscles contributing to an ankle plantarflexion torque (gastrocnemius, recorded in this experiment) are activated to overcome the destabilizing torque of gravity on the whole-body COM. The stabilizing potential of ankle plantarflexion torque is quite limited because the moment of inertia of the whole body about the ankle joint is quite high and the heels will rise with significant plantarflexion torque. Because relatively large ankle torques are required to produce relatively small corrections of the COM using ankle strategy, Kuo’s (1993) optimization model predicts that mixed strategy would be used to correct for translations of all speeds on a flat surface if muscular effort is to be minimized. However, the subjects of this study corrected slow translations with little or no hip torque. This finding is consistent either with the hypothesis that the predominant postural goal during translations slow enough to not compromise stability is to maintain upright alignment or with the possibility that the difference in muscular effort to produce the two strategies is minimal for slow translations. Furthermore, higher ankle torques were associated with larger COM displacements. This suggests that the purpose of the hip torque on a firm, flat surface may be to change the configuration of the body to facilitate torque at the ankle without lifting of the heels to effectively correct the COM position without taking a step (Runge et al., 1999).
Information concerning muscle activation patterns from EMG recordings can provide additional insight into the cause of body movement, and previous experimental observations of body kinematics were paired with EMG recordings (Horak & Nashner, 1986). Nevertheless, EMG recordings can still be misleading, as muscles may act concentrically or eccentrically and the activity of deep muscles cannot be recorded with surface electrodes (Runge et al., 1999).

Summary of Literature Review

Strong evidence from various research groups indicates participants diagnosed with DN have greater postural sway, and may be at a greater risk of a fall than that of a healthy population. It would also appear that participants diagnosed with DN undergo complex nerve damage to multiple types of afferent nerves, impairing their somatosensory system (Simmoneau et al., 1995; Di Nardo et al., 1999). Researchers have inferred from that participants diagnosed with DN make a switch from an ankle based balance strategy to a hip based strategy (Uccioli et al., 1995; Giacomini et al., 1996; Uccioli et al., 1997). Although a range of studies have shown a complex problem at the ankle joint (e.g. muscle weakness and/or various nerve damage) direct research into which of these strategies used by participants diagnosed with DN has rarely been conducted (Bonnet et al., 2009). In light of these statements, the purpose of the present study is to determine what strategies are employed by participants diagnosed with DN versus matched healthy controls during an SOT test. Specifically, EMG data will be measured and compared between an experimental and a control group.
CHAPTER 3

METHODOLOGY

The purpose of this study was to compare muscle latencies for patients diagnosed with diabetic neuropathy versus healthy controls during a perturbed balance task. A secondary purpose for this study was to distinguish postural control strategies the groups used based on the muscle latencies.

Subject Characteristics

To accomplish the purpose of this study, five DN participants (99.7 ± 7.95 kg, 176 ± 9.58 cm, 46.6 ± 16.55 years) and 5 HC (100.36 ± 12.61 kg, 173.76 ± 9.66 cm, 47 ± 13.42 years) participants were recruited from the greater Las Vegas area. Healthy controls were matched to DN participants using Centers for Disease Control and Prevention adult percentiles (Anthropometric Reference Data for Children and Adults: United States, 2003–2006). Healthy controls were matched between ±5% of the DN subjects’ percentile. Participant inclusion criteria for participants diagnosed with DN selection included a history of diabetes mellitus and diabetic neuropathy, ability to walk independently without pain or assistive device, inability to sense 5.07 monofilament, no history of cognitive or orthopedic problems and no subjects over the age of 65 due to alterations in gait caused by aging (Kwon, Minor, Maluf & Mueller, 2003; Sacco et al., 2010). Additionally, all subjects were pre-screened to exclude those individuals using medication that would affect balance, as well as individuals with knee, ankle or hip injuries or other postural instabilities not related to diabetes mellitus (Simmons et al.,
Participants granted institutionally approved written consent before participating in the study.

**Instrumentation**

*Semmes–Weinstein monofilament sensory threshold test*

Cutaneous sensory deficit in each foot, resulting from DN, was evaluated by administering a Semmes–Weinstein monofilament sensory threshold test. A 10-g (number 5.07) monofilament was used to test cutaneous sensation on nine plantar and one dorsal site of each foot (see Figure 2). This monofilament size has a high degree of accuracy, sensitivity and specificity in screening patients predisposed to foot ulceration (Kumar et al., 1991). The 10-g notation represents the common logarithm of 10-times the force in mg to cause the filament to bend.

![Figure 2: Semmes–Weinstein monofilament sensory threshold test](image)

*Posturography*
A NeuroCom (Clackamass, OR) computerized dynamic posturography apparatus was used (100 Hz) in this study as the source of balance perturbation.

**EMG**

A Delsys Trigino Wireless EMG system (2000 Hz) was used to measure the muscle onset in the right leg of all subjects. The following muscles were measured on the right leg and trunk of each participant: tibialis anterior (TA), gastrocnemius medialis (GAS), rectus femoris (QUA), biceps femoris (HAM), rectus abdominis (ABS), and lumbar paraspinals at the iliac crest (PAR) muscles. The Delsys Trigino Wireless EMG system was synced via a synchronization module to the NeuroCom apparatus.

**Procedure**

Participants arrived at the UNLV Bigelow Health Sciences building room 217, where they first read and signed an institutionally approved informed consent form and were given a brief explanation of the procedures.

Subjects not wearing shorts were provided clean laboratory clothing. Subjects were asked to remove their shoes and socks. Cutaneous sensory deficit, possibly due to DN, in each foot was evaluated by administering a Semmes–Weinstein monofilament sensory threshold test to all subjects. Throughout this testing subjects were prone with eyes closed, legs outstretched and their feet extended over the edge of a table. A 10-g (number 5.07) monofilament was used to test cutaneous sensation on nine plantar and one dorsal site of each foot (see Figure 2). This size monofilament has a high degree of accuracy, sensitivity and specificity in screening patients predisposed to foot ulceration.
(Kumar et al., 1991; Lee et al., 2003). The monofilament was applied perpendicular to the surface of the skin and with sufficient pressure to cause the filament to bend. The 10-g notation represents the common logarithm of 10-times the force in mg to cause the filament to bend. Subjects verbally responded if they felt the stimulus and, if so, were asked to point to the location of the sensation. Once completed, subjects correctly identifying and locating four or fewer of the monofilament test sites were classified as having DN (Simmons et al., 1997).

Delsys Trigino Wireless EMG sensors were placed on the following muscles on each subject’s right side of their body: TA, GAS, QUA, HAM, ABS, and PAR. Skin preparation consisted of removing any excessive hair and a light scrubbing with alcohol before application of the EMG sensors. Hypoallergenic double-sided adhesive tape was used to secure sensors to the skin with an interface of electrode cream. Electrodes were placed on the belly of the muscle of interest, with the orienting arrow on the top of Trigino EMG sensor pointing parallel to the muscle fibers. The Anatomical Guide for the Electromyographer fifth edition by Perotto was used to standardize and position electrodes (Kwon, Minor, Maluf & Mueller, 2003).

After all EMG sensors were placed, subjects were suited in a safety harness for the NeuroCom system. Subjects were instructed to take off their shoes before stepping into the NeuroCom apparatus. Subjects were properly suited for safety harness vest ensuring the vest was not too tight or too loose. Subjects then stepped onto the NeuroCom force platform with their feet matching up to the correct foot “size,” small, medium, or large, on the platform. Each subject’s medial malleolus was aligned to the correct placements on the platform. The safety harness was then attached via a carabiner
which was secured to a safety bar (can hold an excess of 500 lbs) located at the top of the NeuroCom.

Once all EMG sensors were properly placed, and the NeuroCom harness properly secured, a baseline of the raw EMG data was taken 200 ms prior to perturbation. This baseline EMG signal was used to determine muscle onset using a 2 standard deviation threshold. Utilizing the Delsys Synchronization Module, both the Delsys Wireless EMG system and the NeurCom were synchronized. Perturbation was set at time 0.

Each subject was given instructions to stare straight ahead and stand as steady as possible. Subjects then performed one condition, three times each, of the SOT VI test. Subjects were given approximately two to five minute breaks when needed between each trial. The following is a brief description of the SOT VI condition used. SOT VI: subjects will have their eyes open and stand on a sway referenced surface while the visual surround moves. After the SOT VI test was completed, all instrumentation was removed, the participant was asked if he or she had any questions, and then the participant was thanked for volunteering and dismissed from the study.

**Treatment of Data**

After the collection process was completed raw EMG data of the TA, GAS, QUA, HAM, ABS, and PAR muscles were analyzed in MatLab using custom written script (APPENDIX I). First, the DC offset was removed followed by full-wave rectification. These data were then filtered through a moving window average algorithm. The moving window average was set to a 35 ms window (Perucca et al., 2014). Perturbation during SOT VI was at time 0. Two-hundred milliseconds of EMG “pre-activation” data were
recorded prior to perturbation. These data were used as a baseline. Latency was determined as the time from the perturbation to the time when the smoothed EMG data exceeded two standard deviations above the baseline for at least 50 ms (Figure 3; Perucca et al., 2014).

![Figure 3: Graphical representation of custom MatLab script locating latency for GAS](image)

**Statistical Analysis**

**Variables**

6 dependent variables were analyzed:

- Muscle latency of the 1) TA, 2) GAS, 3) QUA, 4) HAM, 5) ABS, or 6) PAR muscles during SOT condition VI

1 independent variable was used:
• Two levels: (1) Diabetic neuropathy group and (2) healthy control group

**Statistical Test**

Dependent variables between subjects in the different groups was analyzed in SPSS Statistics 20 software (IBM; Armonk, NY) using a paired t-test. Dependent variables between matched subjects were analyzed in Microsoft Excel using single subject analysis. Muscle latencies were graphed for comparison of the groups and descriptively analyzed.
CHAPTER 4

RESULTS

The purpose of this study was to compare muscle latencies for patients diagnosed with diabetic neuropathy (DN) versus matched healthy controls (HC) during a perturbed balance task. A secondary purpose for this study was to distinguish postural control strategies the groups used based on the muscle latencies.

Table 1 describes the anthropometric measures and age between the DN and HC groups. Independent t-tests were used to assess the differences in age (years), mass (kg), and height (cm). No statistically significant ($p > 0.05$) differences were observed between the DN and HC groups for all anthropometric measures including age ($t(8) = -0.04, p = 0.98$), mass ($t(7) = -0.09, p = 0.93$), and height ($t(8) = 0.33, p = 0.75$). Due to the nature of the study of having matched controls, the lack of significant differences found in anthropometric measures between the groups was beneficial and supported the notion of matched participants.

Table 1: Demographic data for DN and HC groups

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Mass (kg)</th>
<th>Height (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN1</td>
<td>M</td>
<td>44</td>
<td>99.5</td>
<td>185.0</td>
</tr>
<tr>
<td>DN2</td>
<td>F</td>
<td>21</td>
<td>85.7</td>
<td>167.7</td>
</tr>
<tr>
<td>DN3</td>
<td>M</td>
<td>46</td>
<td>110.3</td>
<td>190.0</td>
</tr>
<tr>
<td>DN4</td>
<td>M</td>
<td>58</td>
<td>100.8</td>
<td>167.1</td>
</tr>
<tr>
<td>DN5</td>
<td>M</td>
<td>64</td>
<td>102.2</td>
<td>170.2</td>
</tr>
<tr>
<td>HC1</td>
<td>M</td>
<td>49</td>
<td>102.4</td>
<td>182.3</td>
</tr>
<tr>
<td>HC2</td>
<td>F</td>
<td>25</td>
<td>83.0</td>
<td>163.0</td>
</tr>
<tr>
<td>HC3</td>
<td>M</td>
<td>47</td>
<td>122.0</td>
<td>187.3</td>
</tr>
<tr>
<td>HC4</td>
<td>M</td>
<td>53</td>
<td>98.0</td>
<td>164.2</td>
</tr>
<tr>
<td>HC5</td>
<td>M</td>
<td>61</td>
<td>96.4</td>
<td>172.0</td>
</tr>
<tr>
<td>Mean ± STD</td>
<td>N/A</td>
<td>47 ± 14.2</td>
<td>100 ± 11.1</td>
<td>175 ± 10.2</td>
</tr>
</tbody>
</table>
Subject responses to 5.07 Semmes–Weinstein monofilament sensory threshold tests are illustrated in Table 2 and Table 3. Independent t-tests were used to assess differences in 5.07 Semmes–Weinstein monofilament sensory threshold tests between the DN and HC groups by foot. A significant difference was found between the DN and HC groups for both the right ($t(4) = -14.06, p < 0.01$) and left feet ($t(4) = -14.61, p < 0.01$).

**Table 2: DN group 5.07 Semmes–Weinstein monofilament sensory threshold tests**

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Left Foot</th>
<th>Right Foot</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>DN2</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>DN3</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>DN4</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>DN5</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mean</td>
<td>1.8 ± 1.3</td>
<td>2.0 ± 1.2</td>
</tr>
</tbody>
</table>

**Table 3: HC group 5.07 Semmes–Weinstein monofilament sensory threshold tests**

<table>
<thead>
<tr>
<th>Subjects</th>
<th>Left Foot</th>
<th>Right Foot</th>
</tr>
</thead>
<tbody>
<tr>
<td>HC1</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>HC2</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>HC3</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>HC4</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>HC5</td>
<td>10</td>
<td>10</td>
</tr>
<tr>
<td>Mean</td>
<td>10.0 ± 0.0</td>
<td>10.0 ± 0.0</td>
</tr>
</tbody>
</table>
Figure 4 depicts the direction of mean muscle latencies, from posterior to anterior, for both the DN and HC groups. Muscle latency mean and standard deviation values between the DN and HC groups are given in Table 4. Paired t-tests were used to assess differences between the DN and HC groups for muscle latencies. No statistically significant muscle latency differences ($p > 0.05$) were observed between the DN and HC groups for any muscle (Table 4).

Table 4: DN and HC group latency means ± STD values by muscle

<table>
<thead>
<tr>
<th>Group</th>
<th>GAS</th>
<th>HAM</th>
<th>PAR</th>
<th>TA</th>
<th>QUA</th>
<th>ABS</th>
</tr>
</thead>
<tbody>
<tr>
<td>DN</td>
<td>0.060 ± 0.033</td>
<td>0.091 ± 0.094</td>
<td>0.038 ± 0.053</td>
<td>0.280 ± 0.236</td>
<td>0.443 ± 0.414</td>
<td>0.464 ± 0.473</td>
</tr>
<tr>
<td>HC</td>
<td>0.071 ± 0.056</td>
<td>0.067 ± 0.047</td>
<td>0.075 ± 0.061</td>
<td>0.219 ± 0.350</td>
<td>0.373 ± 0.417</td>
<td>0.248 ± 0.317</td>
</tr>
</tbody>
</table>

Given the clinical nature of the study as well as matched control participants, muscle latencies were compared between participants diagnosed with DN and their HCs using a
within-subject statistical technique (Model Statistic, \( \alpha = 0.05 \)) (Bates, Dufek, & Davis, 1992; Dufek & Bates, 1995). While the Model Statistic has been traditionally used to compare differences between conditions within participants while accounting for within-subject variability, we chose to explore its use on a per-subject basis (between matched pairs), given the nature of this study design. Significant findings were determined by comparing the critical difference (calculated using individual performer variability) versus the observed difference as follows:

1) Critical difference = probability * mean standard deviation (where probability is variable based upon number of trials and significance level),

2) Mean standard deviation = \( \sqrt{\frac{DN\ SD^2 + HC\ SD^2}{2}} \),

3) Comparison of observed difference (DN Mean – HC Mean),

4) If the absolute value of the observed difference was greater than the critical difference, conditions were significantly different at the selected alpha level (\( \alpha = 0.05 \)).
Figure 5: Comparison of matched subjects’ muscle latencies for GAS. * = Statistical Difference

Figure 6: Comparison of matched subjects’ muscle latencies for HAM. * = Statistical Difference
Figure 7: Comparison of matched subjects’ muscle latencies for PAR. * = Statistical Difference

Figure 8: Comparison of matched subjects’ muscle latencies for TA. * = Statistical Difference
Figure 9: Comparison of matched subjects’ muscle latencies for QUA. * = Statistical Difference

Figure 10: Comparison of matched subjects’ muscle latencies for ABS. * = Statistical Difference
Muscle latency mean and standard deviation values by participant-muscle are illustrated in Figures 5 – 10. As seen in Figures 5 – 10, a wide range of significant differences ($p < 0.05$) were observed. HC1 had a significantly ($p < 0.05$) faster TA muscle activation time versus DN1 (Figure 5). No significant differences were observed between DN2 and HC2 ($p > 0.05$; Figures 5 – 10). HC3 had shorter muscle latency ($p < 0.05$) with HC3’s TA, HAM, and QUA muscles having faster muscle activation time versus DN3 (Figures 5, 6, & 9). DN4 had a significantly ($p < 0.05$) shorter PAR muscle activation time versus HC4 (Figure 7). No significant differences were observed between DN5 and HC5 ($p > 0.05$; Figures 5 – 10).
CHAPTER 5

SUMMARY, CONCLUSIONS, & RECOMMENDATIONS

Discussion of Results

The purpose of this study was to compare muscle latencies for patients diagnosed with diabetic neuropathy (DN) versus matched healthy controls (HC) during a perturbed balance task. A secondary purpose for this study was to distinguish postural control strategies the groups used based on the muscle latencies. Specifically, the unique aspect of this study was that muscle latency was examined between muscles of participants diagnosed with DN and their HCs. Another unique aspect to this study was that muscle latency differences between the groups were observed to identify possible balance strategies.

Previous research conducted on participants diagnosed with DN is vast and has shown progressive loss of somatosensory sensitivity, proprioception, and muscular function (Sacco & Amadio 2003). It has been widely studied and recognized that participants diagnosed with DN have greater postural sway compared to HCs (Simmons et al., 1997; Richardson, Ching & Hurvitz, 1992; Cavanaugh et al., 1992; Yamamoto et al., 2001) and that DN have a reduction in nerve conduction velocity and overall nerve function (Richardson et al., 1992; Courtemanche et al., 1996; Butugan et al., 2014; Allen et al., 2014). These results are consistent with the fact that participants diagnosed with DN have a greater risk of falling compared to HCs (Diener et al., 1984; Richardson, Ching & Hurvitz, 1992; Cavanaugh et al., 1992; Hurvitz & Richardson, 1995). As seen, a gamut of studies have been performed on participants diagnosed with DN, however, to
the author’s knowledge no measurement of muscle latency has been conducted for participants diagnosed with DN during a perturbed balance task, and no description of balance strategies have been identified for participants diagnosed with DN based on these latencies.

According to Simmons et al. (1997), Muritz & Dietz (1980), and Giacomini et al. (1996) individuals with cutaneous sensory deficits, similar to that found in participants diagnosed with DN, may rely on hip strategies versus ankle strategies to bring about equilibrium. Sensory information from the vestibular system and from somatosensory afferent fibers in the feet and ankles plays an important role in determining the availability of postural strategies (Horak et al., 1990). Horak & Nashner (1986) and Moore et al., (1986) associated each movement strategy with “stereotypical” muscle activation patterns. The ankle strategy was associated with a distal-to-proximal pattern of ankle-knee-hip muscle activation posteriorly while the hip strategy was associated largely with proximal, hip muscle activation anteriorly (McCollum et al., 1984).

The present study showed participants diagnosed with DN, along with HCs, utilized an aspect of the ankle strategy with a distal-to-proximal pattern of ankle-knee-hip muscle activation posteriorly (see Figure 4). This result is a contradiction to what past researchers have speculated about the balance strategies utilized by participants diagnosed with DN. Although no physical assessment was given, it’s possible the participants diagnosed with DN were more physically active than their HCs allowing for their choice of muscle latency patterns to appear relatively similar to that of the HCs. Research has demonstrated exercise may attenuate the deficits associated with DN. Song et al. (2011) found that that 8 weeks of balance exercises improved static and dynamic
balance. Balducci et al. (2006) specifically examined nerve conduction velocity changes over the course of four years in an exercise group with diabetes versus a control group with diabetes (at the beginning of the study both group lacked symptoms of DN), and found the percentage of participants diagnosed with DN that developed DN during the study was significantly higher in the control than the exercise group. As indicated in the results from Balducci et al. (2006), DN progression may be reduced with exercise. If a physical assessment had been administered to determine the level of activity perhaps the results could be further explained.

It has been commonly observed that motor fibers are affected after somatosensory fibers as DN progresses in a subject (Horak et al., 2002; Zochodone et al., 2008). If the disease was not as advanced in the DN group it’s possible this could account for the similar muscle latency observed in the DN group as the α-motor neurons, myelination, diameter, and motor end plate could have remained unaffected at the time of data collection (Andreassen et al., 2006). As stated by Meijer et al. (2008) motor nerve conduction velocity is largely preserved due to axonal sprouting and reinnervation, moreover, according to Zochodne et al. (2008) who used animal models indicated that the motor neurons seem to be more preserved in the course of the disease.

Nevertheless, the observations made appear in contradiction to the majority of the diabetic neuropathy literature regarding neurophysiology and biomechanics. Examples from recent literature by Watanabe et al. (2013) have indicated that individuals with type 2 diabetes had motor units with lower firing rates. This may have been due to the possibility of delayed repolarization of the neuronal membrane versus healthy controls. This would result in a decrease in action potentials and nerve conduction velocity and
likely affect muscle latency and continues to reinforce the idea of neuromuscular deficits.
It is apparent the way muscle function may deteriorate due to DN is still not completely understood.

Utilizing the single subject Model Statistics procedures (Figures 5 – 10) no significant trends in muscle activation were observed between the participants diagnosed with DN and their matched HCs. Although significant differences ($p = 0.05$) were identified between muscle latencies between some of the participants diagnosed with DN and their HCs, no consistent relationship or pattern was apparent. This is also counterintuitive to previous research that focused on nerve conduction velocity and overall nerve function.

Similar to the logic used to suggest why the DN group versus the HC group may have used similar balance strategies, perhaps for the participants diagnosed with DN who had significantly shorter latencies also had more physically active lifestyle. Song et al. (2011) and Balducci et al. (2006), whose results showed exercise may attenuate DN symptoms, may explain the results from the single subject statistical procedures. In addition, Kluding et al. (2012) identified improvements in neuropathic and cutaneous nerve fiber branching following supervised exercise in participants diagnosed with DN. It would appear in the Kluding et al. (2012) study, exercise may have positively influenced the factors accompanying DN by stimulating microvascular dilation, reducing oxidative stress, and increasing neurotrophic factors.

Although the overall results found in the present study were not suspected, Butugan et al. (2014) studied the nerve conduction velocity of the tibialis anterior, vastus lateralis, gastrocnemius medialis, and biceps femoris and found scarce significant
differences in nerve conduction velocity between healthy controls and four divided
groups of participants diagnosed with DN (absent neuropathy, mild neuropathy, and
severe neuropathy). These results from may illuminate why no trend was seen between
the participants diagnosed with DN versus their matched HCs. Moreover, Butugan et al.
(2014) posited potential neuromuscular deficits not in a distal to proximal fashion, but
rather based on fiber types with type I fibers being affected more than type II. This is
important in that the results from the participants diagnose with DN may have similar
muscle latencies due to muscle stretch reflexes. In other words, the latencies observed
may be an unconscious decision based on a reflex versus a conscious choice in balance
strategies. The results of the present study support the idea that more complex variables
may discriminate neuromuscular deficits in participants diagnosed with DN versus
anatomical location alone, and may challenge the common hypothesis of distal to
proximal evolution of neuromuscular deficits.

Limitations

Limitations of this study included that physicality of the participants diagnosed
with DN and HC was not determined using an activity assessment scale to possibly
further address why certain muscle latencies were observed. Although the 5.07 Semmes–
Weinstein monofilament sensory threshold tests have been found to be very accurate,
using techniques such as fuzzy expert system to further distinguish and stratify the degree
of diabetic neuropathy affecting the DN group would be useful for a deeper analysis of
the data. The fuzzy expert system is used for diagnosing and classifying DN patients into
subgroups: absent neuropathy, mild neuropathy, and severe neuropathy (Butugan et al., 2014).

**Conclusions**

The purpose of this study was to compare muscle latencies for patients diagnosed with diabetic neuropathy versus healthy controls during a perturbed balance task with a secondary purpose to distinguish postural control strategies the groups used based on the muscle latencies. The results of the study lead to the retention of both null hypothesis I and II. The data presented showed interesting and somewhat contradictory results with the DN group exhibiting similar balance strategies, based on muscle latency, to their HCs. No trend of muscle latency was seen between the individual participants diagnosed with DN versus the individual subjects of the HC group. The results of this study may be explained with current research that has challenged the pathophysiology of DN especially regarding whether DN affects the motor system, and if DN may be attenuated by exercise. The results of this study continue to shed light on the complexity of DN.

**Recommendations**

Future research into the differences of muscle latencies of participants diagnosed with DN versus HCs is needed. How these individuals balance themselves with regard to choice of balance strategies also requires further exploration. Future research using a similar protocol to this present study should utilize an increased number of subjects along with more detailed classification for distinguishing the degree of DN exhibited in the groups. Individual exercise regimens, or level of physical fitness, should also be assessed as this may lead to more specifically interpreting the results.
APPENDIX I
CUSTOM DATA PROCESSING PROGRAM

% Kyle Project
% Authors : Ali Pour Yazdanpanah & Kyle Mefferd
clc
close all
clear all

% Parameters

P=dir;
um=size(P,1);
i2=[1 9 17 25 33 41];
j=[2 10 18 26 34 42];
NumMuscles=length(j); % Number of Muscles
count=1; count1=1;

windowSize = 35; % Filter Windows Size
Threshold=0.2; % 200 ms Threshold
Th1=double(0.0006);
Th2=0.05; % 50 ms Threshold
GenCol=1;

% Automatically Find input XLSX Files

for i=3:num
    imname=P(i,1).name;
    img=char(imname);
    k=strfind(img, '_');
    kl=strfind(img, 'DN');
    if k
        c1(count)=i;
        count=count+1;
    elseif kl
        c(count1)=i;
        count1=count1+1;
    end
end

clear k
clear kl

% Create Data Structures for STDs

numxls=size(c1,2);
for il=1:numxls
    eval(['stdData num2str(il) '= zeros(3,6);']);
end

for j1=1:size(c1,2)
    t1=c1(j1);

imname1=P(t1,1).name;  
img1=char(imname1);  
L=xlsread(img1);  
[qq,ww]=size(L);  
tk=find(L(:,1)==Threshold);  
for  
ll=1:NumMuscles  
    avg=mean(L(:,j(ll)));  
    L(:,j(ll))=L(:,j(ll))-avg;  
    L(:,j(ll))=abs(L(:,j(ll)));  
    b = (1/windowSize)*ones(1,windowSize);  
    a = 1;  
    L(:,j(ll)) = filter(b,a,L(:,j(ll)));  
    eval(['stdData' num2str(j1) ' (1,ll)'' = std(L(1:tk,j(ll)));']);  
end  
end  
for  
j2=1:size(c,2)  
t=c(j2);  
imname2=P(t,1).name;  
img=char(imname2);  
L1=xlsread(img);  
for  
ll=1:NumMuscles  
    avg=mean(L1(:,j(ll)));  
    L1(:,j(ll))=L1(:,j(ll))-avg;  
    L1(:,j(ll))=abs(L1(:,j(ll)));  
    eval(['stdData' num2str(j2) ' (2,ll)'' = std(L1(:,j(ll)));']);  
    b = (1/windowSize)*ones(1,windowSize);  
    a = 1;  
    L1(:,j(ll)) = filter(b,a,L1(:,j(ll)));  
    eval(['stdData' num2str(j2) ' (3,ll)'' = abs(std(L1(:,j(ll))));']);  
    eval(['New' img(1,1:end-5) '=L1;']);  
end  
end  
FinalStartingTimes=zeros(qq,(size(c,2)*NumMuscles*(size(stdData1,1)+1)) );  
fileID = fopen('ColInfo.txt', 'w');  
for  
j2=1:size(c,2)  
t=c(j2);  
imname2=P(t,1).name;  
img=char(imname2);  
L2=xlsread(img);  
for  
ll=1:NumMuscles  
    eval(['as=L1(:,j(ll));']);  
    eval(['as2=L1(:,i2(ll));']);  
    for  
NumSTDs=1:size(stdData1,1)  
        eval(['indx=find(as>2*stdData'' num2str(j2) ' ('' num2str(NumSTDs) ',ll));']);  
        eval(['indx=find(L2(:,j(ll))>2*stdData1(l,1,ll));']);  
        kk=as(indx);  
        kk2=as2(indx);
timesvalue = kk2;

if size(indx,1) ~= 0
    mm = timesvalue(1,1);
    TimeSum = zeros(size(timesvalue,1),1);
    StartTimes = zeros(size(timesvalue,1),1);
    for i11 = 1:(size(timesvalue,1) - 1)
        if ((round(timesvalue(i11+1,1)*10000)/10000) - (round(timesvalue(i11,1)*10000)/10000)) < (Th1)
            TimeSum(LL,1) = (timesvalue(i11+1,1) - timesvalue(i11,1)) + TimeSum(LL,1);
            StartTimes(LL,1) = mm;
        else
            LL = LL + 1;
            mm = timesvalue(i11+1,1);
        end
    end
    StartTimes(StartTimes==0) = [];
    TimeSum(TimeSum==0) = [];
else
    LL = LL + 1;
    mm = timesvalue(i11+1,1);
end
StartTimes(StartTimes==0) = [];
TimeSum(TimeSum==0) = [];

if size(indx2,1) ~= 0
    FinalTimes = StartTimes(indx2);
    len = length(FinalTimes);
    FinalStartingTimes(1:len,GenCol) = FinalTimes;
    Colinf = ['std' num2str(NumSTDs) ' (' num2str(NumMuscles) ' - ' num2str(ll) ' - ' img(1,1:end-5) ];
    disp(Colinf);
    GenCol = GenCol + 1;
else
    GenCol = GenCol + 1;
end
else
    GenCol = GenCol + 1;
    eval(['Final=find(as>2*stdData' num2str(j2) ','ll));']);
end
clear kk2
clear kk
clear indx
end
clear as
clear as2
end
xlswrite('FinalStartingTimes.xlsx',FinalStartingTimes)
fclose(fileID);
APPENDIX II

IRB APPROVAL

UNLV
Participant Informed Consent Form

Study Title: COMPARISON OF MUSCLE LATENCIES FOR DIABETIC NEUROPATHY PATIENTS VERSUS HEALTHY CONTROLS DURING A PERBURBED BALANCE TASK

Investigators: Kyle Mefferd, Janet S. Dufek PhD

Departments: Kinesiology and Nutrition Sciences

Contact Information: Principal Investigator Dr. Janet Dufek 702- 895-0702
JDufek@unlv.nevada.edu or Kyle Mefferd, 702-768-0817 MefferdK@unlv.nevada.edu

We invite you to take part in a research study. Please take as much time as you need to read this consent form. You may decide to discuss it with your family, friends, or your doctor. You may find some of the language difficult to understand. If so, please ask questions. If you decide to participate, you will be asked to sign this form.

WHY IS THIS STUDY BEING DONE?

The purpose of this study is to compare how long it takes for a muscle to turn on for patients diagnosed with diabetic neuropathy versus healthy patients during a balance task. A secondary purpose for this study is to distinguish how diabetic neuropathy patients balance themselves.

WHO IS INVITED TO PARTICIPATE IN THIS STUDY?

You are invited to participate in this study because you are between 18 and 65 years old and are apparently healthy or have diabetic neuropathy. You will be eligible to participate if you:

- Can walk independently without an assistive device;
- Are not currently pregnant or think you may be pregnant;
- Have no history of mental/psychological problems;
- Have no history of orthopedic problems.

WHAT IS INVOLVED IN THE STUDY?

If you agree to be part of the study and are eligible, we will ask that you remain on campus for about 1 hour and we will ask your age, gender, and measure your height and weight. If you are not wearing shorts, you will be asked to wear clean laboratory shorts. Next, you will be asked to remove your shoes and socks while we perform a sensory test. Throughout this testing you will lie on your stomach with eyes closed, legs outstretched and your feet extended over the edge of a
table. A monofilament will be used to test sensation on nine parts of the bottom of your foot and one on the top of your foot. You will verbally respond if you feel the stimulus and, if so, will be asked to point to the location of the sensation. After this test, we will apply sensors on the right side of your body at the following locations: lower back, stomach, front of your thigh, calf muscle and front of your lower leg. We will shave any excessive hair and scrub your skin lightly with alcohol before applying the sensors. Hypoallergenic double-sided adhesive tape will be used to apply the sensors. After all sensors are in place, you will be placed in a safety harness which can hold an excess of 500 lbs. You will be asked to step onto a platform which can move while we test your balance and measure muscle activity from the sensors we applied. During the test, which will last for 30 seconds, you will have your eyes open while standing on the platform and a visual surround will move in response to your movement. We will ask you to repeat the test two more times, with approximately with approximately two to five minutes between each repetition. After the third test, all instrumentation will be removed, and you will be thanked and dismissed.

WHAT ARE THE POSSIBLE BENEFITS TAKING PART IN THE STUDY?

There may be no direct benefits to you as a participant in this study. However, knowledge gained through this study may tell us more about how people with diabetic neuropathy balance themselves.

WHAT OTHER OPTIONS ARE THERE?

An alternative would be to not take part in this study.

WHAT ARE THE POSSIBLE RISKS?

There is a very slight possibility that you may fall during the time you are standing; however, a safety harness will be worn throughout the entire study to ensure safety. We will do our best to guard you and keep the area clear so that this doesn’t happen. It is also possible but unlikely that you will get tired or feel sore after standing.

WHAT ARE THE COST AND COMPENSATION?

It is likely the study may take up to an hour. You will be given a UNLV daily temporary permit to park close to the BHS. There will be no financial cost to you to participate in this study. There will be no monetary compensation for your time and participation during this study.

WHOM DO YOU CALL IF YOU HAVE QUESTIONS OR CONCERNS?

If you have any questions or concerns about the study, you may contact Principal Investigator Dr. Janet Dufek 702- 895-0702 or JDufek@unlv.nevada.edu; or Kyle Mefford 702-768-0817 or mefferdk@unlv.nevada.edu. For questions regarding the rights of research subjects, any
complaints or comments regarding the manner in which the study is being conducted you may contact the UNLV Office of Research Integrity – Human Subjects at 702-895-2794 or toll free at 877-895-2794 or via email at IRB@unlv.edu.

WHAT ARE YOUR RIGHTS AS A PARTICIPANT, AND WHAT WILL HAPPEN IF YOU DECIDE NOT TO PARTICIPATE?

Your participation in this study is voluntary. You may refuse to participate in this study or in any part of this study. You may withdraw at any time without prejudice to your relations with the university. You are encouraged to ask questions about this study at the beginning or any time during the study.

WILL YOUR INFORMATION BE KEPT PRIVATE?

Your participation in the study will be kept private and all information gathered in this study will be kept completely confidential. Whenever we write papers or talk about the study in class or workshop presentations, we will only talk about results of the group as a whole and will never identify you by name. All data will be stored in a locked facility at UNLV for at least 3 years after completion of the study. Any forms with your name or de-identified participant number will be shredded after 3 years.

Participant Agreement:

I have read (or someone has read to me) the information provided above. I have been given the opportunity to ask questions and all of my questions have been answered. I understand if I want, a copy of this form, it will be given to me.

By signing this form, I am agreeing to voluntarily take part in this study.

Name of Research Participant                Signature                Date


screening device for identifying diabetic patients at risk of foot ulceration.

Diabetes Research and Clinical Practice, 13, 63-68.


Song, C., Petrofsky, J., Lee, S., Lee, K., & Yim, J. (2011). Effects of an Exercise Program on Balance and Trunk Proprioception in Older Adults with Diabetic Neuropathies. Diabetes Technology & Therapeutics, 13(8), 803-811


CURRICULUM VITAE

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EDUCATION

University of Nevada, Las Vegas – Las Vegas, NV May 2015
Masters of Kinesiological Sciences, Biomechanical emphasis GPA: 3.8

University of Nevada, Las Vegas – Las Vegas, NV May 2012
Bachelor of Kinesiological Sciences GPA: 3.4

THESIS TITLE

COMPARISON OF MUSCLE LATENCIES FOR DIABETIC NEUROPATHY PATIENTS VERSUS HEALTHY CONTROLS DURING A PERTURBED BALANCE TASK

THESIS EXAMINATION COMMITTEE

Chair, Dr. Janet Dufek Ph. D.
Committee Member, Dr. John Mercer Ph. D.
Committee Member, Dr. Richard Tandy Ph. D.
Graduate College Representative, Dr. Szu-Ping Lee Ph. D. D.P.T.

Refereed Poster Presentation

Biomechanical Analysis of Infant Fall Impacts Using HIC to Predict Injury Severity
CMBBE
April 2013
Mefferd, K.C. ¹, Ryan-Wenger, N.A. ² & Dufek, J.S. ¹
¹Department of Kinesiology and Nutrition Sciences, University of Nevada, Las Vegas, Las Vegas, NV; ²Center for Innovation in Pediatric Practice, Nationwide Children’s Hospital, Columbus, OH

Quantify Head Injury Severity Following Pediatric Patient Falls
STEM Summit
November 2012
J.S. Dufek, Ph.D. ¹, N.A. Ryan-Wenger, Ph.D., R.N. ², and K.C. Mefferd B.S. ¹
Biomechanical Analysis of Infant Fall Impacts Using HIC to Predict Injury Severity
University of Nevada, Las Vegas
NORAXON Symposium
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Biomechanical Analysis of Infant Fall Impacts Using HIC to Predict Injury Severity
University of Nevada, Las Vegas
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Quantifying Head Injury Severity Following Pediatric Patient Falls
University of Nevada, Las Vegas
STEM Summit
November 2012
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Biomechanical Analysis of Infant Fall Impacts and Modern Approaches to Predict Future Injury
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RESEARCH EXPERIENCE

Graduate Research Assistant
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Aug 2012 – May 2013

Undergraduate Assistant
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Aug 2011 – May 2012

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HONORS AND AWARDS

Catholic College Campus Achievement Award
Deputy Grand Knight for Council 15061 Awarder High Gross Percentage Increase