A Preliminary Study of Gait in Elderly Women with Diabetic Peripheral Neuropathy

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A Preliminary Study of Gait in Elderly Women with Diabetic Peripheral Neuropathy

By

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Sheri L. Bjornseth

THESIS

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A PRELIMINARY STUDY OF GAIT IN ELDERLY WOMEN WITH DIABETIC PERIPHERAL NEUROPATHY

ABSTRACT

Somatosensory impairment due to diabetic peripheral neuropathy results in decreased environmental input during ambulation, which may compromise feedback mechanisms used for balance control. Individuals with distal symmetrical diabetic peripheral neuropathy provide a model of somatosensory loss in which balance impairments can be studied. Somatosensory input is an important component of standing balance in the elderly. Diabetic peripheral neuropathy has been associated with postural instability and an increased history of falls. This study investigated differences in gait between elderly diabetic subjects with distal symmetrical sensory polyneuropathy and elderly diabetics without neuropathy. To conduct this study the 3-dimensional gait analysis protocol developed at the Center for Human Kinetic Studies (CHKS) was used. In this preliminary study, differences in gait were identified which may reflect a more cautious walking strategy in diabetics with peripheral neuropathy. In stance, subjects with peripheral neuropathy demonstrated an applied extension biased hip torque, decreased hip flexion and decreased pelvic tilt, as compared to non-neuropathic subjects. This distinct hip strategy could reduce forward momentum of the head, arms, and trunk and reduce dynamic balance demands during walking. Total limb support moments during stance in neuropathic subjects were approximately one half as great as those observed for controls. Additionally, neuropathic subjects demonstrated less braking shear force during initial contact and early loading response, with a concomitant increase in horizontal anterior heel velocity, suggesting a slight time lag in weight transfer.
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We wish to thank David Marchinda and Krisanne Bothner for all their help at CHKS, Mary Jean Klebba for recruiting the subjects in this study, Jim Richardson and James Ashton-Miller for their insights on gait and balance and the clinical diagnosis of Diabetic peripheral neuropathy. This study was in part supported by a small research grant from the Michigan Physical Therapy Association.
DEFINITION OF KEY TERMS

Cardinal Planes: anatomical planes
Sagittal Plane: the spatial plane dividing the body into right and left segments.
Frontal Plane: the spatial plane dividing the body into anterior and posterior segments.
Transverse Plane: the spatial plane dividing the body into superior and inferior segments.

Phases of Gait: the motions of walking
Stance Phase: when the foot is in contact with the ground.
  Initial Contact: the instant the stance foot contacts the ground.
  Loading Response: the transfer of the body weight onto the stance leg.
  Mid-Stance: the point at which the body moves ahead of the stance leg.
  Terminal Stance: the point at which the heel of the stance leg rises off the ground.
Swing Phase: when the leg is not in contact with the ground.
  Pre-Swing: the end of stance phase, just prior to swing.
  Initial Swing: the point at which the foot is lifted off the ground.
  Mid-Swing: the point at which the swing leg advances past the body.
  Terminal Swing: the completion of swing leg advancement in preparation for initial contact.

Kinematics: variables describing motion in terms of position, velocity, and acceleration.
Kinetics: variables describing the forces acting on a body.
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CHAPTER 1
INTRODUCTION

Problem Statement

Somatosensory impairment due to diabetic peripheral neuropathy results in decreased environmental input during ambulation, which may compromise feedback mechanisms used for balance control\(^1,2\). Individuals with distal symmetrical diabetic peripheral neuropathy provide a model of somatosensory loss in which balance impairments can be studied\(^3\). Somatosensory input is an important component of standing balance in the elderly. Decreases in static standing balance due to compromised sensory input from the supporting surface have been well documented\(^4,5\). Diabetic peripheral neuropathy has been associated with postural instability\(^6-8\). An increased history of falls, and recurrent falls, has been reported in elderly diabetics with peripheral neuropathy\(^10,11\).

This study will describe the differences in gait between elderly diabetic subjects with distal symmetrical sensory polyneuropathy and elderly diabetics without neuropathy. The analysis focused on kinematic and kinetic patterns of movement which have been reported to be associated with dynamic balance\(^12-14\). These measures include standard temporal and linear parameters of gait, as well as data collected from discrete points of the gait cycle. Although several aspects of gait have been proposed to reflect dynamic standing balance, a definitive set of criteria for assessment does not yet exist\(^15\). A secondary goal of this study was to test the validity of proposed measures of dynamic balance in this model of sensory impairment. A 3-dimensional gait analysis will
provide kinematic and kinetic assessment of the hip, knee, and ankle during self-paced normal walking and at a slow speed.

**Hypothesis**

It is hypothesized that elderly diabetic subjects with distal sensory polyneuropathy will exhibit differences in their gait from elderly diabetic controls.

**Diabetic Peripheral Neuropathy**

Distal symmetrical primarily sensory polyneuropathy is the most prevalent form of diabetic peripheral neuropathy. It is a common complication of diabetes, affecting 30% to 40% of all diabetics. The incidence of peripheral neuropathy increases with age and duration of the disease. Although sensory loss is the hallmark of this complication, diabetic peripheral neuropathy may also involve autonomic and motor nerve fibers. Nerve impairment begins in the most distal parts of the extremities, usually the feet, and progresses proximally. Sensory loss is typically symmetrical in right and left feet. The sensory modalities of vibration, light touch, proprioception, and pain and temperature are affected, indicating involvement of large and small nerve fibers.

Elderly individuals with peripheral neuropathy represent a significant percentage of the population. By the year 2000, 13% of the US population is expected to be over 65 years. The most common form of diabetes in the elderly is noninsulin-dependent diabetes mellitus (NIDDM), sometimes referred to as Type II or adult-onset diabetes. Estimates of the occurrence of diabetes in the elderly population are approximately 20%, depending on the cohort studied. Studies employing diagnostic tests for diabetes such as an oral glucose tolerance test, usually identify an additional 10-20% of
subjects as undiagnosed diabetes. Therefore, a large number of elderly community dwellers have undiagnosed diabetes, and are at risk for complications such as peripheral neuropathy. Since peripheral neuropathy affects approximately 50% of people with NIDDM over 60 years and an estimated 10% of non-diabetic elders, up to 20% of the elderly population in the US may have sensory deficits due to peripheral neuropathy.

Younger diabetics also are affected by peripheral neuropathy since the main predictors for this complication are duration of the disease and the extent of hyperglycemia, whether they have NIDDM or insulin-dependent diabetes mellitus (IDDM). In individuals with IDDM, or Type I diabetes, 20-30% are affected by distal sensory peripheral neuropathy. Peripheral neuropathy in IDDM also increases with age and duration of the disease, and the extent of hyperglycemia. Since the age of diagnosis for IDDM peaks during adolescence, these individuals can have significant peripheral neuropathy in their 30's. Another group affected by distal sensory polyneuropathy are HIV infected individuals. This population may become a larger percentage of rehabilitation patients as survival rates for people with AIDS improve. Diabetic peripheral neuropathy has been associated with postural instability, an increased history of falls, and a decreased perceived safety.

**Gait Analysis**

To conduct this study the 3-dimensional gait analysis protocol developed at the Center for Human Kinetic Studies (CHKS), an affiliation of Grand Valley State University and Mary Free Bed Hospital and Rehabilitation Center will be used. Data generated from the non-peripheral neuropathy subjects will form the basis for a geriatric subject
database for CHKS. The computerized 3-D analysis allows collection and analysis of
kinematic and kinetic data from the hip, knee, and ankle throughout the gait cycle. This
method of gait analysis has been used extensively to describe both normal and
pathological gait\textsuperscript{30,31}. Using this system objective comparisons can be made for any
variable at any point during the gait cycle. Proposed measures of dynamic balance will
be compared between subject groups at self-paced normal walking and at a very slow
speed. Testing subjects at a very slow velocity will likely increase balance demands
during ambulation, and may accentuate any differences in dynamic balance between
elderly diabetics with and without peripheral neuropathy\textsuperscript{32}. 
CHAPTER 2
LITERATURE REVIEW

This literature review will focus on characteristics of the study cohort, elderly diabetics with or without peripheral neuropathy. A brief description of the pathogenesis and outcomes of diabetic peripheral neuropathy is provided. A review of the literature on peripheral neuropathy and falls, postural stability, and gait follows. A synopsis of data on elderly gait and balance is included. Dynamic balance, and proposed measurements of balance during gait will also be discussed.

Study Cohort: Elderly Individuals with NIDDM

One of the goals of this study was to establish a database for geriatric gait for CHKS. Results from this study will provide comparative gait data for future work at CHKS with elderly clients. A database specific for this age group is important because changes in gait have been observed with aging. Therefore, to limit variability in gait due to age and to provide data for CHKS, subjects in this study will be 65 years or older.

Age-associated changes in gait have been well documented. Compared to young adults the elderly exhibit decreases in gait velocity and stride length, and an increase in double limb support time. Changes in these time-dependent measures, or parameters of gait, can be described by many discrete measurements of movement or force during walking. For example, decreases in arm swing, pelvic rotation and ankle power at pushoff may be components of an observed decrease in stride length. Several changes in kinematic and kinetic variables have been reported from a
3-dimensional gait analysis of elderly subjects[^13]. From a task theory perspective, elderly gait reflects adaptation of a safer, more stabilizing pattern of movement[^13].

The majority of elderly diabetics have NIDDM[^23], a disease characterized by defects in the production and action of insulin. The pathogenesis of NIDDM is a long process and only partially understood. Resistance to insulin action in target tissues such as liver, muscle and adipose results in a decreased glucose disposal and an increased hepatic glucose output[^36]. These metabolic disorders lead to a hyperglycemic state. Initially, insulin resistance may be overcome by increased insulin secretion, producing a chronic state of hyperinsulinemia. Hyperinsulinemia has been associated with vascular pathologies such as atherosclerosis and peripheral vascular disease[^37]. Ultimately, the insulin secreting B-cells of the pancreas decline in function, a process termed B-cell exhaustion[^36]. A variety of medications including drugs to stimulate insulin secretion, decrease insulin resistance, decrease gut carbohydrate absorption, as well as insulin replacement are used to treat NIDDM[^38,39].

Although people with NIDDM share a common diagnosis, individuals may present with diverse signs and symptoms. NIDDM is a multidimensional disease involving several pathological processes. Complications include macrovascular and microvascular disease, hypertension, nephropathy, retinopathy, and neuropathy[^40]. In the elderly, NIDDM is an important predictor of stroke[^41] and is associated with adverse risk factor profiles for cardiovascular disease[^25,42].

IDDM can result in many of the same late complications as NIDDM, but has a distinctly different pathogenesis. IDDM is an autoimmune disorder characterized by B-cell destruction which leaves the individual dependent on insulin for life[^43]. Onset of
IDDM is typically seen in younger individuals and is often called juvenile diabetes. In the elderly, only 5-10% of newly diagnosed diabetes cases are IDDM\textsuperscript{23}.

**Pathogenesis and Diagnosis of Peripheral Neuropathy**

Diabetic distal symmetrical peripheral neuropathy is described as a late complication of diabetes with an incidence and prevalence that increases with duration of diabetes and poor glycemic control\textsuperscript{18,20,26}. Two major theories have been proposed for the pathogenesis of peripheral neuropathy in diabetics: 1) nerve hypoxia due to peripheral vascular disease and 2) metabolic nerve disorders resulting from chronic hyperglycemia\textsuperscript{21}. The diabetic microvascular complications of arteriosclerosis and thickening of capillary basement membranes could limit blood flow and nutrient delivery to the endoneurium resulting in nerve hypoxia\textsuperscript{21}. It is unclear, however, whether capillary changes in the endoneurium are consistent with a reduced blood supply. Also, peripheral neuropathies not caused by diabetes and lacking vascular involvement have been documented\textsuperscript{21}. Metabolic defects due to excessive glucose have also been implicated as a cause of peripheral neuropathy. Tight glycemic control in IDDM patients has been shown to increase nerve conduction velocities, suggesting a reversal of neuropathy is possible if glucose levels are controlled\textsuperscript{26}. Specific nerve cell defects have been identified in the myoinositol pathway which may interfere with nerve conduction via the sodium-potassium adenosine triphosphatase pump\textsuperscript{21}. It is quite possible that both of these mechanisms are important in diabetic peripheral neuropathy.

Diabetic peripheral neuropathy is diagnosed clinically by sensory testing and nerve conduction velocity measurements of sensory and motor fibers. Although neuropathy is known to include sensory, autonomic and motor fibers, it is clinically described as a predominantly sensory polyneuropathy\textsuperscript{21}. Histopathological nerve
studies show widespread patchy multifocal lesions of proximal and distal nerve fibers\textsuperscript{21}. Longer fibers are more affected, which may explain greater involvement of the lower extremities. Segmental demyelination and remyelination is common.

Declines in nerve function occur preferentially in the lower extremities and exhibit a distal to proximal pattern of functional loss\textsuperscript{1,44} reflecting axon loss in the peripheral nerve trunk, as opposed to a loss of cell bodies in the dorsal root ganglia or anterior horns. Sensory involvement includes the large myelinated fibers carrying vibration, light touch and proprioception, and small unmyelinated pain fibers\textsuperscript{21}. Clinical examinations for peripheral neuropathy include assessment of all these modalities\textsuperscript{16,45,46}. Autonomic fiber loss involving sympathetic and parasympathetic fibers has also been observed in lower extremity blood vessels in diabetic neuropathy\textsuperscript{21}. Autonomic dysfunction is manifested by reductions in sweating (dry shiny skin), thickened dry skin on the foot and thickened toenails\textsuperscript{47}. Motor nerve involvement is seen as atrophy of foot intrinsic muscles\textsuperscript{3,21}. Clawed toes are a common clinical manifestation of diabetic peripheral neuropathy.

Making the diagnosis of diabetic neuropathy is not well standardized, but typically includes a battery of sensory tests, a history of sensory dysfunction or plantar ulcers, and nerve conduction velocity measurements. Strength testing is sometimes included, but is not considered sensitive enough to diagnose mild neuropathy since muscle weakness is not usually apparent until later stages\textsuperscript{16}. Nerve conduction velocity measurements of the sural nerve (primarily sensory) and the peroneal or tibial nerve (primarily motor) have demonstrated decreased amplitude and increased distal latency in neuropathic individuals\textsuperscript{7,16}.

Sensory function is assessed at the great toe, plantar and dorsal surfaces of the foot, the ankle and calf. Ability to detect vibration has been assessed with both 128 Hz
and 60 Hz devices\textsuperscript{6,18,45,46}. Light touch is commonly tested using a Semmes-Weinstein nylon monofilament\textsuperscript{3,16,45}. The integrity of pain and temperature fibers is determined from detection of a pin prick, or cold stimulus\textsuperscript{16,18,45,46}. Position sense is evaluated at the ankle and great toe\textsuperscript{6,7,16}. Ankle reflexes are commonly assessed\textsuperscript{7,18,45,46}, although Thomson and coworkers demonstrated that at least 25\% of nondiabetic elders had absent ankle reflexes\textsuperscript{19}. Thomson et al. also noted a significant age-dependent decrease in vibration perception in non-diabetic elders, but no significant changes in light touch or pain perception\textsuperscript{19}. Assessment of light touch is a key component in clinical testing for a diabetic peripheral neuropathy diagnosis.

Feldman and colleagues compared diabetic neuropathy diagnostic procedures established by the American Diabetes Association\textsuperscript{16} and Dyck et al.\textsuperscript{46} with their own diagnostic protocol developed at the University of Michigan. The Michigan Neuropathy Program, which uses techniques from both of these diagnostic procedures, was developed to provide a simple screening and staging program for clinically significant diabetic peripheral neuropathy that could be used in an outpatient setting\textsuperscript{45}. All three protocols involved a clinical examination including tests described above, nerve conduction velocity measurements, and a history of sensory dysfunction symptoms and ulcers. The Michigan Neuropathy program consists of a two step process composed of the Michigan Neuropathy Screening Instrument (MNSI), a questionnaire and brief clinical examination, and the Michigan Diabetic Neuropathy Score (MDNS), which involves a more detailed physical examination and nerve conduction velocity testing. A high correlation was found for identifying neuropathy when the three methods were applied to the same group of diabetic subjects\textsuperscript{45}. Additionally, the University of Michigan
procedure, without nerve conduction velocity testing, was shown to be valid for diagnosing diabetic neuropathy.

**Adverse Results from Peripheral Neuropathy: Amputations and Falls**

Peripheral neuropathy is a debilitating and sometimes devastating complication of diabetes. Neuropathic ulcers are a major cause of morbidity and mortality in people with diabetes. Ulcers may result from a loss of protective sensation on the plantar surface, or changes in soft tissue and bone in the foot. In diabetics, peak plantar pressures under the foot while walking have been shown to be elevated and may occur in abnormal areas. Elevated pressures can cause tissue necrosis over bony prominences, such as the metatarsal heads, leading to ulcer formation.

Non-healing neuropathic ulcers can necessitate amputation of the toes, foot, or lower leg. Of the 50,000 nontraumatic lower extremity amputations performed in the US each year, 30,000 involve persons with diabetes. In a recent study of middle aged and elderly NIDDM patients the 7-year incidence of lower extremity amputation was 5.6% in men and 5.3% in women.

Falls are a life threatening event for many elderly. Approximately one third of community dwelling elders fall each year. Falls result in serious injuries such as hip or arm fractures, and head trauma, which can compromise the functional reserve of an older person and exacerbate the effects of the fall. Peripheral neuropathy has been associated with an increased risk of falling. Richardson and co-workers reported significantly more falls in a year for peripheral neuropathy subjects compared to controls (56% vs. 8%). In this study repetitive falling was also higher in neuropathic subjects (40% vs. 4%). Although the average age of subjects was 60 years, ages ranged from 25
to 80 years. In a study of younger IDDM subjects (mean age 32 years), Cavanagh and co-workers found an increase in injuries due to falls and a corresponding decrease in perceived safety during ambulation in subjects with peripheral neuropathy.

**Gait Analysis of Diabetics with Peripheral Neuropathy**

Given that most falls occur during ambulation and diabetics with peripheral neuropathy have an increased rate of falling, it is reasonable to ask if differences in gait have been described for this population. Clinicians have described the gait of diabetics with peripheral neuropathy as shuffling, flat-footed, unsteady, and wide stanced. Few research studies have described the gait of diabetic neuropathic patients and all these studies were limited to an analysis of the sagittal plane. Mueller and colleagues compared self-paced gait in diabetics with peripheral neuropathy and age-matched controls. Compared to the control group, diabetics with peripheral neuropathy had a decreased walking velocity (1.26m/s vs. 1.06m/s) and decreased stride length (1.51m vs. 1.20m). Kinetic ankle data at the terminal stance and pre-swing phases of gait, or pushoff, revealed a decreased plantarflexion moment at the ankle compared to normal. No differences in moments were found at the knee or hip. Similar results were observed for peak ankle power generation during terminal stance. Also noted were decreases in ankle plantarflexion strength and dorsiflexion range of motion in the diabetic peripheral neuropathy group. The authors concluded that significant decreases in ankle strength and mobility were primary factors for the differences in gait observed in diabetics with peripheral neuropathy.

Although not statistically tested, Mueller et al. also noted that many diabetic peripheral neuropathy subjects had greater hip flexion moments and power than ankle
moments and power during pushoff. From this finding the authors proposed that diabetics with peripheral neuropathy used more of a hip strategy than an ankle strategy during walking. In other words, to generate power during pushoff, neuropathic individuals pull their leg into swing phase using hip flexors instead of pushing off into swing phase with ankle plantar-flexors. The authors suggested that employing a hip strategy during walking may reduce peak plantar pressures and possibly reduce neuropathic ulcer formation. In another study by the same group peak plantar pressures were reduced in subjects trained to use a hip strategy during gait. The use of a hip strategy during pushoff into swing phase may be an age-associated change in walking. Winter et al. reported a greater hip flexion power generation and a corresponding decrease in ankle power at pushoff in fit and healthy elderly as compared to young adults.

While providing a significant contribution to the description of gait in diabetics with peripheral neuropathy, these studies have a few shortcomings. No sensory testing was performed to confirm the presence of peripheral neuropathy, or to describe sensory deficits. Diabetic subjects were chosen for the studies on the basis of recent plantar ulcers. The presence of a plantar ulcer may greatly affect gait. Habitual gait deviations acquired during the lengthy healing process may have residual effects on walking after the ulcer is healed. Limitations in strength and range of motion at the ankle may reflect ulcer treatment procedures, the effects of deconditioning, or decreased mobility due to the ulcer. Additionally, controls, although age-matched, were not diabetic.

In a study of postural stability in diabetics with peripheral neuropathy, Simoneau and co-workers identified similar limitations in ankle strength and range of motion in diabetics with and without peripheral neuropathy as compared to nondiabetic controls. Although both diabetic groups had decreased strength and range of motion, only the
peripheral neuropathy group demonstrated decreases in postural stability. While this is a different measure than gait, both tasks involve the use of sensory feedback to maintain balance. It is possible that decreased ankle strength and range is due to other diabetic physiological pathologies than peripheral neuropathy.

Cavanagh and coworkers described initial studies of gait in diabetics with and without peripheral neuropathy\(^3\). The only measurement reported was a sagittal plane description of the variability of the combined kinematic motion of the hip, knee and ankle. This study sought to test the hypothesis that the gait of neuropathic diabetics would be more variable, reflecting a loss in sensory feedback. A "limb angle", formed by a line joining the hip to the head of the fifth metatarsal was used to assess kinematic variability throughout several gait cycles. No differences were noted between diabetics with or without peripheral neuropathy at any point in the gait cycle, but the neuropathic group had slightly greater variability overall\(^3\).

In a recent study, Courtemanche and co-workers described the gait of subjects with diabetic peripheral neuropathy and controls\(^2\). Data from trials of self-paced walking showed decreases in velocity (1.06m/s vs. 1.32m/s), stride length (1.23m vs. 1.43m), and time spent in single limb support phase (65.17% vs. 70.14%) for the diabetic peripheral neuropathy group as compared to controls. These values for velocity and stride length are similar to those presented by Mueller. As in Mueller's study\(^5\), control subjects in this study were not diabetic. However, there were no differences in ankle strength between the neuropathic and control groups.

The effect of an increased attentional demand on gait was also tested in the Courtemanche study by determining reaction times to an auditory signal given during walking and when seated\(^2\). The response in the seated position required a slight movement of thumb and index finger, while an auditory response was used for the
walking task. Reaction times were greater in both groups for the walking task compared to the seated task. Reaction times were greater for diabetics with peripheral neuropathy than controls during the walking task, but not during the seated task. The authors interpreted these results as indicating an increased attentional demand for walking in the diabetic peripheral neuropathy subjects, due to loss of sensory input. Sensory impairments likely increased the demands for balance on the central nervous system as reflected by longer reaction times during walking. They also concluded that decreased gait speed, stride length, and increased single limb support time reflected a more stable gait, which reduces attentional demands, and helps control for decreased sensory input. A more stable gait may be adopted as a compensatory mechanism to control for decreased balance abilities.

Information on the effects of diabetic peripheral neuropathy is limited. Slower walking speeds and reduced stride lengths have been reported in two studies, although different explanations have been proposed for these differences. While these observations of gait are helpful, they do not provide information about the causes of such differences. Studies have suggested that orthopedic issues of strength and range of motion, and neurological issues involving sensory feedback and balance are important. Kinematic and kinetic analysis of gait has focused on only one point of the gait cycle, terminal stance, and only in the sagittal plane. To date, nothing has been reported regarding movement in the frontal or transverse planes during walking. Also, no researchers have investigated measures of dynamic balance during gait in diabetic individuals with peripheral neuropathy as compared to diabetics without peripheral neuropathy, or compared to healthy elderly.
Dynamic Balance: How to Measure It?

Walking has been described as a series of controlled falls in which the body is propelled forward, ahead of its base of support (BOS). The swing limb advances to initial contact and restores the BOS anterior to the body. During walking the center of mass of the body (COM) is outside the limits of the BOS an estimated 80% of the time. The task of maintaining such a dynamic balance is much different from the requirements of static standing balance where the goal of the body is to keep the COM within the limits of the BOS. Although there are no documented single measures of dynamic balance during walking, it may be possible to draw some conclusions by combining several measurements to establish patterns representative of stability and instability.

A few variables have been proposed as specific measures of dynamic balance in gait analysis. These measures involve analyzing movement in the sagittal and frontal planes. In the sagittal plane, at initial contact of stance phase, decreased dynamic balance may be reflected by a greater horizontal heel velocity, suggesting an increased risk of slipping. Foot placement in the peripheral neuropathy group may be more variable due to decreased somatosensation, and result in making initial contact on other areas of the foot than the heel.

In elderly gait at pushoff, Winter and co-workers have demonstrated a decreased generation of ankle power to push the body forward, with corresponding increases in hip flexor power and absorption of power at the knee. Taken together, these data indicate a decreased reliance on pushoff power from the ankle and an increased use of the hip flexors to pull the trailing limb through in swing phase. As plantarflexor power developed at the ankle, the knee absorbed some of it to effectively dampen the propulsive force being generated. This increased absorption of power at the knee may represent a
mechanism by which elderly subjects shortened their stride length, thereby decreasing the balance demands of walking.

Winter has described another kinetic measurement of dynamic balance in the sagittal plane, termed a "covariance" of moments, or forces, at the hip and knee throughout stance phase. Winter suggested that throughout stance phase the hip and knee worked in a coordinated fashion to ensure the maintenance of an adequate support moment to prevent the collapse of the limb. The overall moment of the stance limb, combining moments from the hip, knee and ankle has been defined as the support moment of the limb. It is this support moment which prevents collapse of the limb against gravity. According to Winter, movements of the hip and knee are coupled neurologically in opposite directions. For example, as body weight is supported and balanced over the stance limb, the stance hip may exhibit an internal flexion moment but the knee will have an increased internal extension moment. The stance hip and knee moments are therefore out of phase with each other. While one joint moves more into flexion, producing an applied flexion moment, the other joint moment becomes more extensor. The net effect is a dynamic interaction between the hip and knee to provide balance of the body in the sagittal plane during walking. Winter has termed the covariance of the hip and knee moments an "index of dynamic balance".

In their gait analysis of fit and healthy elderly, Winter and colleagues observed a trend towards a decreased hip-knee covariance, as compared to young adults. Patta and colleagues have suggested that such a decrease in the stance phase hip-knee covariance reflected a decrease in the neural plasticity of an individual's balance control. In other words, an individual with compromised balance, such as what might be expected in a diabetic peripheral neuropathy subject, will likely adopt a more consistent (less flexible) dynamic balance strategy to compensate for impairments like
somatosensory deficits. This type of individual may have less of an ability to adjust for an increased or unexpected demand of dynamic balance during walking.

In the frontal plane MacKinnon and Winter have described dynamic balance in terms of the movement of the body's COM in relation to the BOS. The whole body COM moves slightly from side to side as steps are taken with the right then left leg. This movement was documented by tracing the medial-lateral path of the COM in relation to the center of pressure (COP) beneath the foot. As a step is taken on the right with the left leg in swing phase, the body "falls away" from the right foot and towards the anticipated point where the left foot will make contact. The left foot will contact the ground such that its COP will be lateral to the COM. In this way the COM will travel in a sinusoidal path from left to right within the lateral boundaries of the stance foot, or BOS. Thus, the BOS in the frontal plane is maintained wider than the COM trajectory.

MacKinnon and Winter have shown the hips to be major controllers of balance in the frontal plane. Using the inverted pendulum model proposed by Winter, balance in the frontal plane can be viewed as a task requiring control of the body from the head to the hips. The head, arms and trunk (HAT) represents, on average, two thirds of body weight. MacKinnon and Winter represented this mass as an inverted pendulum balancing on the hips and controlled by the hip muscles. Their theory stems from an analysis of the forces required to control HAT in the frontal plane. MacKinnon and Winter suggested these forces exceeded the capacity of the ankle musculature. If balance control in the frontal plane is determined by actions at the hips, then the hip moment, a measure which describes the forces acting on the hips must be important. The stance hip musculature must provide medial-lateral balance control, especially during single limb support. Muscle activity at the swing hip is important for proper foot placement to ensure the COP is lateral to the COM. Winter has suggested that frontal plane balance
can be studied by an analysis of the hip moments in stance and swing phase, and the progression of the path of COM in relation to COP (Winter, personal communication).

Recently, Besser and co-workers described an alternative method of studying the progression of COM in relation to the COP. The purpose of their work was to provide a measure of dynamic balance during walking which would allow for future comparison of elderly fallers and non-fallers. Data was presented as the distance between the COM and COP, and the difference in the velocity of movement of each. A trajectory of the movement of COM and COP in relation to the other was analyzed for movement in the anterior-posterior and medial-lateral directions. Jian and co-workers have reported on the relationship between COM and COP during initiation and termination of gait and concluded that the interaction between COM and COP is tightly coupled in order to control the body's COM and balance. Researchers studying balance during both walking and transitional movements have explored the dynamic interaction between the whole body COM and its' BOS.

A decrease in dynamic balance during walking may be manifested by a decrease in walking speed and stride length, two interrelated variables. Decreases in these two time-dependent measures of gait have been observed in the elderly population compared to young adults, and in diabetics with peripheral neuropathy as compared to age-matched controls. The parameters of velocity and stride length are composed of many kinematic and kinetic variables. For example, a decrease in stride length may be a product of a decreased ankle pushoff power and a concomitant increased knee power absorption as proposed by Winter et al. An increase in double limb support time has also been equated with a more conservative gait pattern, suggesting the presence of a decreased dynamic balance.
To study dynamic balance researchers have incorporated walking tasks designed to challenge a subject’s balance. A wide variety of perturbations to normal walking such as changing gait speed, unexpected turns, stepping over obstacles, adding attentional demands, and upper body "jostling" have been used. While many techniques have been successfully employed to perturb balance there has yet to be agreement on which ones represent the best test of dynamic balance. The initiation and termination of gait have also been studied to assess the transition of the body from a stable position to a dynamic one.

Clinical Assessment of Dynamic Balance

Functional assessments, which are useful clinical tools, have been developed to assess dynamic balance. They are comprised of a battery of dynamic tasks such as walking, and ascending and descending stairs. These tools do not provide specific information regarding the source of impairments, but are invaluable to the clinician for assessment and treatment planning. Some functional assessment tests have been shown to be reliable and valid to predict an increased risk of falls for a patient. Typically, these tests have not been quantified using motion analysis, force plates, or dynamic electromyography.

Functional assessment tests incorporate tasks which have been associated with falls in elders such as rising from a chair, walking, and changing directions while walking. Based on these functions, the Get-up and Go test consists of rising from a chair, walking a short distance, turning around to walk back to the chair and sit. Tinetti and colleagues developed a test for assessing mobility and gait in elderly subjects that has been correlated with an increased risk of falls. This test includes static and dynamic balance assessment in the seated and standing positions. The Functional Balance
Scale, or Berg Balance Scale, consists of multiple balance-related tasks to evaluate posture maintenance, voluntary movement, and response to external balance perturbations. A tool used with higher functioning individuals is the Dynamic Gait Index, in which all tasks assessed involve gait activities such as stepping over obstacles or changing velocity while walking. The Dynamic Gait Index has been determined to be a valid and reliable tool for determining the risk of falls. Although these functional assessments of dynamic balance are useful tools for clinicians, they do not provide insight into causation. Quantified motion analysis, with a focus on dynamic balance measures or variables, may provide researchers with better information to understand balance deficits.

**Balance in Quiet Standing and Diabetic Peripheral Neuropathy**

During quiet standing a body will sway slightly both forward and back, and side to side, which is reflected in small excursions of the body's COP. Postural stability in quiet standing, a static state, is commonly assessed by measuring body sway as the subject stands on a force plate. Subjects with greater COP excursions are described as having decreased postural stability. (See Shumway-Cook for review)

The demands of quiet standing are different than those of walking. In quiet standing the COM must stay within the BOS maintain balance. Walking requires control of the COM outside of the BOS. Postural stability measured in a static state is not well correlated with functional dynamic tasks. Also, interventions to improve standing balance do not necessarily decrease risk of falls, or increase an individual's perceived safety while walking. In a recent study on the effect of Tai Chi and balance platform training on postural stability, Wolf and coworkers demonstrated an increase in postural stability (decreased COP excursions) in the balance platform trained group, but not in
the Tai Chi group. However, the Tai Chi group, but not the balance training group, reported a decreased fear of falling.

Postural instability in quiet standing in diabetic individuals with peripheral neuropathy has been reported by several authors. Simoneau and coworkers reported greater total COP excursions in diabetics with peripheral neuropathy as compared to diabetic or nondiabetic controls. Subjects were tested with eyes open and closed, and head straight and tilted back. In all conditions, only the peripheral neuropathy group showed a greater instability. Richardson and colleagues reported a greater excursion in COP and a decrease in unipedal stance time in elderly peripheral neuropathy subjects compared with age-matched controls. Peripheral neuropathy subjects maintained a unipedal stance for only 3.83 sec. as compared to 32.3 sec. for the control group. In elderly diabetic subjects with peripheral neuropathy Boucher et al. found increases in COP excursion, both in magnitude of sway and total area of excursion as compared to elderly nondiabetics. These authors also reported an increase in velocity of sway among peripheral neuropathy subjects. Similar findings were reported by Uccioli and associates for IDDM subjects with peripheral neuropathy as compared to non-neuropathic diabetics and nondiabetic controls. Only diabetics with peripheral neuropathy demonstrated increases in length of COP excursion or total area, or velocity of sway. IDDM subjects without neuropathy did not differ from nondiabetic controls in any of these measures. Taken together with Simoneau's data, these data suggest that postural instability in diabetics with peripheral neuropathy is a function of the neuropathy and not diabetes per se.

In a follow-up study of Uccioli's work using the same group of subjects, Giacomini et al. looked at postural control strategies used during quiet stance in IDDM subjects with or without peripheral neuropathy, and in controls. Velocity of COP excursion, as
well as the variability of COP velocity, were greater in diabetics with peripheral neuropathy. Again, no differences were noted between IDDM subjects without neuropathy and nondiabetic controls. Additionally, the authors described the variance of COP velocity, a function of COM movement in the sagittal plane, as a value which could be used to interpret postural control strategies. Diabetics with peripheral neuropathy had a greater variance of COP velocity in relation to their COM movement than the other two groups. The authors concluded that the neuropathic individuals controlled their balance in the sagittal plane from the hip joints, termed a hip strategy, in contrast to the ankle strategies used by the control and non-neuropathic diabetic groups. Assuming an inverted pendulum model for balance, an ankle strategy is normally seen in quiet standing on a firm level surface (see Shumway-Cook for review^64). In IDDM subjects with peripheral neuropathy balance control was directed from the hip, presumably the result of neurological impairments in the distal lower extremities.

While neurological impairments may involve sensory and motor involvement, a case can be made for the importance of sensory dysfunction in postural instability by considering the work of Simoneau^6 and Giacomini^65. Both studies demonstrated postural instability in diabetic subjects with neuropathy, but not in non-neuropathic diabetics. Simoneau reported decreases in ankle plantarflexion strength and dorsiflexion range of motion in the two diabetic groups as compared to nondiabetic controls^6. Postural instability was associated with the presence of peripheral neuropathy, but not with decreased ankle strength or flexibility. Another sensory function, ankle proprioception, has been shown to be diminished in elderly subjects with peripheral neuropathy^67. Peripheral neuropathy subjects had a 4.6 fold larger threshold for detection of ankle inversion and eversion, and a decreased success rate at detecting movement in a standing, weight bearing, position. To summarize, sensory deficits
present with diabetic peripheral neuropathy appear to result in postural instability in quiet standing. This effect is probably due to peripheral nerve impairments, not other pathologies associated with diabetes.

**Summary**

Distal symmetrical primarily sensory polyneuropathy is a common complication of diabetes, affecting more than 50% of elderly individuals with NIDDM. Nerve impairment begins in the most distal parts of the extremities, usually the feet, and progresses proximally. The extent of nerve damage is associated with age and duration of diabetes. Diabetic peripheral neuropathy is diagnosed by symptoms, a battery of sensory tests, and nerve conduction velocity measurements of sensory and motor fibers.

Diabetic peripheral neuropathy has been associated with falls, decreased postural stability during quiet standing, and changes in gait. Gait studies have focused on linear and temporal measures such as velocity and stride length, and ankle function during the terminal stance phase. Authors have attributed differences in gait to decreased strength and mobility, or to impaired sensory feedback. Measures of dynamic balance would be helpful to assess the importance of sensory impairment during walking. While standardized dynamic balance variables during gait have yet to be identified, several quantitative measurements have been proposed to study balance in the stance and swing phases of gait. Functional assessment tools such as the Dynamic Gait Index also offer important, clinically relevant, information on dynamic balance.

The purpose of this study was to describe differences in gait between elderly diabetic subjects with distal symmetrical sensory polyneuropathy and elderly diabetics without neuropathy. A secondary goal was to test the validity of proposed measures of dynamic balance during gait in this model of sensory impairment. A 3-dimensional gait analysis provided an assessment of movement of the hip, knee, and ankle during self-
paced normal walking and at a very slow speed. It was hypothesized that elderly diabetic subjects with distal sensory polyneuropathy will exhibit differences in their gait from elderly diabetic control subjects.
CHAPTER 3
METHODOLOGY

Design

A 3-dimensional gait analysis of community dwelling elderly diabetic female subjects with and without peripheral neuropathy was conducted. Subjects were analyzed during a self-paced walk and a slow walk. The gait test took place at the Center for Human Kinetic Studies (CHKS), Grand Rapids, MI, an affiliation of Grand Valley State University and Mary Free Bed Hospital and Rehabilitation Center, in accordance with established CHKS protocols for collection of kinetic and kinematic data. Subjects were divided into two groups of diabetics, those with peripheral neuropathy (PN) and those without PN. Subjects without PN served as controls.

Subjects

Subjects in this study were limited to women 65 years or older with a diagnosis of NIDDM with and without a diagnosis of peripheral neuropathy. To better control for variability in the disease process, individuals with IDDM were excluded. NIDDM and IDDM differ markedly in pathogenesis and age of onset. The pathology of both types is complex, affecting many physiological systems. Since development of peripheral neuropathy is associated with duration of diabetes and glycemic control, it was important to limit subject variability due to diabetes type.

Seven elderly diabetic women, 65 years and older, with a diagnosis of NIDDM were initially screened for the study. Four of these women participated. Subjects were recruited through Grand Rapids area practitioners who specialize in diabetes and via
advertisement at events sponsored by the Michigan chapter of the American Diabetes Association. Participation in the study required the elder to be a community dweller, ambulate independently without device or limitation due to pain, and have the endurance to walk two blocks. A questionnaire was used (Appendix A) as an initial screening for orthopedic, neurologic or pathological conditions affecting the subject's gait. Results of the questionnaire were applied to the exclusion criteria listed in Table 1, page 29.

Peripheral neuropathy was confirmed using the Michigan Diabetic Neuropathy Score (MDNS) (Appendix C) and the Michigan Neuropathy Screening Instrument (MNSI) (Appendix D) developed at the University of Michigan. The MDNS was used without the nerve conduction velocity portion of the examination, a method which has been determined adequate to diagnose peripheral neuropathy when used in conjunction with the MNSI (Feldman, personal communication). A MDNS score greater than or equal to 7 out of 46, diagnostic for diabetic peripheral neuropathy, was used to determine the presence of peripheral neuropathy in this study. The questionnaire portion of the MNSI was used to describe severity of existing neuropathy.

The exclusion criteria for this study are listed in Table 1, page 27. The list is extensive, however, many variables can influence gait. A history of lower extremity surgery was exclusionary since the procedure may have impacted the individual's gait secondary to the healing process, scar tissue, or resultant weakness. In addition, subjects were devoid of any plantar ulcer history. The rehabilitation of an ulcer could have impacted on strength and flexibility at the ankle, which in turn may have changed a person's gait (see commentaries in Mueller et al.). Leg length was measured since the gait test was done in bare feet and many people with leg length discrepancies greater than one centimeter require a shoe insert to correct for musculoskeletal disorders or pain.
caused by the shorter limb. Individuals with uncontrolled hypertension, defined as a resting blood pressure greater than or equal to 165/95 mm Hg, were excluded. 

The Dynamic Gait Index (Appendix F) was used to screen for cardiovascular effects with activity, endurance, gross vestibular dysfunction, and for later analysis of scores and comparison of subject groups. Activities in the Dynamic Gait Index range in metabolic cost from 2 to 3 metabolic equivalents, or METS, except stair ascension which may require 4 to 7 METS. The gait test required 2 to 3 METS for subjects to walk at a normal speed of 1.0 m/s. A perceived exertion level greater than or equal to 5 out of 10, or described as "hard" following this test excluded subjects from the study. A change in systolic or diastolic blood pressure of greater than or equal to 20 mmHg, or a rise in heart rate greater than 40% above the resting value immediately following the Dynamic Gait Index test were grounds for exclusion, as discussed by the American College of Sports Medicine. This level of activity should not constitute an aerobic exercise session.

<table>
<thead>
<tr>
<th>Table 1. Exclusion Criteria for Participation</th>
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<tr>
<td>-Lower extremity amputation</td>
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<td>-History of plantar ulcers</td>
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<tr>
<td>-Ambulation with assistance or device,</td>
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<td>including braces</td>
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<tr>
<td>-Surgery within one month of study</td>
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<tr>
<td>-Back surgery within one year of study</td>
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<tr>
<td>-History of major lower extremity surgery,</td>
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<td>with subjective report of permanent</td>
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<tr>
<td>change in ambulatory status</td>
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<tr>
<td>-Change in systolic or diastolic blood</td>
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<tr>
<td>pressure by 29 mm Hg or more after</td>
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<tr>
<td>the Dynamic Gait Index test</td>
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<tr>
<td>-Asymmetry in upper or lower extremity</td>
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<tr>
<td>strength as demonstrated by a difference</td>
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<tr>
<td>equal to or greater than one manual</td>
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<tr>
<td>muscle test grade</td>
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<tr>
<td>-Inability to follow verbal command after</td>
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<tr>
<td>3 repetitions</td>
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<td></td>
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<tr>
<td>-Breaks in skin covering feet, at time of</td>
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<tr>
<td>study</td>
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<tr>
<td>-Unstable medical conditions such as</td>
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<td>unstable angina, hypertension or seizures</td>
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<tr>
<td>-History of central nervous system</td>
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<tr>
<td>dysfunction, or detection upon physical</td>
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<tr>
<td>examination</td>
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<tr>
<td>-Leg length discrepancy greater than 1 cm</td>
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<tr>
<td>-Resting blood pressure greater than or</td>
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<tr>
<td>equal to 165/95</td>
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<tr>
<td>-Perceived Relative Exertion greater than</td>
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<tr>
<td>or equal to 5 out of 10 following the</td>
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<tr>
<td>Dynamic Gait Index test</td>
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<tr>
<td>-No active diagnosis of vestibular problems,</td>
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<tr>
<td>or difficulty with items 3 or 4</td>
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<tr>
<td>on Dynamic Gait Index test</td>
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Procedure

Prior to data collection, subjects were contacted by phone and screened for exclusion criteria using the questionnaire in (Appendix A). A clinical examination (Appendix B) was scheduled for those individuals meeting the inclusion criteria addressed in the questionnaire. The physical examination occurred in the subject's home. The clinical examination, lasting approximately 1 hour, and the gait test lasting approximately 3 hours, were conducted on separate days to avoid subject fatigue. An informed consent (Appendix G) was obtained prior to the clinical examination and gait test. Subjects received a copy of the signed informed consent. Prior to the clinical examination, the goals of and procedures used in the study were reviewed with the subject. The clinical examination was conducted, and results recorded on the Clinical Examination form in (Appendix B). Following the examination, a demonstrative video of a gait test was made available to familiarize the subject with the procedures.

A past medical history was obtained from the subject, including duration of NIDDM, medications used, and a history of falls. Specifically, the number of falls within the past year, and the number of injuries incurred from falls during the past year were recorded. If the subject self-monitored blood glucose, the most recent fasting level was requested. Subjects with peripheral neuropathy were asked about the duration of neuropathy and whether a nerve conduction velocity test was performed in the previous year.

Peripheral neuropathy was confirmed using the MDNS (Appendix C) and MNSI (Appendix D) neuropathy assessment tools. The MDNS and MNSI were administered according to instructions supplied by the Michigan Diabetic Neuropathy Program listed in (Appendix E). To familiarize subjects with sensory assessment, each test was first performed on an area of the subject not expected to be affected by peripheral nerve impairments. Vibration testing was demonstrated on the subject's clavicle. Vibration
sensation was tested bilaterally on the dorsum of the great toe. Refer to (Appendix E) for details of the procedure. Light touch with a 10 gram monofilament was tested bilaterally at the great toe per instructions in (Appendix E). Sharp/dull sensation was tested at the same site. A random sequence of 10 trials was used. Prior to testing the great toe, sharp/dull sensation was demonstrated on the dorsum of the subject's forearm. Deep tendon reflexes were tested in bilateral biceps brachii, triceps brachii, quadriceps femoris and achilles. They were scored 0 for present, 1 for present with reinforcement, or 2 for absent.

Additionally, position sense, or proprioception, was determined at the ankle and great toe as part of the clinical examination (Appendix B). Proprioceptive and kinesthetic awareness were assessed by asking the subject to identify the presence of movement, and the direction of movement, of the body segment. To familiarize subjects with this task a researcher first demonstrated with flexion and extension movements of the subject's elbow. With eyes closed, the subject was asked to identify when the elbow was moved, and in which direction, either "up" into flexion or "down" into extension. To test the great toe, a researcher lightly grasped the medial and lateral borders at the IP joint, and flexed and extended the toe in a random sequence through 10 repetitions of varying speed and distance. Correct responses of movement detection and direction in 8 out of 10 trials constituted a negative finding for peripheral neuropathy (Richardson, personal communication). The ankle was tested in the same manner with the researcher's hand placement on the medial and lateral borders, distal to the tarsometatarsal joints of the foot. The ankle was moved through dorsiflexion and plantarflexion. The ankle was tested to rule out upper motor neuron involvement. Correct responses to movement detection and direction fewer than 8 times would raise suspicion of upper motor neuron involvement (Richardson, personal communication).
The clinical examination also included other tests designed to detect neurological dysfunctions not related to diabetic peripheral neuropathy. Some central nervous system deficits are very subtle and may go unnoticed by the subject, and therefore would not be detected in the initial screening questionnaire. It was important to exclude these cases because of the potential influence on gait. Central nervous system (CNS) function testing in the clinical examination included the following; the heel-to-shin and finger-to-nose tests, an assessment of rapid alternating movements in the upper and lower extremities, and the pronator drift test. In the coordination tests the subjects were asked to touch their heel to the shin of the opposite leg. Subjects were instructed to slide their heel up and down the shin 3 times, as fast as possible. The test was repeated with the opposite leg. If asymmetry was noted, an upper extremity finger to nose test was conducted, in which the subject was asked to touch their finger to nose and then to the examiner's finger, 3 times, as fast as possible. Rapid alternating movement tests involved repeated quick tapping of either toes on the floor, or supination/pronation of forearms. The quality and symmetry of movement were assessed during these tests, specifically the coordination, accuracy and speed. Decreased speed of movement, hypermetria, or inability to perform the movement smoothly, when compared to the opposite limb, would be indicative of a possible CNS lesion, and would exclude the subject from the study. Rapid alternating movement tests also screened for Parkinson's disease, as evidenced by significantly slowed movement, or an inability to maintain cyclical movement. For the pronator drift test the subject stood with arms extended to 90° and supinated, and the eyes closed. In persons with a CNS lesion one arm may drift into pronation.

Subjects were then evaluated with the Dynamic Gait Index, described in (Appendix F). This functional clinical assessment tool evaluated the ability of a subject to
respond to additional task demands during walking such as turning and stepping over obstacles. A researcher followed the subject closely during the Dynamic Gait Index test to monitor the subject for loss of balance and to prevent falls during testing. To assist the researcher in guarding, subjects wore a standard physical therapy gait belt during the test. Blood pressure, heart rate, and a rating of relative perceived exertion (RPE), (Appendix J) was obtained prior to the Dynamic Gait Index test (to provide resting values), immediately after completion of the test, and at the end of a three minute rest period. At the completion of the clinical examination, subjects not suspect of CNS lesions or other neurological diseases, who completed the DGI test without demonstrating vital signs that constituted an aerobic activity, were scheduled for a gait test.

On the day of the gait test, the remainder of the physical examination was conducted to assess strength and range of motion (ROM) of upper and lower extremities, and obtain the anthropometric measurements described in (Appendix H). Active ROM of the upper and lower extremities was performed to assess for any gross asymmetries that may influence the subject's gait pattern. Specific manual muscle strength tests, described by Kendall et. al, were performed for ankle dorsiflexion, great toe extension and finger spread strength as part of the MDNS score. Muscle strength was scored as 0 for normal, 1 for a 25% to 50% weakness, 2 for a 75% weakness, and 3 for a 100% weakness. In addition, ROM measurements were taken for knee extension, ankle dorsiflexion and hip extension to use during evaluation of data. Goniometric measurements were used to determine range of motion. Anthropometric measures were necessary for the formulation of kinetic gait data. Measurements were taken at the pelvis and lower extremity as described in (Appendix H). Body weight and height were also recorded.
Following the clinical examination, subjects were prepared for the placement of the reflective targets. The reflectors were held in place by adhesive at the following anatomical landmarks: medial and lateral foot, calcaneus, mid-shank along the tibial crest, mid-calf just below the gastocnemius muscle belly, tibial tuberosity, lateral femoral condyle, lateral thigh, the second spinous process of the sacrum and lateral to the anterior superior iliac spines (ASIS). The targets were placed lateral to the anterior superior iliac spines because excess abdominal soft tissue interfered with camera views of the targets. The lateral thigh reflector was held in place with elastic straps wrapped around the subject's thigh. Reflective markers were placed for either right or left limb data collection as determined by a coin toss. Data were collected for one limb, and then the reflective markers were removed and placed on the contralateral extremity for testing.

Subjects were asked to practice walking through the test space at their normal pace (self-paced) to familiarize themselves with the laboratory, as well as to allow researchers to estimate placement of the force plates. Force plate placement was adjusted by researchers to register the entire stance phase on the first force plate and a second initial contact on the second force plate. Data from force plates were used to determine the path of the center of pressure (COP) and magnitude of the ground reaction force. The coordinate system of the force plate was defined relative to the laboratory coordinate system. Once the testing area was measured for the subject's height, weight, and stride length, the gait test began.

The subject was required to walk a distance of approximately 10 meters, turn and walk back. The subject sat and rested, as needed, between walking trials. Data were collected from 5 self-paced walking trials and 5 slow walking trials for right and left limbs. For a walking trial to be acceptable, the subject must have walked through the laboratory
space uninterrupted, and make contact on both force plates with only the foot of the limb being tested.

Velocity of the slow walk was adjusted to approximately 1/3 of the subject's normal pace, and was in the range of 0.35 meters per second. To determine the target speed for the slow walk, all 5 trials of the self-paced walk were timed with a stopwatch. An average normal velocity was calculated for each limb from which the target slow velocity was calculated as 1/3 of the self-paced speed. Subjects practiced walking at the calculated slow speed a minimum number of times to familiarize themselves with the task. Gait data were then collected from 5 trials at the slow speed with researchers timing each trial. Trials with velocities 50% greater than, or 50% less than the calculated slow velocity were repeated. For example, if a subject's self-paced velocity was 1.0 m/s, the target slow speed would be 0.3 m/s. Any trials at speeds less than 0.15 m/s, or greater than 0.45 m/s were repeated.

A standing trial was performed following the gait tests. With this test the subject stood in the center of the calibration space for approximately 2 seconds while target position data were collected. The standing trial data were used to calculate joint centers of the hip, knee and ankle, and required placement of additional reflective markers on the medial epicondyle of the femur, the medial and lateral malleoli, and directly over both pelvic ASIS. Because the pelvic targets were placed lateral to the ASIS bilaterally during the walking trials, a special processing program was generated to determine hip joint center from the standing file. The distance between the lateral target placement and the position directly over the ASIS was calculated. This difference was applied to the dynamic coordinate system and the hip joint center was calculated. After data were collected for the right and left legs, the subject walked through the test space with reflective targets only on the calcaneus and forefoot of both feet for six additional trials to determine linear and temporal gait parameters.
Blood pressure, heart rate, and RPE were monitored at the onset and throughout the gait test. Blood pressure, heart rate, and RPE were documented after each set of walking trials at self-paced and slow speeds, and for both limbs using the form in (Appendix I). Subjects were educated on RPE and asked for a rating using the 10 point scale described in (Appendix J). The subject was asked to rest if systolic or diastolic blood pressure changed more than 20 mm Hg, or if heart rate rose more than 40% of the resting value.

Instrumentation

Gait analysis occurred through use of motions systems and force plates at the CHKS lab. The Bioengineering Technology and Systems (BTS) Elite Motion Analyzer, and Advanced Medical Technologies, Inc. (AMTI) force plates were used. The BTS Elite Motion Analyzer consisted of four Elite, solid state pixel perfect, CCD high speed cameras. Each camera has light emitting diodes (LEDS) formed in a ring around the lens. The LEDs emit infrared rays, which are reflected back to each camera when they hit the targets that are placed on the subject. Each target was a small wooden ball, covered with 3M Scotchlite Brand High Grain 7610 retroreflective tape. Measurements from the cameras, together with LED impulses were sampled at 100 Hz. An image on a two-dimensional plane was generated by each camera from the reflected signal. Video data were processed simultaneously from each camera through the use of a video processor sending synchronous camera signals to a computer. At least two cameras had to identify each target in order to determine its position in three-dimensional space. Accuracy of the Elite system is reported to be within 3.2 mm of target location.

Cameras were placed in each corner of the designated 2m x 1m x 1m testing volume

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1 Elite, BTS, Milano, Italy
2 AMTI, Advanced Medical Technologies Inc., Newton, MA
and positioned to capture targets as the subject walked through the test space. Prior to data collection, a standardized calibration procedure was used to determine the working volume in relation to each camera position by using a rigid grid system of reflective targets with defined spatial coordinates. Calibration defined the position of each camera relative to the others, and relative to the testing volume and created a laboratory spatial coordinate system.

Two Super Panasonic x20 Digital Zoom VHS cameras\(^4\) were used to video tape the subject in sagittal and frontal views simultaneously, during the gait test. This video record was used for observational evaluation. The images were projected onto a single screen by a Panasonic Digital Effects Generator\(^4\).

Two AMTI force plates were used to determine ground reaction forces and moments, indicating the start and finish of the gait cycle. Three orthogonal forces and moment components were monitored by each force plate using foil strain gauges, which were attached to load cells at the four corners of each platform. Data from the force plates were sampled at 1000 Hz and synchronized with the BTS motion system. The force plate signals were amplified by a signal conditioner/amplifier (AMTI SGA6-4) with a gain of 4000 and filtered.

Other instruments used during the clinical exam included a goniometer, blood pressure cuff and sphygmomanometer, gait belt, stopwatch, tape measure, calipers, a Semmes-Weinstein 5.07 (10g) monofilament, 128 Hz tuning fork, and a reflex hammer.

\(^3\) 3M Health Care, Medical Supply Division, St. Paul, MN
\(^4\) Panasonic Co., Matsushita Electrical Corp., Secaucus, NJ
Data Analysis

Kinematic and kinetic data was processed from all walking trials using the BTS system and CHKS customized software. Three-dimensional joint angles, and applied joint moments were calculated for the hip, knee, and ankle. Joint moments were calculated from joint center position and COP data. Inertial and gravitational forces were not accounted for in this quasi-static analysis of joint moment. Kinematic and kinetic data obtained from walking trials of each subject were splined to convert data from time scale to percent gait cycle. Individual walking trials for each limb were averaged at both self-paced and slow speeds. Additional processing converted force, torque, and power files from percent stance to percent gait cycle.

Subject demographic data: diagnosis of PN, duration of NIDDM, age, and body mass index (BMI) were compared. Also investigated were the number of co-morbidities in addition to NIDDM, the number of current medications, and the number of falls within past year as well as scores on the DGI, MNSI, and MDNS tests. Gait temporal-spatial parameter data from the left and right limbs for self-paced and slow walking speeds were calculated and included velocity, stride length, step width, and percent time spent in stance and in single limb support. Ankle kinematics during stance phase of gait was measured as total range of motion (ROM) and maximum dorsiflexion (DF). Data were represented in degrees as means ± standard deviation (sd) and compared for the right and left extremities for each subject and between subjects. Kinetic measurement of ankle function in preswing was assessed as peak plantarflexion torque (Nm/kg) and associated peak power generation (Watts/kg). Mean values ±sd were used for intersubject comparison. Self-paced and slow velocities for both right and left limbs were evaluated. Right and left hip peak power generation and knee peak power absorption were measured during preswing for self-paced and slow speeds. Intrasubject and intersubject comparisons were made using a mean ± sd.
Pelvis, hip, and knee kinematic motion (degrees) in the sagittal plane was compared within subjects and between the control and PN groups. Data were evaluated for self-paced and slow velocities, and left and right limbs. As means ±sd. Mean kinematic data was also compared between subjects for the left limb at self-paced speed.

Applied sagittal plane torques at the hip, knee, and ankle were analyzed throughout stance on the right and left side, for self-paced and slow walking speeds, and the mean ±sd, as well as the coefficient of variation (CV) were calculated. Total limb support synergy, or support moment, was determined by a summation of moments of the hip, knee, and ankle at each one percent of stance phase. Further analysis of support moment involved comparing the mean hip joint moment and knee joint moment, calculated across stance phase, for both walking speeds. A calculation of hip-knee covariance was utilized to further examine hip and knee synergy for slow and self-paced speeds (Appendix K).

Horizontal heel velocity was measured throughout the gait cycle in the forward direction of body progression (anterior movement) and the medial-lateral direction for self-paced and slow velocities. Mean data were calculated ±sd, in meters per second, and CV was used to determine intersubject and intrasubject variability. Horizontal heel velocity was also investigated at initial contact. Anterior or forward, and medial-lateral directions were evaluated, mean ±sd, for self-paced and slow walking speeds. Stance phase horizontal braking and propulsion forces during self-paced velocity for left and right limbs were compared between control subjects and PN subjects. Force component data were calculated as %body weight per %gait cycle.
**Statistical Analysis**

Independent t-tests with an alpha level of .05 were used for inter-subject comparisons of the following linear and temporal measures of gait: velocity, stride length, step width, percentage of time spent in single limb support. Horizontal heel velocities in the anterior-posterior and medial-lateral directions were compared between subjects at initial contact with independent t-tests and alpha equal to .05. Peak power in the sagittal plane at the ankle, knee and hip during preswing were compared between subjects with independent t-tests and alpha equal to .05. A comparison was made during the entire stance phase, of maximum ankle dorsiflexion ROM and the sagittal plane support moments at the hip, knee and ankle. Hip-knee covariance were compared throughout the gait cycle. During stance phase, variability of mean frontal plane hip torques were examined between subjects. Sagittal plane torque profiles of the hip, knee, and ankle were contrasted during stance phase for left and right sides. The rate of braking force anterior-posterior and the rate of weight acceptance vertically were examined throughout the gait cycle at self-paced velocity and contrasted between subjects.

Data for right and left limb kinetics and kinematics was compared with an independent t-tests using a significance level of alpha equal to .01. This level of significance was used because a large number of t-tests were necessary to determine differences between right and left sides. As the number of t-tests increases the power to reject a null hypothesis decreases. To avoid this problem we chose a more stringent .01 level of significance hence, decreasing the risk of rejecting the null hypothesis. This allowed researchers to combine left and right limb kinetic and kinematic data for subject comparisons when appropriate.
CHAPTER 4
RESULTS

Subject Characteristics

Clinical examinations and gait evaluations were performed on 4 subjects, and demographic data are presented in Table 2. All subjects except subject 3 had a diagnosis of NIDDM for over 20 years. Subject 3 was recently diagnosed with diabetes, however this subject also demonstrated peripheral sensory neuropathy, a late occurring complication of the disease. It is likely subject 3 was diabetic for several years prior to diagnosis, which is a relatively common occurrence for this disease. All subjects were between 65 and 75 years of age and routinely ambulated in the community without an assistive device. All subjects were obese, with body mass indexes (BMI) greater than 25 kg/m². In the geriatric population a BMI above 25 kg/m² has been associated with an increased risk of falls\(^7\). The non-neuropathic (control) subjects 1 and 2 demonstrated higher BMI values than PN subjects 3 and 4 (Table 2).

<table>
<thead>
<tr>
<th>Subject</th>
<th>PN</th>
<th>NIDDM (years)</th>
<th>Age (years)</th>
<th>Height (m)</th>
<th>Weight (kg)</th>
<th>BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>22</td>
<td>65</td>
<td>1.7</td>
<td>81.8</td>
<td>28.3</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>25</td>
<td>74</td>
<td>1.6</td>
<td>91.8</td>
<td>35.9</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>1</td>
<td>75</td>
<td>1.5</td>
<td>59.5</td>
<td>26.4</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>23</td>
<td>71</td>
<td>1.7</td>
<td>72.7</td>
<td>25.2</td>
</tr>
</tbody>
</table>

Table 3 outlines the health, functional, and neuropathic status of the subjects. The number of current comorbidities, aside from diabetes, varied from 3 to 6. Obesity was not considered to be a comorbidity in this study. Non-neuropathic subjects 1 and 2 had lower numbers of comorbidities than the neuropathic subjects. With 6 comorbidities
subject 4 may be at a greater risk for falling than the other subjects due to an increased number of concomitant disease processes. Subject 4 was also taking the most number of medications, 13, compared to 6 for subjects 1 and 3, and 5 for subject 2. Greater than or equal to 4 medications is considered to be a risk factor for falls in the elderly. All subjects would be included in this risk category. None of the subjects in this study had experienced any falls within the past year. Dynamic Gait Index scores were similar for all subjects, however both neuropathic subjects scored lower than controls (20 versus 22). In the DGI all subjects demonstrated mild deficits in stair ascending/descending, requiring the use of a handrail. Additionally, mild deficits in either horizontal or vertical head turns were noted for all subjects. Subjects with PN, 3 and 4, also demonstrated mild deficits in the ability to step over an obstacle (subjects 3 and 4), and around an obstacle (subject 4). The presence of peripheral neuropathy, as measured by a score of 7 or greater on the clinical examination portion of the MDNS, was determined for subjects 3 and 4, each scoring 9 points, a classification of mild PN. MNSI questionnaire results revealed mild PN impairment in subject 4. Subjects 1, 2, and 3 each scored a 4 on the MNSI, a result previously found to be insufficient to discriminate between diabetics with and without PN.

<table>
<thead>
<tr>
<th>Subject</th>
<th>PN</th>
<th>Comorbidities</th>
<th>Medications</th>
<th>Falls</th>
<th>DGI</th>
<th>MNSI</th>
<th>MDNS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>3</td>
<td>6</td>
<td>0</td>
<td>22</td>
<td>4</td>
<td>6</td>
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<td>-</td>
<td>3</td>
<td>5</td>
<td>0</td>
<td>22</td>
<td>4</td>
<td>2</td>
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<tr>
<td>3</td>
<td>+</td>
<td>4</td>
<td>6</td>
<td>0</td>
<td>20</td>
<td>4</td>
<td>9</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>6</td>
<td>13</td>
<td>0</td>
<td>20</td>
<td>7</td>
<td>9</td>
</tr>
</tbody>
</table>

Table 3. Number of current comorbidities in addition to NIDDM, number of current medications, number of falls within the past year. DGI, MNSI, MDNS test scores of function and PN status.
Temporal and Spatial Gait Parameters

Temporal-spatial parameters of gait for both self-paced and slow velocity walking are found in Table 4. Data for right and left limbs were compared for velocity, stride length, step width, and percent time spent in stance and in single limb support with independent t-tests using a significance level of alpha equal to 0.01. A more conservative significance level was used with these measures of gait to reduce the chances of statistically determining differences when using multiple t-tests if none actually exist. Percent time spent in single limb support phase at the self-paced speed for subject 4 was the only gait parameter found to be significantly different for the right and left limbs using this criteria. Single limb support in subject 4 was 37.8%±0.6% of the gait cycle on the right and 33.6%±0.7% on the left (p=.001). Data for right and left limbs combined is reported in Table 4. Data for step width and percent single limb support were not available for subject 1 due to experimenter error.

Average self-paced walking velocities ranged from 0.84m/s to 1.03m/s. Both control subjects exhibited slower self-paced velocities than the neuropathic subjects (0.96±.08m/s and 0.84±.05m/s for controls versus 1.03±.06m/s and 1.03±.04m/s for PN subjects, p=.003), Table 4. All subjects were able to adopt a slow walking velocity within a ±50% range of one third their self-paced speed. Velocity variability at the slow speeds was greater than that seen with self-paced walking. Coefficients of variation were 10% to 25% for slow velocities, while less than 10% at self-paced speeds. The greater variability seen at the slow speeds may reflect the subjects' conscious attempts to adjust their gait to a pre-trained and artificial speed.

Stride length was similar for control and PN subjects (p=.76). Control subjects 1 and 2 had stride lengths of 1.14m±.04m and 0.96m±.04m respectively, while stride lengths for PN subjects 3 and 4 were 0.99m±.03m and 1.14m±.07m respectively at self-paced speeds, Table 4. All subjects exhibited decreases in stride length of
approximately 30% and increased stride length variability at slow walking speeds. Step width was higher, although not significantly, in slow trials for subjects 2, 3, and 4, as compared to self-paced values (subject 1 data unavailable).

The percentage of time spent in stance phase of gait was higher in controls than PN subjects (66.9%±1.3% and 70.0%±2.1% versus 65.7%±1.5% and 64.5%±1.5%, p=.005). Table 4. Single limb support, as a percentage of the gait cycle, was correspondingly lower in control subject 2 than PN subjects 3 and 4. All subjects demonstrated increased time spent in stance phase with decreased single limb support at the slower walking speed.

<table>
<thead>
<tr>
<th>Subject</th>
<th>PN</th>
<th>Speed</th>
<th>Velocity (m/s)</th>
<th>Stride Length (m)</th>
<th>Step Width (m)</th>
<th>% Stance</th>
<th>% Single Limb Support</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>SP</td>
<td>0.96 (.08)</td>
<td>1.14 (.04)</td>
<td>-</td>
<td>66.9 (1.3)</td>
<td>-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Slow</td>
<td>0.33 (.03)</td>
<td>0.78 (.07)</td>
<td>-</td>
<td>77.9 (3.7)</td>
<td>-</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>SP</td>
<td>0.84 (.05)</td>
<td>0.96 (.04)</td>
<td>0.11 (.01)</td>
<td>70.0 (2.1)</td>
<td>29.3 (2.0)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Slow</td>
<td>0.31 (.05)</td>
<td>0.70 (.05)</td>
<td>0.13 (.08)</td>
<td>79.6 (2.1)</td>
<td>23.3 (3.6)</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>SP</td>
<td>1.03 (.06)</td>
<td>0.99 (.03)</td>
<td>0.06 (.01)</td>
<td>65.7 (1.5)</td>
<td>32.8 (2.1)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Slow</td>
<td>0.35 (.09)</td>
<td>0.61 (.06)</td>
<td>0.08 (.01)</td>
<td>79.0 (4.0)</td>
<td>20.7 (4.8)</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>SP</td>
<td>1.03 (.04)</td>
<td>1.14 (.07)</td>
<td>0.13 (.03)</td>
<td>64.5 (1.5)</td>
<td>35.7 (2.4)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Slow</td>
<td>0.45 (.04)</td>
<td>0.85 (.08)</td>
<td>0.16 (.04)</td>
<td>70.0 (2.6)</td>
<td>30.9 (2.5)</td>
</tr>
</tbody>
</table>
Ankle Function During Gait

Ankle function during gait was assessed kinematically by measuring total ankle range of motion (ROM) for the gait cycle and maximum dorsiflexion (DF) in stance. The peak plantarflexion torque generated in preswing and the associated peak power generation in preswing were measured as a kinetic assessment of ankle function. As seen in Table 5, data are presented for both self-paced and slow velocities. No significant differences were noted in total ankle ROM between right and left limbs in any subject using a 0.01 significance level for these multiple measures, and data for both limbs were combined in Table 5. Subject 2 demonstrated the greatest ankle ROM with values of 26°±3° in self-paced trials and 25°±2° during slow trials, Table 5. Subjects 1, 3, and 4 had similar total ankle ROM. No consistent differences were noted of the aforementioned variables between controls and PN subjects at self-paced and slow speeds.

As with total ankle ROM, subject 2 demonstrated the greatest dorsiflexion (DF) in stance during self-paced (23°±2°) and slow (20°±1°) walking trials (Table 5). Right and left limb maximum DF was not different for subjects 1, 3, and 4. Subject 3 had greater DF in the left limb at the self-paced speed (17° versus 13°, p<.01). As was the case with total ankle ROM, no pattern was identified in maximum DF for control subjects versus PN subjects. Clinical examination measurements of passive ankle DF using a goniometer showed increased DF in control subjects versus PN subjects. On the right/left sides, control subjects 1 and 2 had 15°/10° and 20°/20° passive DF, respectively. PN subjects 3 and 4 had 8°/10° and 9°/10° passive DF, respectively, for right/left limbs. Goniometric measurements of passive ankle DF were less than those observed actively during the gait analysis for all subjects.

Peak ankle torque values in Table 5 represent the maximum internal plantarflexion torque produced during preswing, the pushoff phase of stance. No
differences in peak torque were noted between right and left limbs for both control subjects (1 and 2) and for PN subject 4. Subject 3 demonstrated greater ankle plantar flexor torque in the left limb than the right (1.21±.06Nm/kg and 1.03±.04Nm/kg, p<.01). No consistent differences were observed between control and PN subjects. Subject 1 (control) and subject 4 (+PN) had the highest peak ankle torques of 1.49±.04Nm/kg and 1.40±.08Nm/kg, respectively, for self-paced walking trials. Control subject 2 and PN subject 3 had nearly identical peak torques during self-paced walking (1.15±.05Nm/kg and 1.12±.11Nm/kg for subjects 2 and 3 respectively). As expected for slower walking speeds, peak plantarflexion torques were decreased in all subjects compared to self-paced velocities. Overall, peak torques for subjects were an average 30% lower for walking velocities which averaged 63% slower. Comparison of slow speed peak torque between subjects shows a similar relationship to that observed in the self-paced data.

<table>
<thead>
<tr>
<th>Subject</th>
<th>PN</th>
<th>Total ROM</th>
<th>Maximum DF (degrees)</th>
<th>Peak Torque (Nm/kg)</th>
<th>Total ROM</th>
<th>Maximum DF (degrees)</th>
<th>Peak Torque (Nm/kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-</td>
<td>19 (2)</td>
<td>10 (1)</td>
<td>1.49 (.04)</td>
<td>20 (3)</td>
<td>13 (2)</td>
<td>1.10 (.15)</td>
</tr>
<tr>
<td>2</td>
<td>-</td>
<td>26 (3)</td>
<td>23 (2)</td>
<td>1.15 (.05)</td>
<td>25 (2)</td>
<td>20 (1)</td>
<td>0.86 (.16)</td>
</tr>
<tr>
<td>3</td>
<td>+</td>
<td>18 (2)</td>
<td>15 (2)</td>
<td>1.12 (.11)</td>
<td>16 (4)</td>
<td>16 (3)</td>
<td>0.71 (.12)</td>
</tr>
<tr>
<td>4</td>
<td>+</td>
<td>18 (1)</td>
<td>15 (1)</td>
<td>1.40 (.08)</td>
<td>16 (3)</td>
<td>16 (1)</td>
<td>1.01 (.26)</td>
</tr>
</tbody>
</table>

Table 5. Ankle range of motion (ROM), maximum dorsiflexion (DF) in stance, and peak plantar-flexion torque generated during preswing at self-paced and slow velocities. Data are mean (standard deviation) for right and left limbs combined. Maximum DF and peak torque at self-paced velocity for subject 3 were significantly different in right versus left limbs (p<.01, see text for data).
Ankle, Knee, and Hip Power in Preswing

Peak plantarflexion power generated during preswing at self-paced and slow walking velocities was also determined. As depicted in Figure 1, peak ankle power generation in preswing was similar for control and PN subjects during self-paced walking trials. Power generation at the ankle was significantly reduced in slow trials as compared to self-paced trials for all subjects. At the slow speed subject 4 demonstrated reduced peak ankle power in the left limb as compared to the right side (p<.01), although right sided data consisted of only two walking trials.

Peak power generation at the hip, and peak power absorption at the knee during preswing were also measured for self-paced and slow velocities. Winter and coworkers13 described a decreased ankle power generation with concomitant increased hip power generation and knee power absorption during pushoff in fit and healthy elderly as a compensatory stabilizing adaptation to propel the body forward in a more conservative, less destabilizing gait pattern with a resulting decrease in stride length and velocity. Since our subjects did not demonstrate significant differences in peak ankle power generation, we did not expect to find differences in peak power values at the hip or knee. During self-paced walking subject 3 (+PN) demonstrated increased knee power absorption and increased hip power generation as compared to control subjects 1 and 2, and PN subject 4. Subject 3 and subject 4 had similar ankle power values, and identical self-paced velocities (1.03m/s), but subject 3 had a shorter stride length than subject 4 (0.99m+0.03m versus 1.14m+0.07m). The increased hip power generation and knee power absorption at preswing in subject 3 could account for the shorter stride length noted for this subject. An increased cadence was observed in subject 3 compared to subject 4 to compensate for the reduced stride length (124±5 steps/min. versus 109±5 steps/min. for subject 4). Subject 3 was the only subject to demonstrate differences between right and left limb preswing power at the self-paced speed, with knee power absorption and hip
Figure 1. Peak power generation during preswing phase of stance at the hip, ankle, and knee power absorption at self-paced and slow walking speeds. Data are means ±sd for right limbs (solid bars) and left limbs (open bars).
power generation greater in the left limb (p<.01). Data for subject 3 at the slow velocity was unavailable for the right limb, so this same comparison cannot be made at both walking speeds. As observed for ankle power generation, values for hip and knee power were significantly reduced for all subjects at slow velocities compared to self-paced speeds (p<.05).

**Sagittal Plane Kinematics**

Sagittal plane kinematic profiles of the pelvis, hip, knee, and ankle motion for self-paced and slow walking trials for all subjects are found in Appendix L. Data are presented for the left and right limbs, and represents the mean values of walking trials plus and minus one standard deviation (sd). Toe off is depicted as a range of the mean toe off ±1sd. Kinematic profiles for self-paced walking are similar for the right and left limbs of each subject at the hip, knee, and ankle. Intertrial variability, as evidenced by the narrow standard deviation bandwidths, was low at all joints, and consistent from left to right within a subject. Standard deviation bands tended to be wider for ankle motion data, which may be a reflection of the relatively smaller range of motion at this joint.

Sagittal plane kinematic profiles at slow speeds demonstrated increased variability of hip, knee, and ankle motion (Appendix L). Standard deviations were particularly higher for ankle motion. Mean kinematic profiles from left and right sides were similar for subjects 1,2, and 4. Data for the right limb of subject 3 were unavailable due to experimenter error. Knee and ankle ranges of motion were similar between self-paced and slow walking trials in all subjects. An increased hip flexion during stance was observed during slow walking for all subjects.

Mean kinematic data for the pelvis, hip, knee, and ankle were plotted for intersubject comparison. Composite sagittal plane kinematic data for the left limb of all subjects walking at a self-paced speed is plotted in Figure 2. To simplify data
Figure 2. Sagittal plane pelvic, hip, knee, and ankle motion during self-paced walking. Data are mean joint angles from the left limb of all subjects.
presentation and discussion, only one limb was plotted for intersubject comparison. The left limb was chosen for comparison because no differences were noted in the kinematic profiles between left and right limbs, and slow speed data for subject 3 is only available for the left limb. Toe off lines were not included in the graphs since they varied from subject to subject. In general, toe off occurred between 65% and 70% of the gait cycle (Table 4).

Significant differences were noted in pelvic tilt between control subjects 1 and 2, and PN subjects 3 and 4, Figure 2. Control subjects demonstrated an anterior pelvic tilt of nearly 20° throughout the gait cycle. Smaller pelvic angles, less than 10°, were observed in PN subjects 3 and 4. In both groups, pelvic angles in the sagittal plane were fairly constant throughout the gait cycle. Consistent with an increased anterior pelvic tilt, control subjects 1 and 2 also demonstrated more hip flexion than PN subjects, in both stance and swing phases of gait. At initial contact control subjects were in nearly 50° hip flexion, as compared to around 30° for PN subjects. During stance phase only the PN subjects demonstrated any hip extension. Control subjects maintained at least 10° hip flexion throughout stance.

Kinematic profiles at the knee were similar for all subjects, especially during loading response (approximately the first 15% of the gait cycle), terminal stance, preswing, and through swing, Figure 2. Subject 2 demonstrated the greatest knee flexion during stance, a finding consistent with the greater hip flexion and ankle dorsiflexion observed for this subject during stance. Sagittal plane kinematics at the ankle showed more variability in controls versus PN subjects. Overall ankle range of motion was greater in subject 2 as compared to the other subjects (Table 5). No significant differences were observed between subjects 1, 3, or 4.
Sagittal Plane Kinetics

Applied joint torques were also analyzed in the sagittal plane for self-paced and slow walking speeds. Externally applied torques were plotted for left and right limbs throughout stance at the hip, knee, and ankle (Appendix M). Data for the right limb of subject 3 at slow speed were absent due to experimenter error. As with sagittal plane kinematic data, torque profiles were similar between left and right limbs for each subject during self-paced walking (Appendix M). Standard deviations were fairly consistent across stance phase in all subjects. Overall variability, demonstrated by CV, in joint torque was similar at the hip, knee, and ankle. However, in 5 out of 8 limbs studied (4 subjects, left and right), the lowest CV was found at the ankle. No consistent differences were noted in the variability between right and left limbs of an individual subject, or between control and PN subjects. Overall across stance CV's ranged from 8% to 27% for ankle moments, from 14% to 30% for knee moments, and from 12% to 37% for hip moments.

As might be expected, applied joint torques were reduced at the hip, knee and ankle for all subjects at slow walking velocities. In general, the pattern of flexion and extension applied torque observed at slow speeds was comparable to self-paced patterns, especially at the hip and ankle (Appendix M). Applied torques at the knee during loading response and preswing were preferentially reduced, particularly in subjects 1 and 2 in slow walking trials. Variability in sagittal plane torques was greater at slow speeds as compared to self-paced for all subjects, as shown by greater standard deviation bandwidths and overall CV's (Appendix M). This trend, seen at all joints, was especially evident at the ankle where slow speed CV's ranged from 18% to 50%. Slow speed CVs at the knee ranged from 26% to 74%, and at the hip ranged from 20% to 81%.
Applied torque graphs comparing hip, knee, and ankle moments for all 4 subjects are found in Figure 3. Data are presented for the left limb, under the self-paced walking condition, as was the case for the kinematic summary graphs (Figure 2). Ankle torque was similar for all subjects, with no discernible intergroup differences between control and PN subjects. Applied knee torques were more variable, with PN subject 3 tending to have higher knee flexion torques, particularly during loading response and preswing. None of the subjects demonstrated an extension moment at the knee during midstance or terminal stance, as might be expected during these phases of gait.

The pattern of applied hip moment across stance phase was quite different for control subjects 1 and 2 as compared to PN subjects 3 and 4. The two patterns of hip moment over stance phase for control subjects and PN subjects are clearly seen in Figure 3. All subjects demonstrated a rapid rise in applied flexion torque at the hip during the first 5% of stance, i.e. during heel contact and initial limb loading. The rate of increase in flexion torque generation was slightly lower in PN subjects, with peak flexion torque occurring slightly later. Peak flexion torques were also smaller in PN subjects, approximately 0.5Nm/kg as compared to approximately 0.7Nm/kg in control subjects. After peak flexion torque was achieved, hip moments in PN subjects rapidly reversed, becoming an applied extensor torque before the end of the first double support phase, which occurred at 25% of stance for subject 3 and 23% of stance for subject 4. Hip torque in both groups continued to become more extensor in nature through single limb support. However, control subjects demonstrated an applied flexion hip torque which decreased in magnitude throughout single limb support, while an increasing extensor torque was observed for PN subjects during this same period. At the end of the single limb support phase, which was 70%, 75%, and 78% of stance for subjects 2, 3, and 4, respectively, (subject 1 data unavailable), control subjects transitioned from an applied flexor torque to a neutral, or slightly extensor hip moment. PN subjects reached a
Figure 3. Applied joint moments during self-paced walking. Data are mean applied torques from the left limb of all subjects. Positive values represent applied flexion for support, hip, and knee moments and applied dorsiflexion at the ankle.
maximum hip extensor torque at this point and began to transition back to a neutral moment during preswing.

**Support Moment During Gait**

The support moment, or total limb support synergy during stance phase is also represented in Figure 3. The support moment is a summation of the hip, knee, and ankle moments observed at each 1% of stance phase. It is an applied flexor moment, being greater than zero. The functional interpretation of this measure is that the overall applied torques to the hip, knee, and ankle is acting to flex, or collapse the limb. This applied flexor moment must be countered with an overall internal extensor torque in the limbs to maintain an upright position. As seen in Figure 3, support moments across stance were lower for PN subjects compared with controls. This was particularly evident during limb loading and through terminal stance. During this period, the first 25% of stance, the rate of increase in support moment was lower in PN subjects, especially subject 4. Therefore, PN subjects 3 and 4 achieved lower peak support moments during limb loading, and at a slightly later percent of stance than did control subjects 1 and 2. The rate of decrease in support moment during the last 10% of stance, as weight was being transferred to the contralateral limb, was identical for all subjects.

Mean values across the entire stance phase were calculated for support, hip, knee, and ankle torques, Figure 4. Data for right and left limbs were combined in figure 5 since there was no difference between sides (p>.01). For subject 3, slow velocity trials, data was available only for the left limb due to experimenter error. Mean support moments for control subjects 1 and 2 (1.00±0.10Nm/kg and 1.12±0.09Nm/kg respectively) were significantly greater, nearly twice those observed for PN subjects 3 and 4 (0.55±0.07Nm/kg and 0.60±0.06Nm/kg respectively), p<.001. Mean support moments during slow walking were similar to self-paced walking for each subject, p>.05.
Figure 4. Mean joint moments during stance. Data are means ±sd, for self-paced (solid bars) and slow (open bars) walking speeds. Data from right and left limbs were combined since no significant differences were observed between sides. Positive values represent applied flexion for support, hip, and knee moments, and applied dorsiflexion at the ankle.
The difference in support moments was predominantly due to differences in hip moments between control and PN subjects. As seen in Figure 4, the mean applied hip torques over stance phase were flexor in nature for control subjects 1 and 2, but extensor for PN subjects 3 and 4, \( p < 0.01 \) for controls versus PN subjects). Mean hip moments during self-paced walking of control subjects 1 and 2 were not different from each other \( 0.18 \pm 0.09 \text{Nm/kg} \) and \( 0.30 \pm 0.08 \text{Nm/kg} \) respectively, \( p > 0.05 \), nor did the mean hip moments for the PN subjects differ \( -0.26 \pm 0.07 \text{Nm/kg} \) and \( -0.23 \pm 0.08 \text{Nm/kg}, \) respectively, \( p > 0.05 \). During slow walking, hip torques increased positively, that is, either increased in flexion for controls (subjects 1 and 2), or were decreased in extension for PN subjects (3 and 4). Mean hip moments for PN subjects during slow walking were still overall extensor moments and different than controls, \( p < 0.001 \). Mean hip moments were similar in self-paced and slow walking trials for subjects 1, 2, and 4, \( p > 0.05 \). Only subject 3 demonstrated a statistically increased (less negative) hip torque in slow versus self-paced walking, \( p < 0.01 \). Subject 3 exhibited less of an extensor hip torque than did PN subject 4 during slow walking, \( p < 0.02 \).

Mean applied knee torques were flexion in nature over the entire stance phase for all subjects at both walking speeds. No significant differences were noted between subjects at either a self-paced, or a slow velocity (Figure 4). Intrasubject comparisons demonstrated a decreased mean knee moment in slow versus self-paced walking trials for subjects 2 and 3, \( p < 0.05 \). Mean applied ankle moments were also flexion (dorsiflexion) in nature for all subjects at both walking speeds. No significant differences were noted in mean ankle moment between any subjects at either speed, or within any subject for self-paced versus slow conditions. At a self-paced velocity there appeared to be a trend towards an increased applied flexion torque at the knee with a decreased applied ankle dorsiflexion torque. A rank order of subjects, listed from lowest to highest mean knee moment was subject 1 with the lowest moment, followed by subject 2, then 4, and finally subject 3 with the highest knee torque. At the ankle this rank order was reversed. In
other words, the subject with the lowest knee flexion torque had the highest ankle
dorsiflexion torque (subject 1), and the subject with the highest knee flexion torque had
the lowest ankle dorsiflexion torque (subject 3). Therefore, although no statistically
significant differences in ankle or knee moments were noted between subjects for self-
paced walking, there appears to be a consistent tradeoff between moments at the knee
and ankle for all subjects, regardless of neuropathy status.

**Hip and Knee Synergy During Gait**

Another way to compare joint moments during stance phase is to plot the mean
moment of one joint against the mean moment of a second joint. Winter has used this
method to demonstrate the presence of coordinated synergies acting primarily on the hip
and knee, but also at the knee and ankle 30,58,80. Figure 5 includes plots of mean hip
moment versus mean knee moment for the 4 subjects. Data are plotted for individual
walking trials at self-paced and slow velocities to qualitatively assess intrasubject
variability. As seen in the graphs of mean hip moment versus mean knee moment,
subjects 1 and 2 (controls) are grouped tightly together. PN subjects 3 and 4 are also
grouped together, but are distinct from subjects 1 and 2. Control subjects demonstrated
an overall positive (applied flexion) hip moment and a positive (applied flexion) knee
moment. PN subjects 3 and 4 also demonstrated an overall flexion knee moment, but
with a negative (applied extension) hip moment. Winter's theory of a coordinated
movement synergy between the hip and knee proposes these two joints to be coupled in
opposing directions 30,58,80. That is, flexion moments at the knee will be combined with
extension moments at the hip, and extension moments at the knee will be combined with
flexion moments at the hip. Using Winter's schema, PN subjects 3 and 4 demonstrated a
predicted pattern, that of dominant knee flexion with hip extension.
Figure 5. Mean hip moment versus mean knee moment for individual right and left sided walking trials at self-paced and slow velocities.
The coordination between the hip and knee moment strategies was also evaluated by studying the variability of the moments of these joints, individually, and in combination. Table 6 lists coefficients of variation (CV), a measure of the overall variability of joint moments, for hip and knee moments, and the sum of these two (hip+knee) calculated from standard deviations at each 1% of stance. All subjects demonstrated lower variability of the hip and knee when these moments were combined, demonstrating the possibility of a coordination, or synergy between the hip and knee. The variability of the combined hip and knee moment was lower than either individual joint moments in almost all cases (Table 6). The exceptions were the self-paced left limb data for subjects 2 and 3, in which the hip+knee CV was as low as either joint, and the slow left sided trials for subject 4 where the hip+knee CV was higher than the CV for either joint individually.

<table>
<thead>
<tr>
<th>Subject</th>
<th>PN</th>
<th>Limb</th>
<th>Self-paced</th>
<th>Slow</th>
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<td>Hip Knee</td>
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Further analysis of a hip and knee synergy was performed by calculating a hip-knee covariance for self-paced and slow walking conditions (Table 7). With perfect coordination between the two joints acting in opposing directions, that is a one for one trade off of hip extensor torque for knee flexor torque (or visa versa), the hip-knee covariance would be 100% (Appendix K)\(^8\). As shown in Table 7, hip-knee covariances were much less than 100% for all subjects. Overall, there was no difference between control subjects and PN subjects in hip-knee covariances. Covariances were quite different between right and left limbs in all but a few cases. Subjects 1 and 3 demonstrated the greatest hip-knee covariances at both self-paced and slow walking speeds. A negative covariance was calculated for subject 2 for left limb self-paced walking, a finding which resulted mathematically from the relatively low variability of the hip moment in this case. Additionally, data for subject 3, left limb, slow velocity is absent due to experimenter error.

Table 7. Hip-knee covariances at self-paced and slow velocities. Data expressed as a percentage of the maximum possible covariance (100%) if hip and knee torques were perfectly coordinated out of phase during stance.

<table>
<thead>
<tr>
<th>Subject</th>
<th>PN</th>
<th>Limb</th>
<th>Self-paced</th>
<th>Slow</th>
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Horizontal Heel Velocity

Movement of the foot through stance and swing phase was assessed by measuring the velocity of the heel target as the subject walked. Horizontal heel velocities were chosen for analysis because increases in velocity, or an increased variability of foot velocity, may be associated with the risk of slipping, or of unsafe foot placement at initial contact. Horizontal heel velocity was determined for the forward direction of body progression (anterior movement) and for the medial-lateral direction throughout the gait cycle for self-paced and slow walking. As seen in Appendix N, anterior heel velocity increased prior to toe off (with heel rise) and continued to increase through midswing. Peak anterior heel velocities during swing reached were 3m/s to 4 m/s then decreased rapidly during terminal swing to nearly zero at initial contact. Data were similar for all subjects walking at a self-paced speed for either the right or left limb. No consistent differences were noted in anterior heel velocity during self-paced walking between controls and PN subjects. Overall, coefficients of variation (CV), a measure of variability across the gait cycle, were consistently low (<20%) for all subjects at self-paced speeds.

During slow walking trials CV values were higher, ranging from 12% to 222% of mean velocity (Appendix N). Additionally, CV's were different for the right versus left limbs of subjects 1, 2, and 4. Data for the right limb of subject 3 at slow speeds were missing due to experimenter error. The pattern of anterior heel velocity observed throughout the gait cycle also showed more variability in the slow walking trials. Subject 4 reached maximum anterior heel velocity at toe off, whereas maximum velocity in subjects 1,2, and 3 wasn’t achieved until midswing. As with self-paced trials, no consistent intergroup differences were noted in either pattern or variability for anterior heel velocity at slow speeds.

Intrasubject and intersubject variability for medial-lateral heel velocity data was high, in terms of both the pattern of heel motion observed over the gait cycle, and the
intertrial CV's (Appendix N). Overall, maximum medial-lateral velocities reached during swing were \(<0.3\text{m/s}\) and decreased to nearly zero before initial contact. Patterns of medial-lateral heel velocity varied greatly between subjects during self-paced walking. For example, in subject 1 heel velocity increased laterally from terminal stance, reached a minimum at toe off, increased medially through midswing before decreasing to zero at initial contact. In contrast, subject 2 demonstrated lateral movement from terminal stance, a reversal to a medial velocity which peaked at toe off, another lateral motion in swing phase before decreasing to near zero at initial contact. Different patterns of medial-lateral heel velocity were also observed between the right and left limbs of an individual subject. This was particularly true for subjects 3 and 4. Coefficients of variation were significantly greater for medial-lateral heel velocity than anterior heel velocity at self-paced speeds, ranging from 65\% to 230\% of the mean velocity. Part of this increased variability is probably due to the relatively small values for velocity in this direction, which generally were one tenth those observed in the anterior direction.

Medial-lateral velocities at the slow speed were reduced for all subjects, but the general pattern of foot movement was similar to the pattern observed in self-paced trials. CVs were greater for slow trials, as compared to self-paced walking, ranging from 59\% to 376\% of the mean velocity. As was the case for anterior heel velocity data, no consistent differences between controls and PN subjects were observed for magnitude, direction, or variability of medial-lateral velocity measurements.

Horizontal heel velocity at the moment of initial contact in the forward (anterior) and medial-lateral directions was determined for self-paced and slow walking trials and is represented in Figure 6. Data were combined for right and left limbs of subjects since no significant differences were noted between sides (p > .01). At initial contact anterior heel velocities ranged from 0.10m/s to 0.20m/s for self-paced walking and from 0.03m/s to 0.05m/s at slow speeds. Intrasubject variability was high, with anterior heel velocity CVs ranging from 24\% to 43\% of velocity at self-paced speeds, and 18\% to 81\% of
Figure 6. Heel velocity at initial contact in anterior-posterior and medial lateral directions during self-paced (solid bars) and slow (open bars) walking. Data are means ±sd for right and left limbs combined. Subject 2, slow speed, right and left limb data was significantly different (p<.01, see text for data).
velocity at slow speeds. Comparison of control with PN subjects showed significantly increased anterior heel velocity for self-paced trials for PN subjects (p=.02), but no difference between the groups with slow walking. It is difficult to assess the significance of this finding given the high variability of the data and the small number of subjects tested.

The medial-lateral component of horizontal heel velocity was quite variable for all subjects at both speeds (Figure 6). No significant differences were noted between control and PN subjects for self-paced walking, however at slow speeds the heel velocity direction showed lateral motion in controls and medial motion in PN subjects, resulting in a significantly different velocity between the groups (p=.02). Again, the high degree of variability and the small number of subjects makes this significance of the data difficult to interpret.

The horizontal braking and propulsion forces associated with the stance phase of self-paced walking were also investigated (Figure 7). During approximately the first half of stance an anterior shear force, or braking force, is generated. The shear force transitions to a posteriorly directed propulsion force as the body moves into terminal stance and preswing. Inadequate control of this force could increase the risk of slipping, or a fall. As shown in Figure 7, all subjects demonstrated a maximum braking force at 10% to 15% of the gait cycle. The braking force peak magnitude was approximately 10% of body weight for subjects 2, 3, and 4, and 15% for subject 1. It was interesting to note that during the first 5% of stance phase, i.e. during heel contact and initial limb loading, PN subjects demonstrated a decreased rate of braking force generation. Braking rates during the first 5% of stance were (in %body weight/%gait cycle) 1.91 and 0.81 for control subjects 1 and 2, respectively, and 0.43 and 0.51 for PN subjects 3 and 4 respectively, p=.04. An interpretation of this finding might be that PN subjects have adopted a more cautious strategy of foot placement and initial limb loading.
Figure 7. Anterior-posterior shear forces in stance for self-paced walking. Braking (-) and propulsion (+) forces expressed as percent of body weight (%BW).
Horizontal shear forces in PN subjects transitioned from braking to propulsion forces slightly earlier in stance than did control subjects. PN subjects 3 and 4 transitioned to propulsive forces at 47.1% and 47.4% of stance, respectively, compared to 53.6% and 50.7% of stance for control subjects 1 and 2, p=.02. An earlier transition from braking to propulsive force, combined with a decreased rate of braking during the initial portion of stance suggest that PN subjects spent relatively less time in the (potentially destabilizing) braking phase of their gait cycle.

Summary

The gait of four elderly diabetic women, two with and two without peripheral neuropathy, was studied with a 3-dimensional kinematic and kinetic analysis. PN subjects demonstrated an increased self-paced walking velocity and a corresponding decrease in percent time spent in stance phase as compared to controls. No differences in ankle function during walking between PN and control subjects, as may have been expected from previous literature reports. PN and control subjects exhibited markedly distinct hip moment profiles during stance with PN subjects utilizing an extension biased strategy while control subjects exhibited flexion biased hip moments. Differences in hip moment throughout stance between the two groups resulted in significantly lower total limb support moments in PN subjects. The extension biased hip moment observed in PN subjects could produce a decrease in the momentum of HAT, and thereby reduce the dynamic balance demands of walking. Additionally, at initial contact, PN subjects demonstrated increased forward heel velocity and a concomitant decreased braking force, suggesting a slight time lag in weight transfer to the new stance limb.
This study compared the gait of 4 elderly diabetic women, two with and two without peripheral neuropathy. While some differences in the gait strategies used by these two groups have been identified here, the sample size of this study greatly limits the conclusions which can be drawn. The results of this study are probably best interpreted as interesting findings which merit further study in a larger cohort. Increasing the number and diversity of subjects may allow for the confirmation, or the rejection of, conclusions proposed here.

In contrast to previous reports, diabetics with peripheral neuropathy in this study did not demonstrate decreases in self-paced walking velocity, stride length, or percent time spent in the single limb support phase of stance (Table 4). In fact, in this small group of diabetic women, the subjects with peripheral neuropathy actually had greater self-paced walking speeds than the non-neuropathic control subjects. No differences between the groups were observed for stride length or percent single limb support measurements. The subject sample enlisted for this study was not the same cohort used by previous researchers, since all of our subjects were women, aged 65 to 75 years old. Studies conducted by both Mueller et al.\textsuperscript{55} and Courtemanche and coworkers\textsuperscript{2} employed mostly male subjects whose average ages were at least 10 years younger than our subjects. Additionally, neither of these groups used diabetic subjects as controls for comparison with the diabetic PN subjects. The differences in temporal-spatial gait parameters observed by these researchers may not have been due to the presence of peripheral neuropathy, per se, but rather the presence of diabetes, a chronic disease with multi-system involvement.
Ankle function during walking was not found to be different in PN subjects and controls (Table 5 and Figure 1). Mueller et. al. noted decreases in ankle dorsiflexion (passive goniometric measurement), total ankle range of motion observed during gait, and peak ankle torque and power during preswing in diabetic subjects with PN versus non-diabetics\textsuperscript{55,57}. None of these changes in ankle kinetics were observed in the present study. Since decreases in peak ankle power during preswing with a corresponding increase in peak hip power were not observed in PN subjects, these data do not support Mueller's hypothesis of the use of a hip strategy as opposed to an ankle strategy at pushoff of stance by diabetics with PN\textsuperscript{57}.

One of the control subjects (subject 2) did demonstrate greater ankle dorsiflexion and total range of motion than was observed for all other subjects. It is impossible to conclude whether this subject demonstrated excessive dorsiflexion, or if the other control subject (subject 1) had limited dorsiflexion, but it is likely that subject 2 used more dorsiflexion than expected in this population of elderly women. In a study of fit and healthy elderly, Winter reported maximum ankle dorsiflexion values of less than 10°, and total ankle ROM of 20° during the gait cycle\textsuperscript{13}. Additionally, Simoneau et. al. have reported decreases in ankle ROM in non-neuropathic diabetics and well as those with PN\textsuperscript{6}.

Control of foot trajectory during swing phase is a fundamental task of gait\textsuperscript{83}. To assure safe landing and weight transfer at the beginning of stance, foot trajectory must be tightly regulated. Improper placement of the foot, or an increased shear velocity at initial contact could increase the potential for falling. Increased velocity of the heel at initial contact has been suggested as a risk for slipping and possible falls in the elderly\textsuperscript{5,13}. At a self-paced velocity, PN subjects demonstrated a greater heel velocity at initial contact in the anterior direction, which may suggest a less well-regulated foot trajectory strategy during swing phase in these individuals. During self-paced walking,
the variability of the anterior heel velocity throughout the gait cycle, as measured by the overall CV, was similar for control and PN subjects.

Data for heel velocity in the medial-lateral direction across the gait cycle suggested that a wide range of strategies were employed in these subjects for the frontal plane control of foot trajectory. The variability within each subject was very high, with CV's over 200% for an individual limb during self-paced walking. Not surprisingly, CV's during slow walking trials were even larger. Slow walking trials required the subjects to try to match a practiced target speed, which may have encouraged them to think more about their foot movement and placement, thereby increasing variability during swing phase. Although the general pattern of frontal plane heel motion was similar for each subject at the two walking speeds, the directional patterns of heel velocity were highly variable from one subject to another (Appendix N). The pattern of medial-lateral heel velocity, and heel displacement could provide useful information on balance control in future experiments.

Mean support moments of PN subjects were much lower, approximately half that of control subjects (Figure 4). In a study of diabetics with PN compared to non-diabetics, Mueller and coworkers also saw a decreased support moment in diabetics with PN, which occurred over the entire stance phase, but especially during the last half of stance. Support moments for diabetics with PN did not exhibit the characteristic "M" shape, with two peaks in overall applied flexion moment apparent at limb loading and during terminal stance and preswing. Mueller's diabetic PN group lacked the second support moment peak, with support moments decreasing gradually from mid-stance through the end of stance. Comparison of sagittal plane joint moments at the hip, knee, and ankle in Mueller's study demonstrated significant differences only at the ankle where diabetic PN subjects had reduced torques throughout stance, but especially during pushoff. Mueller's findings are consistent with the decreases in ankle torque and power generation observed during pushoff, and consistent with the conclusion that a hip
strategy was being used to pull the limb into swing and move the body forward. As described above, ankle moments of PN and control diabetic women in the present study were indistinguishable during the entire stance phase (Figure 3).

In contrast to Mueller's work, PN subjects in the present study demonstrated lower support moments, compared to controls, during all stance phases except preswing. The initial rise in support moment during limb loading was decreased in magnitude and delayed in PN subjects, and support moments remained much lower than those of controls through mid-stance. Overall, PN subjects in the present study demonstrated a preferential decrease in support moment during the earlier phases of stance as compared to the diabetic controls. This difference in support limb strategies was due to differences in hip moments between the groups.

Sagittal plane kinematic and kinetic data in the present study suggest that PN and control subjects may use two different strategies for walking, and that the hip joint may be the sight of control for both. Data for self-paced walking were primarily considered for this analysis since it should more closely reflect the subjects' natural walking pattern. PN subjects demonstrated an extension hip moment bias across stance whereas control subjects had a flexion hip moment bias (Figure 3). In PN subjects the initial flexion torque produced during limb loading was quickly reversed to an extensor moment. In other words, the ground reaction force vector moved posterior to the hip joint center at a much earlier point in stance. As a result, an applied extension moment was produced by the time the contralateral limb reached toe off, the beginning of single limb support. Consistent with this data, PN subjects demonstrated less hip flexion during stance than controls, and a decreased pelvic tilt as well (maintaining <10% pelvic tilt throughout stance). There were no appreciable differences in either joint motion or torques at the knee or ankle in PN and control subjects. This is in contrast to Winter's work, which suggests that a tradeoff exists between hip and knee moments.
During stance phase, an overall result of these differences at the hip and pelvis would be a relatively more posterior position of the body, particularly of the head, arms, and trunk (HAT). An applied extension moment at the hip throughout most of stance, combined with less hip flexion and pelvic tilt could position HAT more posteriorly in relation to the lower limbs in PN subjects. Such a backward lean at the pelvis and hip could reduce the forward momentum of HAT, and might provide more stability during stance, especially during single limb support.

Momentum of HAT was not directly measured in this experiment, and may not have been different between the two groups. Since momentum is the product of mass and velocity the higher velocities of the PN subjects and the lower body weights could offset any decreases in momentum due to a backward lean during walking. In this experiment PN subjects had an average 13% faster self-paced walking speed and an average 24% smaller body mass. Assuming similar body proportions momentum of HAT would be predicted to be less in PN subjects, even without considering differences in gait patterns. Thus, it seems likely that PN subjects had a decreased momentum of HAT during walking.

Based on the inverted pendulum model of dynamic balance, the momentum of HAT is primarily controlled from the hip in the sagittal (and frontal) plane. HAT represents approximately two thirds of total body mass in normal individuals, and although not directly measured in this study, was probably a higher percentage in our subjects since all 4 subjects were obese (BMI's above 25kg/m², Table 2). Subjects demonstrated significant abdominal adipose tissue based on observation and palpation for pelvic anthropomorphic measurements and target placement. Abdominal adipose tissue would add mass predominantly to the anterior portion of HAT. The backward lean in PN subjects may help them to better control a larger, more anterior HAT.

Control diabetic subjects were also obese, with BMI's higher than those of PN subjects. These subjects, however, demonstrated an applied flexion torque bias at the
hip from initial contact into terminal stance (Figure 3). During stance these subjects maintained at least 10° of hip flexion, (Figure 2 and Appendix L). The increased hip flexion, which would move HAT more anteriorly in conjunction with an increased anterior pelvic tilt, was countered by increase in trunk extension (Figure 2 and Appendix L). The differences observed in gait patterns between PN and control subjects may reflect two alternate strategies for controlling HAT. PN subjects may have adopted a more conservative pattern, allowing them to decrease the forward momentum of HAT, especially if the size of HAT was increased by obesity.

It is unlikely that the difference in gait patterns was due entirely to greater obesity in control subjects. The BMI of control subject 1 was only 2 to 3 kg/m² greater than the BMI's of PN subjects, but was 7 kg/m² less than the BMI of control subject 2 (Table 2). If a change in BMI of 2 to 3 kg/m² was sufficient to produce these distinct walking patterns, one would expect to see some difference between the control subjects since they differed in BMI by more than twice this amount. The diverse gait strategies used by control and PN subjects during stance may instead be due to the altered somatosensory status in PN diabetics.

The backward lean demonstrated by PN subjects may represent a more conservative gait pattern which not only allows them to decrease the forward momentum of HAT, but also to keep more of the body mass over the support limb during terminal stance. This would have the effect of reducing the amount of momentum needing to be controlled at initial contact. In other words, the foot could be set down more cautiously at initial contact. In support of this theory, the forward shear force during initial contact and the first part of limb loading was decreased in PN subjects. While these subjects reached the same magnitude of peak braking force (as a percentage of body weight) and at similar stance times as controls, they demonstrated a lag in the generation of braking force during the first 5% of stance (Figure 7). In agreement with this data, heel velocity in the forward direction was increased at initial contact in PN subjects (Figure 6.
and Appendix N). The total time spent in braking was therefore reduced in PN subjects by delaying the onset of braking and by crossing over from braking to a propulsion shear force sooner.

Taken together, these data describe a gait strategy in PN subjects which allows them at initial contact to momentarily delay commitment of weight transfer to the forward limb. Following loading response, at the end of the first double support phase, the ground reaction force moved posterior to the hip joint center of the stance limb. Combined with a relatively posterior pelvic tilt, this allows the individual to move HAT more posteriorly, decreasing its forward momentum. A more posteriorly positioned HAT during terminal stance keeps more body weight over the trailing stance limb, and decreases the momentum associated with swing limb as it approaches the next initial contact.

Much additional experimental evidence is required to lend credence to this hypothesis. First of all, kinematic data for the trunk in the sagittal plane would demonstrate whether or not HAT was actually positioned more posteriorly in PN subjects. Secondly, measurements of the anterior-posterior acceleration of the head, previously proposed as an indicator of dynamic balance, would be beneficial to describe HAT motion relative to limb motion. Electromyographic measurements of muscle activity during walking should be included to attempt to correlate joint kinematics and kinetics during stance, particularly at the hip and knee. An assessment of muscle strength in the lower extremities would assist with the interpretation of electromyographic muscle activity data.

**Summary**

Although this study is very preliminary, the results suggest that diabetic women with and without peripheral neuropathy utilize different strategies during gait. The major
differences between the two distinct patterns identified here occur at the hip, representing altered patterns of joint action or motor control. This finding is in contrast to previously published reports which identified the ankle as the site of altered function in diabetics with PN. It can be argued from data in the present study that elderly women with diabetic PN have a more cautious gait pattern during stance, evident from initial contact through terminal stance. This altered walking strategy is one which allows the individual to maintain equivalent average temporal-spatial parameters of gait as elderly diabetic women without peripheral neuropathy yet enhance stability especially in single limb stance.

Diabetics with PN have decreased somatosensation in their lower legs which includes a loss of protective sensation on the plantar surface of the foot, and reduced joint proprioception in the foot and ankle. Decreased postural stability and an increased risk for falls have been reported for diabetic individuals with peripheral neuropathy. Falls are highly correlated with greater morbidity and mortality in elderly women. Alterations in gait have been identified in diabetics with PN. While PN subjects in this study did not exhibit the same alterations those previously identified, distinct gait differences were observed and included hip function during stance, horizontal heel velocity at initial contact and braking force during initial contact and loading response. These differences in gait reflect two distinct strategies to control the body during walking, specifically to control HAT, and essential task of dynamic balance. Mean support moment across stance provided the best single variable identified in this study to discriminate between the gait of elderly diabetic women with and without peripheral neuropathy.

Clinical Relevance

The use of an extension biased hip strategy when walking may put an elderly diabetic individual at a higher risk for slips or falls if the center of mass is positioned at
the posterior "edge" of the base of support throughout stance, but especially at initial contact. This strategy affords less control and places greater demands on the body to maintain balance if at initial contact the leading foot slips on ice, or lands on an uneven surface. Diabetic patients with peripheral neuropathy should be educated on the potential hazards of an extension biased hip strategy. Hip musculature should be targeted for strengthening in diabetic patients with peripheral neuropathy to allow them to maintain an extension biased hip strategy, since it is a functional compensatory mechanism developed over time. A gait strategy with a decreased momentum of HAT, such as that employed by diabetics with peripheral neuropathy, is a more energy demanding strategy. Therefore, it will be important to increase the cardiovascular fitness of patients with diabetic peripheral neuropathy to decrease the cardiovascular demands of walking. Lastly, physical therapists should evaluate all elderly diabetic patients for decreased sensation in the lower extremities. A number of clinical examination protocols exist for this specific purpose, and additional community resources include diabetes educators employed at most hospitals. Peripheral neuropathy is a common complication of diabetes in the elderly, affecting at least 50% of those over 65 years old. Peripheral neuropathy has potentially life-threatening consequences, namely falls and amputations, both of which are associated with increased morbidity and mortality.

**Limitations of the Study**

Study limitations included those involving the subject population, the walking tasks assessed, and motion analysis of dynamic balance. The sample size was small, two subjects per group, included only women, and represented a very small segment of the elderly population with NIDDM. A sample of convenience was used which limits the cohort geographically to the Kent County area. All subjects were volunteers, and as such
may not have accurately reflected the population. Extensive exclusion criteria limited participation to community active elders who were in relatively good health. None of the subjects had a recent history of falls. This study only included individuals with mild PN, excluding a significant population of diabetic elders with moderate to severe PN. Since the effects of diabetic complications increase with age, the cohort was comprised of young-old individuals, 65 to 75 years of age. Elderly diabetics with peripheral neuropathy who use an assistive device or have a history of plantar ulcers, two very common characteristics of this population, were excluded from this study.

Walking tasks used in the study were limited to self-paced and slow speeds on a firm, level surface with good lighting. Community dwelling elders encounter many more challenging tasks in their daily lives. Perturbations to walking such as sudden stops or turns, obstacles, uneven surfaces, and poor lighting may affect dynamic balance and increase the likelihood of a fall. Open environments, where attention is shifted away from the task of walking provide a greater challenge to elderly individuals. While important to assess gait deviations and balance under these circumstances, it was beyond the scope of this study.

Data collected in the gait lab may not represent a true picture of a subject's natural walking pattern. Subjects were asked to walk at an artificial slow speed, not directly chosen by them, which should be considered when analyzing slow versus self-paced velocities for differences in kinetics and kinematics. Subjects were walking in an unfamiliar setting, and were required to wear clothing that exposed their legs and some of their abdomen, which may have made them uncomfortable and changed their gait. Walking without shoes could influence the gait pattern. The gait test required bare feet and most diabetics are instructed to always wear shoes when walking. There are many other intrinsic factors besides peripheral neuropathy or sensory loss that affect walking function and dynamic balance in the elderly. Although researchers assessed and
documented some of these intrinsic factors, such as strength and range of motion, these factors were not controlled for and may have impacted group differences.

Due to excess abdominal soft tissue, pelvic targets had to be placed lateral to the anterior superior iliac spines of our subjects. This targeting protocol required an alternative processing method to locate hip joint center, which has not been validated. The data processing did not account for the distance between the target and the actual bony landmark due to excessive soft tissue, which may influence the accuracy of joint motion detection because processing calculations are based on bony landmarks. Electromyographic (EMG) study of muscle function was not utilized in this research design. For this reason, detailed descriptions of muscle synergies utilized by subjects is not available, therefore differences seen must be inferred from other data analysis and may not be as accurate.

Measures of dynamic balance have not been well defined. Conclusions regarding dynamic balance from this study were drawn from a collection of evidence suggested in the literature. Therefore, data generated cannot be compared to a validated standard, or normal performance. The data analysis for joint moments used by CHKS does not employ full inverse dynamics which would account for the inertia and acceleration characteristics of the lower extremity segments during walking. This error is greater for kinetic data at more proximal joints like the hip, since the inertia is proportional to the weight of the limb segment. The weight of the entire leg adds to the inertial component at the hip, while only the foot adds to the ankle inertial component. The research design did not determine the relative position of HAT and therefore the role of head acceleration could not be calculated.
Future Research

To confirm or refute results presented here, this preliminary study needs to be expanded to include more subjects, male subjects, and older subjects. Additionally, subjects with moderated to severe PN should be included. Since a significant portion of this population is obese, a study researching the potential correlation of obesity and changes in gait kinematics and kinetics would enhance data analysis of obese elderly diabetics with and without PN. Instead of using an artificial slow speed, it would be more representative of functional balance challenges if the subject was asked to walk at a fast speed, on varied surfaces, step over obstacles, carry something in their arms, or walk with conflicting or challenging lighting. Use of dynamic EMG would enhance this research design, allowing for analysis of muscle synergies applied during gait. Better control of internal variables could be achieved with more detailed assessment of lower extremity strength and ROM. Further research needs to be done regarding accuracy of target placement, and joint motion detection on subjects with excess soft tissue. An analysis of trunk kinematics, as well as head acceleration may provide information on motion and momentum of HAT, and its role in dynamic balance during walking. Based on the findings of this study future researchers should focus on hip kinetics during stance, and foot trajectory during swing and at initial contact to study walking strategies in elderly diabetics with and without peripheral neuropathy.
REFERENCES


82. The Pathokinesiology Service and the Physical Therapy Department of Rancho Los Amigos Medical Center. *Observational Gait Analysis.* Downey, CA, Los Amigos Research and Education Institute, Inc., 1996.

APPENDIX A

QUESTIONNAIRE FOR INITIAL SCREENING BY PHONE INTERVIEW.

Have you ever had surgery on your legs or feet? (including joint replacements, amputations or hip fractures) yes/no

Have you had any back surgery in the past year? yes/no

Have you had any surgery in the past month? yes/no

Do you have any sores or breaks in the skin on your feet? yes/no
* if "yes" please explain *

Have you ever had any ulcers on the bottom of your feet? yes/no

Have you ever had a condition that affected your ability to walk? yes/no
* if "yes", please elaborate *

Do you walk without help or use of a device like a cane or walker? yes/no

Do you use any braces on your feet or legs when you walk? yes/no
* if "yes", please explain*

Do you walk two blocks, or shop for your own groceries? yes/no

Do you do your own housework or yard work, such as making your bed, doing laundry, mowing the lawn, or gardening? yes/no

Do you have stairs in your home? If yes, do you use them routinely? yes/no

Do you monitor your own blood glucose levels? yes/no

Have you had a nerve conduction velocity test performed? yes/no
* if "yes", when, and what was the result? *

Have you ever had a stroke, head injury, or Parkinson's disease? yes/no
* if "yes", please specify *

Do you have any medical conditions that are not currently controlled by medication, such as high blood pressure, or chest pain? yes/no
* if "yes", please list *

Do you have any dizziness with daily activities? yes/no
### APPENDIX B

#### CLINICAL EXAMINATION

<table>
<thead>
<tr>
<th>NAME</th>
<th>DATE OF EVALUATION</th>
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<tbody>
<tr>
<td>PHONE</td>
<td>DATE OF GAIT TEST</td>
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<tr>
<td>HEIGHT</td>
<td>MDNS SCORE /46</td>
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<tr>
<td>WEIGHT</td>
<td>FALL HISTORY</td>
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<th># FALLS IN PAST YEAR</th>
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<tr>
<td>DURATION OF NIDDM</td>
<td># FALLS WITH INJURY</td>
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<tr>
<td>DURATION OF PERIPHERAL NEUROPATHY</td>
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<td>RESTING HEART RATE</td>
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<td></td>
<td>HR AFTER DYNAMIC GAIT TEST</td>
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<td></td>
<td>PERCEIVED EXERTION</td>
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<td>HR AFTER 3 min. REST</td>
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<td>PERCEIVED EXERTION</td>
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<th>PAST MEDICAL HISTORY</th>
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<td># CO-MORBID CONDITIONS</td>
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</tr>
<tr>
<td>MEDICATIONS</td>
<td>R</td>
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| # CURRENT MEDICATIONS    | LIGHT TOUCH |
| FOOT SKIN INSPECTION     | 10 GRAM FILAMENT |
|                          | -GREAT TOE DORSUM |

<table>
<thead>
<tr>
<th>VIBRATION at: L R SECS</th>
<th>POSITION SENSE at: L R</th>
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<tr>
<td>GREAT TOE</td>
<td>-GREAT TOE /10 /10</td>
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</tbody>
</table>

| LIGHT TOUCH L R        | CEREBELLAR TESTS L R   |
| 10 GRAM FILAMENT       | -HEEL TO SHIN /10 /10   |
| -GREAT TOE DORSUM /10 /10 |

| DEEP TENDON REFLEXES L R | CEREBELLAR TESTS L R   |
| ANKLE                    | -HEEL TO SHIN /10 /10   |
| PATELLAR                 | -FINGER TO NOSE /10 /10 |
| TRICEPS                  |
| BICEPS                   |
RAPID ALTERNATING MOVEMENTS - UPPER EXTREMITY  
- LOWER EXTREMITY

PRONATOR DRIFT + / -

MANUAL MUSCLE TESTS
- PINKY ABDUCTION
- ANKLE DORSIFLEXION
- GREAT TOE EXTENSION

RANGE OF MOTION  L  R
- ANKLE DORSIFLEXION
- KNEE EXTENSION
- HIP EXTENSION

LIST ANY OTHER ROM OR MMT NOT WFL
## Appendix C

### Michigan Diabetic Neuropathy Score

#### Sensory Impairment

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<td></td>
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<tr>
<td>Vibration at great toe</td>
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<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>10-g filament</td>
<td>0</td>
<td>1</td>
<td>2</td>
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</tr>
<tr>
<td>Pin prick on dorsum of great toe</td>
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<td>Not Painful</td>
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<td>10-g filament</td>
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<tr>
<td>Pin prick on dorsum of great toe</td>
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#### Muscle Strength Testing

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<td>Finger spread</td>
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<td>2</td>
<td>3</td>
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<tr>
<td>Great toe extension</td>
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<td>2</td>
<td>3</td>
</tr>
<tr>
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<td>2</td>
<td>3</td>
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<td>Severe</td>
<td>Absent</td>
</tr>
<tr>
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<td>3</td>
</tr>
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<td>Great toe extension</td>
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<td>3</td>
</tr>
<tr>
<td>Ankle dorsiflexion</td>
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#### Reflexes

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<td>Quadriceps femoris</td>
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<th>Present</th>
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<td></td>
<td>Biceps brachii</td>
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<td>1</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>Triceps brachii</td>
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</tr>
<tr>
<td></td>
<td>Quadriceps femoris</td>
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<tr>
<td></td>
<td>Achilles</td>
<td>0</td>
<td>1</td>
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</tr>
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</table>

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**APPENDIX D**

**MICHIGAN NEUROPATHY SCREENING INSTRUMENT**

A. History (To be completed by the person with diabetes)

Please take a few minutes to answer the following questions about the feeling in your legs and feet. Circle yes or no based on how you usually feel. Thank You.

<table>
<thead>
<tr>
<th>Question</th>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Are your legs and/or feet numb?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you ever have any burning pain in your legs and/or feet?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Are your feet too sensitive to touch?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you get muscle cramps in your legs and/or feet?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do you ever have any prickling feelings in your legs or feet?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Does it hurt when the bed covers touch your skin?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>When you get in the tub or shower, are you able to tell the hot water from the cold water?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Have you ever had an open sore on your foot?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Has your doctor ever told you that you have diabetic neuropathy</td>
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<td></td>
</tr>
<tr>
<td>Do you feel weak all over most of the time?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Are your symptoms worse at night?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Do your legs hurt when you walk?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Are you able to sense your feet when you walk?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Is the skin on your feet so dry that it cracks open?</td>
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<td></td>
</tr>
<tr>
<td>Have you ever had an amputation?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Total: 15

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APPENDIX E

HOW TO USE THE MICHIGAN DIABETIC NEUROPATHY PROGRAM

Administration of the Michigan Neuropathy Screening Instrument

**Questionnaire:** The questionnaire is self-administered by the patient. Add only "yes" responses to obtain the total score.

**Clinical Examination:** for all assessments, the foot should be warm (greater than or equal to 30°C).

**Foot Inspection:** The feet are inspected for evidence of excessively dry skin, callus formation, fissures, frank ulceration or deformities. Deformities include flat feet, hammer toes, overlapping toes, hallux valgus, joint subluxation, prominent metatarsal heads, medial convexity (Charcot foot) and amputation.

**Vibration Sensation:** Vibration sensation should be performed with the great toe unsupported. Vibration sensation will be tested bilaterally using a 128 Hz tuning fork placed over the dorsum of the great toe on the bony prominence of the DIP joint. Patients, whose eyes are closed, will be asked to indicate when they can no longer sense the vibration from the tuning fork.

In general, the examiner should be able to feel vibration from the hand held tuning fork for 5 seconds longer on his distal forefinger than a normal subject can at the great toe (e.g. examiner’s DIP joint of the first finger versus patient’s toe). If the examiner feels vibration for 10 or more seconds on his or her finger, then vibration is considered decreased. A trial should be given when the tuning fork is not vibrating to be certain that the patient is responding to vibration and not pressure or some other clue. Vibration is scored as 1) present if the examiner senses the vibration on his or her finger for less than 10 seconds, 2) reduced if sensed for greater than or equal to 10 seconds, or 3) absent (no vibration detection).

**Muscle Stretch Reflexes:** The ankle reflexes will be examined using an appropriate reflex hammer (e.g. Tromner or Queen square). The ankle reflexes should be elicited in the sitting position with the foot dependent and the patient relaxed. For the reflex, the foot should be passively positioned and the foot dorsiflexed slightly to obtain optimal stretch of the muscle. The Achilles tendon should be percussed directly. If the reflex is obtained, it is graded as present. If the reflex is absent, the patient is asked to perform
the Jendrassic maneuver (i.e. hooking the fingers together and pulling). Reflexes elicited with the Jendrassic maneuver alone are designated "present with reinforcement". If the reflex is absent, even in the face of the Jendrassic maneuver, the reflex is considered absent.

Administration of the Michigan Diabetic Neuropathy Score

**Vibration Sensation:** Please follow instructions as under MNSI.

**Pinprick:** Pricking pain sensation will be evaluated subjectively using a disposable safety pin. Patients will be asked to correctly distinguish, with their eyes closed, pricking pain from a simple touch on the dorsum of the great toe.

**10 Gram Filament:** For this examination, it is important that the patient's foot be supported (i.e. allow the sole of the foot to rest on a flat, warm surface). The filament should initially be prestressed (4-6 perpendicular applications to the dorsum of the examiner's first finger). The filament is then applied to the dorsum of the great toe midway between the nail fold and the DIP joint. Do not hold the toe directly. The filament is applied perpendicularly and briefly, (less than 1 second) with an even pressure. When the filament bends, the force of 10 grams has been applied. The patient, whose eyes are closed, is asked to respond yes if he/she feels the filament. 8 correct responses out of 10 applications is considered normal: one to seven correct responses indicates reduced sensation and no correct answers translates into absent sensation.

**Strength:** Strength will be assessed in the designated muscle groups using the Medical Research Council Score. Muscle strength is scored as 0 for normal, 1 for mild-moderate and 2 for severe weakness while complete loss of strength is scored as 3.

**Muscle Stretch Reflexes:** Please follow instructions as under MNSI. In addition to the ankle reflexes, biceps, triceps and quadriceps (knee) reflexes are also elicited. As above, if the reflex is obtained, it is graded as present. If the reflex is absent, the patient is asked to perform the Jendrassic maneuver (i.e. hooking the fingers together and pulling) or clenching the jaw. Reflexes elicited with the Jendrassic maneuver or jaw clenching alone are designated "present with reinforcement". If the reflex is absent, even in the face of the reinforcement maneuvers, the reflex is considered absent.

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APPENDIX F

DYNAMIC GAIT INDEX

Gait level surfaces
Instructions: Walk at your normal speed from here to the next mark (20 feet)
Grading: Mark the lowest category which applies.
(3) Normal: Walks 20 feet, no assistive devices, good speed, no evidence for imbalance, normal gait pattern.
(2) Mild impairment: Walks 20 feet, uses assistive devices, slower speed, mild gait deviations.
(1) Moderate impairment: Walks 20 feet, slow speed, abnormal gait pattern, evidence for imbalance.
(0) Severe impairment: Cannot walk 20 feet without assistance, severe gait deviations, or imbalance

Change in gait speed
Instructions: Begin walking at your normal pace (for 5 feet), when I tell “go”, walk as fast as you can (for 5 feet). When I tell you “slow”, walk as slowly as you can (for 5 feet).
Grading: Mark the lowest category that applies.
(3) Normal: Able to smoothly change walking speed without loss of balance or gait deviations. Shows a significant difference in walking speeds between normal, fast, and slow speeds.
(2) Mild impairment: Is able to change speed but demonstrates mild gait deviations, or no gait deviations but unable to achieve a significant change in velocity, or uses an assistive device.
(1) Moderate impairment: Makes only minor adjustments to walking speed, or accomplishes a change in speed with significant gait deviations, or changes speed but loses significant gait deviations, or changes speed but loses balance but is able to recover and continue walking.
(0) Severe impairment: Cannot change speeds, or loses balance and has to reach for wall or be caught.

Gait with horizontal head turns
Instructions: Begin walking at your normal pace. When I tell you to “look right,” keep walking straight, but turn your head to the right. Keep looking to the right until I tell you, “look left,” then keep walking straight and turn your head to the left. Keep your head to the left until I tell you, “look straight,” then keep walking straight, but return your head to the center.
Grading: Mark the lowest category which applies.
(3) Normal: Performs head turns smoothly with no change in gait.
(2) Mild impairment: Performs head turns smoothly with slight change in gait velocity, i.e., minor disruption to smooth gait path or uses walking aid.
(1) Moderate impairment: Performs head turns with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
(0) Severe impairment: Performs task with severe disruption of gait, i.e., staggers outside 15 inch path, loses balance, stops, reaches for wall.
Gait with vertical head turns
Instructions: Begin walking at your normal pace. When I tell you "look up," keep walking straight, but tip your head and look up. Keep looking up until I tell you, "look down." Then keep walking straight and turn your head down. Keep looking down until I tell you, "look straight," then keep walking straight, but return your head to the center.
Grading: Mark the lowest category that applies.
(3) Normal: Performs head turns with no change in gait.
(2) Mild impairment: Performs task with slight change in gait velocity i.e. minor disruption to smooth gait path or uses walking aid.
(1) Moderate impairment: Performs task with moderate change in gait velocity, slows down, staggers but recovers, can continue to walk.
(0) Severe impairment: Performs task with severe disruption of gait, i.e., staggers outside 15 inch path, loses balance, stops, reaches for wall.

Gait and pivot turn
Instructions: Begin walking at your normal pace. When I tell you, "turn and stop," turn as quickly as you can to face the opposite direction and stop.
Grading: Mark the lowest category that applies.
(3) Normal: Pivot turns safely within 3 seconds and stops quickly with no loss of balance.
(2) Mild impairment: Pivot turns safely in > 3 seconds and stops with no loss of balance.
(1) Moderate impairment: Turns slowly, requires verbal cueing, requires several small steps to catch balance following turn and stop.
(0) Severe impairment: Cannot turn safely, requires assistance to turn and stop.

Step over obstacle
Instructions: Begin walking at your normal speed. When you come to the shoe box, step over it, not around it, and keep walking.
Grading: Mark the lowest category that applies.
(3) Normal: Is able to step over box without changing gait speed; no evidence for imbalance.
(2) Mild impairment: Is able to step over box, but must slow down and adjust steps to clear box safely.
(1) Moderate impairment: Is able to step over box, but must stop, then step over. May require verbal cueing.
(0) Severe impairment: Cannot perform without assistance.

Step around obstacles
Instructions: Begin walking at your normal speed. When you come to the first cone (6 feet away), walk around the right side of it. When you come to the second cone (6 feet past first cone), walk around it to the left.
Grading: Mark the lowest category that applies.
(3) Normal: Is able to walk around cones safely without changing gait speed; no evidence of imbalance.
(2) Mild impairment: Is able to step around both cones, but must slow down and adjust steps to clear cones.
(1) Moderate impairment: Is able to clear cones but, must significantly slow speed to accomplish task, or requires verbal cueing.
(0) Severe impairment: Unable to clear cones, walks into one or both cones, or requires physical assistance.
Steps

Instructions: Walk up these stairs as you would at home (i.e., using the rail if necessary).
At the top turn around and walk down.
Grading: Mark the lowest category that applies.
(3) Normal: Alternating feet, no rail.
(2) Mild impairment: Alternating feet, must use rail.
(1) Moderate impairment: Two feet to a stair; must use rail.
(0) Severe impairment: Cannot do safely.
APPENDIX G

INFORMED CONSENT

Mary Free Bed Hospital and Rehabilitation Center/
Grand Valley State University
Center for Human Kinetic Studies

The Effect of Diabetic Peripheral Neuropathy on Gait in the Elderly

I understand that I am agreeing to participate in a research study designed to characterize parameters of walking, such as joint ranges of motion, forces exerted on the ground, and muscle activity during walking. I understand that the screening physical examination, done by the Physical Therapy student, and gait test will occur on two separate days. I will allow a Physical Therapy student to place adhesive markers on my skin. I understand that a Physical Therapy student will ask about my past medical history condition and perform a physical therapy evaluation on me. If my history and physical examination are not consistent with inclusion criteria for the study, I understand I may not be able to participate in this study.

I understand that during the test I will be wearing shorts and a shirt in order to expose the skin markers and sensors needed to collect data. I understand that I will be photographed and/or videotaped as part of the evaluation. The Center for Human Kinetic Studies (CHKS) will have custody of these data, but will only use the data for the purpose of analysis and/or reporting scientific results. I understand that my record will be kept confidential, as explained to and understood by me.

I understand that all of the procedures involved in this evaluation will take approximately four (4) hours, are non-invasive (nothing will penetrate my skin), and that the risks associated with normal walking, such as tripping or falling, are minimal. I understand that, in the unlikely event of minor injury, first aid will be provided, but further medical care will continue under the direction of my physician in accordance with my own particular financial arrangement.
The benefits of this test have been explained to me. They include assisting the CHKS in establishing data on elderly individuals and providing me with scientifically collected and interpreted data on my walking pattern and balance abilities.

I know that participation in this study is strictly on a volunteer basis and that I may withdraw my participation at any time. I understand that in no way would non-participation or withdrawal from this study affect treatment while at Mary Free Bed nor my educational status at GVSU. There will be no payment for my participation. I know that any questions I have, pertaining to this study, will be answered. I have been given the phone numbers of the principal investigators Roberta Fischer (457-7041) and Sheri Bjornseth (454-5508), and of Paul Huizenga at Grand Valley State University (895-2472) to contact regarding any questions I may have.

_________________________________________  _____________________________
Participant (or legal guardian) / Date             Investigator / Date

_________________________________________
Witness / Date

_________________________________________

I wish to receive a copy of the results of this study.

_________________________________________
Name of participant             Signature / Date

_________________________________________
Address
## APPENDIX H

### ANTHROPOMETRIC MEASUREMENT EXAMINATION

<table>
<thead>
<tr>
<th>Anthropometric Measurement</th>
<th>Value/ Units</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Body Mass</td>
<td>kg</td>
</tr>
<tr>
<td>Height</td>
<td>cm</td>
</tr>
<tr>
<td>Pelvis</td>
<td></td>
</tr>
<tr>
<td>- Width: ASIS to ASIS</td>
<td>cm</td>
</tr>
<tr>
<td>- Depth: ASIS to PSIS</td>
<td></td>
</tr>
<tr>
<td>right</td>
<td>cm</td>
</tr>
<tr>
<td>left</td>
<td>cm</td>
</tr>
<tr>
<td>- Height: Pubic tubercle to height of ASIS</td>
<td>cm</td>
</tr>
<tr>
<td>Leg Length: ASIS to medial malleolus</td>
<td></td>
</tr>
<tr>
<td>-right</td>
<td>cm</td>
</tr>
<tr>
<td>-left</td>
<td>cm</td>
</tr>
<tr>
<td>Foot</td>
<td></td>
</tr>
<tr>
<td>- Length: Calcaneus to end of longest toe</td>
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</tr>
<tr>
<td>-right</td>
<td>cm</td>
</tr>
<tr>
<td>-left</td>
<td>cm</td>
</tr>
<tr>
<td>- Breadth: First metatarsal head to fifth</td>
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</tr>
<tr>
<td>-right</td>
<td>cm</td>
</tr>
<tr>
<td>-left</td>
<td>cm</td>
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## Appendix I

### Gait Test Cardiovascular Data

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<tr>
<th></th>
<th>Blood Pressure</th>
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<th>RPE</th>
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<tr>
<td><strong>Resting values</strong></td>
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<td></td>
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</tr>
<tr>
<td><strong>After 5 walking trials:</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Normal walk side A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow walk side A</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal walk side B</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow walk side B</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
APPENDIX J

RELATIVE PERCEIVED EXERTION SCALE

Instructions to subjects: Please rate your level of exertion at this moment.

0  sitting or laying still, doing nothing at all
1  very light activity
2  light
3  moderate
4  somewhat hard
5  hard
6  becoming very hard
7  very hard
8
9
10 very, very hard
APPENDIX K
CALCULATION OF HIP-KNEE MOMENT COVARIANCE

1. Joint moments from each walking trial were added at every 1% of stance to determine the mean moment and variance for every 1% of stance. Data were analyzed for hip and knee moments, and a sum of the hip and knee moments.

\[ \sigma_h^2, \sigma_k^2, \sigma_{h+k}^2 \]

where \[ \sigma_h^2 = \sum_{i=1}^{100} \sigma_{hi}^2 \]

2. Mean variances across stance were determined.

3. Covariance between the hip and knee, \[ \sigma_{hk}^2 \], was determined across stance.

\[ \sigma_{hk}^2 = \sigma_h^2 + \sigma_k^2 - \sigma_{h+k}^2 \]

Percent covariance between the hip, and knee, COV, was determined across stance.

\[ \text{COV} = \frac{\sigma_{hk}^2}{\sigma_h^2 + \sigma_k^2} \times 100\% \]
APPENDIX L

SAGITTAL PLANE KINEMATIC DATA FOR SELF-PACED AND SLOW WALKING SPEEDS
Figure L-1. Pelvic Sagittal plane kinematic data for subject 1 at self-paced and slow walking speeds. Data are mean (heavy line) ±sd (light lines). Positive values are anterior pelvic tilt. Vertical lines represent average toe off minus 1sd and plus 1sd.
Figure L-1-a. Sagittal plane kinematic data for subject 1 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-1-b. Sagittal plane kinematic data for subject 1 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-2. Pelvic Sagittal plane kinematic data for subject 2 at self-paced and slow walking speeds. Data are mean (heavy line) ±sd (light lines). Positive values are anterior pelvic tilt. Vertical lines represent average toe off minus 1sd and plus 1sd.
Figure L-2-a. Sagittal plane kinematic data for subject 2 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-2-b. Sagittal plane kinematic data for subject 2 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-3. Pelvic sagittal plane kinematic data for subject 3 at self-paced and slow walking speeds. Data are mean (heavy line) ±sd (light lines). Positive values are anterior pelvic tilt. Vertical lines represent average toe off minus 1sd and plus 1sd.
Figure L-3-a. Sagittal plane kinematic data for subject 3 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-3-b. Sagittal plane kinematic data for subject 3 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-4. Pelvic Sagittal plane kinematic data for subject 4 at self-paced and slow walking speeds. Data are mean (heavy line) ±sd (light lines). Positive values are anterior pelvic tilt. Vertical lines represent average toe off minus 1sd and plus 1sd.
Figure L-4-a. Sagittal plane kinematic data for subject 4 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure L-4-b. Sagittal plane kinematic data for subject 4 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Positive values are hip flexion, knee flexion, and ankle dorsiflexion. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
APPENDIX M

SAGITTAL PLANE JOINT TORQUES FOR SELF-PACED AND SLOW WALKING SPEEDS.
Figure M-1-a. Sagittal plane joint torques for subject 1 at self-paced walking speed. Data are mean (heavy line) ± sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-1-b. Sagittal plane joint torques for subject 1 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-2-a. Sagittal plane joint torques for subject 2 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-2-b. Sagittal plane joint torques for subject 2 at slow walking speed. Data are mean (heavy line) ± sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-3-a. Sagittal plane joint torques for subject 3 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-3-b. Sagittal plane joint torques for subject 3 at slow walking speed. Data are mean (heavy line) ± sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-4-a. Sagittal plane joint torques for subject 4 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
Figure M-4-b. Sagittal plane joint torques for subject 4 at slow walking speed. Data are mean (heavy line) ± sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are applied torques of hip flexion, knee flexion, and ankle dorsiflexion.
APPENDIX N

ANTERIOR-POSTERIOR AND MEDIAL-LATERAL HEEL VELOCITY AT SELF-PACED AND SLOW WALKING SPEEDS.
Figure N-1-a. Anterior-Posterior and Medial-Lateral heel velocity for subject 1 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-1-b. Anterior-Posterior and Medial-Lateral heel velocity for subject 1 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-2-a. Anterior-Posterior and Medial-Lateral heel velocity for subject 2 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-2-b. Anterior-Posterior and Medial-Lateral heel velocity for subject 2 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-3-a. Anterior-Posterior and Medial-Lateral heel velocity for subject 3 at self-paced walking speed. Data are mean (heavy line) ± sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-3-b. Anterior-Posterior and Medial-Lateral heel velocity for subject 3 at slow walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-4-a. Anterior-Posterior and Medial-Lateral heel velocity for subject 4 at self-paced walking speed. Data are mean (heavy line) ±sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.
Figure N-4-b. Anterior-Posterior and Medial-Lateral heel velocity for subject 4 at slow walking speed. Data are mean (heavy line) ± sd (light lines). Overall coefficient of variation (CV) of stance phase are depicted for each curve. Positive values are anterior and medial directions. Vertical dashed lines represent average toe off minus 1sd and plus 1sd.