1993

First Ray Joint Mobility, Pressure and Ulceration of the First Metatarsal Head in Diabetic Patients.

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Birke, James Allen, Ph.D.

The Louisiana State University and Agricultural and Mechanical Col., 1993

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FIRST RAY JOINT MOBILITY, PRESSURE AND ULCERATION OF THE FIRST METATARSAL HEAD IN DIABETIC PATIENTS

A Dissertation

Submitted to the Graduate Faculty of the
Louisiana State University and
Agricultural and Mechanical College
in partial fulfillment of the
requirements for the degree of
Doctor of Philosophy

in

The Department of Kinesiology

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August 1993

ACKNOWLEDGMENTS

A sincere appreciation to my "Carville family" who have inspired me to study the neuropathic foot. I have learned much about the foot from both the staff and patients at Carville. The patients, however, have taught me the strongest lessons through their life experiences.

I would like to thank Dr. B. Don Franks, Dr. Elizabeth Hawkins, Dr. Eugene Kennedy, Dr. E. Barry Moser, Dr. Richard Magill and Dr. Ben Sidaway for their encouragement, continual support and critical review of this manuscript. My appreciation to Dr. Peter Cavanagh for the loan of the first ray mobility prototype. A special thanks to Jerry Simmons for his professional assistance with illustrations.

Finally, my loving gratitude to my wife, Sue, who has been my source of strength and motivation in life.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

This dissertation reviews the pathomechanics of plantar ulceration in patients with diabetes mellitus, and studies the relationship between joint mobility, pressure and the location of ulceration at the first metatarsal head.

Neuropathy, mechanical stress and vascular disease have been shown to be the primary causes of ulceration in patients with diabetes. Sensory neuropathy is considered the permissive cause of plantar ulceration. Plantar ulcerations do not occur without loss of sensation in the foot. Other factors including motor neuropathy, autonomic neuropathy, abnormal mechanical stress, foot deformity, joint limitation, and hyperkeratosis are considered important component causes in plantar ulceration. Autonomic neuropathy and vascular disease are trophic factors which cause tissues to be more susceptible to damage and ulceration, but are not direct causes. Motor neuropathy, foot deformity, joint limitation and hyperkeratosis are associated with high foot pressures. Individuals with high foot pressures and loss of protective sensation develop ulcerations from repeated injuries during walking.

Plantar ulcerations commonly occur at the first metatarsal head. This study was conducted to determine if first ray joint limitation was related to ulceration at the first metatarsal head. Measurements of first ray mobility, pressure, and other physical measurements were made on 19 diabetic patients with a history of ulceration at the first

metatarsal head, 20 diabetic patients with a history of ulceration at other locations of the forefoot, 19 matched diabetic, and 19 matched non-diabetic controls.

Analysis of variance showed patients with a history of first metatarsal head ulceration had significantly lower first ray mobility and significantly higher pressure at the first metatarsal head compared to the other groups. Regression analysis showed a strong, negative, linear relationship between limited dorsiflexion of the first ray, and peak pressure and the pressure-time integral.

Analysis of other physical measurements showed duration of diabetes was signicantly higher, and sensation, range of motion at the hip, ankle and foot was significantly lower in patients with a history of ulceration compared to controls.

The results demonstrate that the pathomechanical factors, limited joint mobility and high pressure, are significantly related to plantar ulceration and ulcer location in diabetes.

CHAPTER 1

THE PATHOMECHANICS OF PLANTAR ULCERATION IN DIABETES MELLITUS: A LITERATURE REVIEW

1.1. INTRODUCTION

Foot ulceration in patients with diabetes mellitus is a major public health concern in the United States. In 1990, the Center for Disease Control estimated there were 14 million people in the United States affected by diabetes of whom 25% are expected to develop foot problems.[28] Foot problems account for 20% of the annual diabetic related hospitalizations,[53] and over 50% of the 120,000 non-traumatic lower extremity amputations each year result from complications of diabetes.[63]

Foot ulcerations develop from a combination of causes rather than a single cause (Figure 1.1).[15] Neuropathy, mechanical stresses, and angiopathy (vascular disease) are considered the major causes of foot ulcers in diabetic patients, but a number of other factors have also been cited.[1,9,11,17,36,70] Neuropathy, as compared to angiopathy, has been shown to be a much more important factor in foot ulceration than previously believed.[12,15,35,52,56,75] There is strong cross-sectional and prospective data to show neuropathy and abnormal mechanical stresses are the primary cause of ulcerations on the bottom of the foot (plantar ulceration).[11,59,78] A purely vascular pathogenesis accounts for only 7 - 13 percent of diabetic ulcerations and these cases are typically

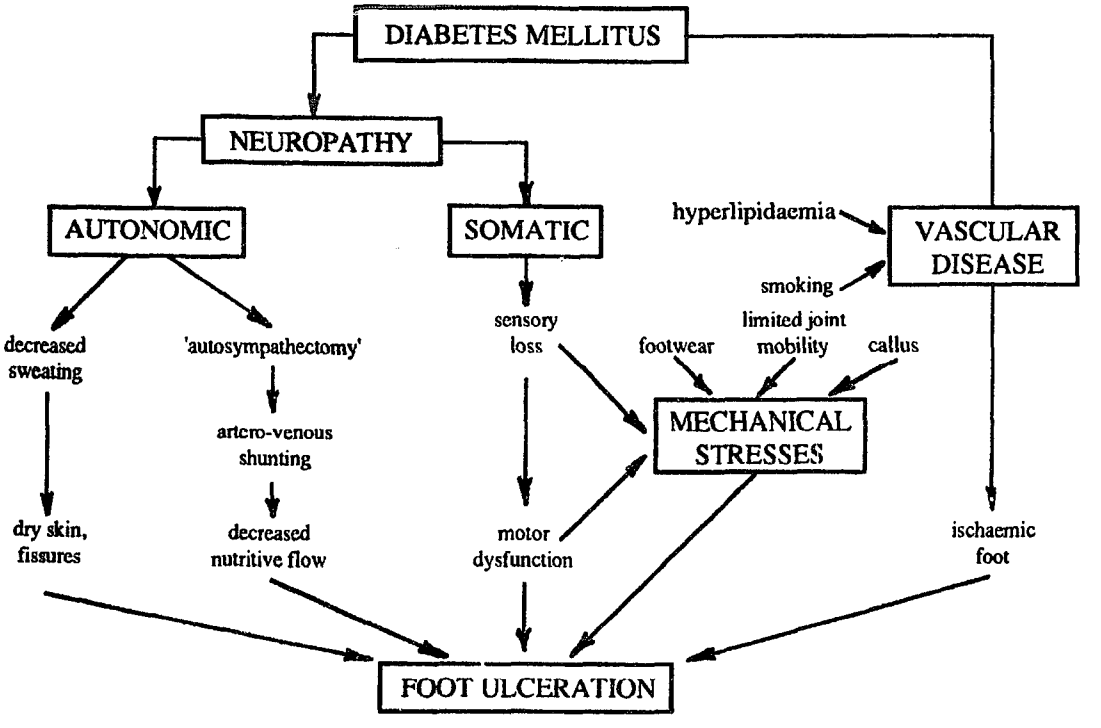

Figure 1.1. Causes of ulceration. Modified from A.J.M. Boulton, 1992.

non-plantar in location.[75] In a recent study neuropathy was also found to be a more common pathway than angiopathy in lower extremity amputation.[63]

1.2. NEUROPATHY: A PERMISSIVE ROLE IN PLANTAR ULCERATION

Neuropathy is considered a necessary factor in the sequela of plantar ulceration formation. This section reviews the permissive role of neuropathy in injury to the foot, and describes the characteristics of neuropathic ulceration.

Altered nerve metabolism, resulting from chronic hyperglycemia, is the likely cause of polyneuropathy in diabetes.[14] The prevalence of diabetic neuropathy has been shown to be higher in diabetic patients with poorly controlled glucose.[64] A distal, mixed sensory-motor-autonomic neuropathy is most common, involving both the large and small diameter fibers. There is a predominance of sensory over motor involvement.[43] Loss of pain and temperature sensation predisposes the area of involvement to repeated injuries from burns and mechanical stresses. Distal motor neuropathy results in weakness of the intrinsic muscles of the foot which leads to the development of claw toe and cavus foot deformities. Muscle atrophy decreases the soft tissue padding of the foot. Weakness of extrinsic peroneal innervated muscles contributes to equinovarus deformities. These deformities cause an abnormal weight-bearing distribution.[36]

The foot of a diabetic patient does not spontaneously ulcerate. Loss of sensation is an essential factor in plantar ulceration, and without insensitivity in the foot ulceration is uncommon.[11,15,63] Brand was the first to describe this important concept, in the development of plantar ulceration in leprosy and diabetes, based on observation data.[16] Boulton et al. found diabetic patients with plantar ulceration had significantly decreased vibratory sensation and increased plantar pressures compared to diabetic patients without ulceration, or normal controls.[11]

Several studies have measured vibratory, pressure and thermal sensory thresholds in the feet of patients with a history of plantar ulceration to determine the threshold level at which injuries occur. The sensory thresholds, in these studies, have been referred to as the level of loss of protective sensation.[6,10,47,60,71] While studies have not agreed on a precise sensory level for protective sensation, the concept appears to be a valuable clinical decision making tool for preventative care programs.[7,45]

The contribution of autonomic neuropathy on foot ulceration has not been well studied, but may be a factor in both ulceration and faulty healing of ulcers. Nielubowicz et al. in studies on dogs found paw ulceration occured only if both sympathetic and somatic nerves were transected.[62] They concluded that sympathectomy causes the opening of arteriovenious anastomoses in the extremity which results in peripheral ischemia, venostasis, and nutritional changes in the tissues. Manley and Darby found that denervated rat pads subjected to repeated stresses ulcerated

at a faster rate than their non-denervated controls.[58] This study would support the view that neuropathy produces trophic effects which contribute to tissue breakdown.

In humans, sympathetic denervation has been shown to result in dilation of the arteries and arterioles increasing blood flow to the foot.[3,34,35,37] This is associated with arteriovenous shunting which rushes blood from the arterial to venous side of circulation, but by-passing the capillary nutrient circulation. Long-term sympathetic denervation may cause structural changes in the artery and lead to medial wall calcification. Reduced capillary flow may increase tissue susceptibility to injury, slow tissue healing, and reduce tissue resistance to infection.[79] Additionally autonomic neuropathy reduces sweat gland function which may contribute to tissue breakdown by the drying and cracking of skin.[16,36]

Young et al. found the relationship between somatic neuropathy and autonomic neuropathy in diabetes was not uniform.[80] Diabetic patients with ulceration had a similar degree of autonomic neuropathy, but had higher somatic neuropathy compared to patients with no history of ulceration. They concluded that it was unlikely that autonomic neuropathy was of prime pathogenic importance in foot ulceration.

The dilation and shunting of vessels increases the blood supply to the bones of the foot. Bone scan studies with radiopharmaceutical agents show increased uptake proportional to increased blood flow and

osteoblastic activity in neuropathic patients compared to non-diabetic controls.[19,35] Accelerated osteoblastic activity results in demineralization and predisposes the bones to damage and deformity (Charcot osteoarthropathy) by minor trauma. Charcot deformity is a severe and relatively common deformity in diabetics that has been associated with midfoot ulceration.[6,61]

1.2.1. Characteristics of Neuropathic Ulcers

Neuropathic ulcers are described as painless, round, surrounded by callus, and located over prominent bony areas of the toes or plantar surface of the foot.[31] A single lesion is more common than multiple lesions. The most common site of ulceration in the diabetic patient is the first metatarsal head.[29] The foot is warm, dry, and pink. The patient is initally unaware of the lesion and may only notice it by the presence of blood or pus. Loss of sensation is an essential predisposing factor accompanied by mechanical, thermal or chemical injury.[16,31,36] In contrast, ulcerations due to poor circulation have been characterized as painful, irregular shaped, without callus, non-plantar, and multifocal.

In summary neuropathy is a primary component in the pathomechanics of plantar ulceration. Somatic neuropathy results in loss of protective sensation which permits injuries to the foot to take place. Atrophy and deformity in the foot, due to muscle weakness, alter the weightbearing distribution which contributes to foot injuries. Autonomic neuropathy causes drying and cracking of skin which may be a factor in tissue

breakdown. Loss of sympathetic vasomotor control promotes dilated arteries, arteriovenous shunting, and abnormal capillary flow which may increase the risk of tissue breakdown, infection, impaired healing, and Charcot deformity. Studies strongly support the conclusion that sensory loss is the primary or permissive cause of plantar ulceration.

1.3. MECHANICAL STRESS AND ULCERATION

This section reviews the relationship of mechanical stress and plantar ulceration, the mechanism of injury to the diabetic foot and methodology in measurement of pressure. Mechanical stress is the most common direct cause of injury in the neuropathic foot. Stresses usually occur at the interface of the foot with the ground or a shoe.

1.3.1. Mechanisms of Injury

Brand described four mechanisms of injury in the neuropathic foot: ischemia, direct trauma, repetitive stress and infection.[16] Ischemia occurs when blood flow to the tissues is blocked by pressures as low as 1 - 5 psi (7 - 35 KPa) over long periods of time. Ischemic injury is most commonly caused by wearing tight shoes. Koziak, in a study on 16 dogs, showed a pressure-time relationship between ischemia and ulceration.[54] Pressures as low as 20 KPa cause ulceration if continuously applied for a long enough duration (12 hours). Ulceration also occurred when higher pressures were applied for shorter periods of time. Low pressure intermittently applied did not cause ulceration.

Patients who change their shoes frequently may protect themselves from ischemic ulcers.

Direct trauma results from a single high pressure greater than 6,900 KPa and only occurs if a patient walks barefoot on a sharp object, or if a nail penetrates a shoe.[16] Patients who never walk barefoot are greatly protected from direct trauma ulceration. Brand observed the most common cause of injury to the neuopathic foot is repetitive stress. Moderate pressures (about 138 KPa) repeated thousands of times a day may cause ulceration.[16] In two separate studies on denervated rat foot pads, repetitive moderate pressure resulted in inflammation, autolysis, and finally ulceration over a 7-10 day period.[4,58] Brand theorized that the human foot is subjected to similar repetitive stresses during walking. A person with normal sensation may develop inflammation from repetitive walking stresses but pain causes him to remove the source of irritation, change the way he walks, or stop the activity. The person with loss of protective sensation, however, continues to walk in the same manner unaware of impending injury.

1.3.2. Abnormal Pressure and Plantar Ulceration

Brand suggested that relatively normal pressures on the foot could cause injury to the neuropathic foot.[16,17] Several studies, however, have shown that plantar ulcerations occur at the sites of highest pressure and that these loads are significantly higher in ulcerated compared to non-ulcerated feet. Stokes et al. measured loads under the feet of diabetic

and non-diabetics subjects using a force plate.[73] Maximal loading was increased in diabetic patients with ulcers compared to those without ulcers, and non-diabetic subjects. The position of maximal loading corresponded to the site of ulceration, and greater than normal loading was found at areas of callus formation. Diabetic patients with ulcers had decreased loading on the toes compared to normals. No differences were found in force due to age or gender, but there was an association between body weight and loading.

Ctercteko et al. studied forces on the feet of diabetic patients with ulceration, those with neuropathy but no ulceration, and normal subjects while walking on a load sensitive platform.[29] Their findings supported those of Stokes et al. Toe loading was found to be lower in diabetic patients compared to normals, and the site of maximum force was found under the site of ulceration. They found foot deformity was a common feature in patients with areas of increased loading. Ulcerated patients were found to be heavier than non-ulcerated subjects.

Rogers, using the Penn State University piezoelectric pressure platform, studied the relationship of body weight, height, foot width, foot length, first ray mobility, arch index (a measurement of arch height), percent body fat and age on foot pressure in 60 normal male subjects.[65] She found weight, arch index, and height were the significant predictors for regional pressure.

Cavanagh et al., using a pressure platform, also found the site of ulceration in diabetic patients corresponded to the location of highest pressure on the foot, and found reduced toe loading in diabetic patients.[23] They determined that structural deformities in the foot resulted in areas of abnormally high pressure, and recommended that pressure assessment be part of routine foot screening in the early stages of diabetes to identify the foot at risk.

High foot pressure may not only be a consequence of diabetes but may also be a pre-existing condition. Cavanagh found in studies on "normal feet" that some non-syptomatic, non-diabetic individuals have high foot pressure.[24] They determined the variation of pressure in the foot was so wide that the use of the mean + 2 standard deviations places patients at risk for ulceration in the range of "normality".

Significantly high foot pressures have been found in diabetic patients with neuropathy compared to diabetics without neuropathy and their non-diabetic controls.[13,77] Abnormal pressure was associated with a decreased toe loading ratio which was suggestive of neuropathy related foot deformity. Veves et al., in a prospective study of 80 patients, found pressure increased over a 2 year period compared to controls. The authors believe high pressure in the diabetic foot results from deformity associated with a progressive motor neuropathy.

In summary, there is convincing data that mechanical stress (abnormal pressure) is related to plantar ulceration. Studies have shown that body

weight, height, foot deformity, and neuropathy may be sources of high pressure in diabetics. Interpretation of these studies is confounded by the interrelationship of the measured variables with pressure.

1.3.3. Measurement of Foot Pressure

Pressure, or more correctly vertical stress, is the force per unit area. Shear stress or horozontal stress is the angular measurement of force per unit area.[74] Vertical stresses on the foot have higher magnitudes but shear stresses may be more important in the causation of tissue injury and ulceration. No satisfactory method is available to measure shear. There are three approaches to pressure measurement: 1) barefoot to ground, 2) shoe to ground, and 3) foot to shoe insole.[24,57] Barefoot to ground measurements are most valuable for understanding the function of the normal and abnormal foot, while shoe-ground and foot-insole measures are more valuable for studying the effect of footwear on the foot. In diabetes barefoot studies may be used to identify the foot at risk, and demonstrate structural foot changes over time. Inshoe measurements are subject to inaccuracies due to imprecise positioning of sensors under the anatomical areas of interest. The limitation due to sensor size, may result in measurement error over bony prominences. Sensors average pressure over their area of measurement and are very sensitive to the placement.[24]

There are basicly two types of pressure devices 1) discrete tranducers and 2) pressure plates or mats.[23,57] Discrete transducers measure

pressure from a limited number of locations where the sensors are placed, and therefore suffer greatest from inaccuracies due to positioning. Pressure plates and mats measure across the entire sole. The resolution of pressure plates/mats is dependent on the number of the sensors arrayed within the device. There are three major commercially available plate/mat systems: the Optical Pedobarograph (Biokinetics, Bethesda, MD), the EMED system (Novel USA, Minneapolis, MN), and the FSCAN device (Tekscan, Inc., Boston, MA). The Pedobarograph uses a mat with tiny dimples overlying a plate of glass illuminated at its edges by a light. The illumination of light is distorted proportionally by pressure during walking, and a video camera records the signal which is digitized and calibrated. A color printout is produced which displays peak pressure for a single walk. The EMED system consists of a platform with capacitance transducers (2 transducers per square cm). An inshoe mat is also available for the EMED System. The FSCAN is only available in an inshoe mat. It utilizes a thin disposable mat (0.004 inch thick) with an array of 950 capacitive sensors.

Pressure systems can record force, pressure, contact time, and contact area. It is not known whether a high magnitude of pressure or a lower pressure acting for long periods of time is more damaging to the foot.[23] Masking software may be used for analysis of regions of interest.

The pedobarograph has been shown to have a lower coefficient of variation than the other systems.[48] The EMED system was found to be highly reliable when at least three recordings were used. Pressure

measurements have been found to increase linearly with walking speed for most sites of the foot.[49,51,69] The reliability of the FSCAN measurments has not been reported.

A pressure threshold for ulceration was first reported by Boulton et al. using pedobarograph measurements in 41 diabetics with neuropathy, 41 without neuropathy and 41 non-diabetic controls.[11] There was a history of ulceration in 22 feet, all in the neuropathy group. Pressure was significantly higher in the ulcerated group, and all had peak pressures greater than 1070 KPa.

Cavanagh and Ulbrecht, using a 1000 element piezoelectric pressure mat, collected regional normative peak pressure values on 27 symptom free elderly males.[24] They determined 750 KPa was a preliminary pressure threshold for injury to the foot, based on a 95% confidence limits for the regions of highest pressure in the sample population. They noted that it is unkown if a single threshold for damage to the foot exists, or if regional norms may be necessary. They speculated that pressure values dangerous to one area of the foot might be easily tolerated by another area.

Veves et al., in a prospective study, found measurements of neuopathic deficit and foot pressure were highly predictive of ulceration.[78] They followed 86 diabetic patients and 28 non-diabetic controls over a 30 month period. Initial and follow-up examinations included a neuropathic deficit score which was a composite of ratings of reflex responses,

pain, touch and vibratory sensation, and foot pressure measurements using the optical pedobarograph. Neuropathic deficit was found in 58 patients. Abnormal pressures (greater than 1205 KPa, based on the mean peak pressure + 1 SD in normal subjects) were found in 43 patients. Fifteen patients developed plantar ulceration. Fourteen of the 15 patients had neuropathic deficit on initial examination and all 15 had abnormal foot pressures. All 15 had neuropathic deficit at follow-up. Diabetic patients had a significant increase in pressure, over time, compared to non-diabetic controls suggestive of progressive diabetes related foot deformity.

Cross-sectional and prospective studies show the relationship of pressure and plantar ulceration. Preliminary studies have published thresholds for abnormal pressure. These values do not consider differences due to foot region, walking speed, and measurement system. Pressure measurements are dependent on which measurement system is used, the speed at which subjects walk, and the region of the foot measured. Studies are needed to establish normal and abnormal thresholds using standardized methods for all commercially available pressure systems.

1.4. DEFORMITY AND PLANTAR ULCERATION

Structural deformities may contribute to high pressure in the foot. This section defines these deformities and briefly describes the pathomechanics related to high pressure and ulceration.

Several foot deformities have been associated with plantar ulceration in diabetes including: claw toes, pes cavus, hallux rigidus (limitus), plantar flexed first ray, equinus, rearfoot varus, forefoot varus, forefoot valgus, and Charcot deformity.[40,70] Claw toes are characterized by hyperextension of the metatarsophalangeal, and flexion of the proximal and distal interphalangeal joints. These deformities have also been associated with a pes cavus (high arch) and callus formation over the dorsum of the proximal interphalangeal joint, metatarsal heads (MTH), and tips of the toes. Hallux rigidus involves loss of metatarsophalangeal joint extension. Inadequate extension during gait results in stresses on the plantar surface of the great toe.

Biomechanical deformities (Figure 1.2) are associated with specific patterns of abnormal weight-bearing stresses on the plantar surface of the foot.[41,46,50,66,76] Rearfoot varus is considered the most common biomechanical deformity in the foot. It is a combination of calcaneal varus and tibial varus. Calcaneal varus is present when the bisection of the calcaneus is inverted relative to the lower one-third of the lower leg with the subtalar joint in the neutral position. Tibial varus is the degree of inversion of the lower one-third of the lower leg relative to the perpendicular weight-bearing line of the limb. Forefoot varus is present when the forefoot is inverted relative to the bisect of the calcaneus with the midtarsal joint fully pronated. Forefoot valgus is present when the forefoot is everted relative to the bisect of the calcaneus with the midtarsal joint fully pronated. There are two types of forefoot valgus: 1) all the metatarsal heads are everted. 2) the

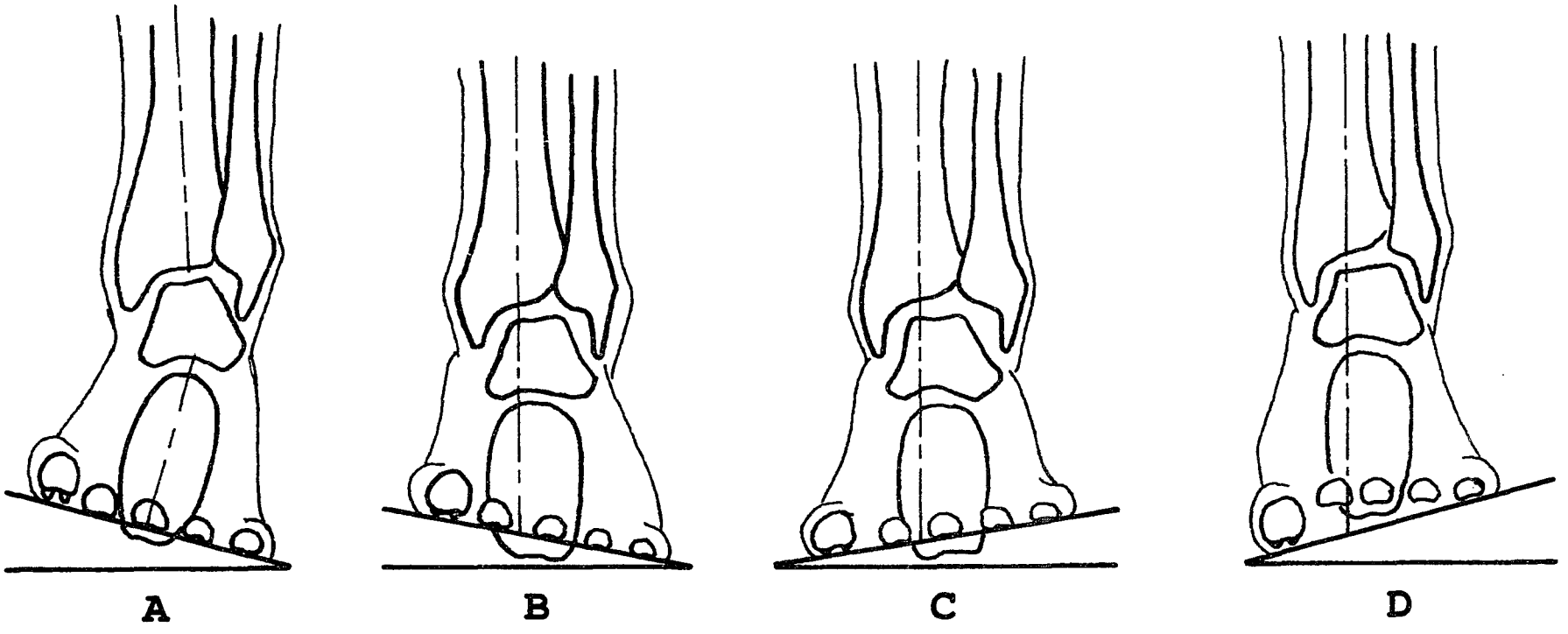

Figure 1.2. Biomechanical deformities in the foot. A, Rearfoot varus. B, Forefoot varus. C, Forefoot valgus. D, Plantarflexed first ray.

first MTH is plantar flexed while the second to fifth MTH's lie in the same plane. Equinus is a deformity resulting from limitation of ankle dorsiflexion. These deformities limit the ability of the foot to rest evenly on the ground. The foot may compensate for structural deformities by abnormal motion at the subtalar and midtarsal joints. If motion is not available the deformities are uncompensated.

Gibbs and Boxer described the relationship of biomechanical deformities of the feet and hyperkeratosis.[41] They observed that rearfoot and forefoot varus were causes of hyperkeratosis along the lateral and plantar aspects of the forefoot in the foot lacking compensatory pronation. In the varus foot with compensatory pronation and normal mobility of the first ray, hyperkeratosis forms on the middle three MTH's. In the varus foot with compensatory pronation and a rigid first ray, keratosis forms over the first and fifth MTH's. Hypermobility of the first ray (excessive dorsiflexion of the first ray during propulsion in gait) may also result in abnormal pressure on the medial aspect of the great toe called a "pinch callus". Equinus results in increased pressure on the first through fourth MTH's because tightness of the achilles tendon forces patients to walk on the balls of their feet. They noted that in patients with diabetes mellitus and loss of protective sensation these deformities may result in ulcer formation and eventually deep sinus tracts. Foot orthotics designed to balance the foot with structural deformities may reduce mechanical stresses and prevent ulceration.[7,45]

Mueller et al. in a retrospective analysis of 40 diabetic patients with forefoot and midfoot ulcerations showed a significant relationship between deformity and ulcer location.[61] Charcot deformity was found in 6 of 7 midfoot ulcers. Compensated forefoot varus was present in 9 of 11, second, third, and fourth MTH ulcers. Ulcers at the first and fifth MTH's, however, were found to have either compensated forefoot varus, uncompensated forefoot varus, or forefoot valgus. The authors recognized that other factors such as limited joint mobility, claw toes, prominent MTH's, or plantar flexed first ray deformities may also contribute to the localization of foot lesions.

The most severe deformities in diabetic patients are associated with Charcot deformities (osteoarthropathies).[19] Minor trauma may cause a Charcot foot in a diabetic with sensory neuropathy, demineralized bone secondary to increased blood flow, or osteoporosis resulting from disuse. Initally, Charcot feet present as swollen, warm and red, and are easily misdiagnosed as infection. Radiological changes soon occur with bone destruction and disruption of articular surfaces. Two well recognized deformities develop: a "rocker bottom", associated with midtarsal bone destruction and subluxation, and a marked pronated foot, resulting from medial dispacement of the talonavicular joint or lateroplantar calcaneocuboid dislocation. Both deformities predipose ulcer formation in the midfoot.

Cavanagh studied structural changes in the foot using radiography in diabetic patients with significant neuropathy and found measurements of

sesamoid height, first and fifth metatarsal angle, and first metatarsal declination angle were significantly related to peak pressure at the first MTH using regression analysis.[26]

Lang-Stevenson et al. found, using pedobarographic pressure studies of non-diabetic neuropathic patients, that high pressure over the area of healed ulcerations was reduced by surgical correction of deformities.[55] Control studies are lacking to validate the effectiveness of surgery in reducing pressure and preventing ulceration.

There is a strong association between deformity and ulceration. Deformities may be pre-existing or directly related to diabetes. They may be the cause of abnormal pressure which leads to ulceration in the neuropathic foot. The location of high stress and ulceration on the foot has been associated with specific deformities. Data to support this linkage are limited and more research is needed to comfirm present theories.

1.5. LIMITED JOINT MOBILITY AND PLANTAR ULCERATION

The finding of joint limitation in diabetic patients is well documented. Grigic et al. found joint stiffness in the hands of 65 of 229 children with insulin dependent diabetes.[44] They noted that short stature, and duration of diabetes were related to joint involvement. They believed that loss of connective tissue elasticity resulted from an increased cross-linking of collagen associated with diabetic metabolic

abnormalities. Rosenbloom et al. studied the hands of 204 patients with insulin dependent diabetes, 336 of their first-degree relatives, and 90 controls.[67] They found limited joint mobility in 21% of patients compared to 3% of the parents, 1% of the siblings, and 1% of the controls. They concluded that joint stiffness was caused by metabolic abnormalities. Buithieu et al. replicated these results in 211 insulin dependent diabetics and 239 controls using quantitative goniometric measurements of finger and wrist range of motion.[21] Campbell et al. found decreased motion in the feet and ankles, as well as, the hands, wrists and elbows of 254 young insulin dependent diabetics compared to 110 controls.[22] Starkman et al. found an association of limited joint mobility in the hand in both insulin dependent and non-insulin dependent diabetes.[72] They also showed limited joint mobility was related to duration of diabetes but only in the insulin dependent group.

There is evidence that both the function and structure of collagen in diabetics are changed as a result of hyperglycemia. Free glucose spontaneously attaches to proteins by a process known as non-enzymatic glycosylation.[18,20] Several investigations have shown that joint limitation may result from increased non-enzymatic glycosylation which leads to the molecular cross-linking of collagen protein and causes thickening and stiffness of periarticular tissues.[32,68] Hyperglycemia in young diabetics may result in the laying down of large amounts of highly glycosylated collagen during the puberty growth spurt.[22]

The relationship of joint limitation and plantar ulceration was established in a study by Delbridge et al.[33] Significant joint limitation at the subtalar joint was found in diabetics with a history of ulceration compared to diabetics without ulceration, and normal controls. There was a significant correlation between joint mobility at the subtalar joint and the first metatarsophalangeal joint in the foot, and the fifth metacarpophalangeal joint of the hand.

Mueller et al. found significantly reduced sensation, ankle dorsiflexion, and subtalar joint motion in diabetic patients with ulceration compared to controls.[60] They demonstrated the linkage of neuropathy and joint limitation with plantar ulceration in patients with diabetes.

Birke et al. demonstrated the relationship of hallux limitus with great toe ulceration. They found significantly decreased great toe extension, using a torque range of motion system, in diabetic patients with a history of great toe ulcers compared to diabetic patients with a history of ulcers at other sites, and non-diabetic controls.[8]

Limited joint mobility has also been associated with high foot pressure. Fernando et al. found, in a study of 30 subjects, that diabetic patients with high foot pressure had significantly decreased joint motion in the hand and foot, compared to patients without high pressure, and controls.[38]

In a later study, Fernando et al. studied the relationship of subtalar and first metatarsophanageal joint mobility, and pedobarographic measurements of pressure in 64 diabetic patients and 15 non-diabetic controls.[39] They found significantly higher foot pressures in patients with limited joint mobility compared to patients, and controls without limited joint mobility. Sixty-five percent of patients with neuropathy and limited joint mobility had a history of ulceration. There was a strong negative correlation between plantar pressures and joint mobility.

Cavanagh et al. measured range of motion in the hand, great toe, and subtalar joint, and foot pressure, using the optical Pedobarograph, in 20 insulin dependent and 30 non-insulin dependent patients.[25] Regression analysis showed decreased subtalar joint mobility and decreased vibratory sensation were related to increased pressure. They believe that limited joint mobility predisposes a patient to high foot pressure, but normal vibratory sensation is protective in avoiding injury to the foot during walking.

These studies demonstrate that joint limitation is an important factor in increased pressure and plantar ulceration. It has been shown that joint limitation is directly related to diabetes. It is not known if joint limitation in the foot is related to hyperglycemia, disuse, motor neuropathy, or other factors. Joint limitation may result in increased foot pressure due to loss of shock absorption, and may focus pressure locally because of motion requirements necessary in gait.

1.6. OTHER FACTORS AND PLANTAR ULCERATION

While deformity and joint limitation have been strongly associated with pressure and ulceration in the diabetic foot, other factors may contribute to plantar ulcer formation. These factors include: obesity, tissue atrophy, and hyperkeratosis.

1.6.1. Obesity

It is reasonable to suspect that individuals with increased weight would have increased pressure on their feet. This is an important issue in diabetes because obesity is a complicating feature of the disease. Boulton found diabetics who developed ulceration had significantly longer duration of neuropathy and were significantly heavier.[11] Studies have shown an association between weight and loading on the foot.[29,65,73] Several studies have not found a significant relationship between body weight and pressure.[12,24,65,77] Early studies[29,73], using force platforms, may have shown a stronger relationship between weight and loading, because force was not measured per unit of area. Weight gain may result in proportional increases in foot mass and tissue padding resulting in a normal pressure distribution. Further study is needed to clarify the relationship between obesity, pressure, and plantar ulceration in diabetes.

1.6.2. Tissue Atrophy

The thickness of the sole pad may also contribute to ulcer formation in diabetic patients. Gooding et al. found, by sonography, that the tissue under the heel and MTH's was reduced in diabetics compared to controls.[42] Thinner tissue padding may be due to atrophy of muscle or connective tissue, or anterior migration of the metatarsal head pads associated with claw toe deformities. Thinning of the tissue over bony areas may result in high pressure which might lead to ulceration.

1.6.3. Hyperkeratosis

Delbridge et al. observed increased non-enzymatic glycosylation of keratin protein in the stratum corneum of skin, in 30 diabetic patients, and proposed that these abnormalities may contribute to hyperkeratosis and plantar ulceration.[32] Repetitive stresses associated with ambulation are the primary cause of callus. Mechanical injuries may develop from neglected, thickened callus which cause local high pressure.[15,31,36] Young et al. measured foot pressures on callused feet of 17 diabetic patients, using the pedobarograph, and found removal of callus from 43 sites reduced pressure an average of 26%.[81] Callus, therefore, is both a cause and effect of mechanical stress.

1.7. SUMMARY AND CONCLUSIONS

Since the early work of Brand there has been a general consensus that sensory neuropathy and pressure are primary causes of plantar ulceration diabetes. Autonomic neuropathy may render the tissues more susceptible to injury, but data in this area are very limited. Brand's observation that repetitive walking stresses are the usual mechanism of injury in the neuropathic foot has been refined by technological advances in pressure measurement. Recent pressure studies have shown that diabetic patients with a history of ulceration have higher pressures, and have a shift in pressure from the toes to the metatarsal heads compared to non-ulcerated diabetic and non-diabetic controls. Plantar ulcers occur at the areas of highest pressure.

Foot deformity and limited joint mobility contribute to high pressure in the diabetic foot. Both pre-existing deformity and deformity related to motor neuropathy have been associated with ulceration. A general pattern of limited joint mobility is found in diabetes, particularly in young patients with insulin dependent patients. In these cases it is likely that joint stiffness is due to the non-enzymatic glycosylation of protein. In older diabetics, limited joint mobility may also be due to other factors such as disuse or motor neuropathy.

Investigators have shown some associations between deformity and location of ulcer. For example, hallux limitus is related to great toe ulceration, and Charcot deformity is strongly associated with midfoot

ulceration. A specific association between deformity, joint limitation, and location of MTH ulceration is an important unresolved issue because ulceration at these locations are so common. Understanding the pathomechanics of specific ulcer locations is central to treatment approaches.

Foot ulcerations are an important contributing factor to the high morbidity in diabetes mellitus. The economic impact of foot problems is substanial. Early identification of the patient at risk of ulceration has been shown to be beneficial in reducing severe foot complications. Foot programs emphasizing preventative care of the feet have significantly reduced the amputation rates at a number of institutions.[2,5,30] An understanding of the pathomechanics of plantar ulceration will assist clinicians and researchers in developing better methods of preventing and treating ulceration.

Studies of the pathomechanics of ulceration have produced valuable direction for the management of the diabetic foot, but a number of issues need further investigation. These issues include:

1. Clarification of the role of autonomic neuropathy in ulceration of the diabetic foot.
2. Identification of the role of obesity in ulceration.
3. Identification of the deformities or patterns of joint limitation responsible for abnormal pressure and ulceration at specific sites on the foot.
4. Determination of the role of biomechanically designed

treatment techniques in reducing foot pressure and preventing plantar ulceration.

5. Establishing instrument and location specific normative pressure data to identify the diabetic at risk of foot injuries.

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CHAPTER 2

FIRST RAY JOINT MOBILITY, PRESSURE, AND ULCERATION OF THE FIRST METATARSAL HEAD IN DIABETES MELLITUS

2.1. INTRODUCTION

Ulceration is a major cause of disability in patients with diabetes mellitus. It has been estimated that twenty-five percent of the fourteen million cases of diabetes mellitus in the United States will develop foot problems.[23] Two hundred million dollars a year is currently spent on hospitalization for diabetic foot ulcerations. Addtionally, 60,000 cases each year require lower extremity amputations at a cost of an additional 50 million dollars.[45,53] It has been shown that many diabetic foot ulcerations can be prevented with early intervention.[1,3]

Foot ulcers have been shown to be a multicausal problem.[10] The major factors which have been shown to contribute to ulcer formation include neuropathy, peripheral vascular disease, and abnormal mechanical stress (pressure).[31] Neuropathy and abnormal stresses are considered the primary causes of plantar ulceration in the diabetic foot.[6,8,12,13,27,62,66,69]

Studies on rats and other observational data have supported the belief that repetitive stress is the most common mechanism of injury to the neuropathic foot.[2,12,13,48,52] Brand theorized that in the neuropathic foot repetitive walking stresses cause injury, tissue inflammation, but not pain. In the absence of normal sensation patients continue to walk in

the same manner, allowing ulceration to develop. Individuals with normal sensation alter their pattern of gait or rest the foot in response to the pain associated with tissue inflammation.

Thompson found that abnormal pressure, which contributes to foot ulceration, can be divided into intrinsic and extrinsic stresses.[66] Intrinsic stresses result from deformities in the foot and are associated with neuropathic plantar ulceration. Extrinsic stresses result from external factors, such as, foreign bodies, poor fitting footwear or heat and are commonly associated with non-plantar ulcerations of a mixed neuropathic and dysvascular etiology.

Boulton proposed that sensory loss was the permissive cause of plantar ulceration, but alone was insufficient for wounds to develop. The additional factors needed for sufficient cause include high foot pressure, callus formation, limited joint mobility and, motor and autonomic neuropathy.[11]

2.1.1. Pressure and Ulceration

Boulton et al. found diabetic patients with a history of plantar ulceration had loss of vibratory sensation in the foot and abnormal patterns of weight-bearing stresses.[8] Several studies have found diabetic patients with a history of ulceration have higher peak pressures compared to diabetic patients without a history of ulceration, or non-diabetic controls, and the locations of ulceration correspond to

the areas of highest pressure.[18,24,65] These studies also show that pressure is lower under the toes and higher under the ball of the feet of ulcerated patients.

Boulton in subsequent research found the "toe loading ratio" was lower in diabetic patients with early evidence of neuropathy compared to controls.[9] This finding supports the view that structural changes in the foot resulting from disease complications contribute to ulceration.

In a recent prospective study of 86 diabetic patients, over a 30 month period, 14 of the 15 individuals who developed plantar ulceration had sensory neuropathy, and all 15 had abnormally high foot pressures on initial examination.[69] These data support the casual relationship of loss of sensation and high pressure in plantar ulceration.

2.1.2. Deformity and Limited Joint Mobility

Structural deformities, including claw toes, pes cavus, equinus, hallux rigidus, rearfoot varus, forefoot varus, forefoot valgus, plantar flexed first ray, have been accociated with plantar ulceration.[34,37,51,62] Claw toes and pes cavus are believe to result in prominence of the metatarsal heads and distal migration of the fat pad, predisposing the metatarsal heads to increased pressure and ulceration. Equinus may also contribute to ulceration on the metatarsal heads by resulting in a prolonged weight-bearing pattern on the forefoot, or compensatory pronation. A hallux rigidus deformity has been associatied with plantar ulceration of

the great toe, as a result of an increased weight-bearing load on the great toe during gait.

Mueller et al. showed a significant relationship between Charcot midfoot deformity, forefoot valgus, compensated forefoot varus, and uncompensated forefoot varus, and ulcer location in a retrospective analysis of 40 diabetic patients with midfoot and forefoot ulcerations.[51] Charcot deformity were present in all 6 midfoot ulcerations, and 9 of 11, second through fourth metatarsal head (MTH) ulcers were associated with a compensated forefoot varus. Ulcers at the first and fifth MTH's, however, were found to have either compensated forefoot varus, uncompensated forefoot varus or forefoot valgus. A specific deformity was not linked to only first or fifth MTH ulceration. The authors noted that other factors, such as limited joint mobility, claw toes, prominent metatarsal heads, or a plantar flexion deformity of the first ray may have contributed to ulcer location.

Structural deformities measured from radiographs have also been associated with foot pressure. Cavanagh found a high declination angle of the first metatarsal and a high first and fifth primus varus angle were related to first MTH pressure.[21]

Gibbs and Boxer described the independent function of the first ray and its influence on the pattern of foot pressure.[37] They proposed that the association of a rigid first ray with a compensated forefoot varus resulted in increased pressure at the first and fifth MTH's whereas,

hypermobility of the first ray resulted in pressure at the middle MTH's. In the neuropathic foot localization of high pressure at these areas could result in ulceration.

Limited range of motion in the ankle and subtalar joints has been shown to be decreased in diabetic patients with a history of ulceration compared to those without a history of ulceration.[29,50] Diabetic patients have limited joint mobility in the upper and lower extremities[16,17,38,58,64] which may be the result of non-enzymatic glycosylation of protein associated with hyperglycemia.[14,15,28,59] It is believed that in diabetes there is an increased cross-linkage of collagen causing decreased elasticity and toughening of connective tissue around the joint similar to that which occurs with age.

It has been shown that joint hypomobility results in high pressure on the foot. Cavanagh in a study of 50 diabetic patients showed that limited subtalar motion and loss of vibratory sensation were predictive for high pressure in the foot.[20] Fernando et al. found subtalar and metatarsophalangeal joint motion was reduced in diabetic patients with high pressure compared to matched diabetic and non-diabetic controls.[32] Joint hypomobility may decrease the shock absorption function of the lower extremity and cause high pressure on the foot. Locally, weight-bearing stresses may be concentrated at the distal segment of a hypomobile joint, which sustains greater resistance to the ground reaction forces, during the forward progression of the body in gait.

The relationship between limited joint mobility and pressure appears to be independent of any relationship between limited joint mobility and neuropathy. In a study by Fernando et al. diabetics with limited joint mobility and neuropathy, or limited joint mobility without neuropathy had significantly higher pressures compared to patients with neuropathy and no limited joint mobility, diabetic controls, and non-diabetic controls.[33]

Limitation of first metatarsophalangeal joint extension has been shown to be related to the development of plantar ulceration of the great toe.[5,25] Birke et al. showed that diabetic patients with a history of great toe ulceration had significantly reduced metatarsophalangeal joint extention compared to diabetics with ulcerations at other locations, and non-diabetic controls. A relationship between limited joint mobility and other locations of ulceration has not been demonstrated.

2.1.3. First Metatarsal Head Ulceration

The first metatarsal head is one of the most common sites of ulceration in diabetic patients.[4,24] The localization of ulcers at the first MTH may be associated with a pattern of deformity or limited joint mobility as has been shown to be the case with the great toe, second MTH and midfoot. A strong case has been made that hypomobility of the first ray may be a critical factor in ulceration that occurs at the first MTH,[37] but investigations have not been made to validate this hypothesis. In a neuropathic foot with a compensated rear or forefoot varus, resulting in

abnormal pronation, the presence of a hypomobile first ray may increase pressure at the first MTH. It has been shown that the repetitive stresses of walking cause ulceration at areas of high pressure.

2.1.4. Purpose

This investigation studied the relationship of first ray joint limitation and first MTH pressure on first MTH ulceration in patients with diabetes mellitus. The purpose of this study was to determine if (1) limited first ray motion, and high first MTH pressure are related to first metatarsal head ulceration in diabetic patients, and (2) if limited first ray motion is related to high first MTH pressure.

2.2. METHODS

2.2.1. Subjects

Foot examinations were made on 19 diabetic patients with a history of ulceration at the first metatarsal head (U1MTH), 20 diabetic patients with a history plantar forefoot ulceration not located at the first metatarsal head (UOTHER), 19 diabetic patients with no history of foot ulceration (DMCONTROL), and 19 subjects with no history of diabetes or symptomatic foot pathology (NCONTROL). Patients in the ulcerated groups were selected from the active 1992 files of the Gillis W. Long Hansen's Disease Center, Carville, LA. A total of 19 patients with a history of first MTH ulceration met the study criteria. Twenty subjects were

consecutively drawn for the UOTHER group. Individuals with a history of ulceration on both feet were only considered once for selection. Subjects in the DMCONTROL group were volunteers referred from the diabetic program, Baton Rouge General Medical Center, Baton Rouge, LA, and local newspaper advertisement, who were matched with the U1MTH group by age and gender (Appendix C). Subjects in the NCONTROL group were volunteer staff and visitors of the GWLHDC matched by age and gender with the U1MTH group.

The U1MTH group included patients with documented insulin-dependent or non-insulin-dependent diabetes mellitus who had been treated over the past 2 years for a first MTH ulceration. The U1MTH group included subjects who had a history of plantar ulcer at another site of the forefoot. The UOTHER group included patients with documented insulin dependent or non-insulin depentend diabetes who had been treated over the past two years for a plantar forefoot ulceration, but had no history of a first metatarsal head ulceration. Subjects were selected for participation in the study only after ulcers were completely healed. Foot surgery other than soft tissue debridement excluded subjects from all groups. The DMCONTROL group included patients with documented insulin dependent or non-insulin dependent diabetes but no history of ulceration on the foot. The NCONTROL group included non-diabetic patients with no history of a foot related disorder. Testing of left and right feet were randomized in control groups. All subjects signed an informed consent prior to participating in the study (Appendix D).

2.2.2. Materials

Degrees of first ray dorsiflexion (RAYROM) and kg/cm of first ray force/displacement slope (RAYSLOPE) were measured using a modification of a measuring device described by Rogers and Cavanagh (Figure 2.1).[56] This instrument stabilized the second through the fifth metatarsal heads and measured the vertical displacement of the first ray when a controlled force was applied to the first metatarsal head. Vertical displacement is assumed to a proportional measurement of dorsiflexion or the saggital plane motion of the first ray. While the first ray moves about all three body planes, vertical displacement is the standard 2measurement.[57] A parallel placed force transducer, and linear variable differential transformer provided simultaneous voltage recordings of the force applied and the vertical displacement of the first MTH. A miniature oscilloscope (SC 501, Oscilloscope, Tectronix, Inc., Beaverton, OR) was used by the tester to visually monitor the force applied to the foot. The signals were balanced and calibrated through an amplifier (Type R/S Dynograph, Beckman Instruments, Inc.). Recordings were stored on a microcomputer (Professional 350, Digital Equipment Corp., Maynard, MA) after computerized analogue to digital conversion (Model ADMPC - A2, Digital Equipment Corp., Maynard, MA). Data were reduced and converted into units of kg of force and cm displacement using custom written software. Good reliability of the instrument has been reported.[56] The reliability of RAYROM for two trials of three repeated measurements on 73 subjects, using an intraclass correlation coefficient (3,1), was 0.93 (Appendix E).[22]

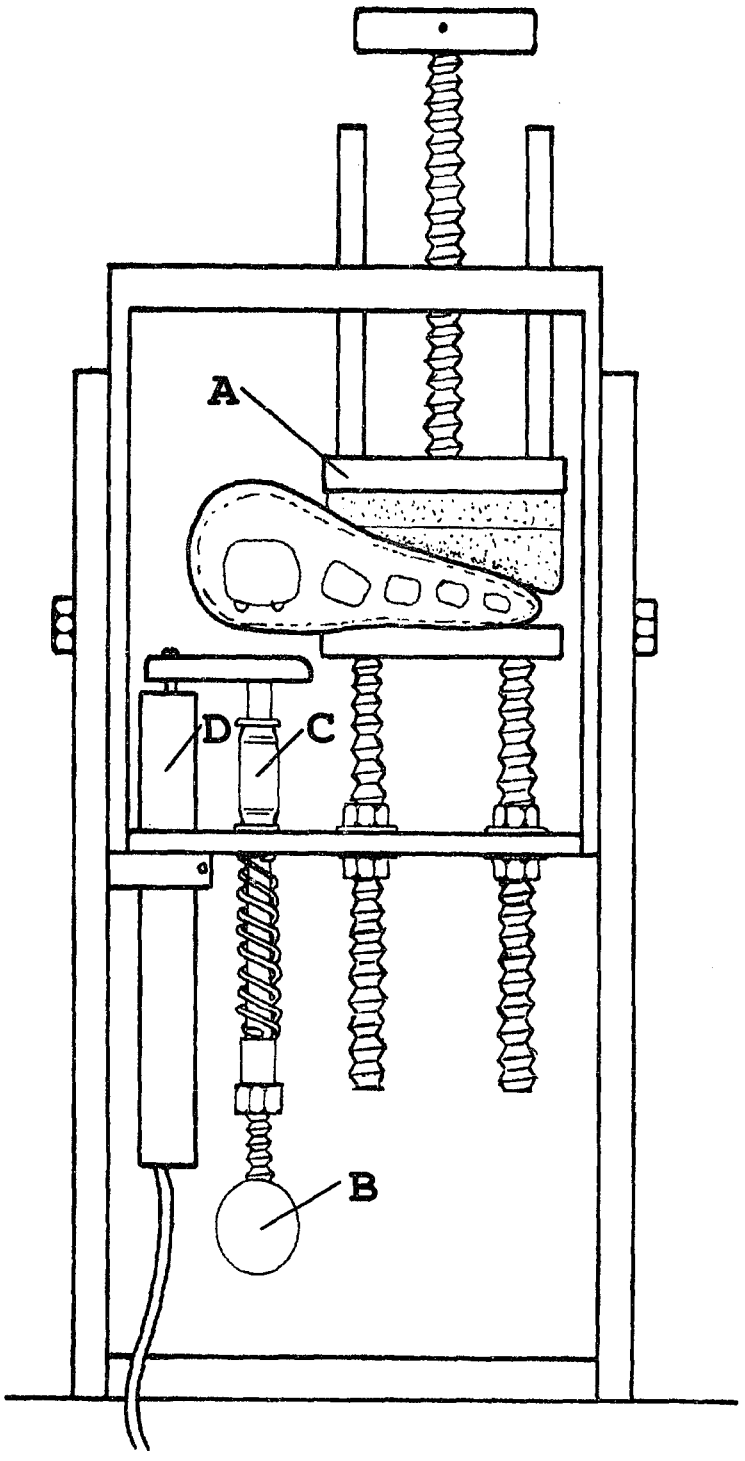

Figure 2.1. First ray mobility device. Modified from M. Rogers and P. Cavanagh. A, Clamp. B, Plunger. C, Force transducer. D, Linear variable differential transformer.

An EMED Pressure System (Novel USA, Minneapolis, MN) was used to measure N/cm^2 of peak pressure (PRESSURE) and Ns/cm^2 of pressure-time integral (PRESSURE-TIME) at the first MTH. The pressure platform has a sensor area of 445 mm X 225 mm containing 2016 capacitive transducers (2 transducers per cm^2) (Figure 2.2). Measurements were made during the second step of three barefoot walks. Patients were provided practice walks until a consistent walking pattern was obtained. The length of the walk was limited to minimize the risk of re-injury in patients with a history of ulceration. The speed of walking was controlled by an infrared photo-electric trigger (Model 49310, Radio Shack, Fort Worth, TX) and electronic counter (DC 503 Univeral Counter, Tectronix, Inc., Beaverton, OR). Data were stored on a microcomputer (Model 286-12D, Data Storage Marketing Inc.), and analysed using commercial software (Multimask EMED Software, Novel USA, Minneapolis, MN). The reliability of the EMED platform has been reported to be good when at least three 2walking trials are used for analysis, and speed of walking is controlled.[42,44,60] The reliability of measurements of peak pressure for three walking trials, on 73 subjects, using an intraclass correlation (3,1), was 0.73 (Appendix F).

2.2.3. Secondary Variables

Recordings were made of years of age, kg of weight (WT), cm of height (HT), gender (GENDER), sensation, ankle ischemic index (INDEX), years of duration of diabetes mellitus (DURATION), type of diabetes (TYPE), joint range of motion, joint position and foot radiographs of all subjects.

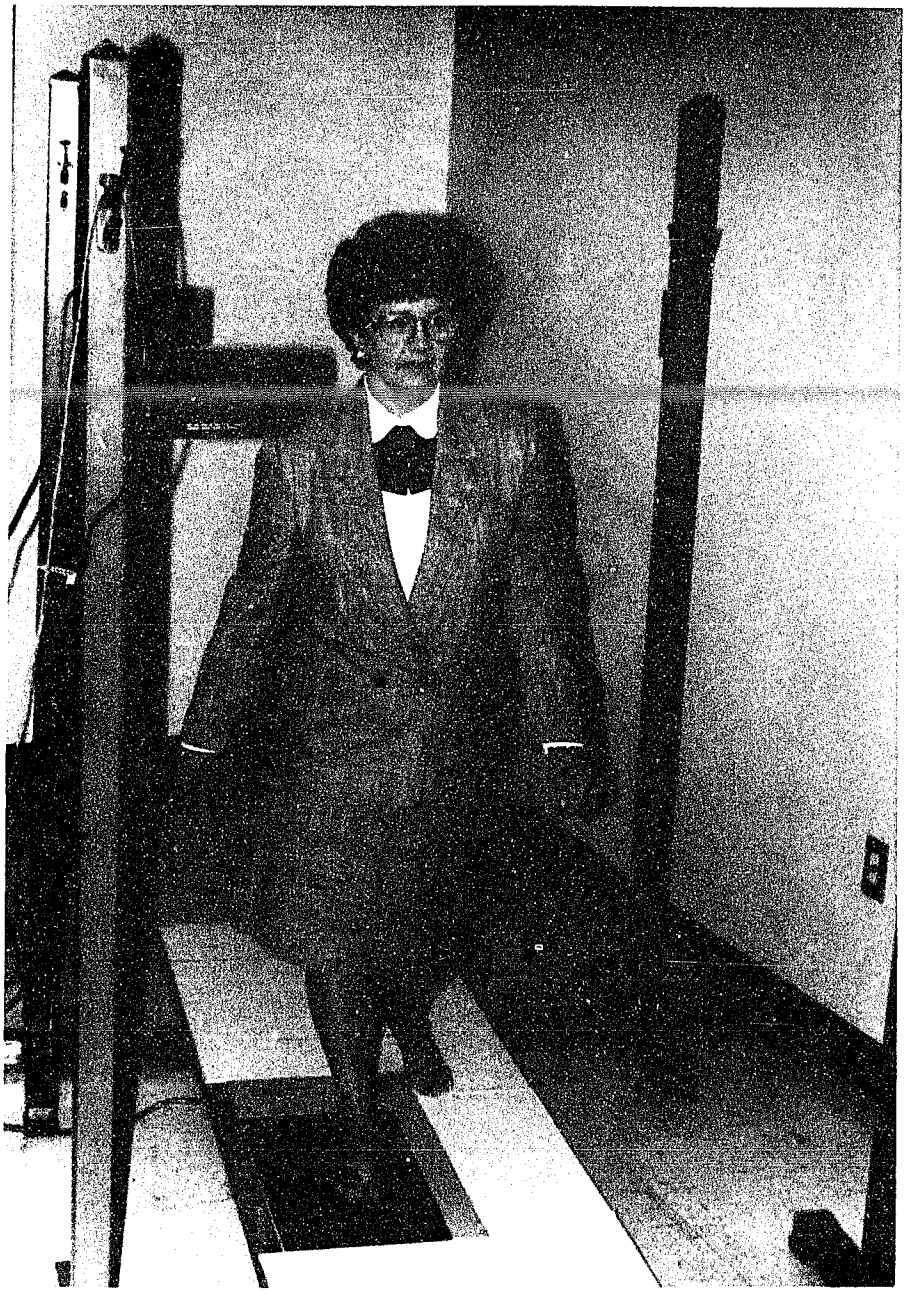

Figure 2.2. EMED system pressure platform.

Degrees of range of motion were measured for first metatarsophalangeal extension (TOEROM), ankle dorsiflexion (ANKLEROM), total subtalar joint motion (STROM) and total hip joint motion (HIPROM). Joint position was measured in degrees of varus for midtarsal joint neutral (MTNEUTRAL), rearfoot neutral (RFNEUTRAL), and subtalar joint neutral (STNEUTRAL). Joint position was measured in degrees of rotation for Hip neutral (HIPNEUTRAL). Radiographic measurements were made in cm for first ray length (RAYLENGTH), and degrees for the first metatarsal declination angle (MTANGLE), and the first and second primus varus angle (PRIMUSANGLE).

The ischemic index is a common clinical measure of blood flow to the foot.[54,70] The index is recorded as a ratio of systolic blood pressure in the ankle divided by the systolic blood pressure in the arm. Measurements were made of touch sensibility (TOUCH) using Semmes Weinstein filaments[4] (Manual Arts Department, Gillis W. Long Hansen's Disease Center, Carville, LA), and vibration sensibility (VIBRATION) using a biothesiometer[7] (Bio-thesiometer, Bio-Medical Instrument Co., Ohio) as previously reported. Both insturments have been used to determine loss of protective sensation in the diabetic foot.[4,7,41,63] Sensation was measured on the distal pulp of the great toe as recommended by Foster.[34] The three filament sizes used were calibrated by the manufacturer and assumed to be log linear.[5] Recordings were made using a 4 level interval scale. Good reliability of sensory testing using Semmes Weinstein filaments has been reported.[50] Recordings from the biothesiometer were in voltage units on a scale of 0-51. Duration

and type of diabetes were obtained from the medical record and patient interview. Type of diabetes mellitus was recorded as either Type I (insulin-dependent) or Type II (non-insulin-dependent). Joint ROM and position measurements were made using goniometer methods as described by Fromherz.[35] Good reliability of range of motion and position measurements in the foot has been reported.[50]

Standardized anterior/posterior and lateral radiographs were taken in a standing weight-bearing position by the radiology technician, Gillis W. Long Hansen's Disease Center, Carville, LA. Measurements of RAYLENGTH, MTANGLE and PRIMUSANGLE were made directly on the x-ray film using a straight edge and compass as described by Weissman.[71]

2.2.4. Procedure

All subjects were interviewed and secondary variables measured and recorded. Measurements of first ray motion were made with the subject sitting at 90 degrees of hip and knee flexion with the foot and leg in their normal stance relationship. The foot was clamped in the first ray mobility device so that the lesser metatarsals were securely immobilized and the first ray was free to move through full range of motion. A dorsiflexion force was applied to the first ray by a manually operated plunger composed of a force transducer and parallel linear variable differential transformer (Figure 2.1). The applied force was displayed on an oscilloscope which provided a visual feedback to the tester, so that a standard, non-injurious force could be applied to all subjects.

The tester attempted to apply the force consistently at a moderate rate. Two trials of three cyclical loadings and unloadings to a maximal force of approximately 10 kg were made on each subject. The mean of six measurements of RAYSLOPE and RAYROM at 8 kg of force were used for analysis. Group differences in first ray length were determined by lateral radiographs. Differences in ray length could result in systematic measurement error of first ray mobility.

Pressure was measured on subjects walking across an EMED System pressure platform. Each subject was given practice trials to ensure a smooth placement of the foot on the pressure platform. A walking rate of .555 $\pm$ .035 m/s was used for all subjects. This rate was determinied during pilot testing to be comfortable for all groups. Data were collected from three trials. The mean of three measurements of PRESSURE and PRESSURE-TIME for the first MTH mask region was used for analysis (Appendix G).

2.2.5. Data Analysis

A multivariate analysis of variance (MANOVA) was used to test the overall treatment group effect for primary and secondary response variables. Analysis of variance (ANOVA) was used to determine which varibles had a group effect. An alpha level of 0.0125 (0.05/4), determined by a Bonferroni procedure, was used for ANOVA of the four primary response variables. This approach controls for Type I experimentwise error by reducing the alpha level proportional to the number of comparisons made. An alpha level of 0.05 was used for

comparison of secondary variables. A Tukey's studentized range test at an alpha level of 0.05 was used for group mean comparisons. The Tukey's test controls for Type I error associated with post hoc comparisons. Group differences in gender and type of diabetes were analysed using a chi-square test which is appropriate for dichotomous scales. A stepwise regression analysis was used to determine the relationship of primary and secondary response variables on the dependent variables PRESSURE and PRESSURE-TIME. The data were further analysed by discriminate analysis to identify variables which best characterize the three diabetic groups from each other.

2.3. RESULTS

MANOVA showed a significant overall group effect (Wilk's Criterion, $p < 0.0001$). ANOVA's (Appendix J) showed a significant group effect for response variables: TOUCH ($p < 0.0001$), VIBRATION ($p < 0.0001$), RAYROM ($p < 0.0001$), RAYSLOPE ($p < 0.0001$), PRESSURE ($p < 0.0001$), PRESSURE-TIME ($p < 0.0001$), TOEROM ($p < 0.0006$), ANKLEROM ($p < 0.0084$), STROM ($p < 0.0005$), HIPROM ($p < 0.0001$), DURATION ($p < 0.0209$). AGE, HT, WT, INDEX, RFNEUTRAL, MTNEUTRAL, HIPNEUTRAL, RAYLENGTH, MTANGLE, and PRIMUSANGLE were not statistically significant. Chi-square tests showed no significant group effect for the categorical variables GENDER and TYPE (Appendix K).

RAYROM was significantly lower in U1MTH compared to UOTHER, DMCONTROL an and NCONTROL. RAYSLOPE, PRESSURE, and PRESSURE-TIME was significantly higher in U1MTH compared to UOTHER, DMCONTROL and NCONTROL (Table 2.1).

TOUCH and VIBRATION, were significantly higher in both ulcerated groups compared to controls, and TOUCH was significantly higher in DMCONTROLS compared to NCONTROLS (Table 2.3). DURATION was sigificantly higher in both ulcerated groups compared to diabetic controls. TOEROM, STROM, and HIPROM were significantly reduced in both ulcerated groups compared to controls, and ANKLEROM was significantly lower in the U1MTH group compared to controls (Table 2.4).

Stepwise linear regression showed RAYROM, PRIMUSANGLE and MTNEUTRAL to be the most significant predictors of PRESSURE, and PRESSURE-TIME over all subjects (Tables 2.7 and 2.8). The coefficient of determination (R^2) and F ratio show RAYROM had a significantly stronger relationship with PRESSURE ($R^2 = .45$, $p < 0.0001$) than response variables PRIMUSANGLE ($R^2 = .04$, $p < 0.0124$) or MTNEUTRAL ($R^2 = .02$, $p < 0.1044$). The coefficient of determination (R^2) and F ratio show RAYROM had a significantly stronger relationship with PRESSURE-TIME ($R^2 = .40$, $p < 0.0001$) than response variables PRIMUSANGLE ($R^2 = .02$, $p < 0.0966$) or MTNEUTRAL (.03, $p < 0.0723$). Plots of residuals showed regressors RAYROM, PRIMUSANGLE and MTNEUTRAL fit a linear model.

A discriminate analysis (Candisc procedure, SAS) showed a pronounced separation between the three diabetic groups relative to the primary

canonical structure (Figure 2.3). The variables which best discriminated the three diabetic groups based on discriminate coefficients were TOUCH (0.8646), RAYROM (- 0.7542) and PRESSURE-TIME (0.6715).

A stepwise discriminate analysis (Table 2.9 and 2.10) (Stepdisc procedure, SAS) showed variables RAYROM, PRESSURE-TIME, and HT were significant discriminators between UIMTH and UOTHER, and response variables TOUCH, RAYROM, TOEROM, DURATION, and RFNEUTRAL were significant discriminators between U1MTH + UOTHER (all ulcerated patients) and DMCONTROL.

Discriminate analysis between ulcer groups showed RAYROM was a significantly stronger discriminator (R^2 = .40, $p < 0.0001$) between ulcer groups than PRESSURE-TIME (R^2 = .17, $p < 0.0085$) or HT (R^2 = .10, $p < 0.0594$).

The discrimation model for UIMTH versus UOTHER resulted in two cases of misclassification into U1MTH and two cases of misclassification into group UOTHER. The discrimination model for group U1MTH + UOTHER versus DMCONTROL resulted in no misclassifications into U1MTH + UOTHER and two misclassifications for entry into DMCONTROL.

Table 2.1
Group Means and Standard Deviations for First Ray Motion and Pressure

Groups	Dorsiflexion cm	Slope kg/cm	Peak Pressure N/cm^2	Pressure-Time Ns/cm^2
U1MTH	.09 ± .26[a]	12.1 ± 2.4[a]	87.1 ± 25.8[a]	38.1 ± 15.1[a]
UOTHER	.52 ± .27[b]	10.3 ± 1.9[b]	49.5 ± 29.8[b]	17.8 ± 11.9[b]
DMCONTROL	.69 ± .18[b]	8.9 ± 1.6[b]	39.2 ± 20.9[b]	13.3 ± 6.3[b]
NCONTROL	.64 ± .26[b]	9.7 ± 2.1[b]	40.7 ± 21.3[b]	15.1 ± 8.8[b]

[a,b] $p < 0.05$

Table 2.2
Group Means and Standard Deviations or Ratios for Physical Characteristics

Groups	Age years	Gender male/female	Weight kg	Height cm
U1MTH	56.3 ± 13.4	7/12	88.1 ± 17.6	26.8 ± 1.7
UOTHER	54.5 ± 11.4	11/9	85.3 ± 17.2	26.9 ± 1.6
DMCONTROL	56.4 ± 13.2	7/12	92.6 ± 26.7	25.9 ± 1.1
NCONTROL	57.1 ± 12.3	7/12	89.0 ± 11.2	26.1 ± 1.2

Table 2.3
Group Means and Standard Deviations or Ratios for Disease Indices

Groups	TYPE I/II	DURATION years	TOUCH grams	VIBRATION volts	INDEX ratio
U1MTH	4/15	20.7 ± 11.9[a]	3.8 ± 0.4[a]	42.3 ± 10.3[a]	0.97 ± .2
UOTHER	5/15	20.3 ± 10.4[a]	3.7 ± 0.5[a]	42.0 ± 9.9[a]	1.03 ± .2
DMCONTROL	9/10	10.9 ± 13.1[b]	1.7 ± 1.0[b]	21.3 ± 13.7[b]	0.94 ± .1
NCONTROL	--	---	1.1 ± 0.3[c]	12.5 ± 7.8[b]	1.03 ± .1

Note. TYPE = type of diabetes, TOUCH = Semmes Weinstein filaments, VIBRATION = bio-thesiometer, INDEX = ischemic index
[a,b,c] $p < 0.05$

Table 2.4
Group Means and Standard Deviations for Degrees of Joint Range of Motion

Groups	TOEROM	ANKLEROM	STROM	HIPROM
U1MTH	34.7 ± 14.2[a]	2.2 ± 4.2[a]	24.7 ± 7.4[a]	76.0 ± 13.9[a]
UOTHER	31.6 ± 13.0[a]	3.6 ± 3.3[ab]	25.7 ± 5.9[a]	77.0 ± 16.2)[a]
DMCONTROL	46.8 ± 10.0[b]	5.9 ± 3.8[b]	32.3 ± 6.8[b]	89.9 ± 15.6)[b]
NCONTROL	47.2 ± 11.1[b]	5.9 ± 4.3[b]	31.7 ± 6.5[b]	94.2 ± 10.9[b]

Note. TOEROM = first metatarsophalangeal extension, ANKLEROM = ankle dorsiflexion, STROM = total subtalar motion, HIPROM = hip motion
[a,b] $p < 0.05$

Table 2.5
Group Means and Standard Deviations for Degrees of Neutral Joint Position

Groups	MTNEUTRAL varus	RFNEUTRAL varus	HIPNEUTRAL external rotation
U1MTH	-2.4 ± 6.5	13.6 ± 3.9	21.2 ± 15.0
UOTHER	0.6 ± 5.9	12.8 ± 4.5	25.5 ± 15.4
DMCONTROL	1.5 ± 6.6	13.2 ± 3.3	26.8 ± 15.3
NCONTROL	1.6 ± 5.3	13.3 ± 4.2	15.8 ± 9.5

Table 2.6
Group Means and Standard Deviations
for Radiographic Measurements

Groups	RAYLENGTH cm	MTANGLE degrees	PRIMUSANGLE degrees
U1MTH	9.0 ± 0.5	20.1 ± 2.7	9.7 ± 3.2
UOTHER	8.9 ± 0.4	20.0 ± 3.8	8.6 ± 3.3
DMCONTROL	8.8 ± 0.4	21.0 ± 3.1	10.3 ± 3.0
NCONTROL	8.9 ± 0.4	21.4 ± 3.3	10.3 ± 2.8

Table 2.7
Summary of Stepwise Regression Analysis for the Dependent Variable Peak Pressure

Step	Entered	Partial R^2	F ratio	Probability
1	RAYROM	0.4558	62.8079	0.0001
2	PRIMUSANGLE	0.0444	6.5731	0.0124
3[a]	MTNEUTRAL	0.0179	2.7047	0.1044

Note. RAYROM = first ray dorsiflexion,
PRIMUSANGLE = primus varus angle,
MTNEUTRAL = midtarsal neutral position
Note. Significance level to enter and stay was 0.15
[a]Step 3 model, PRESSURE = 66.128179 +
RAYROM (-64.142351) + MTNEUTRAL (-0.86027) +
PRIMUSANGLE (2.260636)

Table 2.8
Summary of Stepwise Regression Analysis for
the Dependent Variable Pressure-time Integral

Step	Entered	Partial R^2	F ratio	Probability
1	RAYROM	0.3969	49.3602	0.0001
2	PRIMUSANGLE	0.0222	2.8328	0.0966
3[a]	MTNEUTRAL	0.0253	3.3256	0.0723

Note. RAYROM = first ray dorsiflexion,
PRIMUSANGLE = primus varus angle,
MTNEUTRAL = midtarsal neutral position
Note. Significance level to enter and stay 0.15
[a]Step 3 model, PRESSURE-TIME = 28.655563 +
RAYROM (-28.021392) + MTNEUTRAL (-0.484311) +
PRIMUSANGLE (0.787932)

Table 2.9
Stepwise Discriminate Analysis between Diabetic Patients with a History of First Metatarsal Head Ulceration and Diabetic Patients with a History of Other Forefoot Ulceration

Step	Entered	Partial R^2	F - Ratio	Probability
1	RAYROM	0.4042	25.099	0.0001
2	PRESSURE-TIME	0.1773	7.758	0.0085
3	HT	0.0979	3.798	0.0594

Note. RAYROM = first ray dorsiflexion,
PRESSURE-TIME = pressure-time integral, HT = height
Note. Significance level to enter and stay 0.15

Table 2.10
Stepwise Discriminate Analysis between Diabetic Patients with a History of Ulceration and Diabetic Patients with no History of Ulceration

Step	Entered	Partial R^2	F - Ratio	Probability
1	TOUCH	0.6796	118.798	0.0001
2	RAYROM	0.0674	3.974	0.0512
3	TOEROM	0.0459	2.600	0.1127
4	DURATION	0.0483	2.687	0.1071
5	RFNEUTRAL	0.0479	2.616	0.1118

Note. TOUCH = Semmes Weinstein filaments,
RAYROM = first ray dorsiflexion,
TOEROM = first metatarsophalangeal extension,
DURATION = duration of diabetes,
RFNEUTRAL = rearfoot neutral
Note. Significance level to enter and stay 0.15

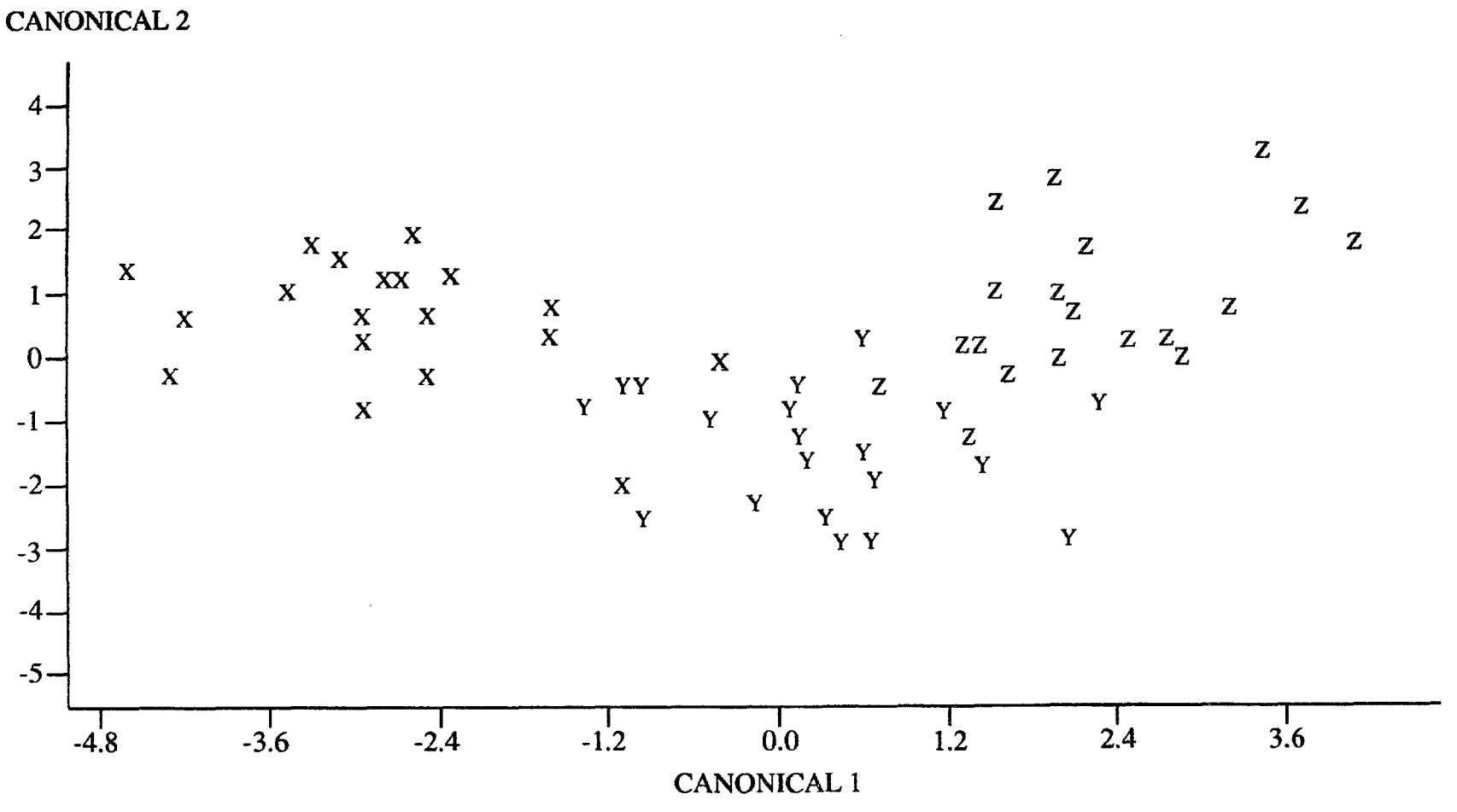

Figure 2.3. Discriminate analysis. Plot of canonical structures for diabetic groups. X, No history of ulceration. Y, Ulceration not at the first metatarsal head. Z, Ulceration at the first metatarsal head.

2.4. DISCUSSION

Mean dorsiflexion was only 0.09 cm for U1MTH compared to 0.52, 0.69 and 0.64 cm for UOTHER, DMCONTROL and NCONTROL respectively which shows the U1MTH group functions in relative plantar flexion (Figure 2.4) compared to the other groups. The first metatarsal is considered plantarflexed when the major portion of total dorsiflexion to plantarflexion is plantar to the relative plane of the lesser metatarsal heads.[57]

Abnormal pressure and ulceration at the first MTH has been associated with plantar flexion deformity in the foot.[37] Slope of the force/motion curve has been shown to be a good indicator of joint stiffness where joint stiffness is defined as the change in motion/change in force.[5] In this study the slope of the force displacement curve was significantly higher in the U1MTH group showing the first ray was stiffer in these patients.

PRESSURE in the U1MTH patients was more than twice that of controls and almost twice that of UOTHER (Figure 2.5). PRESSURE-TIME was two and a half times that of controls and more than twice that of UOTHER. These data support previous studies which have found patients with a history of plantar ulcerations have significantly higher foot pressure, corresponding to the location of ulceration.

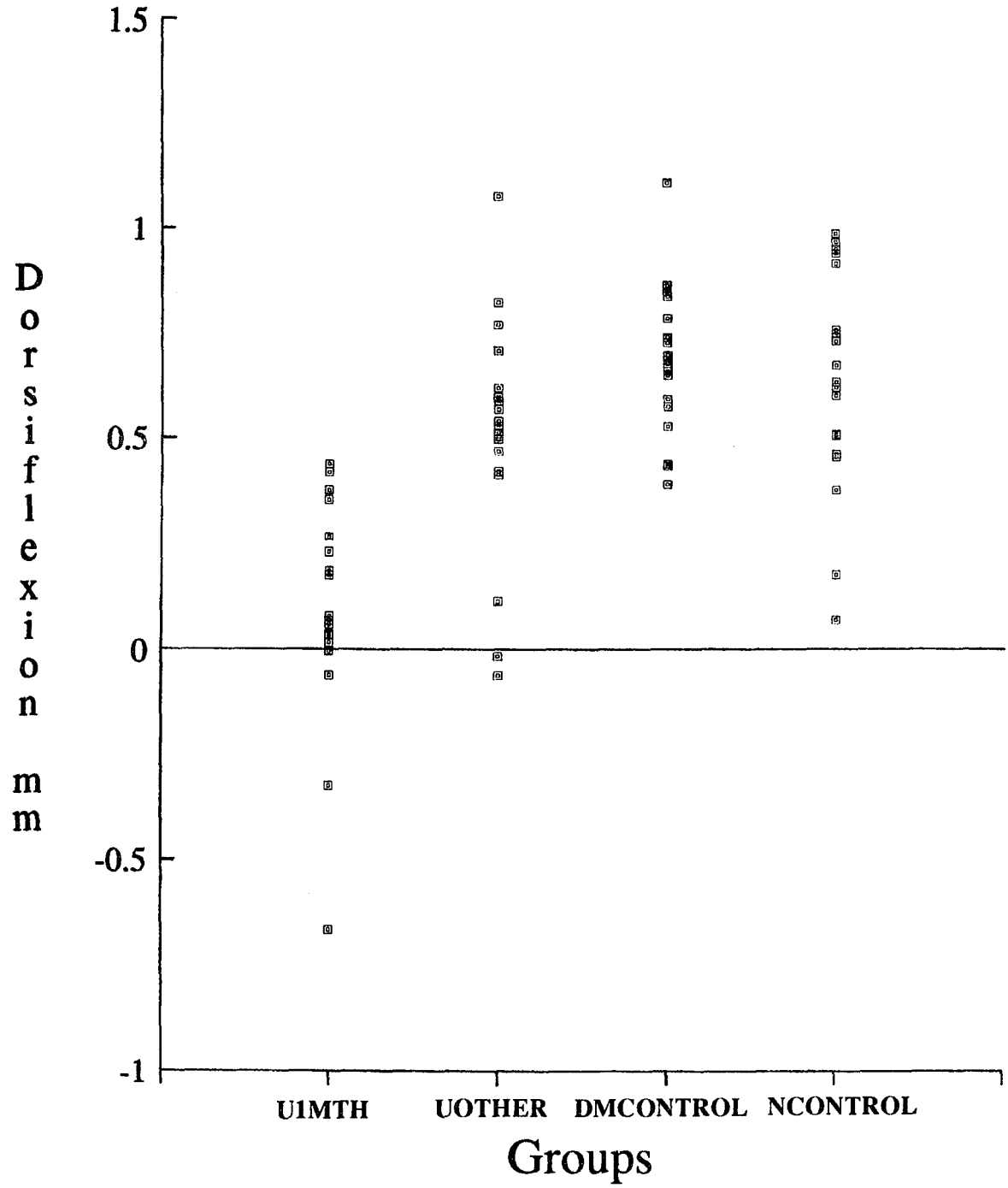

Figure 2.4. First ray motion for groups.

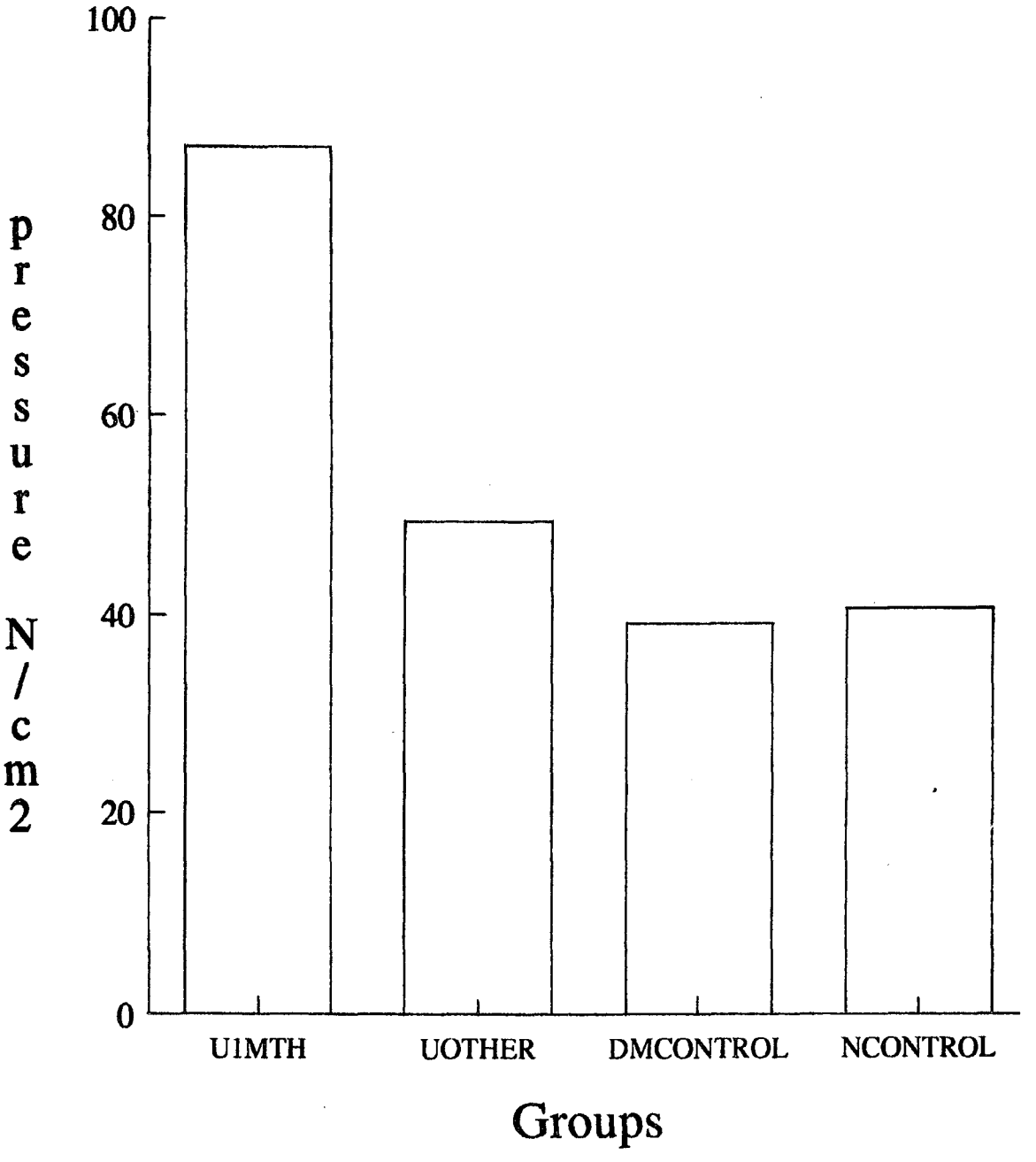

Figure 2.5. Peak pressure first metatarsal head for groups.

Normal and abnormal pressure thresholds have not been determined for the EMED system. The mean + 1 SD in normal subjects has been recommended for abnormal pressure thresholds.[68] In this study using NCONTROL group data the abnormal pressure threshold was determined to be 62.0 N/cm^2 (620 KPa). This value is lower than abnormal thresholds previously reported using the Pedobarograph[68] and Penn State University piezoelectic mat.[19] A lower pressure value using the EMED System may be due to the instruments poorer resolution.[19] Normal and abnormal data for PRESSURE-TIME is not available. In this study, using the mean + 1 SD in normal subjects, the abnormal PRESSURE-TIME threshold was determined to be 23.9 Ns/cm^2 (239 KPa-s). It is not known whether PRESSURE which measures the magnitude of pressure, or PRESSURE-TIME which measures the total pressure acting over a period of time is more damaging to the foot.[18]

These results support the study hypotheses that the first ray is stiffer and limited in dorsiflexion, and the first MTH has higher pressures during walking in diabetic patients with a history of first MTH ulceration. During walking, stiffness in the first ray, may contribute to high pressure and ulceration at the first MTH. The issue of whether limited first ray mobility is a cause or an effect of ulceration must be considered. The retrospective research design used in this study restricts conclusions that a causal link exists. Alternatively, limitation of first ray motion may have resulted from such factors as post-healing scar, treatment immobilization, or disuse. The literature, however, presents strong theory and supportive data that limited joint mobility from a pre-existing condition or secondary disease complication

results in high foot pressures. Limited first ray mobility may result in high stresses on the first MTH which in the neuropathic foot may lead to tissue injury and ulceration.

2.4.1. Discussion of physical characteristics

Age, gender, and obesity are related to diabetes and diabetes complications.[26,47,68] Obesity has been shown to be a risk factor for Type II diabetes and may be a factor in ulceration.[26,36] In this study, age and gender were controlled variables between the UlMTH, DMCONTROL and NCONTROL groups, to eliminate their possible effect on joint mobility or pressure. Weight and height were not significantly different between groups. Weight differences between groups were expected. Veves found diabetic patients were significantly heavier than non-diabetic controls, but no difference was found between those with and without neuropathy.

These findings suggest that weight is not a specific risk factor in plantar ulceration.[68] There is a question as to whether body weight is related to foot pressure. Recent studies have contradicted early reports that weight is a predictor of foot pressure.[22,24,65]

2.4.2. Discussion of disease indicies

There were 3 times more Type II diabetes within the ulcerated groups. (Table 2.3). Foot ulcerations occur in both Type I and II diabetes. Type II diabetes is more common than Type I[26] which explains the distribution

found in this study. The finding of no difference between groups for INDEX supports the belief that poor circulation is not a cause of plantar ulceration. Sensory measurements TOUCH and VIBRATION were significantly higher in both ulcer groups (U1MTH and UOTHER) compared to controls groups. This finding is consistent with numerous studies that have shown sensory loss is strongly associated with ulceration and may be the primary cause of plantar ulceration.

The lowest TOUCH threshold for the ulcerated patients in this study was the #3 filament (75 gram). Several studies have recommended the use of the #2 filament (10 gram) as the threshold level for protective sensation,[4,41,50] while others have recommended a lower threshold level.[46,63] In this study no patient in the ulcerated groups could feel the #2 filament at the great toe. The lowest threshold for biothesiometer testing of the great toe in ulcerated patients was 21 volts. Bloom et al. recommended age adjusted Centile Charts for use in assessing diabetic peripheral neuropathy.[7] They found for biothesiometer testing the mean and standard deviation for the age 50 was 12.1 $\pm$ 7.9 volts. Their values are extremely close to the vibratory thresholds obtained on the NCONTROL group (12.5 $\pm$ 7.8 volts). The upper threshold of normal (mean + 2 SD) using NCONTROL values or Bloom's data is 28 volts. A protective threshold level for the diabetic foot, based on the normal mean + 2 SD, would result in several false negatives in this study. Early detection of loss of protective sensation may identify diabetic patients most at risk of ulceration and in need of preventative care.

TOUCH threshold was significantly increased in DMCONTROL versus NCONTROL. Biothesiometer testing did not show a significant difference between control groups. This finding supports previous studies that found the Semmes Weinstein filaments were more sensitive than the biothesiometer in detecting sensory neuropathy.[46,63]

Duration of diabetes was significantly increased in ulcerated groups compared to DMCONTROL. This finding is consistent with previous data that duration of diabetes is a stong risk factor in diabetic complications.[47]

2.4.3. Discussion of range of motion

Mueller found mean degrees STROM was 25.5 $\pm$ 8 in diabetics with ulceration, compared to 30.5 $\pm$ 10 in diabetic controls, and 34.5 $\pm$ 7 in non-diabetic controls.[50] Mueller found mean degrees ANKLEROM was 2.0 $\pm$ 8 in diabetics with ulceration, compared to 5.0 $\pm$ 3 in diabetic controls, and 6.5 $\pm$ 4 in non-diabetic controls. There is strong agreement between the findings of Mueller and this study (Table 2.4) that diabetic patients with a history of ulcertion have significantly smaller motion compared to their diabetic and non-diabetic controls. Both studies used standard goniometric techniques, and the similarity in data between both studies supports the validity of the methods used.

TOEROM, STROM, ANKLEROM, and HIPROM do not appear specifically related to first MTH ulceration since joint range of motion was not

significantly different between ulcer groups. These data support previous reports which show the association of limited joint mobility and ulceration in diabetics. Hyperglycemia is believed to result in a generalized pattern of joint limitation in the upper and lower extremity. It has been proposed that joint limitation in the foot reduces the shock absorption during walking and increases tissue stresses. Shock absorption is a function of the subtalar joint at the heal strike phase of gait.[30] Limited ankle dorsiflexion more likely contributes to ulceration by high forefoot pressure resulting from an early heel rise or compensatory pronation during the stance phase of gait.[37,40,43,57,67]

This study is the first to show the relationship between limitation of hip motion and diabetic ulceration. Limited hip motion may increase stresses in the foot by abnormal pronatory or supinatory forces on the foot due to a reaction through the closed kenetic chain.[43]

2.4.4. Discussion of neutral position

Differences in MTNEUTRAL and RFNEUTRAL were expected. The literature suggests that rearfoot varus, forefoot varus and forefoot valgus contribute to abnormal patterns of pressure in the forefoot. The U1MTH group had a mean MTNEUTRAL position of 2.4 degrees valgus (indicated by the negative sign) compared to a varus position for the other groups (Table 2.5). A MTNEUTRAL valgus includes subjects with a first through fifth eversion and those with a plantar flexed first ray. The latter may

be the more common in the UlMTH group. The lack of significance of this finding is due to a large variation in the data as noted by the SD values. One source of variation may be measurement error. Fromherz reported that quantifiable forefoot neutral position measurements are difficult to obtain.[35] There may also be true variation in the data. Mueller found both varus and valgus were related to ulceration at the first MTH.[26]

2.4.5. Discussion of radiographic measurements

There was no difference in first ray length among groups (Table 2.6). It is, therefore, assumed that the torque applied to the first ray during mobility measurements was the same among groups.

A larger MTDA was expected in the UlMTH group. MTDA is associated with a cavus or supinated foot, a rigid plantar flexed first ray, and uncompensated rearfoot varus which may contribute first MTH pressure. In contrast, a smaller MTDA and larger primus varus angle is associated with a pes planus or pronated foot, a hypermobile first ray which may contribute to central MTH pressure.[57,67,71] The group mean data do not support these effects.

2.4.6. Discussion of regression analysis

The finding on regression analysis, that reduced RAYROM is a strong, negative predictor of PRESSURE and PRESSURE-TIME across all treatment

groups, supports the theory that limited motion of the first ray is a cause of pressure at the first MTH. These results disagree with the findings of Rogers who in a study of normal male subjects showed weight, the arch index (a measure of arch height) and height were related to regional pressure, but not first ray mobility.[55] There are several possible reasons Rogers failed to show a relationship between first ray mobility and pressure. First, for the purpose of simplifying the analysis, pressures were averaged over general areas of the foot (toes, ball, arch) and first ray mobility was never analyzed with first metatarsal head pressure. Second, her sample included a narrow population (normal males) which provided a limited range of data for regression analysis. Lastly, she analyzed only force discplacement data and did not measure first ray dorsiflexion.

2.4.7. Discussion of discriminate analysis

Discriminate analysis showed: 1) of the two sensory measures TOUCH was a stronger discriminator than VIBRATION, 2) of the two first ray mobility measures RAYROM was a stronger discriminator than RAYSLOPE, and 3) of the two pressure measures PRESSURE-TIME was a stronger discriminator than PRESSURE. Limited dorsiflexion may contribute more to plantar ulceration than stiffness, and pressure over a longer period of time may be more critical in tissue injury that magnitude of pressure.

Discriminate analysis supported the relationship between RAYROM and PRESSURE-TIME to first MTH ulceration which was found in ANOVA and

regression analysis. Ht was also shown to be a discriminator between ulcer groups. Rogers showed a relationship between height and pressure in normal subjects.[55] It may be that tall individuals with large feet have higher plantar stresses, and are more susceptible to first MTH ulceration than shorter individuals. This finding deserves further study.

Both the cases of misclassification in the discrimination model for U1MTH versus UOTHER had a history of fifth MTH ulceration and associated plantar flexion of the first ray deformity. These individuals would be expected to have a high pressure over both the first and fifth MTH's and therefore at risk of first or fifth MTH ulceration.[37] The two misclassifications in the discrimination model for group U1MTH + UOTHER versus DMCONTROL may be cases with a very high risk of ulceration.

2.4.8. General discussion

In this study several methods of data analysis show a strong association between sensory loss, limited range of motion, high pressure and ulceration. Boulton reported that several component causes, including sensory loss, abnormal pressure, and limited joint mobility, were necessary for plantar ulceration to develop. Sensory loss permits injury to the tissues of the foot. Injuries result from high pressures due to such factors as limited joint mobility. These findings suggest that limited first ray mobility may specifically contribute to ulceration at

the first MTH. Prospective studies are needed to validate the causal relationship of limited first ray mobility in first MTH ulceration.

Previous studies have shown that there is a generalized pattern of limited joint mobility associated with the complications of diabetes. In this study range of motion at the great toe, subtalar joint, ankle, and hip was smaller in both ulcerated groups compared to controls. Ulcerated patients were found to have advanced disease based on a longer duration of diabetes and a lower sensory level compared to DMCONTROLS. In contrast, limitation of motion at the first ray and high pressure were found only in the UlMTH group. Limited first ray motion may result from other causes such as a pre-existing biomechanical deformity. Further study is needed to determine the cause of deformity and limited joint mobility in diabetes.

The research design used in this study compared the feet of different individuals. In an alternative design, where both feet of each subject would be measured, comparisons of joint mobility could be made with the uninvolved leg. A finding of lower first ray mobility in the ulcerated limb compared to the non-ulcerated limb would have provided additional support that a relationship between hypomobility and ulceration exists. A study comparing the contralateral limb would have provided control for subject differences due to disease, age and gender. While contrasts with the contralateral limb would have been of interest, the additional time of testing would have limited the availability of volunteer subjects and increased the risk of patients injurying their feet during

barefoot walking. Future studies should focus on the joint mobility of the contralateral limb in ulcerated patients.

Currently, diabetic foot ulcerations are treated with limited success. While the mechanical factors in plantar ulceration are well documented, there remains a strong emphasis on the use of topical agents to promote wound healing. Expensive methods, such as growth factors and hyperbaric oxygen, are promoted by wound care centers while simple, less expensive orthotic devices are under-utilized. Medicare and private insurance often reimburse diabetic patients for topical, wound care agents, hyperbaric treatments and surgery, but not for healing devices and footwear. This study provides data which support the view that limited joint mobility and an associated plantar flexion deformity of the first ray are primary causes of mechanical stresses leading to first metatarsal head ulceration in diabetic patients. Efforts should be made to identify this deformity in early diabetic cases and to develop treatment methods to reduce the deformity or reduce the associated stresses on the first metatarsal head.

Measurement of first ray motion, sensation and pressure may be valuable in identifying early cases who are at risk of first MTH ulceration. Patients with neuropathy, limited first ray mobility and high pressure may benefit from orthotics and footwear which reduce the impact of plantar flexion of the first ray during walking. Orthotics designed with lateral forefoot posts have been recommended to balance a plantar flexed first ray deformity.[40] Footwear with soft, elastic insoles and

outersoles may increase shock absorption resulting from limited first ray mobility. Prospective studies are needed to determine if balanced orthotics and cushioned footwear reduce the risk for first MTH ulceration. Additionally, exercises may be useful in increasing first ray motion. Mobilization of the first ray and passive exercises may increase joint range of motion particularly if the glycosylation of collagen protein is a contributing factor. Exercises may not improve range of first ray motion if limitation is the result of a pre-existing biomechanical deformity. Studies are needed to determine if exercise improves the range of joint motion in the feet of diabetic patients.

2.5. SUMMARY AND CONCLUSIONS

This study showed that diabetic patients with a history of first MTH ulceration had significantly limited first ray mobility, and high pressure at the first MTH compared to diabetics who ulcerated at other locations, and matched diabetic and non-diabetic controls. Limited first ray mobility was shown by regression analysis to be a strong predictor of pressure at the first MTH across all subjects in the study. In combination, these findings support the view that limited first ray mobility is a cause of high stress and a component factor in ulceration at the first MTH in diabetic patients.

Analysis of secondary variables showed duration of diabetes was higher in diabetic patients with a history of ulceration compared to controls, whereas sensation, range of motion at the great toe, subtalar joint,

ankle and hip were significantly lower. Additionally, Semmes Weinstein sensory measurements showed that diabetic patients with no history of ulceration had reduced foot sensation compared to non-diabetic controls. Discriminate analysis showed RAYROM was the strongest discriminator between the ulcer groups. PRESSURE-TIME also provided meaningful discrimination. Sensory loss measured by Semmes Weinstein filaments provided very strong discrimination between ulcerated diabetics and non-ulcerated diabetic controls.

These results demonstrate that the pathomechanical factors, limited joint mobility and high pressure, are significantly related to plantar ulceration and ulcer location in diabetes. Management of joint limitation may be a valuable approach in the prevention and treatment of plantar ulceration in diabetes.

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APPENDIX A

ABBREVIATIONS USED IN TEXT

ANKLEROM	ankle dorsiflexion
DMCONTROL	diabetes mellitus control group
DURATION	duration of diabetes
GENDER	gender
HIPNEUTRAL	hip neutral
HIPROM	hip range of motion
HT	height
INDEX	ischemic index
MTANGLE	first metatarsal declination angle
MTH	metatarsal head
MTNEUTRAL	midtarsal neutral
TOEROM	metatarsophalangeal extension
NCONTROL	normal control group
PRESSURE-TIME	pressure-time integral
PRESSURE	peak pressure
PRIMUSANGLE	primus varus angle
U1MTH	ulcer first metatarsal group
UOTHER	ulcer other group
RAYROM	first ray dorsiflexion
RFNEUTRAL	rearfoot neutral
RAYLENGTH	first ray length
ROM	range of motion
RAYSLOPE	first ray slope
STNEUTRAL	subtalar neutral
STROM	subtalar range of motion
TOUCH	Semmes Weinstein filament sensation
TYPE	type of diabetes
VIBRATION	bio-thesiometer vibration sensation
WT	body weight

APPENDIX B

BIOMECHANICAL CONVERSIONS FOR STRESS (PRESSURE)

1 kg/cm^2 = 14.22 lb/in^2 = 9.807 N/cm^2 = 98.07 KPa = 735.174 mm Hg

1 lb/in^2 = 0.6897 N/cm^2 = 0.07032 kg/cm^2 = 6.897 KPa = 51.7 mm Hg

1 N/cm^2 = 1.45 lb/in^2 = 0.102 kg/cm^2 = 10 KPa = 74.965 mm Hg

1 KPa = 0.145 lb/in^2 = 0.0102 kg/cm^2 = 0.1 N/cm^2 = 7.4965 mm Hg

1 mm Hg = 0.01934 lb/in^2 = 0.00136 kg/cm^2 = 0.01334 N/cm^2 = 0.13339 KPa

APPENDIX C

MATCHING BETWEEN THE U1MTH AND CONTROL GROUPS FOR YEARS OF AGE AND GENDER

Males

U1MTH	DMCONTROL	NCONTROL
40	41	39
44	45	45
55	54	55
56	54	57
59	60	60
72	71	72
73	73	72

Females

U1MTH	DMCONTROL	NCONTROL
29	35	28
34	41	39
47	46	47
48	47	49
51	50	49
55	55	52
64	65	64
65	66	65
66	67	66
68	68	67
70	72	71
74	73	74

APPENDIX D

INFORMED CONSENT FORM

Abstract For Informed Consent
Gillis W. Long Hansen's Disease Center

Title of Study: Limited First Ray Dorsiflexion, Increased Pressure and Ulceration at the First Metatarsal Head in Diabetic Patients

Investigator: James A. Birke

Purpose of Investigation: determine if joint stiffness is a cause of high pressure and ulceration at the first metatarsal head (area behind the big toe) in the feet in patients with diabetes mellitus. High pressure is believed to be an important cause of ulceration (open sores) in the feet of diabetic patients.

Procedures:

1. All diabetic patients will first be examined by a physician of the GWLHDC for medical clearance to participate in the study.

2. Two X-rays will be made of the sample foot to measure the length and inclination angle of the bones behind the big toe.

3. Measurements of the motion of the feet and ankles will be made using a goniometer (angle measuring device) while standing, and lying on a table.

4. While seated in a chair the foot will be secured firmly into a device which will measure motion of the bones behind the big toe. A plunger will push the bones upward and motion will be recorded electronically. The force will be moderate to minimize the risk of pain or injury to the foot during testing.

5. Subjects will walk barefoot on a platform which measures pressure on the foot. This instrument determines if areas of the foot are getting high pressure during walking. The platform is padded with a material used to make shoe insoles to minimize the risk of injury to the feet during walking barefoot.

There are no direct benefits or compensation to you for participating in this study. The data obtained from your participation will be kept confidential and used for the purposes of research.

Date_____

* To Be Retained By The Investigator

EXPERIMENT SIGN-UP FORM

My signature, on this sheet, by which I volunteer to participate in the experiment on Limited first ray dorsifexion, increased pressure and ulceration at the first metatarsal head in diabetic patients

conducted by James A. Birke

Experimenter

indicates that I understand that all subjects in the project are volunteers, that I can withdraw at any time from the experiment, that I have been or will be informed as to the nature of the experiment, that the data I provide will be anonymous and my identity will not be revealed without my permission, and that my performance in this experiment may be used for additional approved projects. Finally, I shall be given an opportunity to ask questions prior to the start of the experiment and after my participation is complete.

_____

Subject's signature

APPENDIX E

RELIABILITY OF THE FIRST RAY MOBILITY DEVICE

Reliability measurements of first ray dorsiflexion (cm) for the mean of three oscillations in two trials of testing on the first ray mobility device in 73 subjects. Four subjects were not included because of missing data.

Trial 1	Trial 2
0.03	-0.06
0.01	0.06
0.20	0.17
0.22	0.24
0.13	0.01
0.13	0.22
0.24	0.29
0.01	0.06
-0.32	-0.33
0.40	0.44
0.38	0.50
0.35	0.35
0.06	0.06
0.01	0.06
0.01	0.03
-0.63	-0.70
0.16	0.07
-0.04	-0.08
0.61	0.63
0.41	0.42
0.92	0.73
0.60	0.45
0.54	0.30
0.60	0.48
0.63	0.57
0.62	0.56
0.59	0.54
0.77	0.48
0.57	0.43
1.12	1.04
0.53	0.49
-0.03	0.00
0.41	0.55
1.00	0.54
0.41	0.53
0.77	0.64
1.03	0.69
0.58	0.29
0.79	0.69
0.66	0.73

0.34	0.53
0.80	0.66
0.44	0.34
1.16	1.06
0.71	0.59
0.86	0.87
0.66	0.66
0.93	0.75
0.51	0.64
0.87	0.83
0.54	0.52
0.59	0.59
0.76	0.82
0.43	0.39
0.60	0.41
0.08	0.06
0.53	0.59
1.02	0.96
0.67	0.67
1.02	0.81
0.45	0.30
1.00	0.90
0.68	0.52
1.03	0.90
0.71	0.81
1.14	0.75
0.67	0.59
0.72	0.75
0.64	0.86
0.08	0.27
0.98	0.89
0.57	0.45
0.61	0.63

Mean and standard deviation
for first ray mobility trials

Trial 1	.52 ± 0.36 cm
Trial 2	.47 ± 0.33 cm

ANOVA summary table for two trials of testing on the first ray mobility device

Source	df	MS	F
Subjects	72	.2293	27.6842
Trials	1	.1063	12.8313
Residual	72	.0082	

Intraclass correlation coefficient (3,1) = .9302

APPENDIX F

RELIABILITY OF THE EMED SYSTEM

Reliability of pressure measurements (N/cm^2) for three trials of walking on the EMED System platform on 73 subjects. Four subjects were not included because of missing data.

Trial 1	Trial 2	Trial 3
111	109	62
112	87	126
70	120	118
61	45	37
124	111	104
95	91	77
73	32	127
56	55	63
90	117	125
82	49	48
110	112	55
107	97	116
80	125	82
21	33	101
110	127	110
126	126	125
83	99	55
39	38	50
65	19	26
30	54	30
52	25	56
126	109	125
33	46	29
43	47	85
36	17	23
26	28	43
16	24	64
30	37	28
21	10	37
16	10	13
37	10	10
99	110	95
24	28	38
57	80	94
72	45	29
39	37	57
19	16	38
96	120	56
18	27	27
20	20	45
25	33	50
31	46	46

87	69	43
31	27	66
33	29	22
24	19	21
42	30	38
31	28	32
22	17	28
42	38	57
14	66	26
21	19	17
20	28	19
28	28	49
39	38	19
87	70	92
55	27	59
24	20	22
22	25	35
32	26	33
44	42	37
22	32	29
22	25	19
36	45	67
19	14	16
26	34	26
43	34	30
33	23	35
19	24	43
116	117	38
17	12	72
109	41	78
31	33	29

Mean and standard deviation for EMED trials

Trial 1	51.67 ± 34.03 N/cm^2
Trial 2	50.01 ± 35.72 N/cm^2
Trial 3	53.73 ± 32.56 N/cm^2

ANOVA summary table for comparison of three trials of walking on the EMED platform

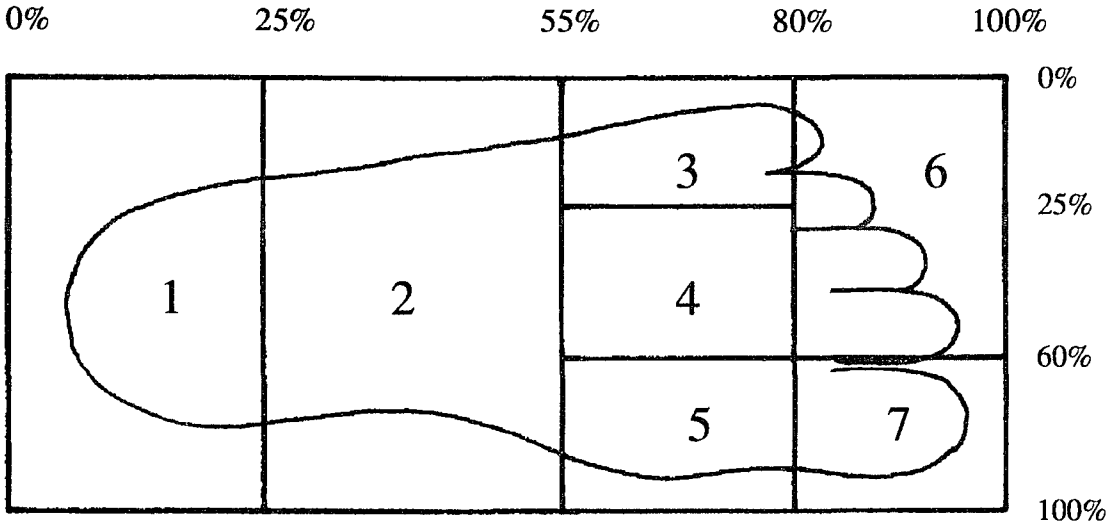
Source	df	MS	F
Subjects	72	2862.647	9.058699
Trials	2	252.473	.7989
Residual	144	316.007	

Intraclass correlation coefficient (3,1) = .7292

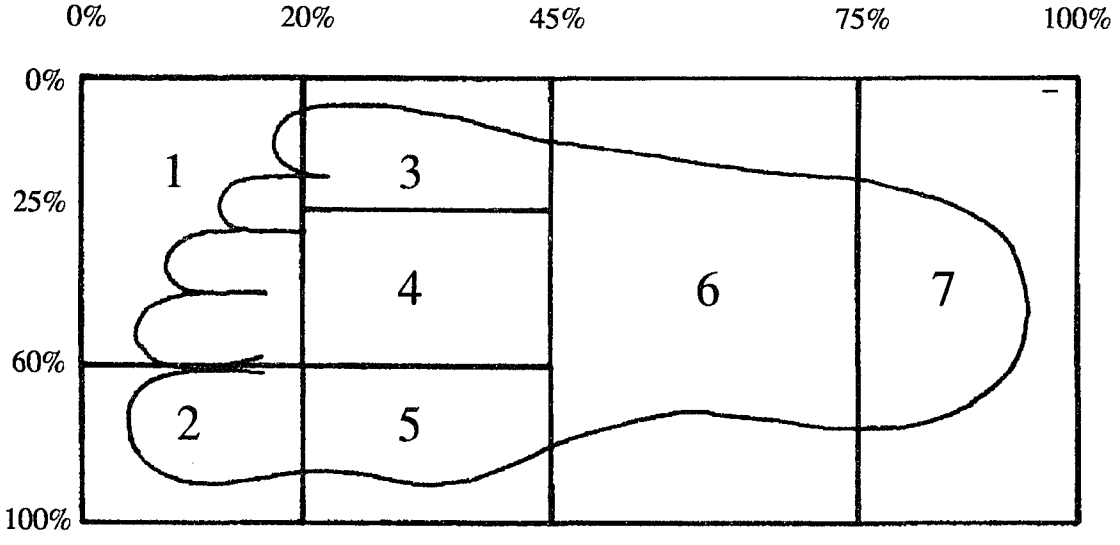
APPENDIX G

MASK DEFINITIONS AND FOOT ORIENTATION
FOR THE EMED SYSTEM MULTIMASK SOFTWARE

The first metatarsal head area is represented by mask 5.



Left Foot



Right Foot

APPENDIX H

RAW DATA BY GROUPS

H.1. U1MTH. Subject characteristics

	GROUP	AGE	GENDER	WT	HT	TYPE	TOUCH	VIB	INDEX	DURAT
1	2	70	F	110	28.0	1	4	39	0.72	24
2	2	55	M	106	29.0	1	4	51	0.96	20
3	2	65	F	65	25.4	2	4	30	1.05	6
4	2	29	F	72	26.8	1	4	51	0.96	16
5	2	64	F	103	25.8	1	4	28	1.18	30
6	2	68	F	73	23.6	1	4	50	0.96	40
7	2	40	M	102	29.5	2	4	51	0.93	3
8	2	55	F	78	26.2	1	4	36	1.09	20
9	2	73	M	92	28.0	1	4	51	1.08	15
10	2	56	M	113	28.4	2	3	51	0.89	14
11	2	59	M	125	29.1	1	4	51	0.75	20
12	2	44	M	64	27.6	1	4	51	0.80	30
13	2	47	F	74	25.4	1	4	51	0.79	38
14	2	74	F	72	24.6	1	4	43	1.17	15
15	2	51	F	86	26.4	2	4	21	1.14	3
16	2	72	M	87	27.8	1	4	51	1.04	20
17	2	66	F	87	26.9	1	3	30	0.81	7
18	2	34	F	88	24.8	1	3	33	1.19	30
19	2	48	F	76	26.6	1	4	34	0.65	42

WT = weight, HT = height,
TOUCH = Semmes Weinstein filaments, VIB = vibration,
INDEX = ischemic index, DURAT = duration of diabetes

H.2. U1MTH. First ray mobility and pressure

	RAYROM	RAYSLOPE	PRESSURE	P-TIME
1	0.080	8.83	40.0	15.0
2	-0.002	11.37	94.0	41.3
3	0.038	14.99	108.3	62.3
4	-0.060	14.96	68.0	22.5
5	0.185	12.15	102.7	31.0
6	0.230	12.04	47.6	16.7
7	0.070	16.11	113.0	46.3
8	0.178	12.11	87.7	33.3
9	0.265	11.83	77.3	64.7
10	0.035	14.16	58.0	31.0
11	-0.325	15.65	110.7	51.7
12	0.418	10.66	59.7	24.3
13	0.376	10.85	100.3	38.0
14	0.437	9.12	92.3	45.3
15	0.353	7.81	106.7	51.7
16	0.060	13.64	95.7	40.0
17	0.036	13.91	51.7	14.3
18	0.018	9.93	115.7	48.3
19	-0.667	10.16	125.7	47.0

RAYROM = first ray dorsiflexion,
RAYSLOPE = first ray slope,
PRESSURE = peak pressure,
P-TIME = pressure-time integral

H.3. U1MTH. Joint mobility and neutral position

	TOEROM	ANKLEROM	STROM	RFNEUTRAL	MTNEUTRAL	HROM	HIPNEUTRAL
1	45	-3	17	5	0	77	17
2	35	-5	25	11	5	65	25
3	35	5	22	15	5	90	10
4	30	-2	29	15	12	85	-5
5	40	5	22	13	-2	60	0
6	18	5	25	14	3	68	12
7	25	3	24	17	5	83	27
8	60	5	25	13	-2	90	30
9	40	0	18	15	-4	55	20
10	25	10	36	19	-5	90	60
11	40	2	24	22	-5	78	18
12	20	2	18	14	-8	92	32
13	30	8	36	15	-9	59	15
14	70	-1	42	9	-10	90	15
15	37	5	28	15	-5	100	30
16	20	3	13	15	0	67	43
17	35	5	20	12	-5	65	5
18	43	-5	29	12	-5	72	28
19	12	-1	17	7	-16	58	20

TOEROM = first metatarsophalangeal extension,
ANKLEROM = ankle dorsifexion, STROM = subtalar motion,
RFNEURTRAL = rearfoot neutral, MTNEUTRAL = midtarsal neutral,
HIPROM = hip motion, HIPNEUTRAL = hip neutral

H.4. U1MTH. Radiographic measurements

	RAYLENGTH	MTANGLE	PRIMUSANGLE
1	9.35	22.75	8.50
2	9.85	15.00	12.50
3	8.60	20.00	11.00
4	9.15	14.00	12.50
5	8.70	17.00	12.00
6	8.75	20.00	10.00
7	9.30	18.50	8.00
8	8.90	23.00	8.00
9	9.35	17.50	6.00
10	9.50	22.50	15.25
11	10.20	21.00	13.50
12	8.75	23.50	8.00
13	8.50	22.50	4.50
14	7.90	19.00	14.00
15	8.90	20.50	10.50
16	9.00	21.50	8.50
17	9.25	22.00	3.50
18	7.80	22.00	11.00
19	9.00	18.75	7.50

RAYLENGTH = first ray length,
MTANGLE = metatarsal declination angle,
PRIMUSANGLE = primus varus angle

H.5. UOTHER. Subject characteristics

	GROUP	AGE	GENDER	WT	HT	TYPE	TOUCH	VIB	INDEX	DURAT
1	1	66	M	108	29.1	1	4	51	.56	21.0
2	1	65	M	73	26.8	1	4	51	1.09	20.0
3	1	52	F	59	26.0	1	4	51	.84	23.0
4	1	66	F	77	25.6	1	3	40	1.26	31.0
5	1	53	F	78	24.2	2	3	35	1.13	18.0
6	1	60	F	67	26.8	1	4	35	.93	35.0
7	1	52	M	103	29.9	1	4	51	.74	22.0
8	1	66	M	86	28.2	2	3	28	.59	20.0
9	1	59	F	110	24.6	2	3	26	1.20	5.0
10	1	42	M	74	27.6	1	4	51	1.13	27.0
11	1	48	F	82	25.8	1	4	51	1.28	29.0
12	1	69	M	90	26.6	2	4	40	1.00	4.0
13	1	47	F	107	26.0	1	4	32	1.26	15.0
14	1	27	M	103	28.4	1	3	40	1.18	11.0
15	1	37	F	109	25.6	2	3	38	1.08	2.0
16	1	62	M	65	26.8	1	4	51	.71	25.0
17	1	44	M	68	28.2	1	3	21	1.17	4.5
18	1	69	M	90	28.0	1	4	51	1.25	30.0
19	1	51	M	95	28.7	1	4	51	1.00	29.0
20	1	55	F	61	25.2	1	4	45	1.25	35.0

WT = weight, HT = height,
TOUCH = Semmes Weinstein filaments, VIB = vibration,
INDEX = ischemic index, DURAT = duration of diabetes

H.6. UOTHER. First ray mobility and pressure

	RAYROM	RAYSLOPE	PRESSURE	P-TIME
1	0.115	12.86	79.0	29.3
2	-0.060	13.68	42.3	15.3
3	0.620	12.88	36.7	11.3
4	0.413	10.00	27.3	8.66
5	0.825	13.20	44.3	12.7
6	0.530	9.93	120.0	40.7
7	0.420	10.34	36.0	11.7
8	0.540	8.78	58.3	24.7
9	0.598	8.36	25.3	6.3
10	0.589	9.56	32.3	19.0
11	0.568	8.43	34.7	5.7
12	0.505	9.13	95.5	35.5
13	0.620	12.68	31.7	15.7
14	0.497	9.29	22.7	9.0
15	1.078	9.59	13.0	2.0
16	0.510	6.55	19.0	4.3
17	-0.015	9.62	101.3	38.3
18	0.772	9.18	77.0	34.0
19	0.468	9.49	48.7	16.3
20	0.708	12.20	44.3	16.0

RAYROM = first ray dorsiflexion,
RAYSLOPE = first ray slope,
PRESSURE = peak pressure,
P-TIME = pressure-time integral

H.7. UOTHER. Joint mobility and neutral position

	TOEROM	ANKLEROM	STROM	RFNEUTRAL	MTNEUTRAL	HROM	HIPNEUTRAL
1	15	2	20	16	-2	38	12
2	25	2	22	13	-5	60	40
3	60	5	32	10	5	85	30
4	35	3	27	14	0	53	18
5	45	15	32	2	7	92	52
6	20	0	29	11	-7	70	20
7	23	2	20	8	5	78	42
8	24	5	27	11	0	80	15
9	37	2	22	20	5	80	40
10	47	3	20	13	-5	90	10
11	22	4	10	16	6	65	35
12	32	3	25	18	-5	77	15
13	25	8	23	10	-5	77	22
14	50	5	34	10	5	95	5
15	10	0	27	20	15	105	45
16	20	0	29	18	-7	83	47
17	38	2	22	14	0	97	7
18	35	4	29	14	0	85	25
19	25	3	33	8	5	60	30
20	45	4	32	11	-5	70	0

TOEROM = first metatarsophalangeal extension,
ANKLEROM = ankle dorsifexion, STROM = subtalar motion,
RFNEURTRAL = rearfoot neutral, MTNEUTRAL = midtarsal neutral,
HIPROM = hip motion, HIPNEUTRAL = hip neutral

H.8. UOTHER. Radiographic measurements

	RAYLENGTH	MTANGLE	PRIMUSANGLE
1	9.60	21.75	9.00
2	9.40	22.50	5.00
3	9.05	18.00	7.00
4	8.60	15.00	12.75
5	8.35	21.00	5.50
6	8.85	18.00	16.00
7	9.25	22.00	3.50
8	9.30	24.50	8.00
9	8.80	13.50	13.50
10	9.00	22.50	6.00
11	8.85	16.50	6.00
12	9.00	22.00	11.00
13	8.40	21.50	9.50
14	8.95	22.50	7.50
15	8.70	9.50	5.00
16	8.50	21.00	8.00
17	9.60	22.00	7.50
18	9.00	19.50	13.50
19	9.50	24.00	10.00
20	7.95	22.50	8.50

RAYLENGTH = first ray length,
MTANGLE = metatarsal declination angle,
PRIMUSANGLE = primus varus angle

H.9. DMCONTROL. Subject characteristics

	GROUP	AGE	GENDER	WT	HT	TYPE	TOUCH	VIB	INDEX	DURAT
1	5	52	F	89	25.4	1	1	19	.60	5.0
2	5	66	F	59	26.0	1	2	24	.93	12.0
3	5	49	F	79	24.8	1	3	20	.98	20.0
4	5	60	M	104	26.2	2	2	21	.87	3.0
5	5	47	F	114	26.1	2	2	14	.98	5.0
6	5	39	F	101	25.2	2	1	15	.90	.5
7	5	49	F	85	26.4	1	4	51	.94	4.0
8	5	64	F	91	26.5	1	1	6	.97	4.0
9	5	71	F	67	24.6	2	1	17	1.00	14.0
10	5	65	F	115	25.0	1	1	11	.89	15.0
11	5	67	F	96	24.6	1	1	17	.89	15.0
12	5	45	M	126	27.4	2	1	13	1.22	2.5
13	5	28	F	113	27.2	2	1	5	1.04	2.0
14	5	72	M	75	26.8	2	2	41	.88	1.5
15	5	74	F	73	24.6	1	1	30	1.00	1.0
16	5	39	M	166	28.4	2	1	11	.94	2.0
17	5	55	M	75	27.0	1	2	29	.98	40.0
18	5	57	M	57	24.8	1	1	9	.96	2.0
19	5	72	M	73	26.4	2	4	51	.98	24.0

WT = weight, HT = height,
TOUCH = Semmes Weinstein filaments, VIB = vibration,
INDEX = ischemic index, DURAT = duration of diabetes

H.10. DMCONTROL. First ray mobility and pressure

	RAYROM	RAYSLOPE	PRESSURE	P-TIME
1	0.686	6.59	87.0	30.30
2	0.858	8.61	24.3	7.00
3	0.433	10.39	90.7	14.30
4	0.740	8.41	24.0	9.00
5	0.697	7.27	28.3	8.30
6	0.437	7.51	36.0	7.00
7	0.728	11.35	41.0	20.30
8	0.390	11.92	66.3	22.67
9	1.110	10.42	41.3	19.33
10	0.650	10.60	28.0	12.00
11	0.865	9.47	21.3	7.30
12	0.660	7.65	36.7	15.00
13	0.840	8.38	30.3	13.70
14	0.575	7.21	22.3	8.70
15	0.850	6.87	45.7	11.70
16	0.678	8.30	45.5	13.00
17	0.527	9.86	35.3	16.30
18	0.593	9.60	19.0	7.30
19	0.788	8.64	22.3	10.30

RAYROM = first ray dorsiflexion,
RAYSLOPE = first ray slope,
PRESSURE = peak pressure,
P-TIME = pressure-time integral

H.11. DMCONTROL. Joint mobility and neutral Postion

	TOEROM	ANKLEROM	STROM	RFNEUTRAL	MTNEUTRAL	HROM	HIPNEUTRAL
1	67	6	40	8	-5	75	5
2	35	11	40	13	7	110	20
3	35	0	30	22	15	110	20
4	44	11	32	13	5	87	37
5	45	10	45	8	0	115	35
6	40	6	32	12	0	75	15
7	56	5	27	16	-5	80	10
8	40	3	35	14	0	97	23
9	55	9	37	15	-6	90	10
10	38	2	28	12	4	70	30
11	40	2	24	12	5	100	20
12	58	10	30	8	7	98	62
13	55	2	33	14	-5	90	50
14	50	5	20	15	0	107	42
15	35	7	42	15	0	90	20
16	40	6	35	12	15	90	40
17	47	5	23	13	-10	67	17
18	65	12	36	16	2	97	13
19	45	0	25	12	0	60	40

TOEROM = first metatarsophalangeal extension,
ANKLEROM = ankle dorsifexion, STROM = subtalar motion,
RFNEURTRAL = rearfoot neutral, MTNEUTRAL = midtarsal neutral,
HIPROM = hip motion, HIPNEUTRAL = hip neutral

H.12. DMCONTROL. Radiographic measurements

	RAYLENGTH	MTANGLE	PRIMUSANGLE
1	8.70	20.00	10.00
2	8.65	21.50	9.50
3	8.95	17.00	14.00
4	8.95	16.00	10.50
5	8.80	19.50	11.50
6	8.45	18.75	11.00
7	8.30	23.00	7.00
8	9.40	20.00	7.25
9	8.35	25.50	8.00
10	8.40	23.50	16.00
11	8.85	17.50	12.00
12	8.95	25.50	6.50
13	8.95	20.00	8.50
14	9.20	23.50	10.50
15	8.15	16.00	17.50
16	9.05	20.00	11.00
17	8.85	24.50	9.75
18	8.05	22.75	8.50
19	9.55	25.00	7.00

RAYLENGTH = first ray length,
MTANGLE = metatarsal declination angle,
PRIMUSANGLE = primus varus angle

H.13. NCONTROL. Subject characteristics

	GROUP	AGE	GENDER	WT	HT	TYPE	TOUCH	VIB	INDEX	DURAT
1	6	65	F	68	26.0	-	1	12	1.23	-
2	6	71	M	73	26.8	-	2	22	1.15	-
3	6	60	M	79	27.2	-	1	11	.95	-
4	6	35	F	86	24.2	-	1	5	1.04	-
5	6	55	F	87	25.5	-	1	9	.98	-
6	6	73	M	104	28.0	-	2	27	1.10	-
7	6	47	F	79	25.0	-	1	5	.99	-
8	6	66	F	64	24.9	-	1	6	1.03	-
9	6	72	F	85	26.0	-	1	12	1.14	-
10	6	68	F	63	26.8	-	1	7	.93	-
11	6	46	F	67	26.8	-	1	14	1.12	-
12	6	45	M	81	26.8	-	1	9	1.02	-
13	6	41	M	81	28.2	-	1	14	1.13	-
14	6	54	M	93	26.0	-	1	6	.69	-
15	6	50	F	68	25.2	-	1	5	1.06	-
16	6	41	F	76	24.4	-	1	7	1.02	-
17	6	67	F	70	24.8	-	1	24	1.02	-
18	6	73	F	95	25.2	-	1	30	.91	-
19	6	55	M	81	28.0	-	1	13	1.09	-

WT = weight, HT = height,
TOUCH = Semmes Weinstein filaments, VIB = vibration,
INDEX = ischemic index, DURAT = duration of diabetes

H.14. NCONTROL. First ray mobility and pressure

	RAYROM	RAYSLOPE	PRESSURE	P-TIME
1	0.505	10.00	32.0	9.67
2	0.070	10.98	83.0	36.33
3	0.460	7.66	47.0	14.00
4	0.987	6.87	22.0	65.00
5	0.673	7.68	27.3	8.33
6	0.915	10.09	30.3	12.67
7	0.375	9.04	41.0	14.67
8	0.950	7.60	27.7	9.30
9	0.600	14.90	22.0	9.00
10	0.967	8.69	49.3	16.33
11	0.758	8.17	16.3	8.67
12	0.940	8.32	28.7	12.33
13	0.633	10.26	35.7	13.33
14	0.453	10.08	54.3	17.00
15	0.738	9.55	30.3	9.67
16	0.750	9.82	28.7	11.33
17	0.176	10.82	90.3	37.00
18	0.508	9.18	76.0	27.70
19	0.620	14.16	31.0	9.30

RAYROM = first ray dorsiflexion,
RAYSLOPE = first ray slope,
PRESSURE = peak pressure,
P-TIME = pressure-time integral

H.15. NCONTROL. Joint mobility and neutral position

	TOEROM	ANKLEROM	STROM	RFNEUTRAL	MTNEUTRAL	HROM	HIPNEUTRAL
1	50	4	25	16	-7	85	35
2	27	3	20	14	0	90	20
3	55	10	35	11	-7	100	30
4	65	16	42	12	5	110	20
5	28	5	30	19	0	75	5
6	37	9	26	13	0	92	12
7	45	0	32	23	0	95	15
8	57	5	33	13	5	107	17
9	50	6	30	12	8	82	2
10	45	0	25	5	5	78	8
11	56	6	45	13	15	103	13
12	40	5	27	14	2	92	16
13	50	6	31	14	5	110	0
14	50	5	26	13	-5	85	15
15	52	10	36	16	0	102	32
16	63	12	40	9	-3	105	15
17	56	-2	37	13	0	102	22
18	40	7	28	18	5	95	15
19	30	5	34	5	3	82	8

TOEROM = first metatarsophalangeal extension,
ANKLEROM = ankle dorsifexion, STROM = subtalar motion,
RFNEURTRAL = rearfoot neutral, MTNEUTRAL = midtarsal neutral,
HIPROM = hip motion, HIPNEUTRAL = hip neutral

H.16. NCONTROL. Radiographic measurements

	RAYLENGTH	MTANGLE	PRIMUSANGLE
1	8.35	21.00	8.50
2	9.50	22.75	7.00
3	9.30	23.75	7.50
4	8.05	16.00	13.75
5	8.95	19.00	10.50
6	9.20	25.00	11.50
7	8.55	17.00	10.25
8	8.65	22.00	12.50
9	8.65	26.00	6.50
10	8.90	27.00	15.00
11	9.05	23.00	13.00
12	8.80	18.00	11.00
13	9.10	20.00	8.50
14	9.45	23.50	10.50
15	8.60	15.50	10.00
16	8.20	23.00	6.50
17	8.65	21.00	9.00
18	8.85	20.00	16.00
19	9.55	23.75	8.00

RAYLENGTH = first ray length,
MTANGLE = metatarsal declination angle,
PRIMUSANGLE = primus varus angle

APPENDIX I

MEANS, STANDARD DEVIATIONS AND RANGES
FOR RESPONSE VARIABLES BY GROUPS

I.1. U1MTH

variable	n	mean	SD	range
Age (years)	19	56.30	13.40	29.0 - 74.0
Weight (kg)	19	88.10	17.60	63.5 - 124.7
Height (cm)	19	26.80	1.70	23.6 - 29.5
TOUCH (filament number)	19	3.80	0.40	3 - 4
VIBRATION (volts)	19	42.30	10.30	21 - 51
INDEX (ratio)	19	0.96	0.17	0.65 - 1.19
DURATION (years)	19	20.70	11.90	3.0 - 42.0
RAYROM (degrees)	19	0.09	0.26	-0.67 - 0.44
RAYSLOPE (kg/cm slope)	19	12.10	2.40	7.8 - 16.1
PRESSURE (N/cm^2)	19	87.10	25.80	40.0 - 125.7
PRESSURE-TIME ($N-s/cm^2$)	19	38.10	15.10	14.3 - 64.7
TOEROM (degrees)	19	34.70	14.20	12.0 - 70.0
ANKLEROM (degrees)	19	2.20	4.20	-5.0 - 10.0
STROM (degrees)	19	24.70	7.40	13.0 - 42.0
HIPROM (degrees)	19	76.00	13.90	55.0 - 100.0
RFNEUTRAL (degrees)	19	13.60	3.90	5.0 - 22.0
MTNEUTRAL (degrees)	19	3.20	5.20	-6.0 - 15.0
HIPNEUTRAL (degrees)	19	21.20	15.00	-5.0 - 60.0
RAYLENGTH (cm)	19	9.00	0.60	7.8 - 10.2
MTANGLE (degrees)	19	20.10	2.70	14.0 - 23.5
PRIMUSANGLE (degrees)	19	9.70	3.20	3.5 - 15.3

I.2. UOTHER

variable	n	mean	SD	range
Age (years)	20	54.50	11.40	27.0 - 69.0
Weight (kg)	20	85.30	17.30	58.5 - 109.8
Height (cm)	20	26.90	1.60	24.2 - 29.9
TOUCH (filament number)	20	3.70	.50	3 - 4
VIBRATION (volts)	20	41.90	9.90	21 - 51
INDEX (ratio)	20	1.03	.23	.56 - 1.28
DURATION (years)	20	20.30	10.50	2.0 - 35.0
RAYROM (degrees)	20	0.51	0.27	-0.06 - 1.08
RAYSLOPE (kg/cm slope)	20	10.29	1.95	6.55 - 13.68
PRESSURE (N/cm^2)	20	49.50	29.80	13.0 - 120.0
PRESSURE-TIME ($N-s/cm^2$)	20	17.80	11.90	2.0 - 40.71
TOEROM (degrees)	20	31.60	13.00	10.0 - 60.0
ANKLEROM (degrees)	20	3.60	3.30	0.0 - 15.0
STROM (degrees)	20	25.70	5.90	10.0 - 34.0
HIPROM (degrees)	20	77.00	16.20	38.0 - 105.0
RFNEUTRAL (degrees)	20	12.80	4.50	2.0 - 20.0
MTNEUTRAL (degrees)	20	3.20	5.20	-7.0 - 15.0
HIPNEUTRAL (degrees)	20	25.50	15.40	0.0 - 52.0
RAYLENGTH (cm)	20	8.90	0.40	7.9 - 9.6
MTANGLE (degrees)	20	20.00	3.80	9.5 - 24.5
PRIMUSANGLE (degrees)	20	8.60	3.30	3.5 - 16.0

I.3. DMCONTROL

variable	n	mean	SD	range
Age (years)	19	56.40	13.20	28.0 - 74.0
Weight (kg)	19	92.60	26.70	56.7 - 166.5
Height (cm)	19	25.90	1.10	24.6 - 28.4
TOUCH (filament number)	19	1.70	1.00	1 - 4
VIBRATION (volts)	19	21.30	13.70	5.0 - 51.0
INDEX (ratio)	19	0.94	0.11	0.60 - 1.22
DURATION (years)	19	10.90	13.10	0.5 - 44.0
RAYROM (degrees)	19	0.69	0.18	0.39 - 1.11
RAYSLOPE (kg/cm slope)	19	8.90	1.56	6.59 - 11.92
PRESSURE (N/cm^2)	19	39.20	20.90	19.0 - 90.7
PRESSURE-TIME ($N-s/cm^2$)	19	13.30	6.30	7.0 - 30.3
TOEROM (degrees)	19	46.80	10.10	35.0 - 67.0
ANKLEROM (degrees)	19	5.90	3.80	0.0 - 12.0
STROM (degrees)	19	32.30	6.80	21.0 - 45.0
HIPROM (degrees)	19	89.90	15.60	60.0 - 115.0
RFNEUTRAL (degrees)	19	13.20	3.30	8.0 - 22.0
MTNEUTRAL (degrees)	19	4.10	5.40	-6.0 - 15.0
HIPROM (degrees)	19	26.80	15.30	5.0 - 62.0
RAYLENGTH (cm)	19	8.70	0.40	8.0 - 9.5
MTANGLE (degrees)	19	21.00	3.10	16.0 - 25.5
PRIMUSANGLE (degrees)	19	10.30	3.00	6.5 - 17.5

I.4. NCONTROL

variable	n	mean	SD	range
Age (years)	19	57.10	12.30	35.0 - 73.0
Weight (kg)	19	79.00	11.20	62.6 - 103.9
Height (cm)	19	26.10	1.20	24.2 - 28.2
TOUCH (filament number)	19	1.10	0.30	1 - 2
VIBRATION (volts)	19	12.50	7.80	5.0 - 30.0
INDEX (ratio)	19	1.03	0.12	0.69 - 1.23
DURATION (years)	--	--	--	--
RAYROM (degrees)	19	0.64	0.26	0.07 - 0.99
RAYSLOPE (kg/cm slope)	19	9.68	2.07	6.87 - 14.90
PRESSURE (N/cm^2)	19	40.70	21.30	16.3 - 90.3
PRESSURE-TIME ($N-s/cm^2$)	19	15.10	8.80	8.3 - 37.0
TOEROM (degrees)	19	47.10	11.10	27.0 - 65.0
ANKLEROM (degrees)	19	5.90	4.30	-2.0 - 16.0
STROM (degrees)	19	31.70	6.50	20.0 - 45.0
HIPROM (degrees)	19	94.20	10.90	75.0 - 110.0
RFNEUTRAL (degrees)	19	13.30	4.20	5.0 - 22.5
MTNEUTRAL (degrees)	19	3.70	3.90	0.0 - 15.0
HIPNEUTRAL (degrees)	19	15.80	9.50	0.0 - 35.0
RAYLENGTH (cm)	19	8.90	0.40	8.0 - 9.6
MTANGLE (degrees)	19	21.40	3.30	15.5 - 27.0
PRIMUSANGLE (degrees)	19	10.30	2.80	6.5 - 16.0

APPENDIX J
ANALYSIS OF VARIANCE FOR RESPONSE VARIABLES

J.1. First ray dorsiflexion

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	4.1983	1.3994	23.24	0.0001
Error	73	4.3952	0.0602		
Total	76	8.5935			

J.2. First ray slope

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	107.4609	35.8203	8.75	0.0001
Error	73	298.7377	4.0923		
Total	76	406.1986			

J.3. Peak pressure

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	28758.4948	9586.1649	15.60	0.0001
Error	73	44848.6883	614.3656		
Total	76	73607.1831			

J.4. Pressure-time integral

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	7563.6148	2521.2049	20.69	0.0001
Error	73	8897.4875	121.8834		
Total	76	16461.1023			

J.5. Age

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	70.409	23.4698	0.15	0.9308
Error	73	11590.4736	158.7736		
Total	76	11660.8831			

J.6. Weight

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	9014.7501	3004.9167	1.71	0.1716
Error	73	127963.2368	1752.92101		
Total	76	136977.9871			

J.7. Height

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	88.8978	29.6326	2.35	0.0797
Error	73	921.8273	12.6277		
Total	76	1010.7251			

J.8. Touch sensation

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	109.7302	36.5767	99.00	0.0001
Error	73	26.9711	0.3695		
Total	76	136.7013			

J.9. Vibration sensation

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	12956.1655	4318.7218	38.39	0.0001
Error	73	8213.0552	112.5076		
Total	76	21169.2208			

J.10. Ischemic index

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	0.1299	0.0433	1.59	0.2001
Error	73	1.9933	0.0273		
Total	76	2.1233			

J.11. Duration of diabetes

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	2	1167.0900	583.5450	4.15	0.0209
Error	55	7731.1901	140.5671		
Total	57	8898.2802			

J.12. First metatarsophalangeal extension

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	3783.7002	1261.2334	8.51	0.0001
Error	73	10821.2868	148.2368		
Total	76	14604.9870			

J.13. Ankle dorsiflexion

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	193.1726	64.3909	4.21	0.0084
Error	73	1116.9053	15.3001		
Total	76	1310.0779			

J.14. Subtalar range of motion

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	889.0566	296.3522	6.66	0.0005
Error	73	3249.6447	44.5157		
Total	76	4138.7013			

J.15. Hip range of motion

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	4834.5072	1611.5024	7.86	0.0001
Error	73	14968.9474	205.0541		
Total	76	19803.4545			

J.16. Rearfoot neutral

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	5.3485	1.7828	0.11	0.9531
Error	73	1166.3658	15.9776		
Total	76	1171.7142			

J.17. Midtarsal neutral

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	11.1554	3.7184	0.15	0.9282
Error	73	1787.8316	24.4908		
Total	76	1798.9870			

J.18. Hip neutral

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	1417.6904	472.3635	2.40	0.0751
Error	73	14399.8421	197.2581		
Total	76	15817.5325			

J.19. First ray length

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	0.5233	0.1744	0.79	0.5018
Error	73	16.0587	0.2200		
Total	76	16.5820			

J.20. Metatarsal declination angle

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	29.8492	9.9497	0.93	0.4322
Error	73	783.5988	10.7342		
Total	76	813.4481			

J.21. Primus varus angle

Source	DF	Sum of Squares	Mean Square	F ratio	Probability
Groups	3	36.3384	12.1128	1.27	0.2917
Error	73	697.3094	9.5522		
Total	76	733.6477			

APPENDIX K

CHI-SQUARE TABLES AND ANALYSIS OF GROUP DIFFERENCES FOR CATEGORICAL RESPONSE VARIABLES

K.1. Type Diabetes

Group	Type I	Type II	Total
U1MTH	4	15	19
UOTHER	5	15	20
DMCONTROL	9	10	19
Total	18	40	58

Chi-square = 3.58, ($p > 0.05$)

K.1. Gender

Group	Male	Female	Total
U1MTH	7	12	19
UOTHER	11	9	20
DMCONTROL	7	12	19
NCONTROL	7	12	19
Total	32	45	77

Chi-square = 2.01, ($p > 0.05$)

VITA

James Birke is a Captain in the United States Public Health Service who is serving as Chief of the Physical Therapy Department, Gillis W. Long Hansen's Disease Center, Carville. Louisiana. His program is responsible for providing clinical care, research and training in the management of neuropathic foot problems.

He received a bachelor's degree from Adelphi University, a certificate in physical therapy from Columbia University, and a master's degree from the University of Kentucky.

He was commissioned in the Public Health Service in 1969 and has had a variety of assignments including Deputy Chief, Physical Therapy Department, U.S. Public Health Service Hospital, New Orleans, Louisiana and Chief, Physical Therapy Department, U.S. Public Health Service Hospital, Baltimore, Maryland. He has made numerous professional presentations nationally and internationally, and has published over a dozen articles on neuropathic foot care.

In the future, he hopes to continue his clinical and research activities, and plans to pursue a faculty position in a physical therapy program.

DOCTORAL EXAMINATION AND DISSERTATION REPORT

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Major Field: Kinesiology

Title of Dissertation: First Ray Joint Mobility, Pressure and Ulceration of the First Metatarsal Head in Diabetic Patients.

Approved:

Major Professor and Chairman

Dean of the Graduate School

EXAMINING COMMITTEE:

Date of Examination:

April 27, 1993