Integrated motion & pressure analysis and its application to normal foot function and diabetes related foot disease

By

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Abstract

The pathogenesis of diabetic foot ulceration has been extensively studied with attention focused on the detection of risk factors for developing a plantar ulcer. The cause of plantar ulceration is multifactorial but the primary aetiology is believed to be excessive repeated pressure particularly under the insensitive foot.

Limited joint mobility, as determined by limited passive range of motion at the ankle joint complex and 1st metatarsophalangeal joint, has been strongly implicated in the generation of high plantar pressures, which have been linked with plantar ulceration seen in diabetics with neuropathy. Although limited joint mobility and high plantar pressures have been shown to co-exist in the presence of diabetic neuropathy, a direct causal link is speculative. This thesis investigated the association between joint mobility, high plantar pressure, and diabetic neuropathic ulceration in more detail. The novel aspect of the present study is that dynamic joint mobility has been assessed using a three-dimensional (3D) motion analysis technique.

Development work was undertaken to establish the feasibility of whether a commercially available electromagnetic tracking system could be applied to study 3D joint movement at the ankle joint complex. Reliability and repeatability experiments were undertaken before the generation of a large database of normative values.

Dynamic joint motion and plantar pressure measurements (using an in-shoe device) were undertaken in a sample of convenience in 3 diabetic groups and a non-diabetic reference group. The study groups consisted of

1) Diabetics with no history of foot ulceration and no clinical evidence of neuropathy
2) Diabetics with no history of foot ulceration but with clinical evidence of neuropathy
3) Diabetics with an active or previous history of plantar intrinsic ulceration
4) A non-diabetic reference group matched for age, taken from the normative database.

Diabetic patients were followed-up for 12 months and development of new ulceration was noted.

No statistically significant differences were found between the study groups for 3D dynamic motion profiles at the ankle joint complex or the 1st metatarsophalangeal joint between the study groups. Furthermore, no statistically significant relationship was found between joint movement data at the ankle joint complex and the peak pressure or pressure time integrals in any of the four study groups. No relationship was found between the passive and dynamic range of motion at the ankle joint complex or the 1st metatarsophalangeal joint. The findings of the present study do not support the current theory that people with a limited passive range of movement will have a limited dynamic range of motion.

Since the present study did not find a relationship between dynamic joint mobility at the ankle joint complex and plantar foot pressures, it is unlikely that there is a cause and effect relationship between limited dynamic movement and the generation of high plantar pressures, with subsequent ulceration in the diabetic neuropathic foot. This suggests that other factors not examined in this study, for example plantar soft tissue properties or the effect of shear forces, may be more important in the pathogenesis of foot ulceration.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

The aim of this chapter is to provide a brief overview of the thesis and to highlight the importance and relevance of the subject matter.

1.1 Relevance

Lower limb amputation represents one of the most devastating end points of complications related to diabetes. Despite extensive work in the area of prevention of ulceration and amputation, diabetes remains to be the leading cause of non-traumatic amputation. Many aetiological pathways have been identified in the formation of diabetic foot ulceration and amputation. A major component in many ulcers is neuropathy. More recently the importance of biomechanics as a means to understand the development and treatment of ulcers has been highlighted. Changes in foot structure, function and gait style associated with diabetic neuropathy have been identified and offer an explanation as to why some patients ulcerate and others do not.

Identification of the foot at risk is one of the main corner stones for the prevention of foot problems. The American Diabetes Association highlight peripheral neuropathy, altered biomechanics, peripheral vascular disease and a history of ulceration or amputation as the major foot related risk conditions (American Diabetes Association 2000). Therefore screening for clinical evidence of neuropathy, circulatory impairment, biomechanical abnormality and the integrity of the skin should be included in screening pro forma.

Assessment for peripheral neuropathy and vascular disease is widely incorporated into screening assessments. Standardised protocols and measurement techniques have been developed and the prognostic value of these measures for determining ulceration risk has been established. Assessment of foot structure is included in many screening programs, identification of digital deformities, bony prominences and Charcot deformities noted for increased ulceration risk. Limited mobility at the ankle, subtalar and first metatarsophalangeal joints has been found in patients with diabetes

and have been linked with high plantar foot pressures and ulceration. Measurements of joint mobility during screening are taken using goniometers, however, literature has shown that taking joint measurements in this way is subject to high errors.

Assessment of foot biomechanics and gait is seldom included in screening programs. As a result there is little quantitative evidence to link the well-accepted biomechanical theory to common foot pathologies and ulceration in patients with diabetes. Subjective assessment of gait occurs in clinical practice, however, the reliability and value of subjective assessment remains unclear. Despite the importance of gait in the aetiology of ulceration, there are relatively few studies in the area, mainly due to arduous methods involved in performing motion analysis.

In recent years the technology has developed vastly reducing the cost, time and technical expertise needed to undertake motion analysis. There is now the opportunity use this technology in the clinical setting as opposed to the research environment. This should increase our understanding as to the role that gait has on the development of ulceration in patients with diabetes with a view to identifying the risk factors which are linked to ulceration so that in future these could be incorporated into foot screening programs.

1.2 Purpose

There have been few studies examining the role that foot function has on the development of ulceration in patients with diabetic neuropathy. Plantar pressure measurement has been used extensively as an indirect measure of foot function; however, many of the assertions made from pressure measurement about foot function have yet to be validated. The purpose of this study is to investigate the differences in foot function between patients with diabetes compared to non-diabetic controls. Three-dimensional analysis of motion at the ankle joint complex will serve as a direct measure of foot function and will be combined with pressure measurement. Diabetic patients both with and without ulceration will be included in the study so that the relationship between foot function and ulceration can be investigated. The hypotheses generated for this study are presented in section 2.7, following the review of the literature material used in the generation of the hypotheses. The aims and objectives of the study will also be presented.

CHAPTER 2

LITERATURE REVIEW

2.1 Diabetes and foot disease

The aim of this section is to present literature on the prevalence, cost and ramifications of diabetes and its complications, with special emphasis on diabetes related foot problems.

2.1.1 What is diabetes?

Diabetes mellitus is one of the most common endocrine diseases in all populations and in all age groups (Mandrup-Poulsen 1998). It is a syndrome which is characterised by high levels of blood glucose resulting from inadequate insulin secretion, impaired insulin action or both (National Institute of Diabetes and Digestive and Kidney Diseases 1999).

Diabetes is classified into two main types: type 1 and type 2. Between 5-10 percent of people diagnosed with diabetes have type 1, which most commonly occurs during childhood or the teenage years. Type 2 diabetes accounts for 90-95 percent of all diagnosed cases and usually occurs after age 40. Other less common types of diabetes can result from surgery, drugs, infections, specific genetic syndromes and other illnesses. These together account for 1-2 percent of all diagnosed cases. Diabetes can occur during pregnancy and is known as gestational diabetes and develops in 2-5 percent of all pregnancies. The diabetes disappears when the pregnancy is over, however, it is thought that there is a greater probability of developing type 2 diabetes in later life (National Institute of Diabetes and Digestive and Kidney Diseases 1999).

2.1.2 Prevalence of diabetes

2.1.2.1 Worldwide prevalence of diabetes

Diabetes is one of the most serious challenges to health care providers worldwide (Mandrup-Poulsen 1998). The worldwide prevalence of diabetes is expected to double between 1994 and the year 2010. It has been estimated that by the year 2010, diabetes will affect 239 million people worldwide (Mc Carty et al. 1994). The regional projections of the prevalence of diabetes in the year 2010, are shown in Figure 2-1. The geographical area highlighted in white represents Europe, which has a high estimated prevalence compared other areas for example North America and Canada, which represent a larger area in terms of square miles but has a lower estimated prevalence of diabetes.

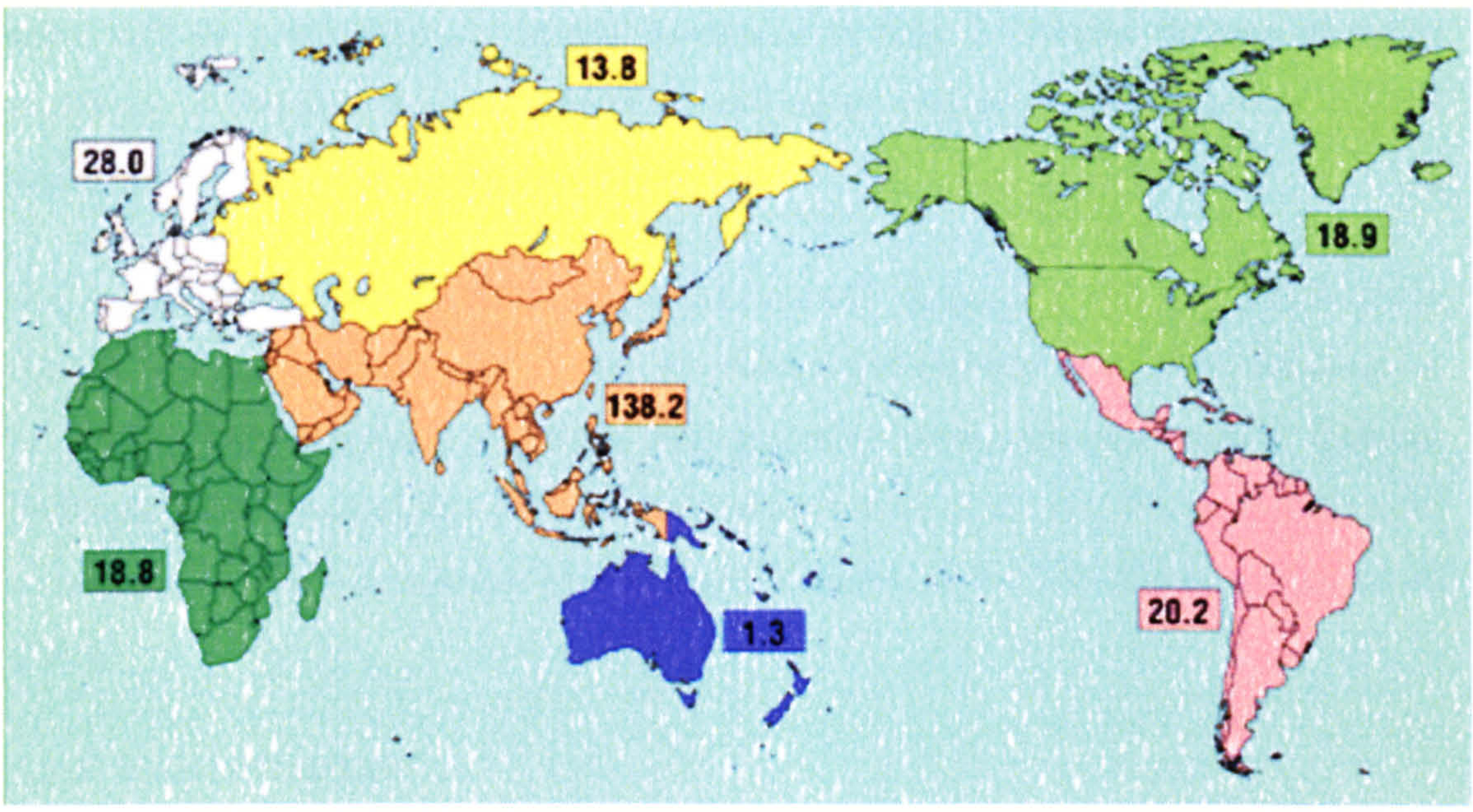


Figure 2-1: Regional projections of the prevalence of diabetes in year 2010, expressed in millions. ***Taken from McCarty D. Zimmet P. Diabetes 1994 to 2010: global estimates and projections 1994.***

2.1.2.2 Prevalence of diabetes in the United Kingdom.

It has been reported that the overall prevalence of diabetes in the UK is three percent (Fox et al. 1999). Diabetes has been reported to be three to five times more common among people of African Caribbean and Asian origin living in the UK (Mather et al. 1985; Simmons et al. 1991). The number of people, who are diagnosed with diabetes each year in the UK, has been estimated to be over 100,000. This equates to one person being diagnosed with diabetes every five minutes (Gatling et al. 1998). A recent report by the Audit Commission says that diabetes now affects one in 30 people in England and Wales, including one in four Asian men over 60 years (Audit Commission 2000).

The prevalence of diabetes increases with age and the current estimate of the prevalence of diabetes in persons aged over 65 years living in the UK is approximately ten percent (Audit Commission 2000). The Audit Commission predicts that the number of diabetic patients in the UK may double to three million by the year 2010. It is anticipated that there will be a dramatic rise in the prevalence of type 2 diabetes because of rising incidence of obesity, an aging population and recent changes in the diagnostic criteria which lowers the threshold for defining diabetes.

It is estimated that over 1 million people are currently diagnosed with type 2 diabetes in the UK and that another 1 million may be undiagnosed (Forrest et al. 1986; Simmons et al. 1991). The significant morbidity and mortality associated with type 2 diabetes highlighted by a recent UK report, means that now more than ever, this type of diabetes is being recognised as a major public health concern in the UK (Diabetes UK et al. 2000).

2.1.3 Cost of Diabetes

Diabetes is a disease, which has major long-term implications on both the individual and the nation as a whole. Adults with diabetes have an annual mortality of about 5.4% (double the rate for non-diabetic adults), and their life expectancy is decreased on average by 5-10 years (Donnelly et al. 2000). The treatment of diabetes and its complications has been estimated to take up 4-5% of the total health care expenditure in the UK (Leese 1992). The costs associated with diabetes can be very difficult to ascertain. The British Diabetic Association (BDA) categorise the costs into three groups: direct costs, indirect costs and intangible or psychological costs (British

Diabetic Association 1995). Direct costs include the costs associated with the detection and treatment of diabetes and its complications. Indirect costs relate to the loss of productive output caused by sickness absence, early retirement and premature mortality. The other costs are non-financial outcomes of the condition including anxiety, pain, suffering and loss in the quality of life. Few studies have been carried in the UK which have mainly concentrated on the direct costs associated with diabetes, as there are inherent difficulties associated with calculating the indirect costs.

The T^2ARDIS survey attempted to examine the "big picture" associated with type 2 diabetes. The objective of the survey was to estimate the total costs of all patient care and to ascertain the impact that the disease has on quality of life.

The Big Picture

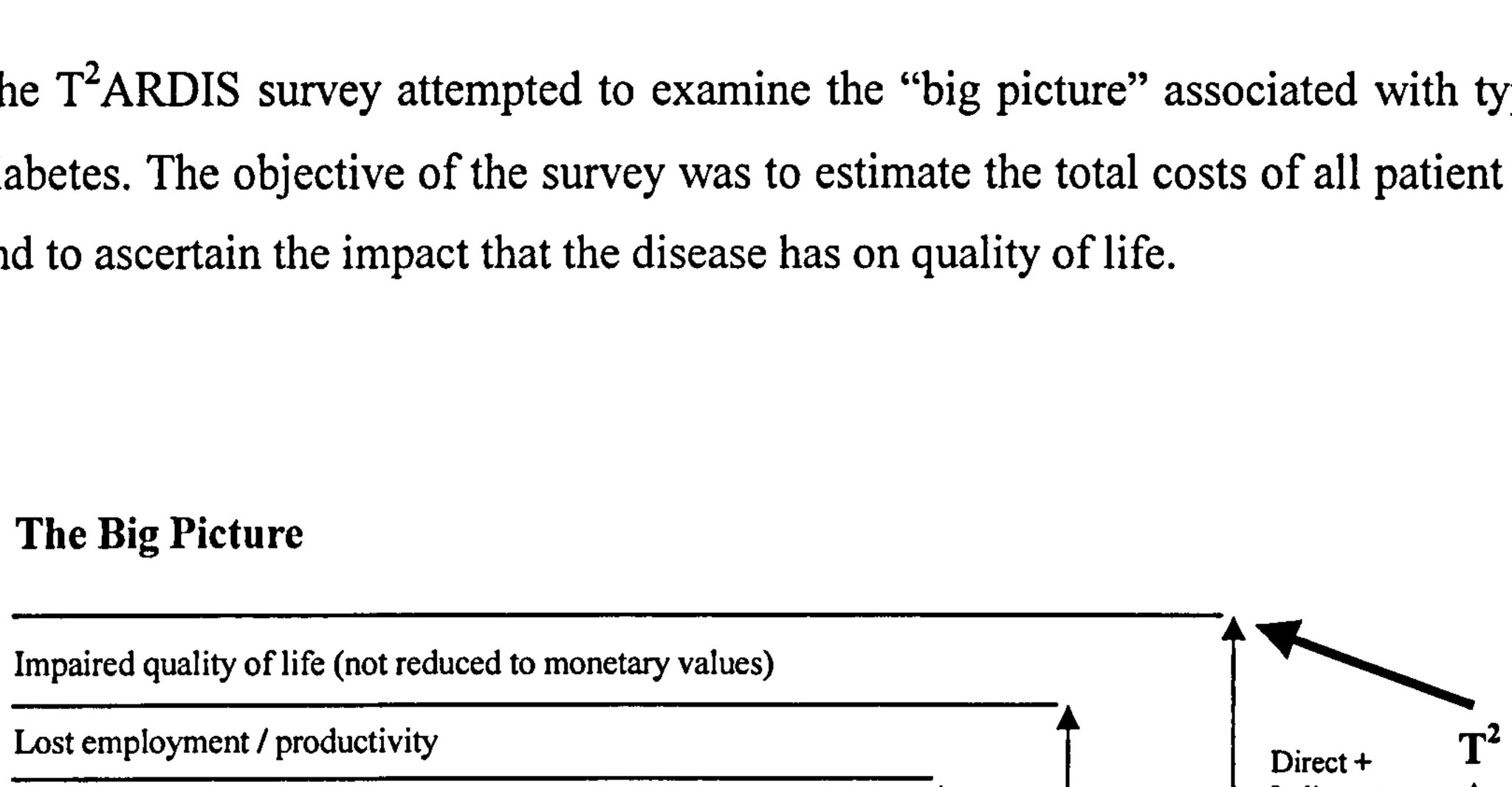


Figure 2-2: A diagrammatic representation of the main components of the costs assessed by theT^2ARDIS survey. ***Taken from T^2ARDIS – the survey abstract, British Diabetic Association, Annual Professional Conference.***

The financial cost associated with type 2 diabetes to the NHS is estimated to be two billion pounds and an additional 36 million pounds is spent on related social services and private health costs (Diabetes UK et al. 2000). The majority of diabetes care is provided in general practice (King's Fund 1996), however, it is estimated that 42% of the overall expenditure on diabetes is spent on inpatient care. The high cost is caused largely by the treatment of the complications associated with diabetes, which is dominated by in-patient care. The T^2ARDIS report found that people with the type 2 diabetes were two to three times more likely to be admitted to hospital than their demographic peers and would stay an average four times as long. Another report also states that people who had diabetes related complications were five times more likely to enter hospital than those people who had diabetes alone (Diabetes UK et al. 2000).

2.1.4 Complications of diabetes

Diabetes can affect nearly every organ system of the body and is the most common cause of blindness among working age adults, the leading cause of end stage renal failure and non-traumatic lower limb amputation. Diabetes was the 7th leading cause of mortality in the United States in 1996 (Centers for Disease Control and Prevention 1999). Persons with diabetes have a reduced life expectancy than their non-diabetic peers and are at an increased risk of developing heart disease, peripheral vascular disease and having a stroke.

2.1.4.1 Lower limb amputations

Foot disease is considered to be the most common complication of diabetes leading to hospitalisation (Kozak et al. 1984) and in the UK more than half of the bed occupancy of diabetic patients are due to foot problems (Waugh 1988). People with diabetes have a 15 times higher risk of lower extremity amputation (LEA) than individuals without diabetes (Most et al. 1983). It is estimated that diabetes accounts for between 20 and 45% of all amputations in the UK (Connor 1987).

Diabetic amputation rates are reported to increase with advancing age (Humphrey 1989; Most et al. 1983). In one study, patients with diabetes aged 65 years or older accounted for 61% of all diabetes related LEAs (Miller 1985). The same study also reported that the extent of amputation increased with age, with a shift from toe amputations to below or above knee amputations

Several studies have shown that there is a higher risk for amputations among males (Reiber et al. 1995). The risk of an amputation is approximately two fold, and is reported to be more pronounced in younger male diabetic patients (Centers for Disease Control and Prevention 1991).

Racial and ethnic differences in amputation rates have also been reported. It is generally accepted that there is a higher rate of diabetes related amputations in nonwhite races compared to white race. One study reported that black diabetic patients had an amputation rate 2.3 times greater than white diabetic patients (Most et al. 1983). The increased prevalence of LEA in these races may be due to socioeconomic and health care factors, which the population-based findings were not able to control (Reiber et al. 1995). In the UK it has been shown that rates of LEA in individuals with diabetes of Asian ethnic origin, were lower than that for Caucasians, after adjusting for age and sex. The rates were 3.4/10 000 in diabetic patients of Asian ethnic origin compared to 14.2/10 000 in Caucasian diabetic patients (Gujral et al. 1993). It has also been noted that there is a lower rate of neuropathic and/or ischaemic ulceration in patients of Asian ethnic origin living in the UK. The lower rate of ulceration of may be due to differences in joint mobility and may be a consequence of cultural differences, leading to better self-care foot practices. A further explanation may be that many diabetic patients of Asian origin may die from ishaemic heart disease before ulceration and the need for amputation develops (Chaturvedi et al. 2002).

2.1.4.2 Consequences of an amputation

Amputation represents the most devastating endpoint of all diabetic foot disorders. The long-term prognosis for the diabetic undergoing an amputation is poor. The mortality rate among diabetic individuals following an amputation, varies considerably between countries and is dependent on the amputation site (Reiber et al. 1995; Reiber 2001b). Many studies have examined the death rate after an amputation. Survival rates are approximately 50% for the following 3 years and about 40% during the 5 years after the amputation (Palumbo et al. 1985).

It is very common for a patient to require successive amputations, after an initial LEA, of the same limb or of the contra-lateral limb. Levin stated that out of 485 patients studied, 42% required an amputation of the opposite leg in the first three years following the first amputation and 56% in 3 to 5 years (Levin et al. 1998). The overall

cost of an amputation is difficult to assess, many figures exclude the cost associated with rehabilitation, loss of future earning power and an increased reliance on social services (Mackey et al. 1986). Most of the work on the economic impact of diabetic foot problems has centred on the estimation of direct health care costs, the costs involved with the identification, treatment and care of the patients with these problems (Williams 1994). Cost of care estimates for lower limb amputations in the US in 1992 ranged from $24,000 to $27,000 and from $14,500 to $21,500 for rehabilitation (Reiber et al. 1995).

The emotional and social cost, both to the patient and their families, cannot be measured easily. The morbidity associated with a LEA is dependent on the site of amputation. With a major LEA there is a loss of independence due to functional impairment. This has an impact on both the quality of life of the patient and their family and increases the economic burden on the individual, the health care service providers and social services.

2.1.4.3 Foot Ulceration

It is estimated that 15% of all diabetic patients will develop an ulcer of the foot or ankle at some time during their lifetime (Palumbo et al. 1985). It has been estimated that 4-24% of people with diabetic foot ulcers will require a lower limb amputation (American Diabetes Association 1999). In the United States it has been reported that foot ulcers precede 85% of amputations in people with diabetes (Pecoraro et al. 1990). The estimated cost for chronic skin ulceration in the United States in 1986 was $150 million, this figure represents1.3 percent of the total direct costs spent on diabetes as a whole in that year (Reiber 1992).

In a population study of 1077 patients with diabetes, 7.4 percent had active or previous history of foot ulceration (Walters et al. 1992). Currie and associates studied the frequency and in-patient care costs for peripheral vascular disease, neuropathy, foot infection and foot ulceration in people with diabetes (Currie et al. 1998). The highest average length of stay was for chronic ulceration of the skin. More than 75% of the total bed days used for patients with diabetes were for chronic skin ulceration and other peripheral vascular disease. From this data Williams estimated that the annual number of admissions for foot ulceration in people with diabetes in the UK was around 115000 (Williams et al. 1999).

2.1.4.3.1 Consequences of foot ulceration

The prognosis for diabetics with foot ulceration is poor, in a prospective study 725 people with diabetes were followed for an average length of just under two years. The relative risk of death for those with an ulcer was 2.39 versus those without an ulcer at the start of the study (Boyko et al. 1996).

Foot ulceration has been shown to have a major negative effect on the quality of lives of both the patients and their families (Carrington et al. 1996). Ulceration has been shown to have a negative impact on social activities and psychological well-being. Limitation of activity levels as a result of the ulceration lead to a reduction in productivity, lack of earnings and a negative impact on overall health.

2.1.5. Summary

Diabetic foot problems are a major source of morbidity, the figures related to amputation and ulceration rates and their associated direct and intangible costs highlight the need to develop strategies to try and prevent the complications. The St Vincent's Declaration in 1989 aimed to reduce the number of amputations from diabetic gangrene by fifty percent. Despite much effort directed towards prevention of amputation in the last decade the incidence of diabetes related lower extremity amputations have continued to rise (American Diabetes Association 1999). There is a need for the development of strategies aimed at the prevention of foot ulceration and to improve the level of care for patients who develop ulceration. A thorough understanding of the different mechanisms by which ulceration occurs is needed in order to achieve this aim.

2.2 Diabetic foot ulceration.

This section provides an overview of the aetiological pathways and classification of foot ulceration. A description of risk factors associated with foot ulceration will be presented with more detailed discussion of the factors associated with plantar intrinsic ulceration

2.2.1 Aetiology of diabetic foot ulceration

Foot ulcers are one of the most costly aspects of the treatment of diabetes, in the UK. Many patients with ulceration are treated in the community and, district nurses may need to visit to redress ulcers up to three times a week. The prognosis for diabetic patients with foot ulceration is poor, it has been estimated that between four and twenty-four percent will require an amputation (American Diabetes Association 1999). Reoccurrence rates for diabetic foot ulcers are high, in one study 35-40% of patients reulcerated over three years and this increased to 70 percent over five years (Apelvist et al. 1993). Figures like these highlight that it is imperative that the causes of ulceration are identified and addressed by appropriate management strategies to prevent ulceration.

As medical technology has improved the understanding of the causative pathways leading to ulceration have increased. The breakdown of the diabetic foot was traditionally thought to be a consequence of peripheral vascular disease, peripheral neuropathy and infection (Boulton 2000), however, now many other factors have been identified which have been shown to play an important role in the pathogenesis of foot ulceration.

It is widely recognised that diabetic foot ulceration is the result of a combination of several risk factors. A model for causation leading to diabetic amputation and foot ulceration, has been described by Pecoraro and Reiber (Pecoraro et al. 1990; Reiber 2001a). The model is based on the concept of component and sufficient cause. A component cause in isolation will not cause ulceration (the neuropathic foot will not

spontaneously ulcerate). However, if component causes act together, they may result in sufficient cause for ulceration (trauma which is not detected due to neuropathy and results in ulceration). Alternatively, the pathway to ulceration and amputation has been described as an interaction of, predisposing, precipitating and aggravating factors (Faris 1991). Predisposing factors include neuropathy and vascular disease, precipitating factors include physical trauma and aggravating factors include infection, poor wound healing and poor compliance.

Foot ulceration then, has a multifactorial aetiology and many different pathways to ulceration have been identified. Levin in a review article compiled a comprehensive diagram to highlight major pathways in the pathogenesis of ulceration, shown in Figure 2-3 (Levin 1995). This model is generally accepted by many podiatrists working in the area of management of the diabetic foot, however, it fails to include the importance of foot biomechanics in the formation of ulceration (this area will be covered in more detail in section 2.4 and 2.5).

2.2.2. Classification of ulceration

Foot ulceration can be classified in a number of different ways, by aetilogy, location, the extent of ulceration or by the presence/absence of infection. A widely used classification system divides ulceration into, purely neuropathic, purely ischaemic or neuro-ischaemic.

Neuropathy is regarded as the most important and most common complication of diabetes leading to foot ulceration (Levin 1995). A large percentage of ulcers seen in diabetic clinics and/or in the community are neuropathic in origin. In one study neuropathy was present in 60% of all patients with foot ulcers and was cited to be the major contributory factor (Gavin et al. 1993). A similar percentage (62%) of neuropathic ulcers was reported by Edmonds, however, Boulton and associates found neuropathy to be the major aetiological factor in 87% of new foot ulcers (Boulton et al. 1986; Edmonds 1987).

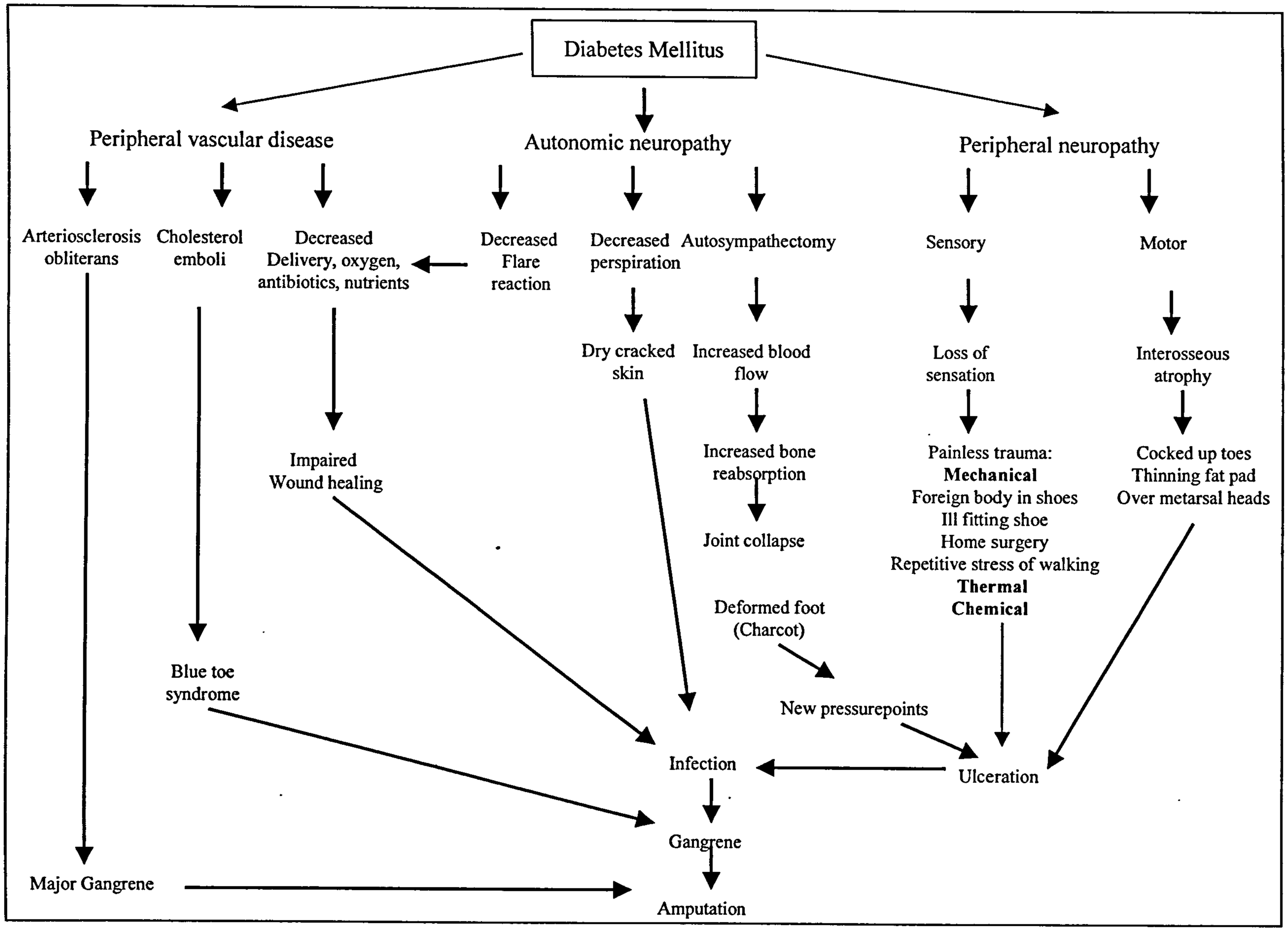


Figure 2-3: Pathogenesis of diabetic foot lesions. *Taken from, Levin M.E. (1995). Preventing amputations in the patient with diabetes. Diabetes Care, 18; 10: 1383-1394.*

Peripheral vascular disease has long been implicated in the formation of diabetic foot ulceration, and is regarded as a major contributory factor in the pathogenesis of ulceration and amputation (Pecoraro et al. 1990). The percentage of ulceration due to pure ischaemia has been reported to be between 7 and 20% (Thomson et al. 1991; Edmonds 1987). Although peripheral vascular disease has long been implicated in the formation of diabetic foot ulceration, neuropathy should be emphasized as the major component in the pathogenesis of ulceration and ischaemia considered a secondary factor (Cavanagh 1999). A combination of neuropathy and vascular disease can result in ulceration. This type of ulceration (neuroischaemic) is where both components are cited to be equally as important in the formation of the ulcer. Ulceration of mixed origin has been reported to be between 20 and 45% (Gavin 1993; Thomson et al 1991).

Ulceration can also be broadly defined as extrinsic or intrinsic ulceration. Extrinsic ulceration is the result of injury due to external pressures or trauma. This type of ulceration commonly results from wearing ill-fitting footwear or results from injuries sustained during barefoot walking (standing on a nail etc). Effective patient education and appropriate footwear should in theory be able to prevent extrinsic foot ulcers. Therefore the greater challenge for clinicians is the prevention of intrinsic ulceration. This type of ulceration occurs on the plantar surface of the foot and is the result of normal levels of pressure experienced under the foot during daily activities. This level of pressure would not normally cause ulceration, but due to loss of protective sensation, leads to excessive tissue damage and subsequent ulceration.

Intrinsic plantar foot ulcers usually develop as the result of repeated moderate stress on the foot, which due to lack of protective sensation goes unnoticed by the patient, although there are no prospective studies to support this. At the sites of repetitive stress, areas of callus develop, which can increase the local pressure by around 30% (Masson et al. 1998). The ulcers normally occur at the site of maximum pressure and are often found over the metatarsal heads and on the plantar surface of the hallux. An interaction between altered sensation, biomechanical abnormalities and mechanical trauma is the most common pathway to developing intrinsic foot ulceration.

2.2.3 Risk factors associated with diabetic foot ulceration

The early recognition and management of risk factors for ulcers and amputations can prevent or delay the onset of these adverse outcomes (American Diabetes Association 2000). Risk identification is fundamental for effective preventative management and the best method is to identify and manage risk factors before foot ulceration occurs. There are a number of factors that have been implicated in the formation of diabetic foot ulceration which are listed in the Table 2-1.

Table 2-1: List of potential and well accepted (bold type) risk factors for foot injury and amputation. ***Taken from Cavanagh and Ulbrecht 1991. Biomechanics of the diabetic foot: A quantitative approach to the assessment of neuropathy, deformity and plantar pressure.***

Peripheral vascular disease
- **Macrovascular**
- Microvascular

Peripheral neuropathy
- **Sensory**
- **Motor**
- **Autonomic**

Foot deformity
- **Subsequently poorly fitting footwear**
- **Subsequent increased plantar pressures**

Inadequate footwear
Inadequate footcare
Neuropathic (Charcot) fracture
Abnormal foot function
Impairment of vision
High activity level
Infection
Hyperglycaemia
- **Presumed cause of neuropathy**
- **Presumed cause of macrovascular disease**
- **Cause of connective tissue glycosylation**
- **Possibly cause of impaired wound healing**
- **Cause of hypercoagulability**

Race
Gender
Duration of diabetes
Previous ulceration
Previous amputation

A full discussion of each risk factor in Table 2-1, is beyond the scope of this thesis and can be found elsewhere (Boulton 2000; Boulton 2001; Levin 1995; Levine 1993). Some of the most common pathways to diabetic foot ulceration are illustrated in Figure 2-4. This section will focus on the risk factors associated with the development of intrinsic foot ulceration from the literature; neuropathy, limited joint mobility, foot deformity, callus formation and a history of previous ulceration / amputation.

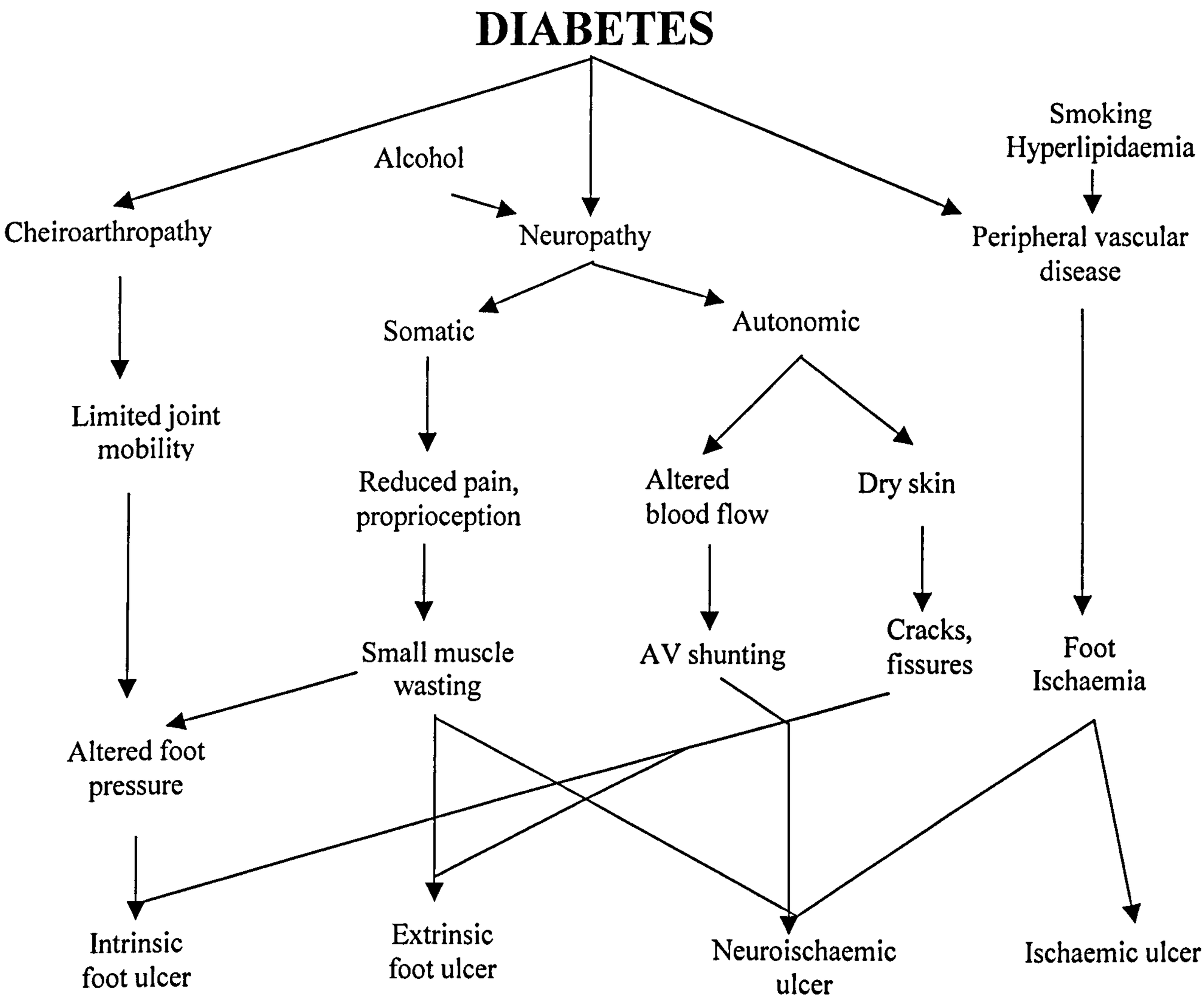


Figure 2-4: Pathways to foot ulceration in diabetic patients. ***Taken from Boulton A.J.M. (1992). Peripheral neuropathy and the diabetic foot. The Foot, 2:67-72.***

2.2.3.1 Diabetic neuropathy

Diabetes is the most common cause of neuropathy in the western World. A number of different types of neuropathy occur in diabetes and they include manifestations in the somatic and/or autonomic parts of the peripheral nervous system (American Diabetes Association et al. 1988). Diabetic neuropathy is classified into discrete clinical syndromes, shown in Table 2-2.

Table 2-2: Classification of the diabetic neuropathies. ***Taken and adapted from neuropathy in the diabetic foot: new concepts in etiology and treatment, Greene, D.A., Feldman, E. L., Stevens, M. (1998).***

Syndromes of diabetic neuropathy

1. **Diffuse neuropathies** (common, insidious onset, usually progressive)
    a) Distal symmetrical sensorimotor polyneuropathy
        I Acute sensory
        II Chronic senorimotor
    b) Autonomic neuropathy
2. **Focal neuropathies** (rare, sudden onset, usually transient)
    a) cranial neuropathy
    b) Radiculopathy
    c) Plexopathy
    d) Mononeuropathy / mononeuropathy multiplex
        I Entrapment neuropathy
        II Other mononeuropathies

Distal symmetric sensorimotor polyneuropathy is the most commonly recognised form of diabetic neuropathy and both sensory and autonomic neuropathy, are associated with foot ulceration. Focal neuropathies are relatively uncommon and there is no evidence to suggest that mononeuropathies lead to foot ulceration (Boulton 1994).

2.2.3.1.1 Sensory neuropathy

Sensory neuropathy is the most common of all diabetic neuropathies. The prevalence of neuropathy varies widely in the literature. This is the result of differences in diagnostic criteria and different methods used in the assessment of neuropathy. The prevalence of neuropathy has been shown to increase with age and the disease duration. Pirart found that 10% of patients had neuropathy at the time of diagnosis of diabetes. The percentage of patients with neuropathy after having diabetes for 25 years increased to 50% (Pirat 1978). The reported prevalence of chronic sensorimotor neuropathy in a UK study of 6500 patients attending diabetic clinics was 28.5% (Young et al. 1993a). In one study patients with peripheral sensory neuropathy were shown to have a sevenfold increased risk of developing foot ulcers in the following three years (Boulton et al. 1983).

2.2.3.1.2. Autonomic neuropathy

Autonomic neuropathy is considered to be an important risk factor for foot ulceration, because it results in a reduction in sweating causing dryness of the skin. Dry skin is thought to be less compliant and more likely to crack and fissure than moist skin. Once cracked there is the portal of entry for a bacterial infection. Skin conductivity/resistance measurements can be used as a valid measure to monitor the effect of systemic control on eccrine gland activity. Decreased sweating assessed by skin resistance and other methods has been shown to be strongly associated with foot ulceration (Boulton et al. 1983; Ryder et al. 1988).

2.2.3.2. Limited joint mobility

A number of studies have shown that diabetic patients have smaller ranges of movement at joints in the upper and lower extremity. This limitation of joint mobility is a presumed by product of non-enzymatic glycosylation. Limitation of movement at the ankle joint, subtalar joint and the first metatarsophalangeal joint have been demonstrated in diabetic patients and have been associated with the development of foot ulceration (Delbridge et al. 1987; Fernando et al. 1991). Limited joint mobility

and its relationship to diabetic foot ulceration will be discussed in more detail in section 2.4.

2.2.3.3 Foot deformity

Minor foot deformities, especially clawing of the toes are common in people with diabetes (Borssen et al. 1990). The long-term effects of sensorimotor neuropathy are thought to lead to a characteristic posture of the foot, which may lead to an abnormal distribution of weight under the foot (Boulton et al. 1987b). Foot deformities due to neuropathy frequently lead to ulceration (Boyko et al. 1999; Levin 1995). The key element in the linkage between foot deformity and ulceration is elevated peak pressure (Cavanagh et al. 1994). A full description of the changes in foot structure associated with diabetes and the relationship to foot ulceration will be discussed in section 2.4.

2.2.3.4 Callus

A wide variety of dermatological conditions are recognised as occurring more commonly in people with diabetes. It has been suggested that neuropathy could predispose the patient to an excessive production of plantar hyperkeratoses (Sage 1987). It has been suggested in the literature that it is more likely to be a consequence of changes in foot structure associated with diabetic neuropathy, which predispose the foot to elevated plantar foot pressures and subsequent callus formation (Cavanagh et al. 1993).

The process of non-enzymatic glycosylation of connective tissue proteins in the skin occurs in diabetes and results in changes, which make the skin thick and less flexible. These changes directly affect collagen, making it stiffer and not able to deform as quickly in response to a given load. It is more susceptible to cracking and splitting in response to rapid deformation associated with high velocity foot impact (Landsman et al. 1995). These changes may make the skin more prone to tissue damage (Faris 1991).

Callus has been shown to be strongly predictive of subsequent ulceration, retrospective analysis by Murray and associates, found plantar ulceration was 77

times more likely to occur at a site of previous callus (Murray et al. 1996). Plaques of keratin are thought to develop in response to mechanical stresses and tissue breakdown may occur underneath the plaque. Inflexibility of collagen tissue as a result of glycosylation may potentiate tissue breakdown in areas exposed to high shearing forces and may facilitate the formation of cavities and ulcers (Delbridge et al. 1985). Reduction of plantar callus has been shown to significantly reduce the mean peak plantar pressures in the forefoot by an average 29 percent (Young et al. 1992a).

2.2.3.5 Previous foot ulceration / amputation

Previous ulceration and or amputation is a leading risk factor for future ulceration as the patient has a combination of risk factors that together produce ulceration. More recently it has been suggested that altered mechanics of the scar tissue may increase the risk for ulceration. Scar tissue is not strong and is thought to be more vulnerable to the shearing forces during walking (Levin 1995). It has been suggested that the scar tissue is less able to dissipate mechanical stress and transmits large concentrated loads to the underlying softer tissue (Cavanagh et al. 2001a). Previous amputation may result in changes in the pressure distribution under the foot, resulting in excessive pressure and subsequent tissue damage.

2.3 Screening Techniques to identify the "At Risk Foot".

This section reviews the literature concerning screening techniques employed in the assessment of the foot at risk of developing ulceration, focusing on methods to assess neurological and vascular status. A description of screening methods and rationale will be presented, together with a discussion of scoring systems used in clinical practice. The literature reviewed in this section will form a basis to support the screening techniques employed in the methods section

An international working group has produced practical guidelines for healthcare workers on the management and the prevention of the diabetic foot problems. Identification of the foot at risk is one of the main corner stones for the prevention of foot problems. Recognising important risk factors and making a comprehensive assessment of the diabetic limb requires a thorough and consistent diagnostic approach. A useful evaluation will involve identification of contributory problems and assignment of relative risk (Harkless et al. 1991). The first step is to take a patient history and, an examination of the foot is of paramount importance to assess the potential for future problems. The American Diabetic Association position statement for the clinical recommendations for preventative foot care highlight peripheral neuropathy, altered biomechanics, peripheral vascular disease and a history of ulceration or amputation as the major foot related risk conditions (American Diabetes Association 2000). Therefore screening and assessment for clinical evidence of neuropathy, circulatory impairment, biomechanical abnormality and skin disorders is important to determine who is at risk for ulceration and to initiate an appropriate preventative management strategy. A number of different screening methods are used in the assessment of the diabetic foot. Standardisation of methods has not been adopted. Many specialist centres use different scoring systems to ascertain risk status. The aim of this section will review the different methods used to screen for neuropathy, peripheral vascular disease, biomechanical abnormality and scoring systems used to ascertain risk status.

2.3.1 Assessment of neuropathy

The reported prevalence of neuropathy has previously ranged from 5-80%, due to differences in diagnostic criteria and the type of population studied (Eastman 1995). Diffuse symmetrical sensorimotor neuropathy is the most common type of neurological dysfunction found in diabetics, the prevalence varies according to the diagnostic criteria used. In 1988 a joint conference of the American Diabetes Association and the American Association of Neurology adopted standardised nomenclature and criteria for diagnosis of neuropathy in diabetes. This classification recognises subclinical and clinical neuropathy. Subclinical neuropathy is defined by an abnormal electrodiagnostic test, quantitative sensory threshold or autonomic function test in the absence of clinical signs or symptoms. Clinical neuropathy is defined as signs or symptoms together, or as symptoms or signs alone plus an abnormal test results. The preferred criteria for diagnosis of distal symmetrical polyneuropathy are abnormalities in two of three areas: symptoms, signs, and quantitative sensory tests or electrodiagnostic studies (American Diabetes Association et al. 1988).

2.3.1.1 Electrodiagnostic tests

Electrophysiological techniques have the advantage of being the most objective, sensitive, specific and reproducible method to detect and localise neuropathic changes (Zeigler 1994). However, they have a relatively low specificity in detecting diabetic neuropathy and correlate poorly with clinical findings. Electrophysiological techniques measure function in the largest, fastest myelinated fibres and many small myelinated fibres and unmyelinated fibres are not assessed. The function of small unmyelinated fibres, which carry autonomic signals and awareness of pain are not reflected in any of the routine electrophysiological tests (Guy et al. 1985). Measurements from electrophysiological tests do not directly predict symptoms or signs of neuropathy (Cavanagh et al. 1991b). A detailed examination of neurological function using these methods is not always necessary or feasible and is not used for routine screening for diabetic neuropathy

2.3.1.2 Quantitative sensory testing

The Peripheral Neuropathy Association recommend detection of thresholds of touch pressure, vibration, coolness, warmth, heat pain, cold pain and mechanical pain to determine cutaneous sensation (Peripheral Neuropathy Association 1993). The advantages of quantitative sensory testing are that the procedures are relatively simple, non-invasive and non-aversive and can be valuable in screening large numbers. Variables such as room and skin temperature and skin condition must be controlled and all devices used should be calibrated frequently to ensure consistency (Kahn 1992b). The instructions given to patients should be simple and clear and procedures of testing standardised. A major limitation associated with this type of testing is that the results are very much dependent on the cooperation and alertness of the patient which can result in high intra-individual variability. Adequate standardisation of testing (forced choice methods) may be time consuming and lead to decrease in concentration of patient and diagnostic error.

2.3.1.2.1 Touch Pressure

The Semmes-Weinstein filaments are used for evaluating touch-pressure sensation. The system consists of a series of graded, pressure sensitive nylon filaments of increasing calibre that buckle at a reproducible stress and can measure a patient's cutaneous pressure perception threshold. The filaments are pressed perpendicularly onto the skin until buckling occurs. The thicker the filament the greater the force required for buckling.

The Semmes-Weinstein filaments are a simple, inexpensive and effective screening method for the detection of loss of protective sensation. The vibration perception threshold and Semmes-Weinstein filaments have been shown to be the most effective methods of measuring sensory deficits in the hand and foot (Perkins et al. 2001). Birke and Sims found the 4.17 filament represented the approximate lower limit of normal sensation. To characterise the insensate foot they recommended the use of the 4.17, 5.07 and 6.10 filaments, bending with 1, 10, and 75g of force respectively (Birke et al. 1986).

Pressure perception has been shown to be strongly associated with foot ulceration. Sosenko and associates found a strong association between impaired pressure perception and foot ulceration in patients with diabetes (Sosenko et al. 1990). Boyko and Rith-Najarian identified increased ulcer risk in patients unable to detect the 5.07 filament (Boyko et al. 1999; Rith-Najarian et al. 1992). Monofilament testing has been shown to be an important prospective predictor of foot wounds (Litzelman et al. 1997). The filaments have been shown to be reliable in screening for patients at risk of foot ulceration, (100% sensitive and 77.7% specific) and found to be more sensitive in identifying patients with foot ulceration than biothesiometry (Kumar et al. 1991).

A standardised methodology for the use of monofilaments has not been widely adopted. The sensitivity and specificity of monofilament testing for the prediction of neuropathy is highly dependent on the methodology used (McGill et al. 1998). It must also be noted that not all commercially available 10g monofilaments are the same. Booth and Young found differences in the accuracy and durability of commercially available monofilaments (Booth et al. 2000). Continual use of a filament can lead to reduced ability to accurately detect peripheral neuropathy. The filament can become less rigid and buckling can occur at a decreased force therefore becoming potentially hypersensitive in the prediction of loss of protective sensation. This may have important ramifications on finite resources if patients with normal sensation are incorrectly identified as having loss of sensation, limited resources may be used inappropriately (Booth et al. 2000; McGill et al. 1998; Yong et al. 2000).

Despite the limitations outlined monofilaments are inexpensive and are simple to use. They have been reported to provide the quickest and best method of confirming and measuring loss of protective sensation. In the clinical setting sensory examination with a 5.07 monofilament remains to be the single most practical measure of risk assessment (McNeely et al. 1995).

2.3.1.2.2 Vibration

Measurement of vibration perception threshold is widely used as a sensitive and reproducible test for assessing peripheral large myelinated fibres. Deficit in this

function tends to correlate with, but often preceeds abnormality in tendon reflexes, light touch and position sense (American Diabetes Association et al. 1988). A number of instruments are commonly used for detection of vibration perception thresholds (VPTs), including the biothesiometer, neurothesiometer and vibraton. It has been shown that VPTs correlate significantly with peripheral nerve function, demonstrated by nerve conduction parameters from the sural nerve and clinical scoring systems of neuropathy status (Franklin et al. 1990). Determination of the VPT with the neurothesiometer has been shown to be less variable than with the vibraton (Bril et al. 1997) and has been shown to compare favourably with the biothesiometer (Young et al. 1993b).

Raised vibration perception thresholds are closely associated with the presence of foot ulceration (Boulton et al. 1983; Boulton et al. 1986; Guy et al. 1985). An investigation found that VPT was the most discriminative test for neuropathic changes in the lower extremity, and a VPT of greater than 35 volts (outside the normal limits of vibration perception for the study group) was significantly associated with the presence of neuropathic foot ulceration (Boulton et al. 1983). In a prospective study VPT was able to predict diabetic patients at increased risk of developing foot ulceration. A VPT of greater than 25 volts carried a sevenfold risk of foot ulceration compared with a VPT of less than 15 volts (Young et al. 1992b). It has been shown that VPT increases with age and there is a positive correlation between duration of diabetes and an increase in VPT (Young et al. 1993b).

Wide variability in VPT at different sites in the same subject has been documented, which reduces the diagnostic value of this variable. Williams and associates found differences of up to 30% existed between contralateral and ipsilateral sites in diabetic subjects with a single observer (attempts had been made to standardise the technique) (Williams et al. 1988). Differences in the tissue characteristics locally and patchy asymmetric neuropathy were proposed as possible explanations for the wide variability in VPT among sites. The study concluded that readings at single or unilateral sites may be unrepresentative and highlighted the need for bilateral examination in conjunction with other neurological tests and clinical observation to determine if neuropathy is present. It has been shown that the assessment of vibration perception has limited value when screening for neuropathy in elderly people. Loss of vibration perception has been shown to be marked in the elderly (people over 70

years) and therefore makes the distinction between reduced perception as a result of the normal ageing process and neuropathy very problematic (Thomson et al 1993). On this basis measurement of vibration perception alone for the assessment of neuropathy is not recommended.

2.3.1.2.3 Temperature

Abnormalities in cold and/or warm thresholds may constitute the earliest evidence of neurological deficit (American Diabetes Association et al. 1988). Detection of thresholds for cold and warm sensations provides a quantitative assessment of small nerve fibre function. A variety of commercial instruments are available to evaluate thermal sensitivity. Some instruments consist of two thermal plates and others have only one thermal plate. In two plate systems, one plate acts as a reference and the temperature of the other plate is varied. The smallest temperature difference between the two plates correctly identified by the patient is the perception threshold. With one plate systems the absolute maximum temperature at which the patient can detect cold and the absolute minimum temperature at which the patient can detect warm are recorded.

Impulses induced by cooling stimuli are conducted in different fibres from those concerned with the sensation of warmth (Le Quesne et al. 1991). Significant differences between hot and cold perception thresholds have been demonstrated. It has been stated that measurement of cooling thresholds will not detect minor variations in small nerve fibre function in diabetic subjects therefore assessment of warm threshold is preferred (Sosenko et al. 1987; Sosenko et al. 1988).

Thermal sensitivity has been shown to be grossly abnormal in the feet of diabetic subjects with neuropathic ulceration and Charcot joints (Guy et al. 1985). Temperature sensation is often lost in tandem with pain sensation (both small fibres), this can occur without evidence of abnormality in vibration perception thresholds or touch pressure sensation (large fibres) (Brown et al. 1984). It has been suggested that the small fibres are more susceptible to damage than large fibres (Guy et al. 1985).

2.3.1.2.4 Pain

Pain sensation is subserved by two groups of nerve fibres: the rapidly conducting, high-threshold mechanosensitive myelinated Aδ fibres responsible for sharp, "first" pain and the polymodal nonmyelinated nociceptive C fibres responsible for dull, burning pain (Le Quesne et al. 1991). A variety of types of stimulation (thermal pain, cold pain, pinch and pressure pain) have been used to determine the threshold for pain appreciation. Although information on pain appreciation in normal subjects has been obtained using elaborate psychophysical techniques, there is little information on pain appreciation in the diabetic foot. Quantitative testing for pain appreciation in the high-risk foot is complex, the stimulus needs to be carefully controlled so that there is no risk of causing undue trauma which could lead to serious foot injury. Quantitative testing of this type is not used for routine screening.

The pinch pain threshold has been measured in diabetic and control subjects, using a "pinchometer" on the dorsum of the foot. In some diabetic patients pain was not appreciated at one or more points, when the maximum force (2.8kg) was used. In other diabetic subjects the quality of pain was altered, patients reported they felt a dull ache rather than sharp pain appreciation. The authors interpreted the loss of sharp pain perception in some diabetic patients as being due to a loss of Aδ fibres (Le Quesne et al. 1986).

2.3.1.3 Autonomic function tests

Diabetic autonomic neuropathy may manifest as dysfunction of several different organ systems. The diagnosis of autonomic neuropathy was first based on symptoms but now is dependent on various objective reflex tests. Autonomic function testing is used to document diabetic autonomic failure which can be divided into two categories; autonomic failure, in which there is a structural lesion of the peripheral autonomic neuron and functional autonomic failure in which no structural lesion occurs (Kahn 1992c).

A number of objective measurements have been developed to evaluate autonomic function. Typically testing is performed to detect cardiovascular abnormalities, motor disturbances of gastrointestinal tract, genitourinary tract disturbances, sudomotor sympathetic function and endocrine tests for functional autonomic failure. The tests measure end organ response to activation of the neural reflex arcs and can be non-invasive or invasive. Some tests whilst useful for physiological studies are impractical for use in a diabetic clinic. Invasive tests are not suitable for routine screening or monitoring progression of neuropathy. Non-invasive tests have been shown to be reliable, reproducible and correlate with other tests of peripheral nerve function. Cardiovascular tests (for example measurement of change in blood pressure on standing) are generally accepted as the 'gold standard' for assessment of autonomic dysfunction in diabetics, however tests in other systems can be useful.

Evidence regarding the relationship of autonomic neuropathy and foot ulceration has been conflicting. Corbin and associates found no clear association between autonomic neuropathy and the degree of abnormal blood flow in diabetic patients with and without symptoms of neuropathy and recurrent foot ulceration (Corbin et al. 1987). Young and associates found that peripheral somatic electrophysiological tests were significantly higher in patients with foot ulceration compared to diabetic patients without ulceration, whereas autonomic function tests were not (Young et al. 1986). McFadden and associates compared peripheral sensory nerve function and cardiac autonomic reflexes in diabetic patients with foot ulceration compared to diabetic controls, using discriminant analysis they concluded that an abnormal autonomic score was the best predictor of foot ulceration in diabetic patients (Mc Fadden et al. 1991). It has been noted that the cardiovascular autonomic function tests assess only central autonomic function and do not necessarily correlate with autonomic denervation in the feet (Ryder et al. 1990).

A test for peripheral autonomic denervation in the feet has been developed. The test measures the sweating response in the skin to intradermally injected acetylcholine. The sweating response needs an intact sympathetic nerve supply to the sweat glands. If there is denervation of the sweat glands there would not be a sweating response to the acetylcholine. Using this test Ryder and associates tested 19 patients with a history of foot ulceration, they found that all but one patient with a foot ulcer had peripheral autonomic denervation of the feet and suggested that autonomic

neuropathy was a major factor in diabetic neuropathic foot ulceration (Ryder et al. 1990). The mechanism by which autonomic neuropathy can result in foot ulceration is that decreased sweating as a consequence of autonomic neuropathy can lead to cracking and fissuring of the skin, which can progress to ulceration. Although a high proportion of patients with foot ulceration have been shown to have autonomic neuropathy, this does not imply cause and effect.

2.3.1.4 Clinical Measures of neuropathy

Clinical measures are defined as the medical and neurological history and a physical examination (Kahn 1992a). These measures are used to classify and grade clinical neuropathy and are used as a secondary outcome measure in clinical studies. Most practitioners in the absence of quantitative neurological testing equipment will use clinical measures as a first line screen to determine if further investigation is warranted. Directed scored histories and physical examination of the sensory, motor and autonomic systems are considered as clinical measures which show a strong correlation with physiological and morphological abnormalities (Kahn 1992a). Clinical measures are relatively subjective and are dependent on the aptitude of the examiner. Limited reliability and reproducibility of clinical measures and the lack of sensitivity to change restrict their use as primary outcome measures. They form an essential part of clinical studies but other more objective tests are required in addition to purely clinical measures.

2.3.1.4.1 Assessing symptoms

Several different symptom questionnaires have been developed, some are administered by health care professionals and others are patient administered. Reproducibility of questionnaires is generally enhanced if the symptoms are classified as present or absent, rather than attempts to grade severity of symptoms.

The Neuropathy Symptom Score has items related to motor, sensory and autonomic neuropathy, and symptoms are scored in a binary fashion. The Neuropathy Symptom Profile is a true or false questionnaire with the questions grouped into sub-scales to reflect motor, sensory or autonomic dysfunction (Dyck et al. 1986). A diabetes symptom checklist has been developed for type 2 diabetes, this consists of 34

questions to measure the occurrence of physical and psychological symptoms related to type 2 diabetes and its complications (Grootenhuis et al. 1994). Patients are asked how often symptoms have occurred during the past month, the total score gives a value for neuropathic pain and sensory alteration. When performed by expert examiners, the reproducibility of the Neuropathic Symptom Score and the Neuropathic Symptom Profile is reported to be acceptably high (Dyck et al 1991).

It must be noted that prediction of polyneuropathy from neuropathic symptoms alone is not advocated. The San Luis Valley study considered patients to have neuropathy if they had two of the following; bilateral symptoms, bilateral absent or decreased ankle reflexes and absent or altered cold perception (Franklin et al. 1990). Using the criteria they classified 27.8% of the sample as having definite neuropathy. The percentage of the sample with a history of neuropathic symptoms was 97%. In a study by Feldman and associates they found that equal numbers of patients with and without neuropathy answered up to six relevant questions about neuropathic symptoms positively and concluded that symptoms may not always indicate underlying neuropathy (Feldman et al. 1994).

2.3.1.4.2 Physical Examination

Neurological evaluations attempt to assess the distribution and severity of motor, sensory and autonomic deficits. Clinical neurological examination of the lower limb is a fundamental part of diabetic assessment. The examination includes assessment of the sensory, motor and autonomic systems.

2.3.1.4.2.1 Sensory examination

The traditional methods of sensory examination include evaluation of pain (pin prick), touch pressure (cotton wool, monofilaments), vibration (tuning fork), temperature, reflexes, two-point discrimination and proprioception (joint position sense). The testing is performed at multiple defined sites on the lower limb, with reference testing on other sites of the body (trunk, face). Clinical practice recommendations for the standardisation of sensory examination states that the sites to be examined should include the distal toe and distal finger (Kahn 1992a). Results of sensory tests are more reproducible if classified as normal or abnormal, however, a limitation of

assessing deficits in this way is the lack of sensitivity to change once they have become abnormal. These tests are easily applied to the clinical outpatient setting for screening large numbers of patients. Pinprick, light touch sense, vibration sense and ankle reflex, are validated and shown to be adequate for use in daily practice (Valk et al. 1992; Valk et al. 1997; Valk et al. 2000). Descriptions on how to perform these tests are documented elsewhere and will not be discussed in this chapter (Cavanagh et al. 1991b; Tanenberg et al. 2001). The rationale and clinical evidence to support these methods will be briefly summarised. It must be noted that all psychophysical tests of sensory perception are dependent on patient cooperation and motivation and are open to interpretation by both the examiner and the patient and must be utilised in this context.

2.3.1.4.2.1.1. Pain Assessment

The sensation of superficial pain can be tested by pinprick using a sharp pin or neurotip (sharp and dull side). The sites to be examined should include the distal toe and distal finger. Limitations associated with this type of test include difficulty in standardising the amount of force applied to the skin. The neuropen has been developed in attempt to allow the examiner to standardise the amount of force used when applying a neurotip to the skin. The superficial pain test has been shown to have comparable sensitivity and specificity with the 5.07 monofilament and vibration perception testing (Perkins et al. 2001). Valk and associates compared bedside clinical examination with neurophysiological examination and concluded that impairment of pin prick sense was an early indicator of neurological dysfunction and was an important parameter in the clinical diagnosis of diabetic polyneuropathy (Valk et al. 1992). The superficial pain test using the neurotip can be confidently used in the annual screening for diabetic neuropathy (Perkins et al. 2001).

2.3.1.4.2.1.2 Touch pressure

Light touch sense can be evaluated by using cotton wool and testing is performed by gently touching the skin surface of the foot with a cotton wool wisp. The clinical significance of light touch sense using cotton wool has been confirmed (Valk et al. 1992). It has been demonstrated that light touch sense indicated changes associated with impaired or absent sural nerve function, which is considered to be an important

indicator of diabetic polyneuropathy. Diminished light touch sense indicated changes in sural nerve function better than vibration sense (Valk et al. 1992).

2.3.1.4.2.1.3 Vibration

The 128Hz tuning fork is commonly used to assess patient's ability to detect vibration. The inability to feel the tuning fork at the great toe carries the same significance as the inability to detect the 5.07 monofilament (Tanenberg et al. 2001). Vibration testing with the tuning fork using the on-off method involves the tuning fork being placed in contact with the patient's skin, the tuning fork may or may not be vibrating. The patient is asked to report when they feel vibration and when the vibration sensation ceases as a result of attenuated oscillation. This method is preferred over the timed method as it is much quicker to perform and the results are more valid in their interpretation. Vibration testing by the on-off method is recommended as an accurate method to predict the likelihood of neuropathy (Perkins et al. 2001).

2.3.1.4.2.1.4 Temperature

Commercial systems are available to test temperature perception, however, they are not usually available in most out-patient settings. Temperature perception is usually tested in the clinical situation by using test tubes filled with hot and or cold water. The patient has their eyes closed and is asked to identify the temperature as the test tubes are placed in contact with the skin in a random order. This method is not standardised and there is a lack of evidence to support this as a method to screen for the diagnosis of diabetic neuropathy.

2.3.1.4.2.1.5 Reflexes

The prevalence of absent ankle reflexes in the normal adult population is uncertain, however there is an increase in the absence of ankle reflexes after the age of 70 years (Bowditch et al. 1996). It has been reported that the frequency of decreased or absent ankle reflexes exceeds 5% in healthy subjects older than 50 years (Dyck et al. 1995). In screening of diabetic neuropathy the reflexes are usually classified as present, present with reinforcement or absent. The reflexes at the ankle and knee are usually

tested. The ankle reflex has been shown to be reproducible and has moderate agreement with the Semmes-Weinstein monofilament (Smieja et al. 1999). McNeely and associates found absence of the Achilles tendon reflexes to be a significant independent predictor for foot ulceration (McNeely et al. 1995).

2.3.1.4.2.1.6. Two-point discrimination

Two-point discrimination is used as an estimate of nerve fibre density, it is a complex process that involves not only the peripheral nervous system but also cerebral processing of information. A set of dividers with two dull points in which the distance between them can be altered are used for this type of test. The smallest distance of separation the patient can correctly identify is recorded at the index finger and great toe. Normal values for index finger and great toe are 2mm or less. A value of greater than 2mm is consistent with nerve fibre loss (Tanenberg et al. 2001).

2.3.1.4.2.1.7 Joint position

Joint position sense is usually assessed first at the interphalangeal joint of the hallux. If the patient is unable to detect changes in joint position at this joint, more proximal joints are tested (the 1st metatarsophalangeal joint and the ankle joint). There is no standardised procedure to test joint position sense. Most clinicians will demonstrate, moving the hallux up, down and a reference position (neither up or down). The patient is asked to close their eyes and report if they think the position of the hallux is up, down or in the reference position. It has been noted that joint position sense is preserved until late stages of neuropathy. Valk and associates found that joint position sense was normal in 96.4% of patients who had impaired sural nerve function. They found a significant difference in the number of normal findings in this patient group between joint position sense and light touch, pin prick and vibration perception (Valk et al. 1992).

2.3.1.4.2.2 Motor assessment

Symptoms of muscle weakness in the lower limb are difficult to evaluate, symptoms such as weakness, unsteadiness, falling could as easily be related to sensory loss or joint problems as to muscle weakness (Cavanagh et al. 1991b). There are usually no

symptoms associated with weakness of the intrinsic muscles of the foot. Motor assessment of the lower limb is assessed by manual muscle testing in the clinical situation. The examiner has to manually resist or break a particular voluntary movement. Although there are specific tests for almost every muscle, in the lower limb muscle strength is usually evaluated around a joint. A typical assessment involves evaluation at the hip, knee, ankle and metatarsophalangeal joints. In diabetic neuropathy muscle weakness appears late and usually involves the intrinsic muscles of the foot and the ankle dorsiflexors, more proximal muscles are only involved in severe cases (Kahn 1992a).

A number of different grading systems have been developed to test and grade muscle power. In the UK one of the most commonly used grading system is the Medical Research Council scale. In this system muscle power is graded on an eight-point scale (values ranging from zero for no movement up to five for normal power).

Manual muscle testing is useful for assessment of major deficits but has limited value for the assessment of minor decrements in motor function. The technique has poor inter and intra tester reliability. A number of mechanical devices have been developed to aid quantification of muscle strength and improve methods for manual muscle testing. The mechanical devices are still subject to large inter-tester variability attributed to differences in technique and placement of device (Soderberg 1997).

Muscular weakness of the intrinsic muscles of the feet is postulated to lead to foot deformity and increased foot pressures and an increased risk of foot ulceration. Several studies have shown an association between impairment of motor nerve conduction velocities with foot injury and ulceration (Boulton et al. 1983).

2.3.1.4.2.3 Autonomic assessment

Autonomic neuropathy can cause dysfunction in many different organ systems. Symptoms of autonomic neuropathy include disturbances in sweating, urinary and faecal incontinence, urinary retention, constipation, diarrhoea, gastroparesis, impotence and postural hypotension. A set of questions designed to reveal autonomic symptoms have been developed and are included in the direct scored histories (Cavanagh et al. 1991b).

In the lower limb decreased sweating and increased blood flow through the foot are a frequent clinical finding with autonomic neuropathy. Both can be easily detected by clinical observation (dry skin, warm foot with bounding pulses and venous distension) and graded as present or absent for a semi-quantitative assessment of autonomic function.

2.3.1.4.3 Neurological scoring systems

It has been recommended that assessment of clinical symptoms, clinical examination, electrodiagnostic studies, quantitative sensory tests and autonomic function tests should all be used to diagnose neuropathy (American Diabetes Association et al. 1988). These recommendations are not applicable to routine clinical settings and mass screening requirements have lead to development of simpler screening techniques for diabetic neuropathy.

Although clinical practice guidelines recommend annual screening for neuropathy, they are unable to recommend a specific screening modality (Perkins et al. 2001). The optimal method for the detection of neuropathy in patients with diabetes have been based on expert opinion rather than on clinical trial evidence (Perkins et al. 2001). A number of neurological scoring systems have been developed, some for specific use in detection of diabetic neuropathy. Frequently used systems include the Neuropathic Disability Score and various modified versions, the Neuropathy Deficit Score, the Michigan Neuropathy Screening Instrument, the Michigan Diabetic Neuropathy Score, the Neuropathy Impairment Score in the Lower Limbs and the Clinical Examination Score of Valk.

The Neuropathic Disability Score (NDS), was originally designed for neuropathy in general, it is a comprehensive scoring system but is difficult to perform in clinical practice on patients with diabetic foot problems (Dyck et al. 1980). A number of modified versions of the NDS have been developed to specifically assess for distal symmetrical polyneuropathy (Young et al. 1993a). The modified version of the NDS by Young has been used in a number of cross sectional and prospective studies (Abbott et al. 1998; Cabezas-Cerrato 1998; Calle-Pascual et al. 2001; Kumar et al. 1994; Pham et al. 2000) and is often used in conjunction with a simplified version of

the Neuropathic Symptom Score (NSS). The modified NDS is derived from the evaluation of ankle reflexes and vibration, pin-prick and temperature sensation at the great toe. Neurological symptoms are determined using the simplified NSS, a standardised questionnaire which considers the distribution and intensity of typical symptoms such as burning, numbness, paraesthesia, fatigue, cramping or aching.

The modified neuropathy disability score has been shown to be associated with foot ulceration (Kumar et al. 1994). Recent studies have stated that NDS was the best predictor of foot ulceration (Calle-Pascual et al. 2001; Meijer et al. 2000). The modified NDS has not been validated and although it has been identified as a predictive measure of foot complications there is no information regarding its use as a screening instrument for diagnosis of neuropathy (Meijer et al. 2000).

The Neuropathy Impairment Score in the Lower Limbs (NIS-LL) is another variation on the NDS, which has not been validated. The scoring system was developed for assessment of diabetic polyneuropathy, however, it focuses more on motor activity grading (Abbott et al. 1998; Bril 1999). The Michigan Neuropathy Screening Instrument (MNSI) has been developed as a simple screening technique for peripheral diabetic neuropathy (Feldman et al. 1994). It consists of inspection of the foot, examination of the Achilles reflex and determination of the vibration threshold. The maximum score for the MNSI is 8 and a score of 2.5 or more is considered as positive for peripheral neuropathy and further neurological examination is required. The MNSI does not recognize involvement of the autonomic nervous system or patient symptoms. It has been shown to be reproducible and reliable and has good correlation with the Michigan Diabetic Neuropathy Score (Lunetta et al. 1998). The Michigan Diabetic Neuropathy Score (MDNS) is based on an objective neurological examination (Lunetta et al. 1998). Vibratory sensitivity is determined with a tuning fork, pin-prick sensation is tested on the dorsum of hallux and the 10g filament is tested on the back of foot. Muscle strength is evaluated in the upper and lower limb and reflexes tested in upper and lower limb. A total clinical score of more than 6 is considered abnormal. This scoring system does not take into account patient's neurological symptomology.

The clinical examination score of Valk is a scoring system is based on tendon reflexes, muscle strength and sensory testing including pin prick, light touch,

vibration sense, and joint position sense. The scoring system includes an evaluation of the patient's history regarding neuropathic symptoms in the lower extremities (burning, aching or stabbing pain and paraesthesiae) (Valk et al. 1992). Scoring is dependent on the intensity and duration of symptoms and impact on daily activities. A good correlation between clinical examination and neurophysiological examination has been demonstrated (Valk et al. 1992). The scoring system has been validated and can easily be performed in clinical practice.

2.3.2 Assessment of vascular status

Peripheral arterial disease is four times more prevalent in diabetics than in non diabetics (Kannel et al. 1979). There is a substantial predispostion to premature and accelerated macrovascular disease associated with diabetes (Levin 2001; Shaw 1996). The anatomical distribution of atherosclerosis is altered in diabetics compared to non diabetics. Arterial disease in a person without diabetes usually involves the more proximal vessels (femoral, iliac and aorta), whereas diabetic patients tend to have more disease in the tibial and peroneal arteries and less in the arteries of the foot. Kumar and associates found that the absence of two or more foot pulses or a history of previous peripheral revascularisation was a significant predictor of foot ulceration (Kumar et al. 1994). It has also been reported that absent dorsalis pedis pulse was associated with a 6.3 fold increased risk of foot ulceration (Walters et al. 1992). More recent literature suggests that vascular disease is a more important risk factor for delayed wound healing and subsequent amputation than the actual development of ulceration.

The presence of vascular disease is evaluated by combination of clinical signs and symptoms plus abnormal results on noninvasive vascular tests. Signs and symptoms of vascular disease are cold feet, blue toes, intermittent claudication, rest pain, night cramps, poor healing, sparse hair growth on lower limb, skin atrophy, muscle wasting and thickened nails. Simple clinical evaluation of the lower limb provides useful information on the arterial circulation. Cold extremities, absent pulses, pallor on elevation and rubor on dependency are all indicative of significant peripheral vascular disease (Levin 2001). Abbott and associates in a prospective study found that the relative risk of developing new foot ulcers was 2.9 fold among patients who had loss of pedal pulses at baseline (Abbott et al. 1998). Palpation of pulses is susceptible to

variation between observers and the pulses may be masked by the presence of oedema. Non-invasive measurement of blood pressure in peripheral arteries using Doppler ultrasound provides an objective measurement of vascular status. The most commonly used measurements are the ankle pressure index and the toe pressure index. Absolute toe pressures of less than 30mmHg have been shown to be a risk factor for amputation (Apelqvist et al. 1992).

Atherosclerosis in diabetes is characterized by early calcification, which results in hardening of the arterial walls. The relative incompressibility of the arteries in diabetes influences the interpretation of blood pressure recording in the foot. The difficulty in interpreting ankle pressure index due to calcification has produced some controversy regarding the validity of the measurement. Non-invasive tests have been faulted for underestimating severity of arterial insufficiency (Caputo et al. 1994) and Doppler pressures have been noted to correlate poorly with symptoms and angiographic findings (Mercer et al. 2000). The most reliable non-invasive investigations are toe pressures and analysis of the Doppler waveform (Mercer et al. 2000).

Elaborate vascular examination is in general not required for routine screening (Cavanagh 1999). The use of vascular testing equipment is not always readily available in the out-patient setting. Initial screening is based on palpation of pulses, appearance of limb and patients symptoms. If an abnormality in vascular status is suspected, further investigation is indicated.

2.3.3 Assessment of the foot

One of the most effective mechanisms for preventing diabetic foot complications is regular inspection (Armstrong et al. 1998a). Self reported preventative practices have been linked with decreased risk of lower extremity complications. The reason for this is that it may prompt early treatment intervention of foot problems. Examination of the foot remains to be the most neglected part of the diabetic assessment (Levin 2001). Low rates of foot inspections have been reported during out-patient and in-patient consultations (Cohen 1983; Wylie-Rosett et al. 1995; Bailey et al. 1985; Peters et al. 1996). In a recent study of health care providers in the USA it was reported that healthcare providers regarded foot examination in diabetic patients to be

a very important process and gave a high rating for their responsibility for conducting foot examination. Fourteen percent of respondents stated that examination of feet was time consuming and approximately one-quarter of the providers agreed that forgetting to examine feet was a practice pattern barrier to patient care (Chin et al. 2001).

2.3.3.1 Examination of Skin and nails

Surface examination of the foot is performed to identify any callus, haemorrhage into callus, scarring from previous ulceration, breaks or cracks in the skin. Callus builds up in response to high vertical and horizontal pressures, it has been shown to be a predictor of ulceration, (Murray et al. 1995). Haemorrhage into callus is recognised as a precursor to foot ulceration. It implies that enough trauma has occurred to cause tissue damage (Cavanagh et al. 2001b). Scar tissue from previous ulceration may have altered mechanical properties, which increases the risk of further ulceration. Subjective assessment of the skin surface temperature is used as an indicator of inflammation due to tissue damage or charcot joints. Examination of the nails is performed to identify in-growing toenails, thickening of nails and subungual haematoma due to trauma or ill fitting footwear.

2.3.3.2 Foot deformity

Identification of significant foot deformity, which may affect foot function or which may make foot wear ill fitting is performed. Common foot deformities which have been identified as increasing the risk for ulceration are prominent metatarsal heads, claw / hammer toes, hallux valgus, hallux rigidus, prior amputation and Charcot foot deformity. Foot deformities are believed to be more common in diabetic patients due to wasting of the small muscles in the feet. An increased risk of ulceration has been shown to be associated with foot deformity (Rith-Najarian et al. 1992). Most diabetic foot ulcers are located over areas of bony prominence (Armstrong et al. 1998b).

2.3.3.3. Assessment of joints

Assessment of joints can involve determination of range of movement or determination of joint stiffness. To measure joint stiffness the joint motion and the force required to cause the motion needs to be recorded simultaneously, so the

quantity of force needed for each unit of angular displacement can be calculated (Cavanagh et al. 1991b). Measurements of joint stiffness have been performed in research studies, but are not easily performed in routine clinical assessment clinics.

During clinical examination many clinicians assess the quality, direction and symmetry of joint motion in a non-weight bearing position. They estimate the range of motion by moving the joint through its whole range and document joint movement as normal, restricted or severely restricted. This method is very subjective and is not conducive to either good assessment or good documentation (Cavanagh et al. 1991b). Range of movement at joints can be assessed using a goniometer to measure end point of the range of motion in both directions. The end range of motion at a joint can be achieved by either the clinician moving the joint to its end range or the patient voluntary moves the joint to its end range. Limited range of movement at the subtalar, ankle and 1st metatarsophalangeal joints have been associated with the development of foot ulceration (Delbridge et al. 1987; Fernando et al. 1991; Veves et al. 1995).

2.3.4 Systems to ascertain risk status

There is no risk classification system, which is universally accepted to predict future ulceration. An understanding of clinical risk factors for developing foot ulceration will help clinicians categorize patients by their risk status and may indicate appropriate intervention to prevent ulceration (Lavery et al. 1998). Guidelines for the assessment and treatment of diabetes related foot problems have been developed by a number of different professional bodies. The American Diabetes Association recommends screening for peripheral neuropathy, altered foot biomechanics, peripheral vascular disease and a history of ulceration or amputation when assessing for future risk of ulceration. The consensus on the Diabetic Foot suggest that the risk classification system should be based on the presence of sensory neuropathy, signs of peripheral vascular disease and foot deformities (International consensus on the Diabetic Foot 1999).

Detection of patients at risk of foot ulceration remains problematic at the primary care level (Jirkovská et al. 2001). Low rates of foot inspections have been reported during out-patient and in-patient consultations (Bailey et al. 1985; Cohen 1983; Wylie-Rosett et al. 1995; Peters et al. 1996). Edelson and associates reported that less than 15% of

patients admitted to a University teaching hospital with diabetes related foot pathology receive a minimally competent lower extremity examination (Edelson et al. 1996). In a retrospective survey of diabetic patients receiving hospital care and undergoing a non-traumatic amputation only 50% had undergone a complete foot examination in the year preceding the initial ulceration or gangrene (Deerochanawong et al. 1992). Simple physical examination and clinical diagnosis of neuropathy and angiopathy based on the patient's medical history is not sufficient to identify those at risk of ulceration (Jirkovská et al. 2001). The use of simple standardised non-invasive testing methods to determine neurological and vascular status greatly improves the accuracy of identifying patients at risk of developing diabetes related foot problems at the community level (Jirkovská et al. 2001).

Lavery and associates evaluated risk factors for foot ulceration using a stepwise logistic regression model (Lavery et al. 1997). They identified neuropathy, foot deformity, high plantar pressures and a history of amputation as significantly associated with the presence of foot ulceration. They found that patients with neuropathy alone were at approximately 1.7 times greater risk of presenting with and developing foot ulceration than diabetic patients without neuropathy. This risk increased to 12.1 times when the patients presented with foot neuropathy and foot deformity. Patients with neuropathy, deformity and a history of previous ulcer or amputation were approximately at 36 times greater risk of developing another ulcer (Armstrong et al. 1998b; Lavery et al. 1997). Based on these findings a treatment based classification system for assessment and care of the diabetic foot has been proposed (Armstrong et al. 1996). The University of Texas Diabetic foot classification system, has four categories for risk of ulceration, illustrated in Table 2-3. The University of Texas Diabetic Foot classification System does not include risk classification for presence of peripheral vascular disease. A risk classification system, which addresses the significant risk associated with ischaemia has been proposed (Frykberg 1991). This classification recognises five categories outlined below in Table 2-4. This system places any person with diabetes at risk level of 1, as it recognises the presence of diabetes alone to be a potential risk factor.

A similar risk classification scheme using the 5.07 monofilament has been used in the primary care sector in the USA. The risk for foot ulceration is determined by taking a brief history of previous foot problems and performing a simple foot examination.

A history of previous ulceration or amputation is noted and the foot is inspected for deformity and callus. The examination includes checking the foot pulses for evidence of ischaemia and assessing loss of protective pain sensation using the 5.07 monofilament. The system recognises four categories of increasing risk. The first category zero, is assigned to a patient with a normal foot shape who has full detection of 5.07 monofilament. Category one is assigned to a patient who is insensitive to the 5.07 monofilament but does not have any foot deformity. A patient who is insensitive to the 5.07 monofilament and has foot deformity will be in risk category two.

A patient who has a history of previous ulceration or amputation is deemed to be at highest risk of developing foot ulceration and is category three. Kumar and associates used this risk classification system in a prospective study of 358 patients followed over 32 month period. They found that incidence rates of ulceration correlated positively with increasing risk category and all the amputations occurred in risk groups 2 and 3 (Kumar et al. 1991).

Ideally assessment of the diabetic foot should be standardised and be a valid, specific and sensitive prognostic measure of foot ulceration. In reality a number of different assessment scoring systems have been developed, each system having inherent merits and limitations for use within the clinical environment. Many assessment systems have not been validated and the prognostic value for prediction of ulceration has not been established. Most systems fail to include an assessment of gait, despite the increasing awareness that the impact that biomechanical issues has on the development and treatment of foot ulceration. Financial and time constraints have inhibited the wide implementation of gait analysis tools in clinical screening programmes. A number of research groups have used gait analysis to increase knowledge about the aetiology of ulceration, however, adapting this knowledge to the outpatient screening environment remains problematic.

Table 2-3: The University of Texas Diabetic Foot classification System: Treatment based classification system for assessment and care of diabetic feet.

Risk Category	Clinical features	Possible treatment interventions
No Pathology 0	Protective sensation intact Ankle brachial Index > 0.80 and toe systolic pressure > 45mmHg Foot deformity may be present No history of ulceration	Two to three visits per year to assess neurovascular status and foci of stress Patient education Possible shoe accommodation
Neuropathy No deformity 1	Protective sensation absent Ankle brachial Index > 0.80 and toe systolic pressure > 45mmHg No foot deformity No history of ulceration No history of Charcot's joints	Same as category 0 plus: Possible shoe gear accommodation (orthotist consultation) Quarterly visits to asees shoe gear and monitor for signs of irritation
Neuropathy with deformity 2	Protective sensation absent Ankle brachial Index > 0.80 and toe systolic pressure > 45mmHg Foot deformity present (focus of stress). No history of neuropathic ulceration No history of Charcot's joints	Same as category 1 plus: Orthotist consultation for molded insoles/ shoe accommodation Possible prophylatic surgery to alleviate focus of stress
History of Pathology 3	Protective sensation absent Ankle brachial Index > 0.80 and toe systolic pressure > 45mmHg Foot deformity present (focus of stress) History of neuropathic ulceration History of Charcot's joints	Same as category 2 plus: Orthotist consultation for custom molded insoles/ extra depth shoe accommodation Possible prophylatic surgery to alleviate focus of stress More frequent visits may be indicated for frequent monitoring

Table 2-4: Risk status classification proposed by Frykberg, taken from Diabetic foot Ulceration, in The High Risk Foot in Diabetes Mellitus 1991.

Risk status	Clinical features
1	Normal sensation with no deformity
2	Normal sensation with deformity (or high plantar pressure)
3	Insensitivity without deformity
4	Ischaemia without deformity
5	Complicated Combination insensitivity, ischaemia, and/or deformity Prior history of ulceration Charcot deformity

2.4 Alterations of foot structure and function in diabetes

The following provides a description of the types of changes to foot structure associated with diabetes, including changes in the soft tissue characteristics, muscle and joint structure and function. The impact that these changes have on the development of foot ulceration will be discussed.

There are three mechanisms by which foot injury can occur in the diabetic foot with loss of protective sensation. The first mechanism for tissue injury is by direct mechanical disruption of the tissue caused by a high force concentrated over a small area, it takes an approximate pressure of 100Kg/cm^2 to penetrate normal intact tissue (Jenkin et al. 1991). This type of injury is usually sustained as the patient is walking barefoot and stands on a sharp object (pin, glass). Ideally this type of injury should be virtually eliminated if the patient takes appropriate measures to protect their feet. The second mechanism for injury occurs when a low pressure is sustained for a long period of time. Capillary blood flow is occluded leading to tissue ischaemia and ulceration occurs after several hours. This type of injury is usually related to tight fitting footwear, resulting in excessive pressure from circumferential tension (Jenkin et al. 1991). The majority of all wounds on insensate feet are caused by repeated intermittent moderate stress generated during normal walking (Brand 1988). Following multiple repetitions of moderate stress traumatic inflammation occurs and with loss of protective sensation, the subject proceeds to walk upon the injured area without altering gait and continues to traumatise the inflamed tissue which subsequently ulcerates (Jenkin et al. 1991).

Ulceration has been shown to occur at sites of high pressure most frequently located in the forefoot (Oyibo et al. 2002), with approximately equal distribution on the dorsal and plantar surfaces (Edmonds et al. 1986). Ulceration on dorsal areas of the foot often result from external pressure from ill-fitting footwear and in theory should easily be prevented with the use of appropriate footwear and patient education. Ulceration on the plantar surface is usually located at sites of high pressure resulting from mechanical problems that pre-exist within the intrinsic foot type (Schoenhaus et al. 1991). It is believed that diabetes may alter both musculoskeletal and soft tissue

mechanics in a manner that elevates plantar pressure and makes tissue damage more likely (Cavanagh et al. 2001b). Biomechanical factors thought to be responsible for the development of foot injury in the neuropathic diabetic foot can be attributed to changes in soft tissues, foot structure, function and gait style.

2.4.1. Soft tissue changes

Skin and soft tissue serve as a mechanical protective medium, shielding the body from external stresses (Thompson 1988). Soft tissue failure (ulceration) occurs as a result of an abnormal interaction between an environmental stress and the soft tissues over a given period of time. When sensation is intact environmental stress will be perceived as noxious and there will be a withdrawal response. The magnitude of stress that is perceived as noxious is lower than that which would produce soft tissue breakdown (Jenkin et al. 1991). With loss of protective sensation the stress is not perceived as noxious, withdrawal does not occur and tissue failure results.

The soft tissue under the foot is very strong and extremely resilient to failure under stress, the viscoelastic nature of soft tissues allows the applied forces to be dispersed or dissipated efficiently. The viscous component of soft tissue will mould to applied stress, increasing the surface area of contact and decreasing the pressure. The elastic element of soft tissue will absorb energy and return it to the environment via elastic recoil (Jenkin et al. 1991). Viscoelasticity allows the soft tissues to adapt to stresses applied in both space and time. The ability to adapt in space is afforded by moldability of soft tissues and ability to adapt in time is afforded by thickness of soft tissue. Resilience to breakdown would be impaired if any deleterious changes were to occur in either of these characteristics (Jenkin et al. 1991).

Non-enzymatic glycosylation (NEG) of many proteins in the body have been demonstrated in patients with diabetes. This process may contribute to significant alterations in physical and functional properties of the soft tissue (Hashmi 2000). Glycosylation of collagen results in a number of chemical reactions, the end result is the formation of advanced glycation end products (AGEPs). AGEPs are highly cross-linked and are thought to cause significant alterations in the physical properties of collagen-rich tissues. With NEG the soft tissue becomes less flexible and thus less able to distribute pressure through deformation (Hamlin et al. 1975).

The structure and thickness of soft tissue vary, dependent upon the mechanical demand at a given location (Thompson 1988). Areas that are subject to high mechanical forces, for example the heel have increased thickness of soft tissue compared to areas subjected to minimal orthostatic stress. Cavanagh and associates examined the relationship between bony structure of the foot and plantar pressure. They identified structural parameters associated with high pressure using stepwise multiple regression. The thickness of soft tissue between the sesamoids and the ground determined from standardised weight-bearing x-ray was found to be one of the strongest predictors of pressure under the first metatarsal heads (Cavanagh et al. 1997; Morag et al. 1999).

In diabetes the amount of soft tissue between the skin and the bones may be decreased in many areas of the foot thus increasing the risk of soft tissue failure and subsequent ulceration. The decrease in soft tissue thickness has been attributed to atrophy or displacement of the sub metatarsal fat pad associated with foot deformity. Atrophy of the fat pad is associated with the normal ageing process but is also associated with chronic diabetes (Cavanagh et al. 1993). Plantar fat pad thickness determined by ultrasound was found to be thinner in patients with diabetes compared to non-diabetics. A decrease in soft tissue thickness has been linked with increased pressures under the foot (Morag et al. 1999). It has also been noted that the fat pads of diabetics with previous foot ulcers were thinner than in diabetics who had not ulcerated (Gooding et al. 1986). This observation was made on a cross sectional study and does not show a causal relationship between decreased soft tissue and the formation of ulceration.

Previous ulceration is regarded as a leading risk factor for further ulceration, this has been confirmed in a prospective study by Murray and associates. They found the relative risk of developing a new ulceration in patients with a history of ulceration was 56.8, confirming that a previous history of ulceration is the most important risk factor for predicting subsequent ulceration (Murray et al. 1996). Reported rates for re-ulceration over a period of five years have been reported to be 70% (Apelvist et al. 1993). The recurrence rate for ulceration after two years in patients wearing inappropriate footwear has been reported to be as high as 83% (Edmonds et al. 1986). The high risk linked to prior ulceration could be attributed to changes in mechanical

properties of scar tissue and decreased tissue thickness. The viscoelastic properties of repair tissue from previous ulceration is significantly inferior in terms of moldability and thickness making subsequent ulceration more likely (Jenkin et al. 1991). It has been theorised that scar tissue may act like callus, by transferring large concentrated loads to underlying softer tissues (Cavanagh et al. 2001b).

One of the key events in the pathway to neuropathic ulceration is the development of hyperkeratosis (callus). A direct correlation has been demonstrated between the presence of plantar callus and ulcer formation (Murray et al. 1995). The relative risk of ulceration for a callused area was found to be 11.0. Under the influence of intermittent compressive stress the normal process of keratinisation, which maintains the stratum corneum as a protective cover, becomes over-stimulated (Murray et al. 1996). Large plaques of hyperkeratotic tissue form in areas of high mechanical stress. These plaques may further increase the pressure and concentrate the stress to underlying soft compliant tissue (Murray et al. 1996;Thompson 1988).

Hyperkeratosis is generally attributed to abnormalities in load distribution resulting from disturbances to normal foot function associated with structural abnormalities in the lower limb (Bevans et al. 1999). Callus formation in diabetes may be due to NEG, altered mechanical exposure, autonomic dysfunction or a combination of these factors (Hashmi 2000). NEG of keratin the major protein component of the stratum corneum has been demonstrated to occur in patients with diabetes (Delbridge et al. 1985). This process is thought to contribute to the increased skin stiffness seen in patients with diabetes and may play a role in the formation of callus (Hashmi 2000).

It has been noted that callus in people with diabetes may have different mechanical properties to callus in non diabetics. It has been reported that the callus in diabetics is stiffer and unusually hard (Buckingham et al. 1984; Delbridge et al. 1985). Using durometry (a device which applies an indentation load on soft tissues and can determine relative tissue hardness) Piagessi and associates evaluated skin hardness in people with diabetes compared to non-diabetic controls. They determined skin hardness in areas of the foot exposed to stress and areas not usually exposed to mechanical stress. A higher degree of skin hardness was found in neuropathic feet compared to non-neuropathic feet and non diabetic controls in both areas which were exposed to stress and those areas which are not usually exposed to stress (Piaggesi et

al. 1999). The increase in skin hardness in people with diabetes could be due to NEG, alterations in foot function associated with diabetes or decreased sweating associated with autonomic neuropathy. Autonomic neuropathy has also been implicated in the formation of ulceration, dry skin resulting from dyshidrosis is not as mouldable as hydrated skin therefore the ability to dissipate forces will be impaired and the resilience to trauma decreased (Jenkin et al. 1991).

2.4.2 Foot structure

The alterations to foot structure related to diabetes are thought to be primarily the result of neuropathy (Faris 1991). The characteristic "intrinsic minus" foot associated with diabetes has claw toes, a high medial longitudinal arch and prominent metatarsal heads. Foot deformity in people with diabetes can be categorised as primary, secondary or iatrogenic (Frykberg 1995). Some deformities (primary) are independent to diabetes and are often present before the onset of the disease. Secondary deformities are directly linked to diabetes and associated with changes in neurological function, joints and soft tissue. Iatrogenic foot deformities are most commonly the result of previous amputation.

In the presence of diabetic neuropathy structural deformities are considered to be major risk factors for ulceration. Foot deformity has been shown to be associated with an increased risk of foot ulceration (Boyko et al. 1999; Rith-Najaran et al. 1992). The link between foot deformity and ulceration is increased pressure. Deformities and bony prominence tend to focus stress onto a smaller area of distribution, thereby increasing the pressure and the risk for tissue failure. Mueller and associates found a significant relationship between foot deformity and location of ulceration in diabetic patients with neuropathy (Mueller et al. 1990).

Clawing of the toes is a common clinical finding, although it is seen in people without diabetes it is believed to occur more frequently in people with diabetes. However, the author could find no literature to support this statement. Clawing of the toes is believed to result from motor neuropathy, causing atrophy of the intrinsic muscles responsible for stabilising the digits. There is no evidence to support this theory and a causal relationship between motor neuropathy and clawed digits has not been validated. Furthermore, recently an alternative hypothesis for the development of

claw toes has been proposed. Rupture of the plantar fascia has been demonstrated in a small number of diabetic patients with clawing of the toes. No rupture was found in matched control diabetic patients without clawing of the digits (Taylor et al. 1998). Using quasi-static cadaver models Hamel and Sharkey, demonstrated the importance of the plantar fascia for the efficient transmission of force through the toes. They found that without the tethering effect of the plantar fascia on the proximal phalanges, the toes were pulled into a clawed position and lost their ability to effectively transmit plantar force (Hamel et al. 1999). The veracity of the finding of plantar fascia discontinuity by magnetic resonance imaging has been questioned when alternative imaging techniques are employed. Masson and Taylor investigated the integrity of the plantar fascia using ultrasound, in ten diabetic patients with claw toe deformity. The plantar fascia was found to be intact in eight patients with claw toe deformity (Personal communication, Masson and Taylor 2003).

Regardless of its aetiology this type of deformity increases the risk of dorsal ulceration from footwear. It is also associated with anterior displacement of the plantar metatarsal fat pad that decreases the amount of soft tissue under the metatarsal heads, thus increasing pressure and the risk of ulceration at this site. Dorsal contracture of the digits at the metatarsophalangeal joints produces an increasing retrograde plantar-flexory force through the metatarsal heads, which increases the pressure under this area during propulsion (Schoenhaus et al. 1991).

Foot structure has been shown to play a large part in determining the plantar pressure exerted by the foot during walking (Cavanagh et al. 1997). Metatarsal inclination and a reduction in soft tissue thickness are directly related to increased plantar pressures under the first metatarsal head. Using finite element analysis, biomechanical models have been developed to evaluate the relationship between foot structure and plantar pressure (Cavanagh et al. 2001b). The detrimental changes in quality and quantity of soft tissue under the foot associated with diabetes were discussed earlier. The impact that these changes have on foot pressures has been described using the biomechanical model approach. The foot with adequate cushioning under the metartarsal head is predicted to show low pressure under the area. The model predicts the foot without adipose tissue will show pressures in the area of the metatarsal head that are over five times higher than those in the normal foot, thereby increasing the risk of ulceration (Cavanagh et al. 2001b).

2.4.3 Foot function

The important changes in foot function associated with diabetes are related to muscle weakness attributed to motor neuropathy and changes in joint mobility. Muscle weakness and limited joint mobility can have major detrimental effects during the whole gait cycle, dependent on which muscle groups / joints are affected.

2.4.3.1 *Muscle weakness*

Andersen and associates examined muscle strength in a group of 56 insulin dependent diabetics with duration of diabetes greater than 20 years compared to age, weight and height matched non-diabetic controls. They found that the diabetic group had a 21% reduction of muscle strength of both the ankle dorsal and plantar flexors and a 16 percent reduction in knee extensor strength compared to the control group. A correlation was found between neuropathy rank-sum score and the muscle strength of the ankle dorsal and plantar flexors and knee extensors and flexors (Andersen et al. 1997).

2.4.3.1.1 Effect of muscle weakness on the ankle joint complex

When the heel contacts the ground a strong external plantarflexor moment is created at the ankle joint, resulting from the placement of the ground reaction force vector (directed in a superior posterior direction behind ankle joint axis). Eccentric contraction of the anterior muscle group creates an internal dorsiflexor moment that decelerates plantar flexion movement at the ankle and decreases the forces encountered in the forefoot upon reaching the supporting surface. If weakness is present in the anterior muscle group, the foot will plantarflex too quickly and this will result in an increased force under the forefoot, which could contribute to ulcer formation. It has been suggested that the rate of tissue deformation may be a critical factor in the formation of diabetic ulceration. High rates of tissue deformation have been shown to result in endothelial cellular death or injury, while low rates are much less likely to produce lasting injury (Landsman et al. 1995). With anterior muscle group atrophy the forefoot will reach the ground with increased velocity resulting in a high strain rate of tissue deformation. High strain rate tissue deformation has been

proposed as an alternative explanation for the aetiology of foot ulceration in patients with anterior muscle group atrophy. This theoretical model has yet to be supported by any clinical investigations.

Weakness in the anterior muscle group can allow the posterior group to gain a mechanical advantage, clinically seen as hypertrophy of the calf muscles. This causes an increased plantarflexion pull on the calcaneus and progressive limitation of ankle joint dorsiflexion can occur, which increases the forces under the forefoot. It has been suggested that limitation of dorsiflexion at the ankle joint will result in compensation at the subtalar joint. This compensation will produce excessive frontal plane motion of the rearfoot during the stance phase of gait, which will contribute to hyper-mobility within the forefoot during propulsion and increased shearing forces. The effect that limited ankle joint dorsiflexion has on rear foot frontal plane motion in non-diabetics during stance phase has been investigated by Cornwall and McPoil. They studied two groups, one group had a passive ankle dorsiflexion range of less than or equal to ten degrees the other group had a dorsiflexion range of more than fifteen degrees. Three-dimensional motion analysis at the ankle joint complex was performed during over ground walking. They concluded that slight to moderate limitation of ankle dorsiflexion did not alter the magnitude of frontal plane motion, but significantly altered the timings of heel lift and time to reinversion. The stance phase duration was not significantly different between the two groups but heel lift was significantly earlier in the group with limited ankle joint dorsiflexion (Cornwall et al. 1999a). It is reasonable to assume that an earlier heel lift would cause the forefoot to be loaded for a greater proportion of the stance phase, and that the pressures in the forefoot may be increased in the group with limited ankle dorsiflexion. The study did not examine plantar pressure, a combination of motion analysis with pressure measurement would have provided a greater insight into the impact that limited ankle joint movement had on foot function.

At heel strike the subtalar joint is inverted and the ground reaction force vector is directed lateral to the joint axis. This produces an external eversion moment about the subtalar joint axis. Internal inversion moments are created about the subtalar joint axis by tendons of tibialis posterior, flexor digitorum longus, flexor hallucis longus and triceps surae which decelerates the subtalar joint eversion (Otis 2000). If myopathy is present the transfer of load from the lateral side of the foot to the medial side is much

faster. The subtalar joint is thought to reach its end range of motion abruptly causing jamming of the joint (Schoenhaus et al. 1991). The jamming of the joint is not recognised by the neuropathic diabetic patient due to decreased proprioception and loss of protective sensation. Osteoarthritic changes at the subtalar joint could occur which decreases the range of movement (Schoenhaus et al. 1991).

2.4.3.1.2 *Intrinsic muscle atrophy*

Stability of the toes is essential during propulsion and is achieved by balance of muscle groups within the foot and the tethering effect of the plantar fascia. When the lumbricals and interossie muscle groups contract they create a flexion force on the metartarsophalangeal joints. The flexion force is antagonised by the muscles superior to the deep transverse metatarsal ligament, extensor digitorum brevis and extensor digitorum longus (Schoenhaus et al. 1991). The relationship between muscles is perfectly balanced so that the digits remain parallel to the supporting surface. Instead of causing movement, the muscle forces create compression and stability across the joints.

In diabetics with neuropathy atrophy of all the intrinsic muscles of the foot is thought to be common but has not been validated. This is thought to cause disruption of the muscle balance and loss of stability in the forefoot during propulsion. The muscle forces now cause dorsiflexion of the proximal phalanx, which causes plantar flexion of the metatarsal head. Force is no longer efficiently transmitted through the toes and the force under the metatarsal heads increases. Due to loss of stability during propulsion the shearing forces are also thought to increase.

2.4.3.2 Joint mobility

A generalised limitation of joint mobility has been demonstrated in patients with diabetes. Limited joint mobility (LJM) accompanying diabetes has been described in the hands, elbow, shoulder, subtalar joint and 1st metatarsophalangeal joint. (Campbell et al. 1985; Delbridge et al. 1987; Fernando et al. 1991; Schulte et al. 1993; Starkman et al. 1986). The exact pathogenesis of LJM in diabetes is unclear, increased collagen deposition in the periarticular connective tissues, increased cross-linking of collagen and non-enzymatic glycosylation (NEG) have been described in patients

with limited joint mobility. NEG causes significant alteration in the physical and mechanical properties of collagen rich tissues (Hashmi 2000). Tendons and ligaments have a high collagen content and therefore changes in their mechanical properties may affect the range of motion available at joints (Cavanagh et al. 1991b).

Limitation of joint movement in the foot has been shown to increase the risk of foot ulceration in the neuropathic foot (Delbridge et al. 1987). A significant decrease in the range of motion at the subtalar joint in patients with diabetes has been demonstrated and is widely recognised as a risk factor for the development diabetic foot ulceration (Delbridge et al. 1987; Fernando et al. 1991; Veves et al. 1995). Fernando and associates found a strong correlation between range of movement at the subtalar joint and plantar foot pressures (Fernando et al. 1991). They found that peak foot pressures were significantly higher in patients with LJM compared to neuropathic patients without LJM.

Ulceration on the plantar aspect of the hallux is common in neuropathic patients (Cavanagh et al. 2001b). It has been noted that diabetic patients with a history of ulceration at the hallux have limited dorsiflexion at the first metatarophalangeal joint (Birke 1988). The same group also identified that diabetic patients with a history of ulceration at the first metatarsal head had significantly lower first ray mobility and significantly higher pressure under the first metatarsal head (Birke et al. 1995).

The ramifications of LJM at the subtalar joint are claimed to be lack of shock absorption during heel strike due to restricted subtalar joint pronation, and an alteration of the progression of the forces in the foot resulting in higher forefoot pressures and ulceration (Hiss 1949; Simmons et al. 1997; Root et al 1977). Yingling and associates examined the impact that restriction of subtalar joint pronation using a medial wedge had on impulse waves at the tibia during treadmill running. They found no significant difference in the impulse wave characteristics at the level of the tibia when motion at the subtalar joint was restricted by medial wedging during the initial 15% of stance phase (Yingling et al. 1992). This finding does not support the previous theories about the impact that LJM has on the diabetic foot function (Payne 1998).

More recently it has been suggested that normal foot function is dependent on an adequate range of motion at the 1st metatarsophalangeal joint during walking (the sagittal-plane facilitation of motion model) (Payne 1998). This model claims to offer an alternative explanation for correlation between LJM and increased plantar pressures in the diabetic foot. The model states that if a sufficient dynamic range of motion at the 1st metatarsophalangeal joint is not available there will be an unloading of the medial side and an increase in weight bearing under the lateral forefoot. Although this pattern of pressure loading in patients with diabetes has been reported by a number of workers (Stokes et al. 1975), this theoretical model has not been supported by any clinical investigations.

Clinical evidence suggests that Asian and African-Caribbean diabetic patients have a lower prevalence of foot ulceration (Clarke et al. 1998; Gujral et al. 1993). There are many potential explanations for the lower prevalence including differences in culture, in health belief systems, self-care foot practices and differences in joint mobility (Greenhalgh et al. 2001; van Schie et al. 2000). Veves and associates investigated differences in joint mobility and foot pressures using an in-shoe pressure measurement system in African-Caribbean and Caucasian diabetic patients. They found that Caucasian diabetic patients had significantly reduced subtalar joint mobility and significantly increased plantar peak pressures without shoes when compared to African-Caribbean diabetic patients. A significant difference in peak pressure was not found between these groups in-shoe (Veves et al. 1995). In this study patients wore their own footwear, hosiery and insoles, the differences in footwear would significantly alter the plantar foot pressures recorded, therefore changes in pressure cannot be directly related to changes in joint mobility. Another limitation of the study is that the pressure measuring insoles were taped to the foot to record pressure without shoes, the authors did not provide any information regarding reliability or repeatability of using the in-shoe system in this manner. Movement of the insole during the walking trial, bending and stretching of the insole could influence the pressures recorded.

Most workers investigating LJM have found relationships between reduced joint mobility at the subtalar and metatarsophalangeal joint is associated with increased foot pressures and prevalence of ulceration. The measurement of joint mobility has been based on the static assessment of joints using goniometers. Many studies have

shown that taking joint measurements in this way is subject to large errors (Menz 1995; Elveru et al. 1988a) and that there is poor correlation between static measures of the ankle joint complex and dynamic foot function. (Hamill et al. 1989; McPoil et al. 1994a; McPoil et al. 1996a). A recent study has evaluated dynamic ranges of motion at the ankle, subtalar and first metatarsophalangeal joints in a small number of European and diabetic patients of Asian origin (van Schie et al. 2000). They found an association between the rate of plantar flexion after heel strike and history of ulceration. This finding is in agreement with Landsmann's theoretical model that the rate of tissue deformation may be a critical factor in the formation diabetic ulceration (Landsman et al. 1995).

A greater understanding of the impact that limited joint mobility at the ankle joint complex and first metatarsophalangeal joint is needed to establish its role in the aetiology of foot ulceration. Work in this area could be enhanced if LJM was predicted from dynamic motion (with comparison against normal parameters) rather than static joint assessment. The relationship between joint mobility during walking and static ranges of joint mobility needs to be investigated to determine if static ranges of movement should remain a valid part of diabetic foot assessment.

2.5 Gait changes in diabetes with reference to normal gait

This section provides a detailed description of normal gait, with specific reference to the ankle foot complex. The changes in gait related to diabetes and neuropathy will be discussed together with the relationship to ulceration.

In order to understand abnormal gait, it is necessary first to understand normal gait, since this provides the gold standard against which everyone else's gait can be judged (Whittle 1997). The history of gait analysis has shown a steady progression from early descriptive studies through to increasingly sophisticated methods of measurement. Until recently most gait analysis has been performed in a research environment rather than a clinical setting. The low utilisation in the clinical environment can be explained by the high equipment and labour costs combined with arduous data collection and analysis procedures which make gait analysis difficult to justify in the routine clinical setting. With the advent of new technologies providing automated motion analysis techniques and the increased availability of competitively priced commercial systems there has been an attempt to take gait analysis out of the research laboratory and into the clinical environment.

The gait cycle is defined as the time interval between two successive occurrences of one of the repetitive events of walking. Although any event could be chosen for definition of the start of the gait cycle it is usually convenient to use the instant at which one foot contacts the ground (Whittle 1997). The gait cycle can be divided into two phases, the stance phase in which the limb is in contact with the supporting surface and the swing phase where the limb is not weight bearing. Both the stance phase and swing phase can then be further subdivided and the terminology used to describe these phases can vary considerably from one publication to another. For the purposes of this study seven phases of gait outlined by Whittle (1997) will be used to describe the gait cycle. The seven events of the gait cycle are shown in Figure 2-5, this diagram shows the position of the right limb during a single gait cycle.

A comprehensive description of the gait cycle including timing of events, kinematic and kinetic activity at all the joints in the lower extremity is beyond the scope of this thesis and has been reported elsewhere (Inman et al. 1981; Root et al. 1977; Rodgers

1995). This section will focus on the ankle foot complex, which is the terminal section in the lower kinetic chain and is considered to be the most important joint complex during locomotion.

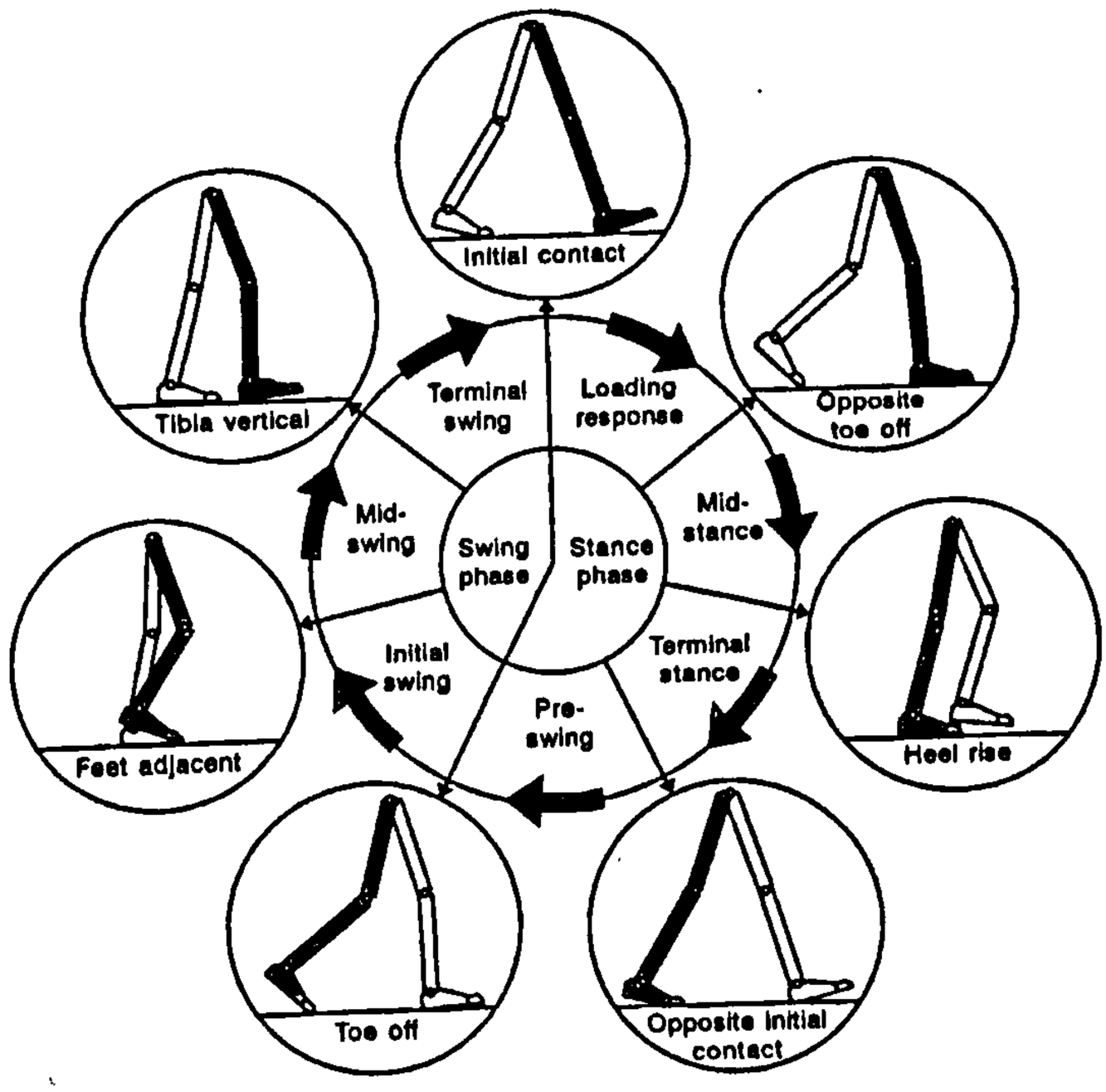


Figure 2-5: Position of the legs during a single gait cycle by the right leg (shaded). *Taken from Whittle (1997).* ***Gait Analysis; an introduction. Second edition. Butterworth-Heinemann, Oxford.***

2.5.1. The ankle foot complex

The ankle foot complex consists of 28 bones with more than 70 articulating surfaces and should be able to distribute and dissipate the compressive, tensile, shearing and rotatory forces encountered during the stance phase of gait (Donatelli 1990). Movement between the foot and leg is produced by a composite of motion occuring at two joints, the talocrural (ankle) and the talocalcaneal (subtalar) joints. The subtalar joint comprises of three articulations between the superior surface of the calcaneus and the inferior surface of the talus. The ankle joint consists of three articulations, the tibiotalar, fibulotalar and tibiofibular articulations. Movement at the subtalar joint

throughout the gait cycle has been extensively documented (Root et al. 1977; Whittle 1996). The characterised movement of the subtalar joint described by Root and colleagues is well accepted and used as a basis to diagnose and prescribe for lower limb pathologies. Assessment of subtalar joint motion forms an essential part of a podiatric biomechanical examination.

The range of frontal plane motion at the subtalar joint is thought to play an important role in the overall function of the lower limb during locomotion. It has been reported that the minimum total range of frontal plane motion at the subtalar joint for normal locomotion is 8-12 degrees (Root et al. 1977). The static range of motion at the subtalar joint varies considerably between subjects, the reported range of frontal plane motion in the literature varies between 10 and 53 degrees (Alexander et al. 1982; Manter 1941; Nigg et al 1992; Ball et al 1996; Inman 1976). The total frontal plane range of motion at the subtalar joint decreases by around twenty percent with age (Alexander et al. 1982; Ball et al. 1996; Nigg et al. 1992). The total range of motion at the subtalar joint during gait is much less than non-weight bearing range of motion.

The rationale for taking lower limb static measures during a biomechanical evaluation is to see if an abnormality exists which could affect the dynamic function of the lower limb (McPoil et al. 1994b; McPoil et al. 1996b). It is an assumption that structure dictates function and numerous structural and functional characteristics have been identified as been related to alterations in foot function (Hamill et al. 1989; Hlavac 1977). It is generally accepted that high arched feet are rigid and poor shock absorbers, whereas flat feet are hyper-mobile. Very few studies have attempted to evaluate the relationship between static foot type and dynamic lower extremity kinematics. Recent literature suggests that static lower limb measures are poor predictors of dynamic foot function (Hamill et al. 1989; Knutzen et al. 1994; McPoil et al. 1994a; McPoil et al. 1996a).

When podiatric biomechanical assessment techniques are evaluated a significant inadequacy become apparent (Menz 1995). Recent literature has identified key problems associated with current techniques.

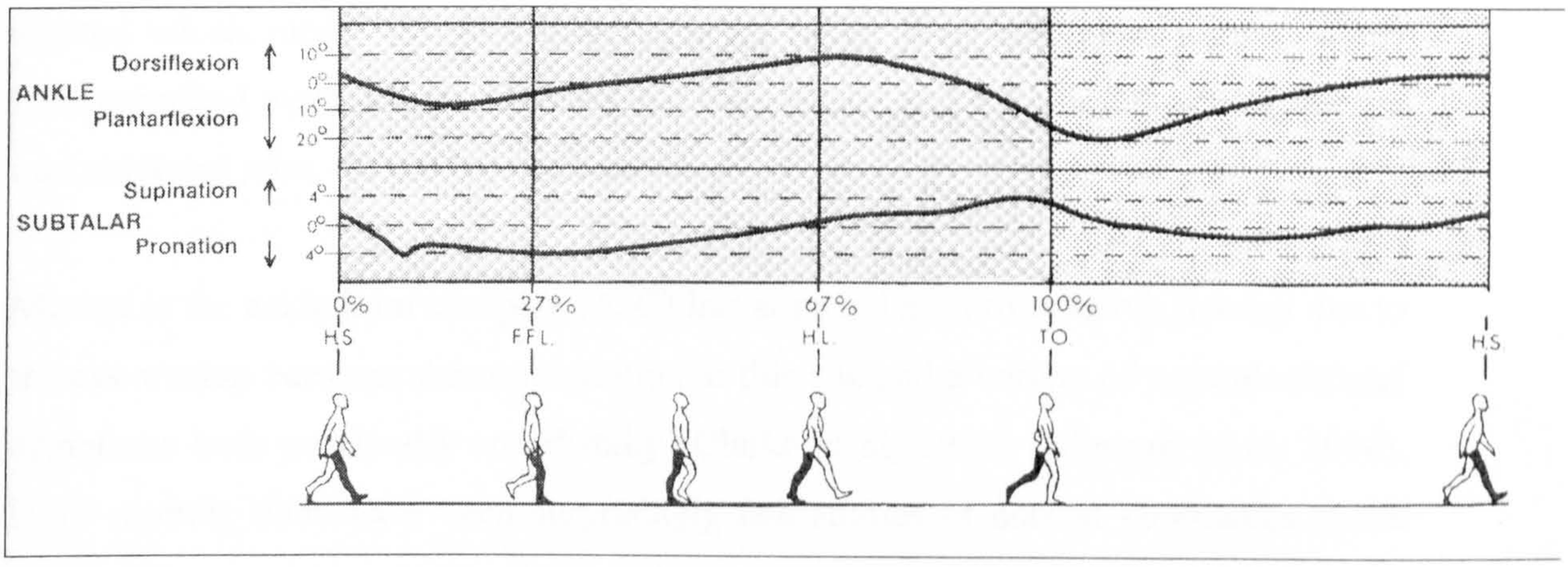
1) Static foot measurements have been shown to be unreliable in clinical practice (Ball et al. 1993; Pierrynowski et al. 1996).

2) Static clinical measurements are poor predictors of dynamic function (Hamill et al. 1989; Knutzen et al. 1994; McPoil et al. 1994a; McPoil et al. 1996a).

In light of recent literature, current techniques used in the podiatric biomechanical evaluation appear to be unsatisfactory. The limitations associated with static lower extremity joint evaluation suggests that dynamic motion analysis is the preferred method.

2.5.2 Movement at the ankle and subtalar joints during gait

The joint movement at the ankle and subtalar joint during walking as described by Root in 1977 is depicted in Figure 2-6. The Root paradigm for foot function is well accepted, however, it must be noted that the descriptions of joint movement are based on observation and clinical experience rather than quantitative joint movement data.



H.S. – Heel strike, F.F.L.- Forefoot loading, H.L.- Heel lift, T.O.- Toe Off

Figure 2-6: Sagittal plane motion at the ankle joint and frontal plane motion at the subtalar joint. *Taken from Root M.L., Orien W.P., Weed J.H. (1977). Normal and abnormal function of the foot. Clinical Biomechanics; Volume II, Clinical Biomechanics Corporation, Los Angeles.*

Advances in biomechanical methods for dynamic analysis have facilitated a more quantitative and precise description of foot function during gait (Rodgers 1988). The

primary movement of normal gait occurs in the sagittal plane and therefore the majority of kinematic data at the ankle foot complex is within the sagittal plane, joint movement in the other two planes is less commonly reported. Many earlier studies analysing rearfoot motion have utilised two-dimensional (2D) methods in both barefoot and in shod walking. The limitations associated with 2D systems have been cited by many workers (Areblad et al. 1990; Cornwall et al. 1995;Soutas-Little et al. 1987) namely projection errors. Direct comparisons between 2D and three-dimensional (3D) video based systems for analysing rearfoot motion in walking and running indicate that 2D analysis is essentially the same for the initial 60% of the stance phase (Areblad et al. 1990; Soutas-Little et al. 1987; Cornwall et al. 1995). Therefore, 2D analysis has its uses in clinical practice but 3D studies are the preferred method to accurately analyse rearfoot motion throughout all of the stance phase.

Study of joint motion at the ankle foot complex is complicated by the fact that no part of the talus is directly observable externally, as a consequence direct observation of kinematics at the ankle can only be made with invasive techniques (Lundberg 1997). In order to try and overcome this problem kinematic models of the foot have been created which model the foot into segments rather than individual joints. Many biomechanical models have assumed that the subtalar and ankle joints act together as one universal joint, the ankle joint complex.

Motion at the ankle joint complex (AJC) has attracted a lot of research interest due to the association between abnormal motion at this site and a variety of musculoskeletal symptoms both proximally and distally (Clarke et al. 1983; Lafortune et al. 1994). Until recently there have been surprisingly few studies of normal kinematics at the ankle joint complex during free walking. With recent technological advances researchers have overcome the complex measurement issues associated with the measurement of frontal plane motion at the AJC and a number of studies have been published. Many studies have small sample sizes and different methods, data collection procedures, reference positions, sampling rates and filtering techniques, which make comparisons between data sets difficult (Allard et al. 1997). The gait patterns of the left and right limb are assumed to be symmetrical and in fact many gait analysis studies only collect data on one limb. The literature is divided in its support of gait symmetry. Numerous studies document gait symmetry in terms of temporal parameters and kinematics (Arsenault et al. 1986; Hamill et al. 1984; Hannah et al.

1984). Allard and associates (1996) collected temporal and 3D bilateral kinematic data on 19 male subjects, they found that both limbs had the same walking speed but they reported statistically different mechanical energies for the right and left limbs (Allard et al. 1996). Very few studies have performed simultaneous kinematic measurements at the AJC they have assumed that the gait is symmetrical and relied on unilateral data.

A summary of kinematic data at key events in the stance phase from a number of referenced sources related to the AJC in normal healthy adults during walking is provided in Table 2-5. The average range of motion from the referenced sources in all three planes is similar, however, there is wide variability in the absolute angular rotation position of the AJC at the key events in the stance phase. This can be explained by the absence of a standardised reference zero / neutral position for the AJC . The zero positions used in the reference sources varies from seated to standing, weightbearing to non-weightbearing and relaxed calcaneal standing position to subtalar joint neutral position. Despite the differences in the angular rotation position between referenced sources there is a general agreement for the pattern of motion at the AJC.

In the sagittal plane there is an initial period of plantarflexion as the forefoot comes into ground contact. During midstance the ankle joint dorsiflexes as the tibia moves forward over the stationary foot and reaches a peak just after heel rise. The ankle joint then starts to plantarflex again before initial contact of the contra-lateral limb, the plantarflexion continues for the rest of the stance phase. In the frontal plane most reference sources describe eversion from heel strike through to mid stance, during late midstance the motion changes to inversion and this reaches a peak at the end of the stance phase. This pattern of frontal plane motion is contrary to the concept of foot function proposed by Root et al 1977, which states that the subtalar joint is positioned in the neutral position at midstance. In the transverse plane there is a pattern of internal rotation from heel strike to midstance and for the remainder of the stance phase external rotation.

Table 2-5: Comparison of kinematic data at the ankle joint complex at key events during the stance phase of normal gait. *Taken and adapted from Woodburn J., Turner D.E., Helliwell P.S., Barker S. (1999). A preliminary study determining the feasibility of electromagnetic tracking for kinematics at the ankle joint complex. Rheumatology; 38: 1260-1268*

	Woodburn *et al 1999* *(n=10)*	Mosely *et al1996* *(n=14)*	Kobayashi *et al1997* *(n=1)*	Reinschmidt *et al1997* *(n=5)*	Kepple *et al1990* *(n=5)*	Cornwall *& McPoil 1999* *(n=153)*	Rattanaprasert *et al 1999* *(n=10)*
Age of subjects	20-35	20-24	39	Mean 28.6	22-30	Mean 26.2	22-45
Measurement technique	EMT	Video	EMT	Video	Video	EMT	Video
AJC Sagittal plane							
ROM	15.2	14	17	21	13.6	17.0	20.2
Angular position at							
HS	-3.8	2.3	2.0	-3.6	-2.0	17.0	-3.0
FF	-7.9	-6.3	-	-	-	-10.0	
MS	0.6	3.0	4.3	2.1	0	3.0	4.0
HL	2.6	5.0	-	-	-	4.0	4.0
TO	-0.5	-2.6	8.0	-15.7	-5.5	-10.0	-12.0
AJC Frontal plane							
ROM	8.1	11.1	17.0	17.3	13.0	8.7	13.7
Angular position at							
HS	-2.7	-1.8	6.4	1.8	1.4	2.5	1.5
FF	-3.3	-3.8	-	-	-	2.0	-3.0
MS	-7.4	-7.0	-10.7	4.5	-2.0	-2.0	-2.0
HL	-7.9	-7.0	-	-	-	-1.8	2.0
TO	-0.2	3.8	0	6.9	10.2	5.8	8.0
AJC Transverse plane							
ROM	7.7	9.9	17.1	13.7	12.3	10.8	10.3
Angular position at							
HS	6.6	2.0	15.0	-3.6	6.8	-0.5	0
FF	8.8	4.5	-	-	-	3.7	3.0
MS	8.1	6.2	25.0	2.3	6.8	1.0	0
HL	7.6	7.7	-	-	-	0.5	-3.0
TO	1.8	-2.8	17.0	-3.6	-1.4	-7.0	-5.0

EMT, elcctromagnetic tracking; Sagittal plane dorsiflexion (+) / Plantarflexion (-); Frontal plane, Inversion (+) / eversion (-); Transverse plane, Internal (+) / External (-); - data not presented in reference material.

2.5.3. Spatio-temporal characteristics of gait

Walking speed or velocity is a product of both stride length and cadence, consequently changes in walking speed may be achieved by altering either or both of these components. Many studies have reported the spatial and temporal aspects of gait, however, in many cases the sample sizes are small and the age ranges are limited. Differences in methodologies and instrumentation across studies make comparison between data sets difficult. Craik (1995) provides an excellent overview of spatial and temporal characterisitics of gait in which she summarises the findings of recent studies in tabular form (Craik et al. 1995). Table 2-6 taken and adapted from Craik (1995) provides a summary of data from a number of studies. In general females tend to walk slower with shorter step lengths and the natural cadence for females is around six to nine steps per minute higher than that of males and this is probably related to the difference in limb length (Chao et al. 1983).

2.5.4. Age related changes in Gait

It is generally accepted that the freely chosen speed of walking in elderly adults will be slower compared to that of young adults (Elble et al. 1991; Hageman et al. 1986; Himman et al 1988). Age related decreases in walking velocity are not always present, chosen walking velocities are influenced more by customary activities than by age (Imms et al. 1981). One has to make the distinction between chronological age and biological age (presence of co-morbidity), taking into account the effect of health and exercise (Grabiner 1997). Martin and associates found that the preferred walking speed of active older adults was similar to that of sedentary younger adults (1.43 and 1.41 metres per second respectively) (Martin et al. 1992). The age related changes in gait are thought to be associated with gaining an increased sense of security during walking. The characteristic smooth, cyclic and reproducible normal gait patterns appear to be retained until at least the seventh decade (Donaghue et al. 1996). It is generally accepted that there is a general decrease in stride length and stride frequency with increasing age (Himman et al. 1988; Hirasaki et al. 1993; Kaneko et al. 1991). There is an increase in walking base and an increase in the cycle time, which leads to a reduction in the percentage time of the gait cycle spent in single limb support.

Table 2-6: Temporal and spatial characteristics of gait: a summary of findings. *Taken and adapted from Craik & Dutterer 1995 in Craik & Oatis; Gait analysis theory and application; Mosby- Year Book, In, St Louis.*

Author	Sample Demographics	No. of subjects	Walking speed (m/s)	Cadence (steps/min)	Cycle time (sec)
Blanke& Hagemen (1989)	**M** 20-33 **M** 60-74		1.31 1.38	- -	- -
Cunningham et al (1982)	**M** 19-49 **M** 55-66	43 41	1.39 1.33	108 109	1.11 1.10
Himann et al (1987)	**M** 19-39 **F** 19-39 **M** 40-62 **F** 40-62		1.37 1.26 1.34 1.27	108 114 107 -	
Murray et al (1964) (1969)	**M** 20-55 **M** 60-65 **M** 60-87	32 12 32	1.52 1.47 1.26	111 115 111	1.03 1.04 1.13
Chao et al (1983)	**M** 19-32 **F** 19-32 **M** 32-85 **F** 32-85	21 20 32 37	1.20 1.02 1.27 1.12	100 102 104 112	1.20 1.18 1.15 1.07
Finley et al (1964)	**F** 18-38 **F** 64-84	12 23	0.82 0.70	105 109	1.14 1.10
Gabell et al (1984)	**?** 21-47 **?** 66-84	32 32	1.37 1.19	108 112	1.10 1.08
Hageman et al (1986)	**F** 20-35 **F** 60-80	13 13	1.60 1.32	119 120	1.01 1.00
Jansen et al (1982)	**M,F** 20-29 **M,F** 60-69	20 20	1.10 1.10	131 135	
Oberg et al 1993	**M** 20-29 **M** 30-39 **M** 40-49 **M** 50-59 **M** 60-69 **M** 70-79 **F** 20-29 **F** 30-39 **F** 40-49 **F** 50-59 **F** 60-69 **F** 70-79		1.23 1.32 1.33 1.25 1.28 1.18 1.24 1.29 1.25 1.11 1.16 1.11	119 120 121 118 117 115 125 128 130 122 124 122	

A general decrease in static joint range of motion is associated with the ageing process and a decrease in joint motion during walking has also been documented. Reduced sagittal plane motion at the hips, knees and ankles have been documented in groups studied over 65 years of age (Murray et al. 1964). Previous studies have highlighted that older women demonstrate significantly smaller maximum range of ankle joint motion than their younger counterparts during walking (Hageman et al. 1986; Kaneko et al. 1991). Winter and associates reported a decrease in the plantarflexor work in late stance in older adults (Winter et al. 1990). These changes may be related to a shorter stride length and a slower walking velocity.

Bendall and associates reported that plantar flexor strength was significantly related to walking velocity in older men and women (Bendall et al. 1989). It is debatable as to whether a reduction in plantarflexor work is a causative factor in the reduction of step length in the elderly or is merely a reflection of the mechanical requirement of a reduced step length (Grabiner 1997). Martin and associates examined the effects of a 16-week program to strengthen plantar flexion strength on the preferred walking speed in younger and older adults. They found that the training program significantly increased the plantar flexion strength in the groups, however, they did not find any change in the preferred walking velocity from pre to post training (Martin et al. 1992).

2.5.5. Gait characteristics associated with Diabetes

It is widely acknowledged that the majority of ulcers on the plantar surface of the foot in diabetics are sustained from repetitive loading of tissues during walking (Katoulis et al. 1997a; Cavanagh et al. 1991b). Losses of protective sensation combined with changes in gait characteristics associated with diabetes are thought to be responsible for the increased level of tissue damage, failure and subsequent ulceration. Despite the importance of gait in the pathogenesis of foot injury there have been relatively few studies on the gait characteristics of patients with diabetes. Changes in postural stability, spatial and temporal parameters, joint movement and joint momentshave been associated with diabetes and diabetic neuropathy.

2.5.5.1 Postural stability

Diabetic neuropathy is thought to result in poor postural control and instability during gait, which is related to an increased risk of injury during walking. In a retrospective study Cavanagh and associates reported increased levels of injury in patients with neuropathy compared to non-neuropathic controls. They found that the neuropathic group had an odds ratio of 15 for injuries during gait compared to the non-neuropathic group (Cavanagh et al. 1992a). Many studies have reported increases in body sway associated with increased neurological deficit (Boucher et al. 1995; Courtemanche et al. 1996; Uccioli et al. 1995). The increase in body sway in patients with neuropathy has been attributed to changes in prorioception and instability of foot posture related to muscle weakness. Instrumentation for the accurate assessment of joint movement and position perception is not widely available, therefore many studies utilise body sway / balance tests as an indirect measure of joint movement position sense. Balance tests are assumed to provide a good assessment of proprioceptive function, however, these tests are influenced by other factors such as muscle strength and reaction time (Simoneau et al. 1996).

Simmoneau and associates developed apparatus to measure ankle joint movement perception in the sagittal plane in a weight-bearing position. They measured the joint movement perception threshold in diabetic patients with and without cutaneous sensory deficit as determined by monofilamant testing and they found that diabetic patients with neuropathy demonstrated a significant loss of ankle joint movement perception (Simoneau et al. 1996). The study showed that patients with sensory deficit had significant postural instability compared to the controls and they reported a shift in the usage from a predominantly ankle based method of compensation in balance to a hip correction strategy as the difficulty in maintaining balance increased. They found that the joint movement perception threshold was a good predictor of instability during stance (Simmoneau et al 1994). A decrease in the joint movement perception for subtalar joint eversion and inversion in diabetic patients with peripheral neuropathy has also been reported (Van den Bosch et al 1995).

Significant differences in body sway in the frontal plane have been reported between patients with neuropathy and foot ulceration compared to diabetics with neuropathy (Katoulis et al. 1997a). The authors attributed the significant increase in body sway in

this group to detrimental changes in proprioception and instability of foot posture related to weakness of the intrinsic muscles of the foot. A possible explanation is that the treatments for ulceration (limited weight bearing and prolonged disuse of lower limb) might cause muscle weakness, changes in joint mobility and increase the instability within the foot. The role of proprioception in the pathogenesis of ulceration needs to be confirmed in a prospective study.

2.5.5.2 Spatio-temporal characteristics of gait in diabetes

Patients with diabetic neuropathy show a more conservative gait pattern than their non-neuropathic counterparts. Their gait is charcterised by a marked decrease in walking velocity, a shorter stride length, a longer cycle duration and a slower cadence (Katoulis et al. 1997b; Mueller et al. 1994a; Courtemanche et al 1996; Shaw et al. 1998). Mueller and associates reported that the mean walking velocity for a group of diabetic patients with neuropathy with a mean age of 57.7 years was 1.06 metres per second. The walking velocity in the non-diabetic age matched control group was 1.26 metres per second. A smaller percentage of the gait cycle is spent in single limb support and a greater proportion of time is spent in double limb support (Mueller et al. 1994a). This gait pattern is thought to be a compensatory mechanism for the increased postural stability associated with neuropathy. It has been reported that diabetic neuropathic patients have a low level of perceived safety when walking, they lack confidence and have a fear of falling during gait (Cavanagh et al. 1992a).

2.5.5.3 Kinematics

2.5.5.3.1. Motion at the AJC

There have been surprisingly few studies on dynamic joint motion at the AJC during walking in patients with diabetes. A small number of studies have reported limited ankle joint dorsiflexion and plantar flexion during gait in patients with diabetes compared to age matched diabetic controls (Mueller et al. 1994a; Mueller et al. 1994b). The mean total range of ankle joint motion during gait was 30.6 (SD 4.1) degrees and 22.1 (SD5.4) degrees for the non-diabetic and diabetic groups respectively. It has been suggested that the reduction in ankle joint motion during gait can result in a shorter step length and reduced ankle moments during gait (Mueller et

al. 1995). Limited movement at the ankle joint has also been offered as an explanation as to why some diabetic patients with neuropathy use the hip correction strategy for the maintenance of balance. Restriction in ankle joint flexibility reduces the limits of sway available and this compounds the postural instability problems, therefore, maintaining balance using the hip strategy is preferred.

Limited dorsiflexion at the ankle joint has been associated with foot ulceration. Holewski and associates found that patients with a history of foot ulceration or amputation had a significantly higher prevalence of limited dorsiflexion at the ankle joint when compared to diabetic patients without a history of ulceration (Holewski et al. 1989). Limited dorsiflexion at the ankle joint has also been reported to result in abnormal subtalar joint pronation, which causes hypermobility in the forefoot during the propulsive phase of gait. The abnormal subtalar joint pronation is thought to increase both the vertical and shearing forces under the foot. Limited joint mobility determined from static joint assessment has been demonstrated at the ankle and subtalar joints in patients with diabetes (Delbridge et al. 1987). Limited joint mobility at these sites has been associated with increased plantar foot pressures and ulceration (Fernando et al. 1991; Mueller et al. 1989). Only one study to date has examined dynamic ranges of motion at the AJC and its impact on plantar foot pressures and ulceration (van Schie et al. 2000). The total range and timings of joint motion in all three planes at the AJC is not reported. The authors found an association between the rate of plantar flexion after heel strike and a history of ulceration.

2.5.5.3.2. Motion at the 1st metatarsophalangeal joint

Sagittal plane motion at the 1st metatarsophalangeal joint (MPJ) is very important for normal walking. The minimum range of dorsiflexion necessary for normal locomotion is reported to be approximately 65-75 degrees (Root et al. 1977). More recently it has been suggested that normal foot function is dependent on an adequate dynamic of range of motion at the 1st MPJ (Payne 1998). Restriction of dorsiflexion at the 1st MPJ is associated with high pressures under the hallux and subsequent ulceration in the presence of diabetic neuropathy (Birke 1988). Severe restriction of sagittal plane motion at the 1st MPJ determined by static joint measurements have been reported in patients with diabetes (Larsen & Hostein 1987; Birke 1988; Delbridge et al. 1987; Fernando et al. 1991; Duffin et al 1999). Very few studies have

attempted to evaluate the range of motion at the 1st MPJ during walking using gait analysis techniques. Cavanagh and associates presented joint motion at the 1st MPJ in one subject with diabetes and limited joint mobility. The maximum dorsiflexion at the joint was 33 degrees, approximately 90 percent of the range determined by static joint assessment (Cavanagh et al. 2001b). The sagittal plane facilitation model suggests that dynamic range of motion at the 1st MPJ is independent to the range of motion determined during clinical examination. The relationship between static joint range of motion and dynamic range of motion needs to be investigated in a larger sample size.

2.5.5.4. Kinetics

A joint moment is a measure of the net effect of all the muscle activity which causes rotation about a given joint and is used as an indicator of overall muscle performance (Mueller et al. 1995). Joint moments at the ankle during the stance phase of walking typically include a small dorsiflexor moment followed by a large plantar-flexor moment, the power generated by the plantar flexors at the end of the stance phase is typically the largest muscle power burst recorded during gait. The moment pattern at the hip usually shows an extensor moment followed by a flexor moment. Ankle moments and power are known to be limited during walking in patients with diabetes and peripheral neuropathy (Andersen et al. 1997; Mueller et al. 1994a; Mueller et al. 1994b). Limited joint mobility at the AJC and loss of muscle function associated with motor neuropathy are linked with the decrease in joint moment at the ankle. Mueller and associates compared the gait characteristics of patients with diabetes and a history of neuropathic ulceration to age matched non-diabetic controls. They found that the diabetic patients had less ankle joint motion in the sagittal plane, a slower walking velocity, a shorter stride length, lower peak ankle moments and lower peak ankle power (Mueller et al. 1994a). They concluded that the decreased plantar flexion strength reduced the amount of push off at the end of the stance phase, this resulted in shorter steps and a decrease in the walking velocity in the diabetic group compared to the controls. The diabetic group had greater hip moments and power than the ankle moments and power during terminal stance. The diabetic group appeared to use the hip flexor muscles to pull the limb forward (hip strategy) rather than the pushing the limb into swing with the ankle plantar flexor muscles (ankle strategy). The study found that ankle plantar flexion strength and mobility rather than walking velocity,

sensory loss or other complications of diabetes were the primary factors contributing to the changes in gait style in the diabetic group. The authors suggested that the hip strategy might place lower pressures under the forefoot, due to the generation of lower shear forces and minimal push off. A further study by the same group was performed to examine the effect of hip and ankle strategies on peak pressures under the feet in diabetic patients with a history of ulceration (Mueller et al. 1994b). The results showed that the adoption of the hip strategy resulted in a 27% reduction of peak pressures in the forefoot.

Motion analysis has facilitated a greater understanding of the mechanics of gait in patients with diabetes and its relationship to the pathogenesis of ulceration. A major limitation of previous work is that sample sizes were very small and usually only motion in the sagittal plane has been studied. Abnormal frontal plane motion at the AJC has been related to a number of musculoskeletal problems including high plantar pressures and ulceration in the neuropathic foot. The dynamic range and pattern of motion at the AJC and 1st MPJ in all three planes is needed in order to gain an insight into the pathogenesis of ulceration.

2.6 Pressure Measurement in the diabetic foot

This section aims to briefly summarise the relevant literature on plantar pressure measurement and its relationship to the development of foot ulceration. The prevalence and distribution of high foot pressures and the mechanisms by which pressures become elevated in the diabetic foot will be explored before a discussion of the limitations associated with pressure measurement.

In recent years pressure measurement has become widely available and has been used in both a research and clinical environment. The increased use of pressure measurement techniques has facilitated a greater understanding as to why people with diabetes tend to have high pressures under their feet and the causative role high pressure has on the formation of foot ulceration. Many commercial systems are available for the measurement of barefoot and in-shoe plantar pressures. Both in-shoe and barefoot pressure measurement systems have inherent advantages and disadvantages, which have been discussed elsewhere in the literature (Cavanagh et al. 2001b). The technological and methodological issues associated with plantar pressure measurement have been extensively reviewed elsewhere (Cavanagh et al. 1991b; Cavanagh et al. 1992b; Lord 1981; Lord et al. 1986; Cavanagh et al. 2001b). Pressure measurement as an indicator of overall foot function in the diabetic foot has been studied extensively and has been used as both a diagnostic and an outcome measurement tool. A detailed critical review of all the literature in this area is beyond the scope of this thesis. This section will provide a brief overview of the key findings of pressure measurement in the diabetic foot and the limitations associated with pressure measurement.

2.6.1. Prevalence of high pressures and its relationship to foot ulceration

Abnormally high plantar foot pressures are commonly found in patients with diabetes, especially in the presence of neuropathy (Boulton et al. 1987a; Veves et al. 1992a). The prevalence of high foot pressures in patients with diabetes and in non diabetic control has been reported (Boulton et al. 1983). In this study Boulton and associates found 51% of neuropathic feet studied had abnormally high pressures underneath the metatarsal heads compared with 7% in the non-diabetic control subjects. Veves and

associates using the same pressure measurement system reported high pressures under 30% of all diabetic feet. The prevalence of high foot pressures in the diabetic group without neuropathy was similar to the non diabetic control group, 15% and 14% respectively (Veves et al 1988).

It is widely recognised that high plantar foot pressures have a contributory role in the ulcerative process. Frykberg and associates performed a cross sectional study to ascertain the risk of ulceration with high foot pressures and neuropathy in a large group of patients with diabetes. They found that patients with high foot pressures (greater than 6Kg/cm^2) were twice as likely to have ulcerated than those without high foot pressures (Frykberg et al. 1998). The role of high pressures in the development of foot ulceration has been confirmed in a prospective study (Veves et al. 1992a). In this study 86 diabetic patients were followed for a mean period of 30 months. At the baseline measurements, 50% of diabetic patients had abnormally high foot pressures and 67% of patients had established peripheral neuropathy associated with their diabetes. The mean peak foot pressure in diabetic patients was higher at the follow up visit when compared to the baseline measure, no such difference was identified in the non diabetic control group. During the study 17% of diabetic patients developed ulceration, all of these patients had abnormally high foot pressures at the baseline measurement. Murray and colleagues investigated the relationship between callus formation, high pressures and foot ulceration in diabetic patients with neuropathy (Murray et al. 1996). They found that presence of callus at the onset of the study was predictive of ulceration. The relative risk for ulceration in a callused area was 11.0, compared to a relative risk of 4.8 for a high pressure area. Callus is known to form at areas of high mechanical stress and has been shown to increase plantar pressures (Young et al. 1992a).

Foot ulceration tends to occur at sites of maximum force and pressure (Ctercteko et al. 1981; Stokes et al. 1975; Boulton et al. 1983). High pressures have also been reported at the sites of previous ulceration (Boulton et al. 1987a). Although high pressures have been identified as a major aetiological factor in the formation of foot ulceration it must be noted that high pressures in the absence of established neuropathy do not lead to ulceration (Masson et al. 1998).

2.6.2. Why are pressures elevated in the diabetic foot?

In early literature, obesity has been cited as an explanation for increased foot pressures in diabetes. Previously it has been noted that many diabetic patients are heavier than their age and height matched non-diabetic counterparts and this may increase foot pressures. More recently it has been suggested that foot structure is the dominant factor in determining plantar pressure and that body mass is a poor predictor of plantar peak foot pressures (Cavanagh et al. 1991a). In a later book chapter the same author concluded that a gain in body mass may or may not increase plantar pressure, depending on whether there is deposition of adipose tissue in the plantar tissues of the foot (Cavanagh et al. 2000).

Diabetic neuropathy has been postulated to be one of the main factors, which contributes to the development of high foot pressures (Boulton et al. 1983). It has been reported that early changes in the distribution of pressure under the feet may precede the development of clinical neuropathy (Boulton et al. 1987a). A mixture of sensorimotor and autonomic nerve dysfunction in the foot are probably responsible for changes in both foot structure and function which disrupts the normal gait cycle and increases plantar foot pressures. The intrinsic minus foot shape (retracted toes, prominent metatarsal heads and a high medial longitudinal arch) is associated with diabetic neuropathy. In this foot type there is a decrease in the weight-bearing area of foot thus less area to spread force and the pressure increases. It must be noted that foot structure (not related to neurological dysfunction) and joint mobility plays a large part in determining the plantar pressure exerted by the foot during walking (Cavanagh et al. 1997; Morag et al. 1999).

Limited joint mobility at the ankle, subtalar and 1st MPJ has been associated with high plantar foot pressures and subsequent ulceration in the diabetic foot. Racial differences in joint mobility have been reported (Frykberg et al. 1998). Caucasian people with diabetes have been shown to have significantly reduced mobility at the ankle, subtalar and first metatarsalphalangeal joint, significantly higher foot pressures and an increased prevalence of foot ulceration compared to Asian, Hispanic, and Afro-Caribbean patients (Clarke et al. 1998). The presence of foot deformity is associated with increased plantar foot pressures and a greater risk of developing foot ulceration. Prominent metatarsal heads are associated with higher foot pressures

under the metatarsals. Hallux valgus and hallux limitus foot deformities have been shown to be associated with higher pressures under the hallux but lower pressures under the metatarsal heads (Frykberg et al. 1998).

Cavanagh and associates have examined the relationship between foot structure and function and high plantar pressures during barefoot walking in fifty non-diabetic healthy subjects (Cavanagh et al. 1997). In the study, standardised weight-bearing radiographs were taken and 27 radiographic measurements were taken in order to characterise foot structure. Plantar pressure measurements were recorded using the optical pedobarograph and peak pressure at the heel and first metatarsal head was determined. Stepwise multiple regression was used to identify the structural factors associated with high plantar foot pressures. The compressed soft tissue thickness (determined by calcaneal or sesamoid height) and the height of the medial longitudinal arch were identified as the strongest predictors of plantar pressure both under the heel and the first metatarsal head (Cavanagh et al. 1997).

Cavanagh and associates found that using only structural characteristics in the regression allowed them to predict only 31% and 38% of the variance in peak plantar pressure at the heel and the first metatarsal head respectively. In a further study many other functional variables related to gait style were included in the regression model (Morag et al. 1999). With the addition of the functional characteristics (for example range of motion at the ankle joint) in the regression, they found that 50% of the variation in plantar pressure could be predicted. In the rearfoot both structural and functional variables were important in prediction of peak pressure. Under the first metatarsal head structural variables were most important at predicting peak pressure at this site.

The work by Cavanagh's group highlights the importance of combining pressure measurement with gait analysis techniques in order to fully understand why pressures are elevated in some individuals and not others (Cavanagh et al. 1997; Morag et al 1999). The findings from their study may help clinicians to predict areas of high pressure under the feet during walking. A similar study in a diabetic population is needed to gain a greater insight into what causes high pressures under the diabetic foot and to identify any additional possible mechanisms by which plantar pressures can be reduced.

2.6.3. Plantar pressure distribution in the diabetic foot

The pattern of pressure distribution under the normal foot during walking has been extensively documented. There is a lack of agreement as to the pattern of loading in the forefoot. Peak pressures have been reported to be under the second metatarsal (Betts et al. 1980; Rodgers et al. 1989) under the third metatarsal (Soames 1985) and under the first metatarsal (Stokes et al. 1975; Stott et al. 1973). There is wide variability in the magnitude and location of peak pressure in the normal population. Regional pressure parameters taken from a number of papers are presented in Table 2-7. Although the same measurement equipment is used in the majority of papers presented wide variability can be seen in the magnitude of peak pressures in anatomical areas. The wide variability is most probably due to different methodologies used and wide inter-subject variability in the normal population.

The literature regarding the distribution of pressure under the diabetic foot is conflicting. Stokes and colleagues studied the plantar pressure distribution in diabetic patients with and without ulceration and in non diabetic controls (Stokes et al. 1975). They found that patients with diabetes who had ulcers had significantly greater loads under the foot than both patients without ulcers and non-diabetic controls. They reported a significant decreased load bearing on the toes and more lateral distribution of pressure in diabetic patients when compared with the control group. Ctercteko and associates studied diabetic patients with and without ulceration and non-diabetic controls. They reported a medial shift of the load under the forefoot and less pressure under the toes in patients with diabetes (Ctercteko et al. 1981). More recently Veves and colleagues reported that they did not find a transfer of pressures either medially or laterally under the diabetic foot (Veves et al. 1992b). A general consensus in the literature has noted several changes in the distribution of pressures under the diabetic foot. The changes include a rise in pressures under the forefoot and a transfer of pressures from the heel to the metatarsal heads. Lesser toe retraction or clawing, results in a shift of pressure from the toes to the metatarsal heads.

Table 2-7: A summary of the pressure distribution under the plantar surface of the foot

Authors	Bryant et al 2000	Hayafune et al 1999	Cavanagh et al 1991	Bennet et al 1993
Sample Demographics	N= 30 M-12, F -18 Aged 23-68	N= 42 M 19, F 23 Aged 20-59	N= 27 M= 27 Middle aged	N = 86 M= 30, F=44 Aged 18-30
Equipment	EMED-SF Platform	EMED-SF Platform	EMED-SF Platform	Musgrave Footprint ®[1]
Method	Second step 3 Trials analysed	Mid gait 1 trial analysed Free walking	First step - Trials	Midgait Cadence controlled 3 Trials analysed
Heel	PP 350 (7.8) MP 167 (2.4) PTI 80 (2.1)	PP - MP- PTI -	PP 341 (98) MP - PTI -	PP 441.3 (49.0) MP - PTI -
Midfoot	PP 73 (3.1) MP 39 (2.5) PTI 21 (1.2)	PP - MP- PTI -	PP 64 (18) MP - PTI -	PP - MP - PTI -
First Metatarsal	PP 290 (110.6) MP 122 (33) PTI 91 (35)	PP 372.8 (171.9) MP - PTI -	PP 319 (101) MP - PTI -	PP 294.2 MP - PTI -
Second Metatarsal	PP 420 (147) MP 188 (41) PTI 126 (40)	PP 435 (162.6) MP - PTI -	PP 533 (222) MP- PTI -	PP - MP - PTI -
Third Metatarsal	PP 366 (114) MP 154 (32) PTI 119 (38)	PP 340.7 (100.3) MP - PTI -	PP - MP- PTI -	PP - MP - PTI -
Fourth Metatarsal	PP 251 (103) MP 114 (39) PTI 88 (3.8)	PP 213.5 (79.3) MP - PTI	PP - MP- PTI -	PP - MP - PTI -
Fifth Metatarsal	PP 249 (207) MP 89 (4.3) PTI 7.5 (5.5)	PP 128.5 (78.5) MP - PTI -	PP - MP - PTI -	PP 225.6 (98.1) MP - PTI -
Lateral metatarsals	PP - MP- PTI -	PP - MP - PTI -	PP 446 (183) MP - PTI -	PP - MP - PTI -
Hallux	PP 442 (197) MP 139 (38) PTI 110 (63)	PP 462.4 (200.9) MP - PTI -	PP 511 (185) MP - PTI -	PP 343.2 (107.9) MP - PTI -
Second Toe	PP 223 (93) MP 78 (2.5) PTI 50 (26)	PP 214.9 (99.5) MP - PTI -	PP 238 (128) MP - PTI -	PP - MP - PTI -
Lateral Toes	PP 159 (78) MP 50 (18) PTI 38 (20)	PP 139.6 (85.1) MP - PTI -	PP 206 (116) MP - PTI -	PP - MP - PTI -

PP= Peak pressure in kPa, MP= Mean Pressure in kPa, PTI= Pressure Time Integral in kPa .sec,
Standard deviation in parenthesis.

2.6.4. Pressure threshold for Ulceration

Despite extensive work in the area of pressure measurement a universal threshold level at which ulceration will occur in the diabetic foot has not been identified. Boulton and associates have defined the upper limit of normal pressure (mean pressure plus 1 standard deviation) as 1,207kPa and have suggested a danger threshold for ulceration to be 1080kPa using the optical pedobaragraph (Boulton et al. 1983). Cavanagh and associates have reported ulceration at much lower pressure values using the Emed SF Pressure Platform. They regard peak pressures under the metatarsals and hallux greater than 500kPa as possible danger of ulceration (Hsi et al. 1993).

Several factors identified in the literature complicate the development normal pressure values and a threshold value for diabetic foot ulceration and are summarised below;

- Different foot regions may have different thresholds for ulceration. The heel for example is specially adapted to withstand high pressures during walking and will have a high pressure threshold, whereas an area in the mid-foot that does not normally bear weight may not be able to withstand high pressures and is more likely to ulcerate.
- Different pressure measurement systems and methodologies will yield different results. Comparison between pressure data recorded on different systems is not valid.
- There is wide variability of peak plantar pressures in the normal population.
- Ulceration has been show to occur at pressure levels that can be seen in normal healthy subjects.
- Many other factors in addition to plantar pressures have been identified as important in the pathogenesis of ulceration.

A number of different pressure parameters can be determined from most pressure measurement systems, however, most literature focuses on the peak plantar pressure value. There is no standardised method on how to collect pressure data or how to analyse and present pressure data. More recently it has been recognised that both the magnitude and the duration of pressure is important in determining tissue damage. The development of ulcers is dependent on the duration of exposion, rather than the magnitude of pressure alone (Cavanagh et al. 1991a). Pressure time integrals are a

measure of both the magnitude and duration pressure and can be calculated for specific regions of the foot. Until recently, very few authors have quoted the pressure time integral in research articles, the value of this variable as a screening measure to predict for ulceration in the diabetic foot has yet to be fully investigated.

2.6.5. Limitations of Plantar Pressure Measurement in the study of diabetes related foot disease

A major limitation associated with both barefoot and in-shoe pressure measurement is the large range of values encountered in normal healthy subjects, which results in a high standard deviation. For other data sets, normal values are defined as the mean plus or minus two standard deviations. When examining pressure data sets the upper value of normal using the normal criterion would result in pressure values previously shown to be associated with ulceration being defined as normal. Many authors have attempted to define normal pressure values under specific regions of the foot (summarised in Table 2-7). In all of the studies, wide variation in plantar pressures between individuals was reported. In one study by Cavanagh and associates they found that one area of the foot (the medial midfoot region) had a coefficient of variation of 118% and the range between the upper and lower limits of pressure in some areas was more than 800kPa (Cavanagh et al. 1987).

It has been shown that plantar pressure measurement is sensitive but not specific for predicting foot ulceration. Armstrong and colleagues found pressure measurement was 100% sensitive at predicting ulceration in patients with neuropathy but had a low specificity (45%) (Armstrong et al. 1998c; Stacpoole-Shea et al. 1999). In contrast to the previous study, Pham and associates found that plantar foot pressures offered the best specificity for a single factor in a more recent prospective study of 248 patients followed for a mean period of 30 months. They found that combining foot pressures with the Neuropathy Disability Score offered the best combination for specificity and sensitivity (Pham et al. 2000).

Plantar pressure measurement has been shown to be useful in the assessment of the diabetic foot; however, the wide variability encountered in the normal healthy population hampers its use in clinical practice. It has been shown that both the structure of the foot and aspects related to gait style are important factors when

attempting to predict high foot pressures and ulceration. A combination of pressure measurement with gait analysis techniques would facilitate a greater understanding of the mechanisms that lead to diabetic foot ulceration.

2.7 Literature summary

This section provides a brief summary of the relevant literature and justification to the aims and scope of the study. The hypotheses to be tested during this study are presented.

The prevalence of diabetes is rising rapidly and it is estimated that in the year 2010 diabetes will affect 239 million people worldwide. The management of diabetes and its complications is costly both in economic and human terms. Diabetic foot ulceration has been shown to have a negative impact on the quality of life of patients and their families. The prognosis for patients with foot ulceration is poor, many will ulcerate again and some may require an amputation.

Foot ulceration has traditionally been considered to be the result of vascular disease, peripheral neuropathy and infection. More recently advances in technology have provided a mechanism by which to increase our understanding of the role that abnormal foot function and structure has on the pathogenesis of ulceration. Abnormally high plantar foot pressures are commonly found in patients with diabetes and ulceration has been linked to high plantar pressures in both retrospective and prospective studies. Although plantar pressure measurement has facilitated a much greater understanding as to why many diabetic patients with neuropathy ulcerate there are several limitations inherent with using pressure studies alone. The main limitation is that in a normal healthy population there is a wide variability in pressure values making definition of normal and abnormal pressures difficult, as many diabetic patients ulcerate at pressures that would be defined as within normal limits. For this reason it has not been possible to define a pressure threshold which if exceeded would result in ulceration in patients with diabetic neuropathy.

The measurement of pressure between the terminal part of the foot and its interface with the supporting surface reflects a mix of joint movement, muscle activity and lower limb structure all intricately coordinated by the central nervous system. All these factors will have an influence on the magnitude and pattern of pressure seen under foot. In an attempt to try and explain the wide variability of pressure found in

normal healthy population a number of workers have studied aspects of foot structure and gait style and its influence on plantar pressures. They found that both structural and functional variables were important in prediction of peak plantar pressure. Gait parameters have been shown to be important in predicting pressures under foot and can explain help to explain why pressures are elevated in some individuals and not others.

Many characteristic gait patterns have been identified in people with diabetes especially in those with diabetic neuropathy and ulceration. Postural stability, spatial and temporal parameters, joint movement and joint moments can all be altered in the presence of diabetic neuropathy. Gait style has been offered as a reason as to why some patients ulcerate and others do not and it has been proposed that training patients to alter their walking pattern may help to prevent ulceration.

A generalised limitation of joint mobility has been demonstrated in patients with diabetes. Most workers investigating joint mobility have found relationships between reduced joint mobility at the subtalar and metatarsophalangeal joints and increased foot pressures and prevalence of ulceration. The assessment of joint mobility has been based on the static assessment of joints using goniometers. Many studies have shown that taking joint measurements in this way is subject to large errors and that there is poor correlation between static measures of the foot and dynamic foot function. Work in this area could be enhanced if the range of joint motion at joints in the feet were determined during gait.

Assessment of gait in screening programs remains to be a neglected area. In clinical practice subjective assessments of gait if any invariably occur. Objective assessment of gait is beset by financial and time constraints. Recent advances in technology have reduced the cost and time needed to perform gait assessment and has provided the opportunity to accurately and objectively assess gait within the confines of a clinical environment.

2.7.1. Aims and scope of the present study

This study aims, for the first time to describe motion at the AJC in all three planes of motion during the stance phase of gait in a diabetic population. This study will investigate the differences in the joint motion at the AJC and the 1st metatarsophalangeal joint during gait in the following groups; diabetic patients who have neuropathic foot ulceration, patients with diabetic neuropathy but no history of ulceration and patients with diabetes without neuropathy or a history of ulceration. Differences in dynamic joint motion highlighted between the groups may help to explain why some patients develop ulceration.

It is generally assumed that if there is a limited passive range of motion at the AJC and 1stMPJ, the range of motion during gait will also be limited and this explains the mechanism by which limited passive joint motion is associated with high plantar pressures and ulceration. The aim of the present study is to examine the relationship between the passive ranges of motion at the AJC and 1st MPJ and the ranges of motion during gait.

Very few papers have combined plantar pressure measurement with gait analysis techniques. As there are few data regarding dynamic function at the AJC and plantar pressure, this study aims to explore the inter-relationships between the variables in more detail.

Finally, patients will be followed for 12 months following gait assessment and their ulceration status will be monitored. The relationship between motion at the AJC and 1st MPJ and subsequent ulceration will be investigated.

2.7.2. Hypotheses

The following hypotheses are proposed to be tested during this program of work.

- There is a statistically significant difference in the 3D motion time curves at the AJC during stance phase between the following groups;

A) Diabetics with active or previous history of ulceration,

B) Diabetics with established peripheral neuropathy,

C) Diabetic controls (not neuropathic or history of ulceration)

D) Age matched, non-diabetic controls.

- Patients with a history of ulceration will have a significantly lower range of motion at the AJC and 1st MPJ during walking, than the neuropathic and diabetic control groups.

- There is a correlation between the dynamic range of motion at the AJC, in all three planes of motion and the location and magnitude of peak pressures in the forefoot.

- There is a correlation between the range of motion at 1st MPJ and the location of peak pressures in the forefoot.

- Reduced dynamic motion at the AJC and the 1st MPJ will be a positive predictor of ulceration.

In order to test the above hypotheses the present investigation has identified four core elements to the program of work;

1) To develop a method by which a commercially available motion analysis system can be successfully applied to study motion at the AJC in a diabetic population.

2) To determine motion time curves and plantar pressure for a normal healthy control group.

3) To employ a method of screening by which to identify diabetic patients with neuropathy.

4) To determine and analyse the motion time curves and plantar pressure in diabetic groups.

A sample of convenience gained from three clinical sites will be used. Using neurological screening and an assessment of the foot, diabetic patients will be assigned to the control, neuropathic or ulcerated group. No formal method of stratification will be used, however, attempts will be made to balance the groups for age, height, gender and disease duration. The non-diabetic reference group will be taken from the normative database matched for gender and age within decade. Electromagnetic tracking will be used to determine the passive range of motion at the AJC and 1stMPJ. The range of motion during gait at both sites will be measured. In shoe plantar pressure measurements will be recorded. Patients in the diabetic groups will be followed for 12 months and their ulceration status monitored.

CHAPTER 3

METHODS AND THEIR DEVELOPMENT

3.1 Development of kinematic technique for measuring three-dimensional movement at the AJC

The aim of this section is to introduce the technology behind 'electromagnetic tracking' and to highlight the potential benefits and limitations of using this technique to study three-dimensional lower extremity kinematics. Electromagnetic tracking has been successfully applied to the AJC using two contrasting methods, however, a review of the literature showed that some key methodological areas needed to be addressed before the technique was deemed suitable for the purposes of the present study. This section will provide a brief description of the electromagnetic tracking system used in this study and how the system was tested and applied to monitor 3D motion at the AJC in the clinical environment.

3.1.1 Background information

The application of gait analysis techniques has contributed to a better understanding of foot biomechanics. Many different techniques exist to measure kinematics at the AJC, of which video-based systems predominate. Motion analysis systems have been critiqued for under-fulfilling initial potential in the clinical setting, with most criticism levelled at high costs coupled with demanding technical and time consuming features inherent with most systems. Recently, electromagnetic tracking (EMT) has been adaptedto measure kinematics in the lower limb during gait. This technique is very convenient to use and overcomes many of the technical problems associated with camera based systems. Motion analysis using camera-based systems is costly with respect to time, (due to lengthy equipment set up), calibration procedures, and post data collection tracking of lost marker points. Motion analysis using EMT requires no digitisation of markers, and the kinematic variables can be calculated within minutes of the data collection; for this reason it has the potential to become a valuable clinical

and research tool. There are several limitations associated with EMT including restricted measurement area, electromagnetic interference from metallic objects in or near to the operational field, relatively slow sample rate when using a number of sensors and cumbersome trailing cables.

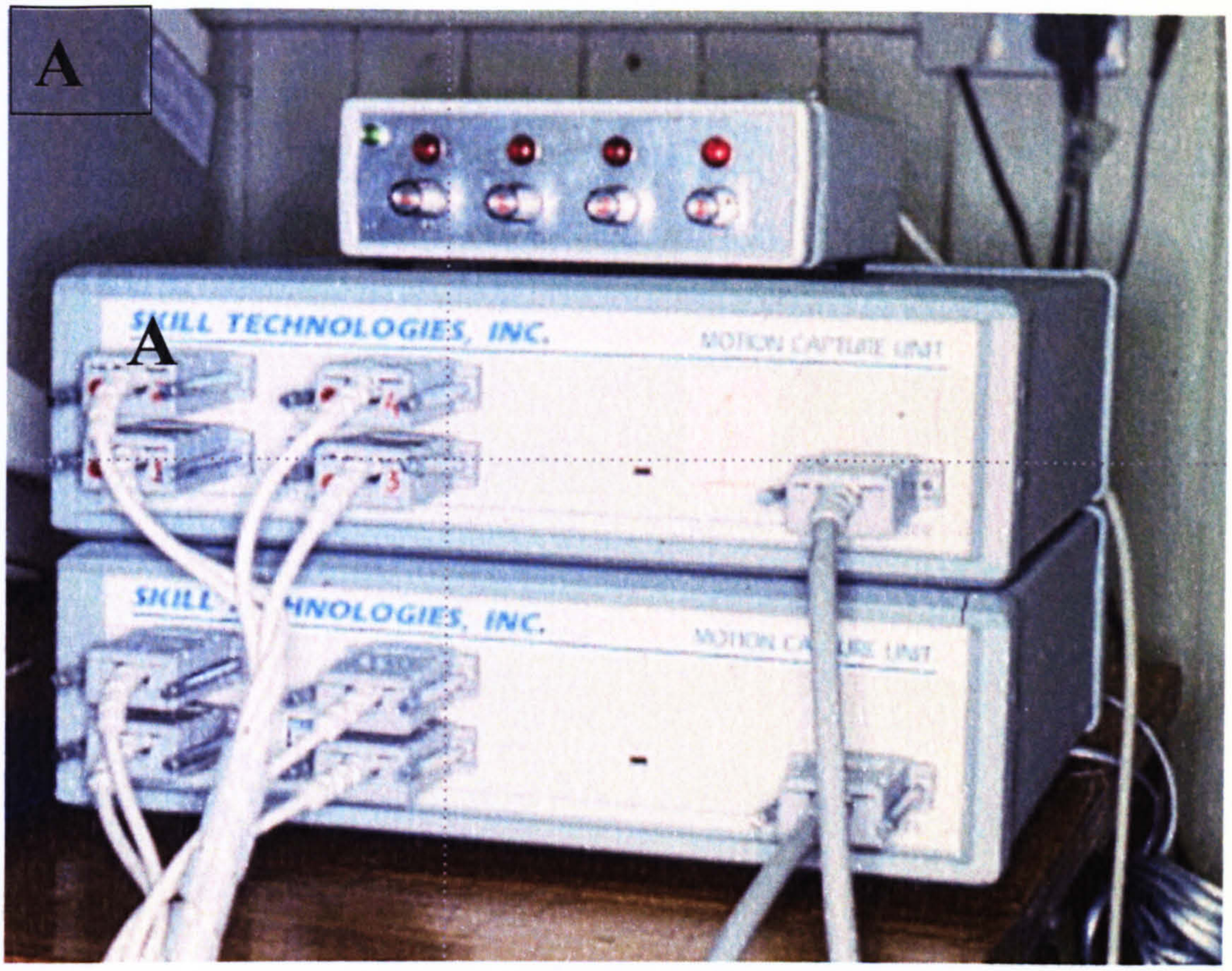
EMT motion analysis systems consist of three major components: a source (transmitter), a sensor and a systems electronic unit. The source emits a low frequency electromagnetic field and the sensors detect this field. Both the source and the sensors are connected to the systems electronic unit. All the information from the source and the sensor is processed in the systems electronic unit and calculations are performed using dedicated in-built software to work out the position and orientation of the sensor. The use of EMT is well established for measuring spinal, knee and in vitro foot kinematics. However, two contrasting techniques have been described for measuring lower limb kinematics during gait. In most studies the subject walks past a fixed transmitter, (Cornwall et al. 1999b; Kobayashi et al. 1997; Mannon et al. 1997) whereas in one study both the transmitter and sensor have been attached to the lower limb (Abboud et al. 2000). Both techniques have been reported as accurate and repeatable.

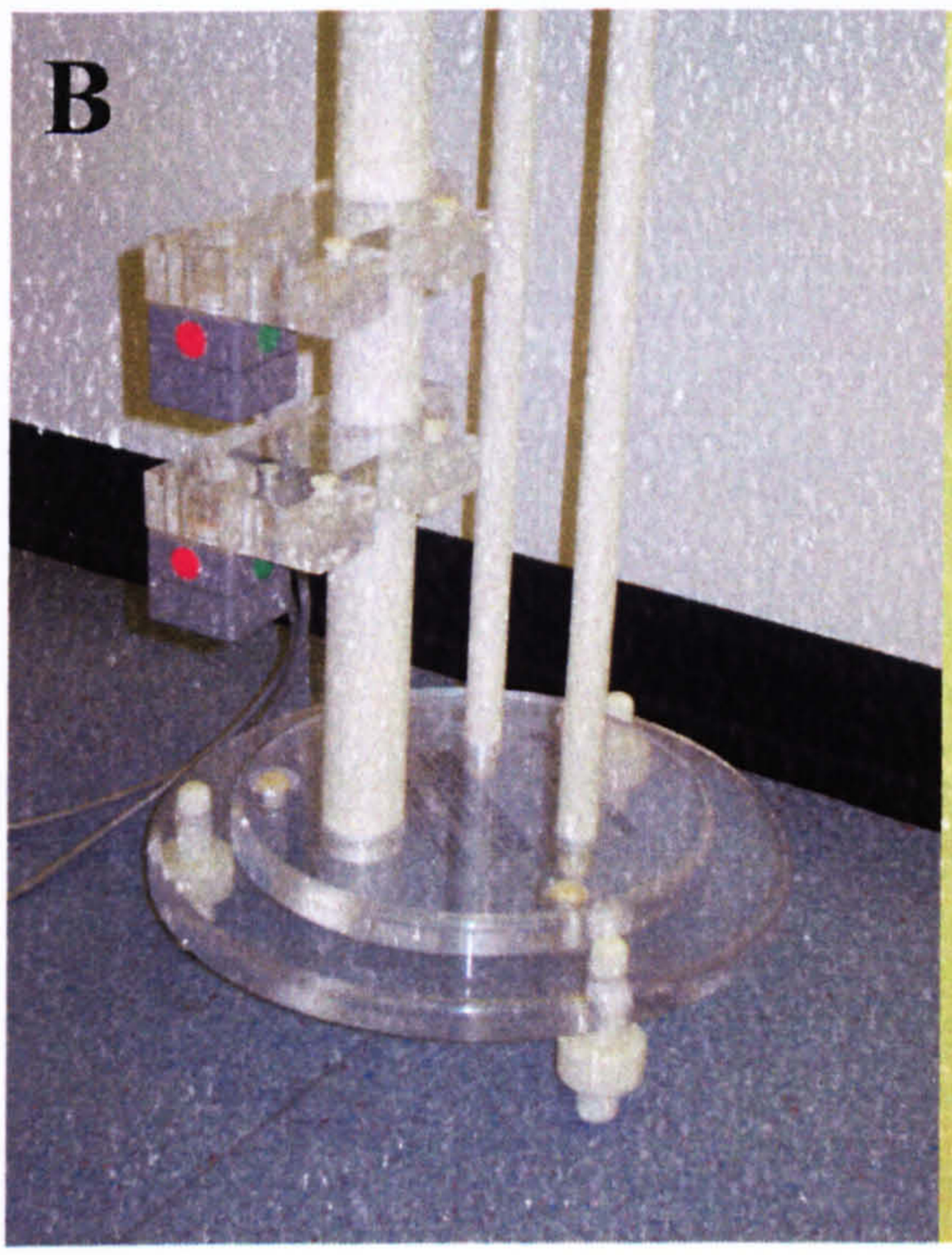
The 6D RESEARCH system (Skills Technologies Inc, Phoenix, AZ, USA) is a commercially available electromagnetic tracking motion analysis system using FASTRAK sensors (Polhemus Inc., Colchester, VT, USA). The system consists of three main components; a motion capture unit (systems electronic unit), a transmitter unit and four Polhemus sensors (Figure 3-1). The transmitter unit emits a low frequency magnetic field (in the region of 1-4000 x 10^{-9} Tesla). The system detects changes in position and orientation of the sensor by updating the previous values by the difference in the magnetic fields detected by the sensor (An et al. 1988). The motion capture unit communicates with custom software on a host PC via a RS-232 COM port. The 6D RESEARCH software uses predefined kinematic models to calculate motion at one or more joints. To define a joint, a sensor must be placed both proximally and distal to the joint; each sensor will be represented by a coloured triad or animated skeletal icon as defined by the user. Data collection is triggered by activation of the capture icon within the software. During data collection gross movement of the sensors can be viewed in real-time. A number of different

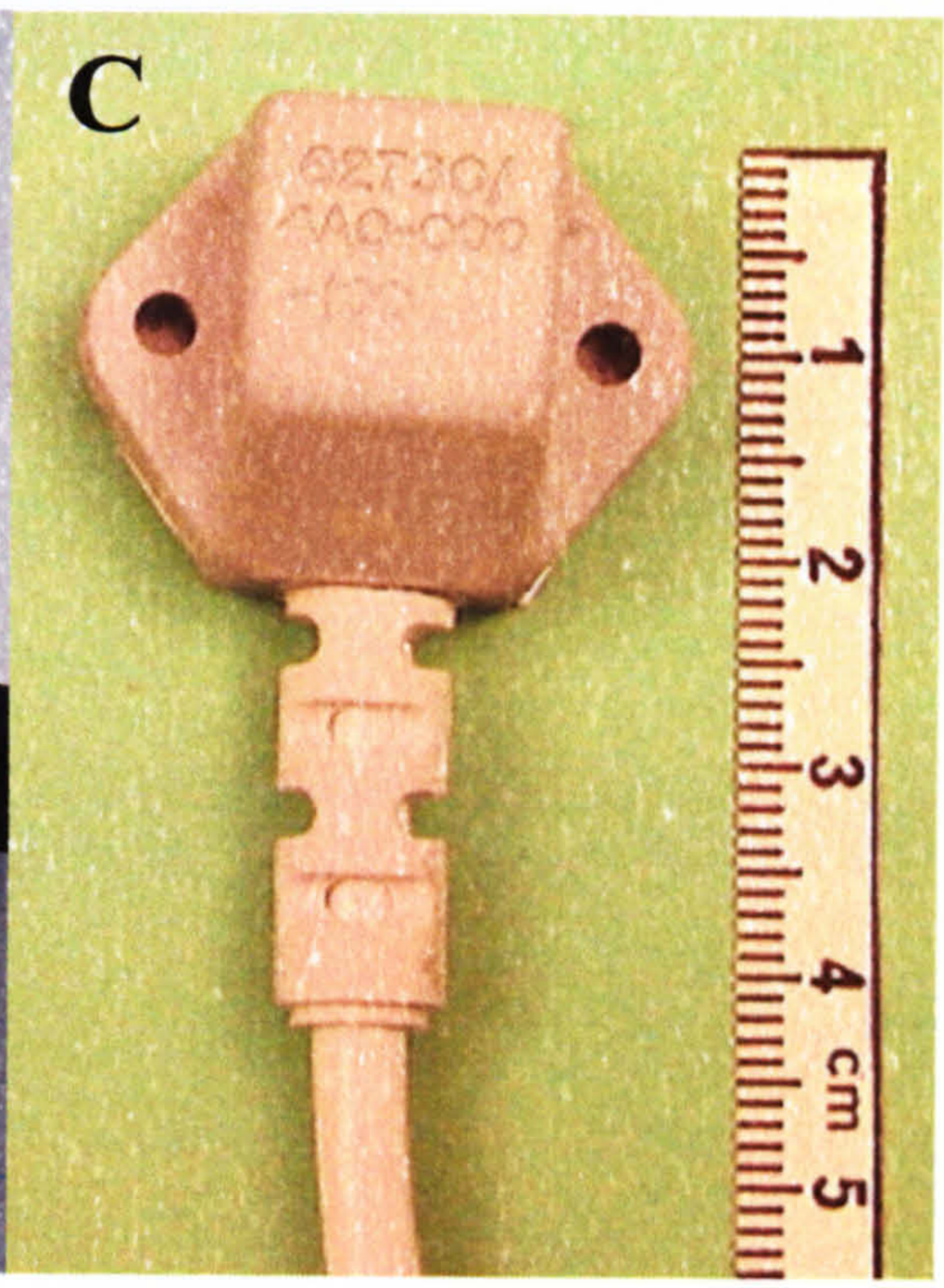
parameters including Euler, projected and joint co-ordinate system angles can then be calculated according to the user specifications within the model.

3.1.1.1 Joint Co-ordinate System for the ankle joint complex

The position of a point in space can be determined with any three fixed axes. Relative motion of two adjacent segments of a joint can be described using six independent variables (degrees of freedom) three for translation and three for rotation. There are several methods for determining the orientation between two body segments in three-dimensional space, each with inherent advantages and limitations which have been discussed in the literature (Nigg et al. 1999). One approach that is widely employed in the field of biomechanics to quantify relative segmental motion is the Joint Coordinate System (JCS). Grood and Suntay in 1983 originally described this method with specific application to the knee (Grood & Suntay 1983). Two segment-fixed axes and a floating axis are used to describe segmental motion. The first axis is embedded in the proximal segment and the second axis is embedded in the distal segment, the third axis is floating and is orthogonal to the first and second axis. More recently the JCS has been used as a method to describe three-dimensional movement at the ankle joint complex. The International Society for Biomechanics Standardisation and Terminology Committee has defined the terminology for describing the JCS for the AJC (Allard et al. 1995) (Figure 3-2), and this will be used for the purposes of this study.



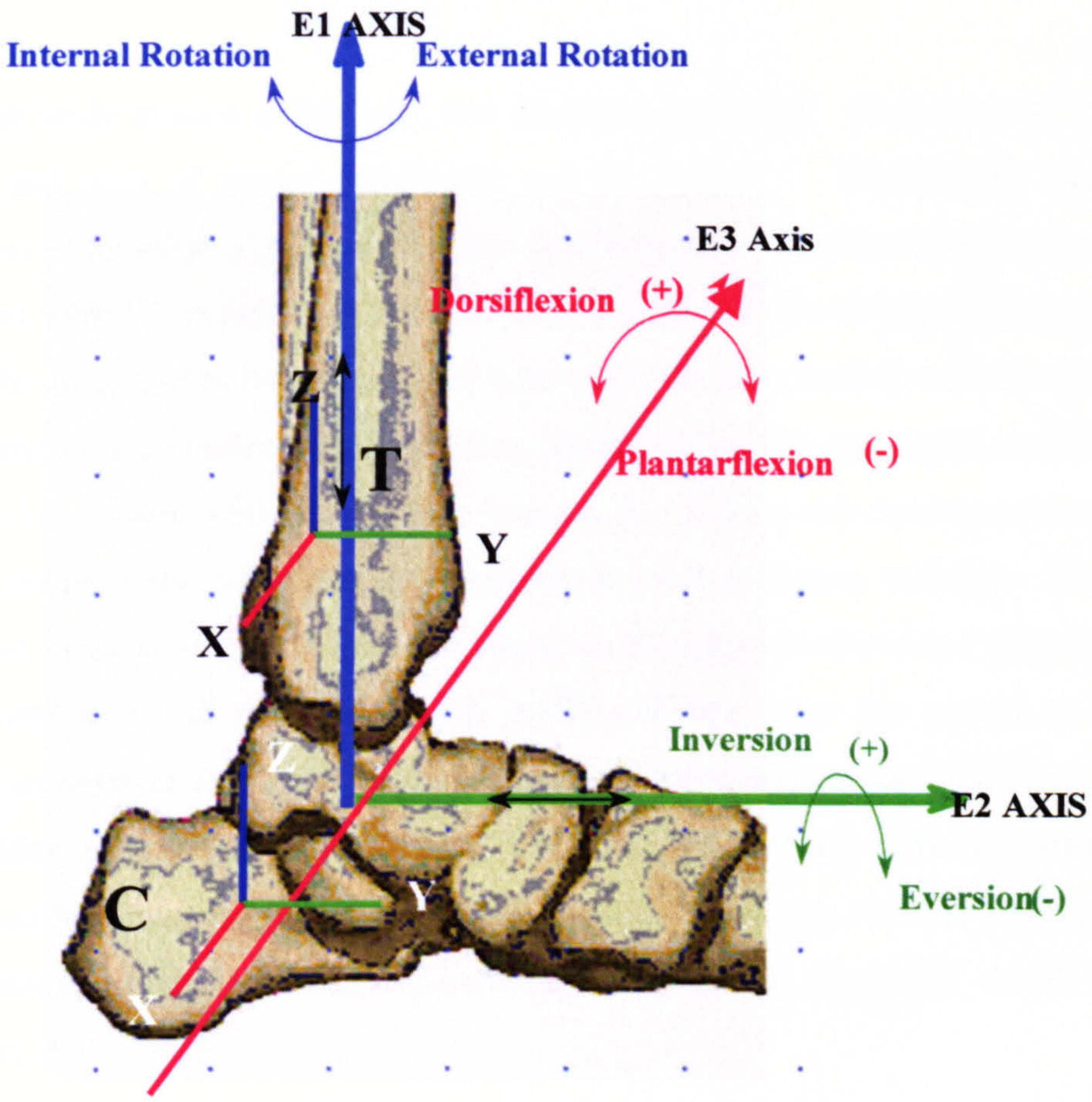




A- Two motion capture units each connected with four sensors and one transmitter, four channel events unit on top of the motion capture units B- Transmitter units on height adjustable stand

C- A Polhemus motion sensor close up

Figure 3-1: Hardware components of the electromagnetic tracking system.



T= Tibia/fibular (proximal segment), C= Calcaneus (distal segment), XYZ (in white)= body fixed anatomical frame of the calcaneus, XYZ (in black)= body fixed anatomical frame for the tibia/fibula, E1 axis = axis fixed to the proximal segment and coincides with the X-axis of the tibia/fibula frame, E3 axis = axis fixed to the distal segment and coincides with the Z-axis of the calcaneal frame, E2 axis = floating axis which is perpendicular to E1 and E3 axis.

Figure 3-2: Joint Coordinate System for the AJC *(Taken and adapted from Allard et al 1995).*

3.1.2. Application of electromagnetic tracking to study kinematics at the ankle joint complex.

3.1.2.1. Sensor attachment and placement

In order to study motion at the AJC, two sensors are needed. A number of different methods and sites of sensor attachment can be chosen. Preliminary work was undertaken to establish a protocol for sensor attachment. The sites selected for sensor attachment were (1) proximal to the joint complex on the anterio-medial aspect of the tibia (at the midpoint between the tibial tubersosity and the medial malleolus) and (2) distal to the joint complex on the posterior aspect of the calcaneus (the central third) (Figure 3-3). These sites were chosen because of minimal sub-cutaneous tissue thus reducing errors associated with skin movement artefact during walking. The most convenient method of attachment was found to be using double-sided adhesive tape between the skin and the sensor with additional tape over the sensor to further minimise movement at the site. Elastic bandage placed around the thigh was used to retain cables, which were passed into a waistband Preliminary data collected using this method had good face validity compared with the findings of other workers using similar motion analysis systems (Cornwall et al. 1999a; Cornwall et al. 1999b; Kobayashi et al. 1997).

In order to be able to identify the temporal parameters of gait, foot switches were interfaced into a four-channel events detection unit, to place a mark in the collected data when loaded. Small thin flexible switches (Interlink Electronics, Santa Barbara, USA) were placed under the plantar heel and under the 1st MPJ to allow identification of heel-strike, foot flat, heel lift and toe-off (Figure 3-4). As many diabetic patients have toes that are not weight bearing during stance phase the 1st MPJ was chosen for identification of toe-off (Ctercteko et al. 1981; Stokes et al. 1975).

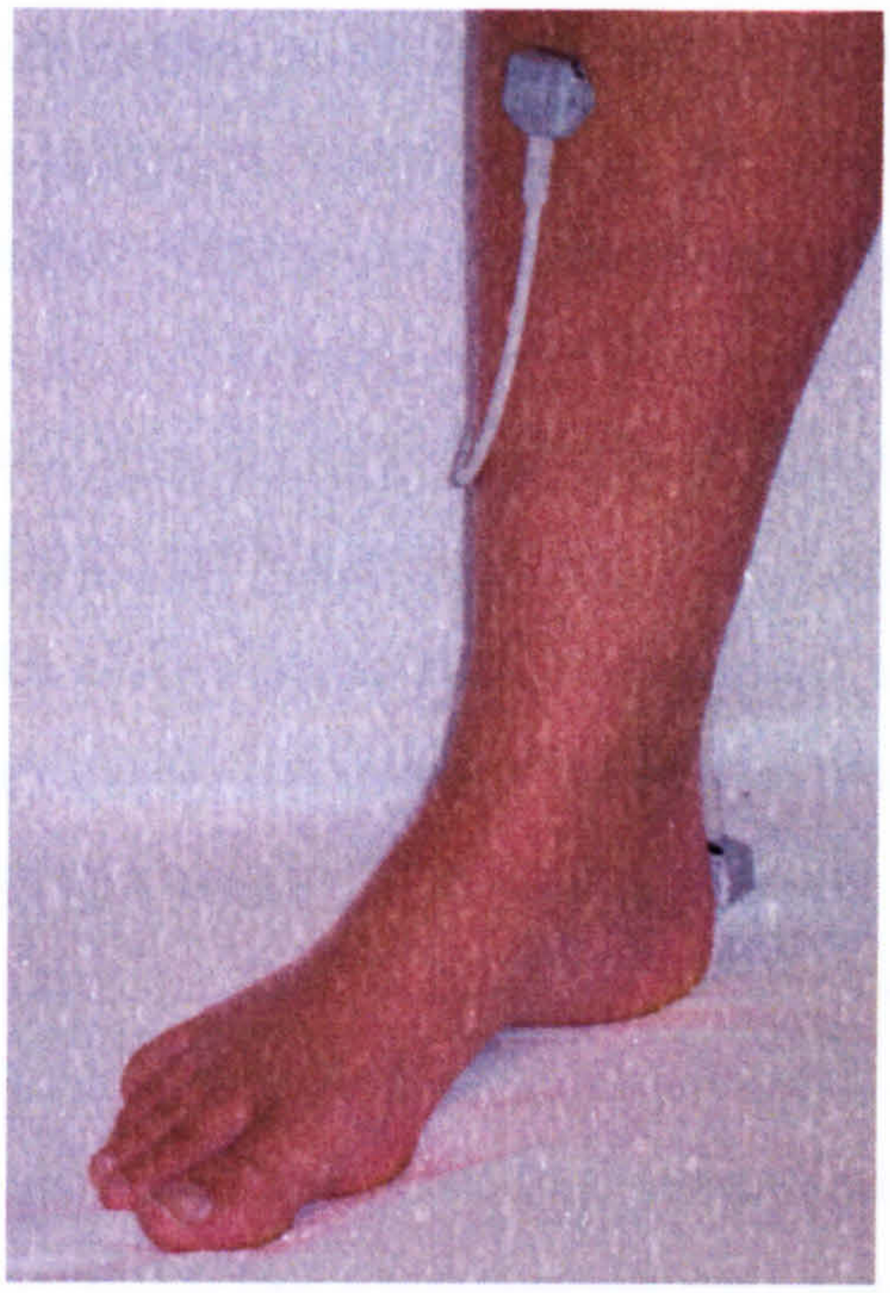


Figure 3-3: Sensor placement for data collection.

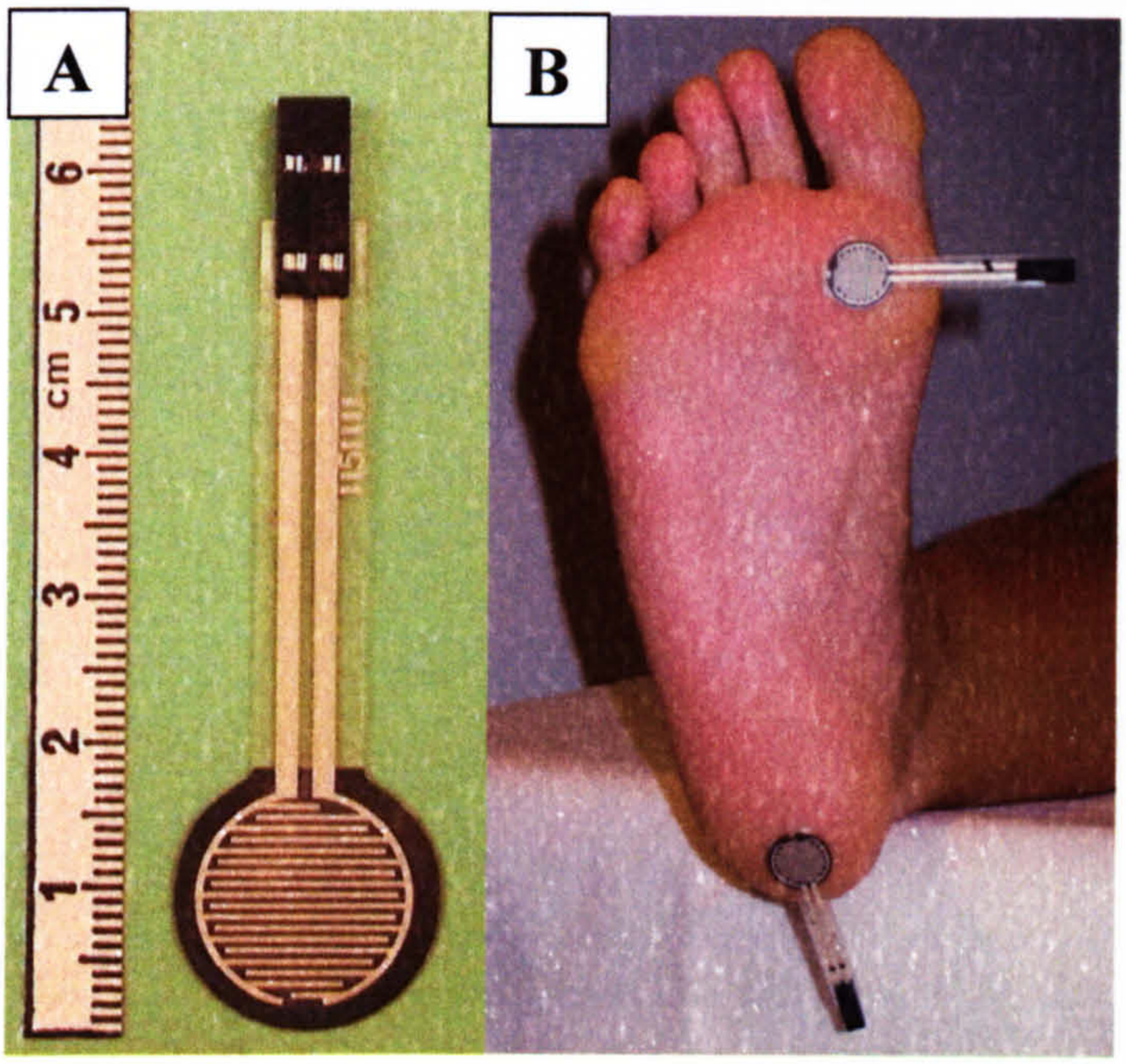


Figure 3-4: Location of footswitches

A- Interlink pressure switch B- Location of footswitches, placed over heel and 1st MPJ .

3.1.2.2. Description of in-shoe measurement

The pressure measurement system used in this study was in-shoe; therefore, it was necessary to be able to measure kinematics at the AJC in shod condition for comparison. Previous studies have positioned sensors/markers from motion analysis systems on to the heel counter of footwear to determine motion at the rearfoot in shod conditions (Eng et al. 1994; Nawoczenski et al. 1995). Recent studies have compared external markers on shoes to bone markers, for measuring rearfoot motion (Reinschmidt et al. 1997b; Reinschmidt et al. 1997a) and conclude that markers on shoes are not a satisfactory method to estimate rearfoot motion. It has been suggested that markers tracked through windows cut into the shoe might provide a better representation of rearfoot motion (Lundberg 1996). Preliminary work was undertaken to try and develop a shoe, which would allow in-shoe motion analysis via a window in the heel counter without unduly affecting the heel counter stability. The final design comprised a standard shoe, which had a window cut out of the heel counter but with a Velcro fastening at the top, which could be tightened to maintain the stability of the shoe (Figure 3-5). Sets of standard shoes (male and female UK sizes from 3-12) were modified to allow in-shoe measurement. The Velcro fastening also allowed the shoe to be put on and removed without disturbing the EMT sensors or altering the alignment position.

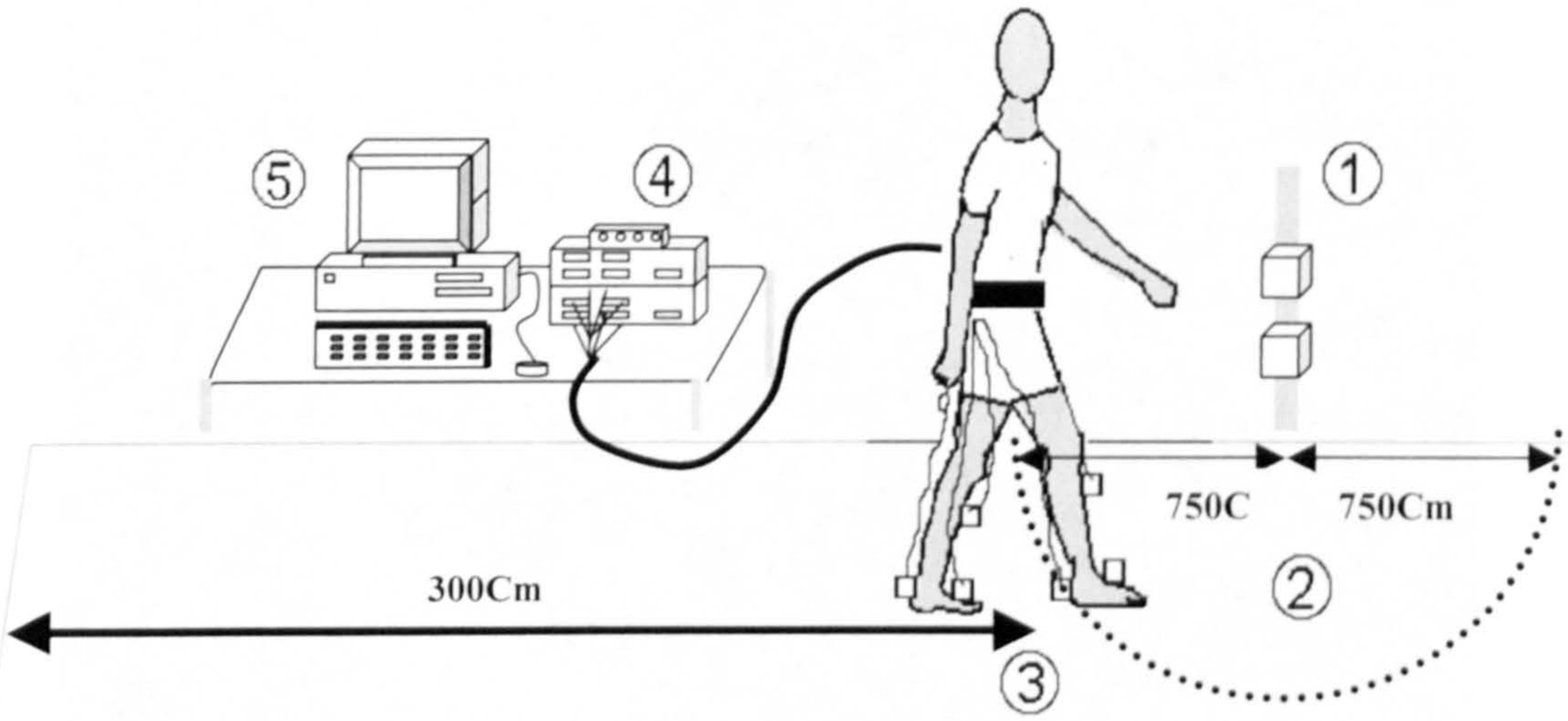
Figure 3-5: Footwear modification to allow in-shoe motion analysis

3.1.2.3. Description of experimental procedure

The system used consisted of two transmitter units and two motion capture units each having four motion sensors, to allow measurements to be taken from both limbs simultaneously. A walkway (dimensions, width 2000mm, length 8000mm,) was constructed at a height of 650mm to decrease the influence of metal pipes in the floor of the gait laboratory. The transmitter units were mounted on a stand made of Perspex, the height of the lower unit was 300mm above the walkway with a vertical separation distance of 248mm between the 2 transmitter units. The transmitter stand was placed 3 metres from the start of the walkway, to allow subjects to initiate walking and reach normal walking speed before entering the electromagnetic field. Discrete markings on the walkway were used to allow the operator to restrict data capture to within the accurate operational field of the EMT system (±7500mm from the transmitter units) in accordance with the manufacturers recommendations and the literature. A schematic representation of the arrangement of the electromagnetic tracking system can be seen in Figure 3-6.

3.1.2.4. Data acquisition and analysis

The 6D RESEARCH software uses predefined kinematic models to calculate motion at one or more joints. To define a joint, a sensor must be placed both proximally and distally to the joint. A coloured triad or animated skeletal icon represents each senor as defined by the operator. A person has sensors applied and positioned at the sites previously described; prior to data collection the subject stands next to the transmitter unit in a pre-determined standardized position and the sensors are aligned to a zero position. Activation of the "bore-sight" (alignment) icon within the data collection screen aligns the sensors to zero. Data collection is triggered by activation of the "capture" icon within the data collection screen. During data collection, gross movement of the sensors can be viewed in real-time (Figure 3-7). A number of different parameters including Euler, projected, and joint co-ordinate system angles can then be calculated after data collection according to the user specifications within the model. The system uses a 4^{th} order Butterworth filter with a low pass cut off frequency of 6Hz.



1 Transmitter units on plastic support stand with a defined vertical separation distance. 2 Accurate Data collection area, as determined by discrete floor markings. 3 Subject set up with sensors attached to the lower limb, trailing cables into a waist band 4 Motion capture units with a four channel event detection unit. 5 Personal computer, running the 6D RESEARCH software.

Figure 3-6: Experimental set up for data collection

Figure 3-7a: Screen shot of 6D Research software during data collection.

Example shows motion sensors on the tibia, calcaneus and navicular on both limbs.

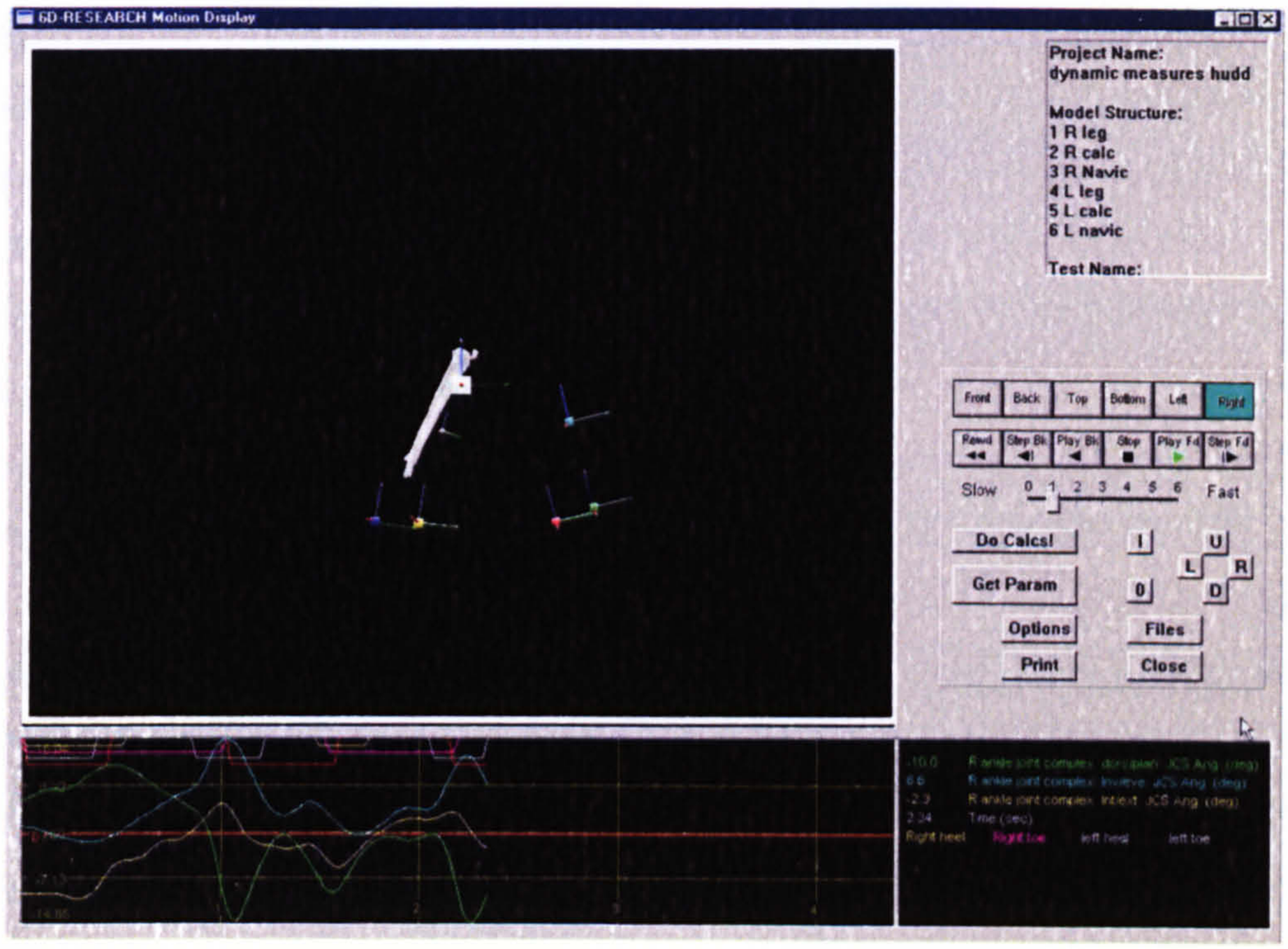


Figure 3-7 b: Screen shot from 6D Research software showing graphical display of joint motion post data collection. This example shows motion at the ankle joint complex in all 3 planes for the right limb, along with footswitch data.

3.1.3 Suitability of 6D RESEARCH system to study ankle joint complex motion

Preliminary data collected using the method described in section 3.2.1 had good face validity compared with the findings of other workers using similar motion analysis systems (Kepple et al. 1990; Mosely et al. 1996; Reinschmidt et al. 1997a). However a number of key areas concerning the clinical application of EMT to study the AJC required further investigation. A number of studies were proposed with the aim to determine:

- The effect that electromagnetic tracking has on gait characteristics
- The optimum sampling rate
- The optimum "bore-sight" (Alignment) procedure
- The repeatability of electromagnetic tracking in a normal population

3.1.3.1 The effect that electromagnetic tracking has on gait

One potential disadvantage of EMT is that the subject is tethered to the motion capture unit via a series of cables from each sensor. It is possible that the presence of the electromagnetic sensors and associated devices necessary for data collection could affect the way that people walk. This issue had not been addressed in the literature.

The measurement of temporal parameters of gait are outcome measures of overall gait performance that reflect the net result of all dynamic activity occurring during locomotion (Winchester et al. 1996). Perry (1991) stated that a subject's customary temporal distance factors arise from a mix of joint mobility, muscle strength, neural control and energy (Perry 1991). Assuming reliable testing equipment, changes in temporal distance factors can be reasonably attributed to changes in walking pattern. If a test subject is free from pathology it is justifiable to use temporal distance factors to indicate the effect that body attached instrumentation has on their characteristic walking pattern.

In order to investigate if the presence of electromagnetic sensors and associated cabling had any influence on temporal and spatial parameters of gait a small study was performed on ten subjects who met the following inclusion / exclusion criteria:

pain free motion at the AJC, no past history of trauma to the lower limb and no past or current history of organic disease likely to affect foot posture or gait.

The study used a telemetry footswitch system to record temporal distance measures of gait (MT8, MIE, Medical Research Ltd, Leeds, West Yorkshire, UK). The foot switches were placed under the heel and 1st MPJ of both feet, and connected via a data cable into a pack on a waistband. Speed information was collected using a passive infrared measurement (PIR) system, an established method for measuring walking speed. The system used for this study was a modified version of that described by Hendry and associates (Hendry et al. 1990). Two thermal infrared detectors (used in security alarm systems) were placed on an 8000mm walkway at a height of 1000mm. Opaque masking tape was applied to the front window of each alarm terminal, to narrow the beam of the thermal infra-red detectors to 1cm. The distance between the two detectors was 6000mm. Both detectors were connected to an electronic stopwatch via the alarm terminals. The stopwatch was triggered when the first detector was activated and stopped when the second detector was activated. A diagram of the experimental set up can be seen in Figure 3-8. Data collection was performed during a one-week period with restricted access to the calibrated area so as to ensure the detectors were not moved. However, prior to any data collection the infrared detectors were checked to ensure that the point at which they triggered was 6000mm apart.

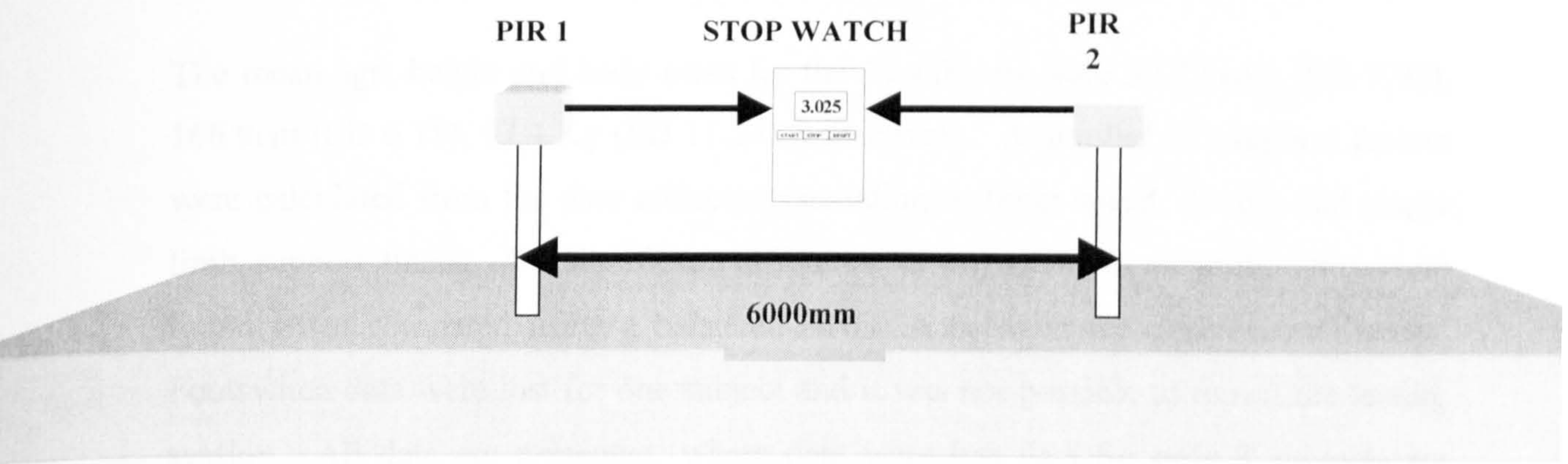


Figure 3-8: Passive infra-red detector placement used during data collection.

In an attempt to reduce the effect that fatigue would have on the measurements the subjects were divided into two groups. In the first group there was a progressive build up of switches and cabling. The subjects in the first group were asked to walk at their normal walking speed down the 8000mm walkway, three times barefoot and three times in a standard pair of shoes. Recordings were taken from the stopwatch for each pass. The footswitches were then applied and three trials were recorded barefoot and shod. When the first infrared detector was triggered, data were collected from the foot switches for five seconds at a sampling rate of 60Hz (enough time to allow the subject to pass between the two PIR sensors). The time taken to pass between the PIR was recorded, this would then be used when analysing the foot switch data. Sensors from the 6D-Research system were then applied to both limbs, in accordance with the protocol outlined previously; however, data recorded from the sensors were not analysed. Three trials were recorded barefoot and shod as outlined in the previous trial. In the second group all the switches and cables were applied initially and the subjects were instructed to walk at their normal walking speed down the walkway. The switches and cables were then systematically removed during the subsequent trials and the timing data were recorded. Data from the foot switches were downloaded into Myo-dat software (MIE, Medical Research Ltd, Leeds) on a host PC. Timings for walking speed (metres per second), double limb and single limb support times were calculated for each trial (% of stance phase).

Results and discussion

The mean age, height and body mass for the 10 subjects were 30.7 years (SD 7.92), 166.9cm (SD 6.18), 67.4 Kg (SD 11.24) respectively. A number of temporal factors were calculated from the data collected, including walking speed, double and single limb support times. No significant difference in any of these measurements were found when compared using a balanced ANOVA between the experimental states. Footswitch data were lost for one subject and it was not possible to repeat the testing session. All data are presented; where data were lost data for only 9 subjects are presented. Table 3-1 and Table 3-2 show the mean walking speed and percentage spent in double limb support respectively.

Table 3-1: Mean walking speed (m sec^{-1}) for 3 trials in ten subjects, together with mean and standard deviation for the group.

Subject	Barefoot			Shod		
	None	FS	FS + Cables	None	FS	FS + Cables
1	1.25	1.39	1.35	1.41	1.33	1.26
2	1.56	1.51	1.44	1.43	1.57	1.51
3	1.21	1.30	1.23	1.37	1.34	1.42
4	1.35	1.26	1.15	1.36	1.37	1.26
5	1.10	1.27	1.29	1.30	1.37	1.36
6	1.12	1.18	1.20	1.32	1.27	1.18
7	1.41	1.49	1.39	1.51	1.40	1.40
8	1.29	1.35	1.35	1.43	1.74	1.67
9	1.26	1.39	1.48	1.37	1.53	1.39
10	1.42	1.35	1.30	1.42	1.44	1.35
MEAN	1.30	1.35	1.32	1.39	1.44	1.38
SD	0.14	0.10	0.11	0.06	0.14	0.14

Table 3-2: Mean percentage of time (% of stance phase) spent in double limb support for 9 subjects, together with mean and standard deviation for the group.

Subject	Barefoot		Shod	
	FS	FS + Cables	FS	FS + Cables
1	17.3	16.3	22.2	24.7
2	21.6	20.8	23.8	22.1
3	10.6	23.1	21.1	24.9
4	17.8	18.6	27.7	26.3
5	24.0	20.8	25.5	27.7
6	21.4	23.2	21.6	22.5
7	20.7	17.9	29.8	22.4
8	23.1	21.8	25.8	24.7
9	17.4	16.8	21.5	21.4
MEAN	19.3	19.9	24.3	24.1
SD	2.13	3.07	2.61	4.09

None- no foot switches. FS- foot switches attached. FS + cab- foot switches and motion sensors from the 6D RESEARCH system attached.

The application of electromagnetic sensors did not significantly alter the gait pattern of normal adults. The temporal distance measures found in this study are similar to those reported in studies of larger populations (Winchester et al 1996).

3.1.3.2 The optimum sampling rate

Winter and Wells (1978) have stated that a sampling rate of 25Hz is adequate when performing motion analysis at normal walking speed (Winter et al. 1978). However, previous studies for the AJC, have sampled at rates between 50-100Hz, depending on which system has been used. Despite an extensive literature search in this area no material was found to substantiate a specific sampling rate for the AJC. The 6D Research motion analysis system has four sampling rate outputs of, 30, 40, 60 and 120Hz, depending on how many sensors are being used. The sampling rate is 120Hz when using one sensor, 60Hz when using two sensors, 40Hz for three sensors and 30Hz when all four sensors are been used. A study was designed with the aim of determining the lowest accurate sampling rate for the ankle joint complex with the 6D-Research system. A lower sampling rate would allow more sensors to be used thus more joints to be evaluated.

In this study the 6D Research motion analysis system was set up so it would record positional data from the AJC, at the three different sampling rates 30, 40 and 60Hz. Sensors from the 6D-research system were placed on the tibia and the calcaneus of the right limb in accordance with the protocol previously outlined (section 3.2.1). The zero alignment position was taken in the relaxed calcaneal stance position. Flexible foot switches were placed under the heel and the 1stMPJ. These were connected to an events channel, which was connected to the PC. Whenever the foot switches were activated during data collection, a mark was placed in the 6D-Research data. This enabled accurate identification of the key events of stance, heel strike, foot flat, heel lift and toe off. Fifteen subjects were included in the study meeting the inclusion / exclusion detailed previously (section 3.1.3.1). Each subject was given a period of acclimatization whilst connected to the system, prior to data collection.

Five walking trials were recorded at each of the sampling rates; the data were digitally filtered with a 4th Order Butterworth filter with a low pass cut off frequency of 6Hz to

eliminate noise. Joint coordinate system calculations were performed for motion at the AJC. Three-dimensional positional data were recorded at heel strike, foot flat, heel lift and toe off, for each sampling rate. The data were exported in an ASCII (American Standard Code for Information Interchange) format and imported into Excel and then Mintab for statistical analysis.

Tables 3-3, 3-4 and 3-5 show the absolute angular position of the AJC at heel strike, foot-flat, heel lift and toe off in the sagittal, frontal and transverse planes respectively. A balanced analysis of variance (ANOVA) showed there was no significant difference between the 3 different sampling rates at each event of stance in all three planes of motion, with the exception of heel strike in the sagittal plane. Due to equipment limitations it was not possible to perform simultaneous measurements at the AJC in all three sampling rate options. As a consequence differences in bore-sight positions between trials and natural variation of walking pattern would influence the absolute angular positions at key events during the stance phase. The motion time curves generated from each of the three sampling rate outputs appeared similar. No significant difference in the total range of movement in all three planes of motion was found between the three sampling rates. In conclusion the results of this study show that motion at the AJC can be accurately measured at the lower sampling rate of 30Hz, which means that the additional sensors can be used in order to study motion at other joint complexes (for example the 1st MPJ) and gain more information about overall dynamic foot biomechanics.

Table 3-3: Mean angular position of the ankle joint complex of 3 trials for 15 subjects in the Sagittal plane at sampling rates of 30, 40 and 60Hz together with mean and standard deviation for the group.

Subject	Sampling Rate 30Hz				Sampling Rate 40Hz				Sampling Rate 60Hz			
	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)
1	-1.52	-3.52	9.38	-2.34	-0.76	-4.06	8.7	-1.98	-3.02	-5.98	9.18	-4.24
2	0.12	-3.24	-13.6	-0.14	6.04	1.6	-6.12	7.92	4.62	1.22	-8.36	5.2
3	2.94	-3.76	-1.76	2.4	3.16	-0.38	0.12	4.64	2.54	-0.92	-2.04	3.92
4	-6.08	-3.28	-1.56	-5.78	-5.12	-2.58	-0.3	-4.1	-4.5	-4.82	-0.46	-3.28
5	-4.38	-5.3	1.16	-1.96	-5.12	-2.58	-0.3	-4.1	-2.66	-3.98	1.52	-0.82
6	-1.35	-0.7	2.4	3.55	-1.2	-0.56	4.22	0.4	-1.48	-1.76	2.96	0.96
7	-5.78	-6.56	1.38	-1.5	-5.34	-6.94	0.48	-0.72	-5.08	-7.94	0.22	-0.14
8	-0.8	2.16	6.84	3.98	2.84	3.8	9.52	4.66	3.28	3.64	9.02	6.5
9	-5.72	-6.14	1.64	-7.78	-5.32	-7.7	1.64	-9.2	-4.32	-7.48	1.48	-7.66
10	-1.14	-2.04	0.18	-3.18	-1.02	-2.8	0.46	-1.76	-0.28	-2.92	0.36	-0.76
11	-7.78	-5.6	4.86	-7.88	-7.32	-9.94	4.16	-3.68	-9.14	-12.1	0.12	-9.02
12	-1.78	-0.12	1.7	0.4	-1.08	-0.18	1.16	-0.98	-0.16	0.48	1.9	2.26
13	0.1	-2.68	3.3	-1.06	0.68	-3.16	3.44	-1.24	-0.1	-3.5	3.18	-0.88
14	-1.44	-3.42	3.82	0.48	-1.34	-4.02	3.92	-0.08	-1.16	-4.16	4.12	-0.36
15	7.14	10.62	0.76	-2.78	7	11.46	-0.52	-2.256	6.54	11.48	0.22	-2.82
Mean	-1.83	-2.24	1.37	-1.57	-0.93	-1.87	2.04	-0.83	-0.99	-2.58	1.56	-0.74
SD	3.81	4.26	5.08	3.63	4.31	5.12	3.86	4.16	4.09	5.54	4.20	4.32

Table 3-4: Mean angular position of the ankle joint complex of 3 trials for 15 subjects in the frontal plane at sampling rates of 30, 40 and 60Hz together with mean and standard deviation for the group.

Subject	Sampling Rate 30Hz				Sampling Rate 40Hz				Sampling Rate 60Hz			
	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)
1	4.06	1.48	0.9	9.44	2.86	0.54	1.64	7.38	4.6	1.84	1.12	9.8
2	-0.42	1.38	-0.34	-7.2	-1.54	0.46	-0.08	-9.22	-1.5	0.88	-0.14	-8.52
3	-2.74	-0.46	-1.02	-10.28	-4.8	1.08	-0.38	-12.08	-3.54	1.82	0.52	-8.5
4	3.54	2.42	2.4	4.34	4.18	2.62	2.26	4.22	4.48	0.98	0.42	3.52
5	2.04	0.7	0.7	5.64	4.18	2.62	2.26	4.22	2.26	0.6	1.04	5.18
6	4.7	4.05	-0.85	5.58	5.7	4.44	0.46	10.16	3.7	2.82	-0.7	7
7	-3.18	-4.18	-3.1	1.76	-3.3	-4.56	-0.72	0.88	-2.92	-4.78	-4	0.82
8	2.32	-0.76	-2.88	6.2	6.1	2.64	1.06	10.26	6.38	1.76	0.38	8.3
9	2.12	1.3	-0.4	5.1	1.66	1.58	-1.14	5.88	2.04	1.48	-0.88	5.76
10	-0.94	-1.6	-3.74	2.64	-1.48	-1.96	-3.08	2.24	-1.62	-1.88	-3.1	1.84
11	1.14	0.7	-2.48	2.96	0.48	-	-1.3	2.8	0.42	-1.1	0.16	2.78
12	1.52	-1.52	-1.94	5.84	1.86	-0.6	-1.24	5.92	2.12	-1.46	-1.9	4.86
13	1.2	0.48	-0.66	4.64	1.22	0.1	-1.16	4.14	0.8	0.24	-0.64	4.48
14	-0.1	-0.1	-1.28	2.86	-0.02	-0.32	-1.684	3.42	0.06	-0.14	-2.4	3.36
15	0.88	3.62	2.9	-1.32	0.92	4.16	2.62	-2.16	0.5	3	2.16	0.54
Mean	1.07	0.5	-0.79	2.55	1.20	0.91	-0.03	2.54	1.19	0.40	-0.53	2.75
SD	2.27	2.1	1.93	5.21	3.14	2.41	1.69	6.27	2.87	2.04	1.69	5.24

Table 3-5: Mean angular position of the ankle joint complex of 3 trials for 15 subjects in the transverse plane at sampling rates of 30, 40 and 60Hz together with mean and standard deviation for the group.

Subject	Sampling Rate 30Hz				Sampling Rate 40Hz				Sampling Rate 60Hz			
	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)	HS (Degrees)	FF (Degrees)	HL (Degrees)	TO (Degrees)
1	2.12	0.78	3.96	5.2	2.28	1.58	4	4.12	3.08	1.58	3.8	5.44
2	3.28	3.58	0.92	-5.26	0.44	-1.14	-3.06	-6.86	2.06	1.66	0.42	-5.34
3	3.56	0.56	2.48	-9.18	3.92	0.22	2.18	-10.78	8.08	3.72	4.44	-7.78
4	-0.26	1.96	2.32	0.64	1.2	2.54	2.62	1.12	1.04	0.3	0.84	1.66
5	0.08	-1.9	-0.14	5.02	1.2	2.54	2.62	1.12	-0.5	-2.8	-0.38	3.4
6	3.98	1.88	0.05	6.33	1.32	0.52	0.48	5.96	1.46	-2.22	-0.2	5.88
7	-3.82	-4	-0.68	-0.14	-3.84	-3.96	-2.28	-1.06	-2.06	-4.22	-2.16	-0.58
8	1.3	-0.4	-2.38	3.94	1.72	-1.2	-2.52	2.72	1.7	-1.38	-2.42	1.92
9	0.16	-1.94	0.76	6.66	-0.14	-2.78	-0.96	6.62	1.06	-2.02	0.28	7.94
10	-3.92	-4.08	-6.1	2.3	-4.98	-5.24	-4.38	1.64	-5.3	-4.94	-4.4	0.14
11	-1.92	-2.22	-0.06	2.7	-0.22	-0.66	1.18	2.96	0.66	-0.06	1.42	4.24
12	-0.7	-1.88	-1.34	1.88	0.12	-1	-0.86	2.3	-0.08	-0.7	-0.54	1.84
13	-2.54	-6.16	-2.2	1.02	-1.68	-3.6	-2.28	-0.2	-2.52	-6.18	-2.24	0.8
14	-3.53	-3.938	-2.72	0	-2.63	-4.08	-2.44	1.76	-2.68	-3.8	-2.4	2.44
15	0.8	3.28	1.74	-2.74	1.04	4.88	1.98	-2.96	0.54	2.74	1.72	-2.3
Mean	-0.09	-0.97	-0.23	1.22	-0.02	-0.76	-0.25	0.56	0.44	-1.07	-0.12	1.31
SD	2.66	2.93	2.5	4.4	2.37	2.87	2.56	4.59	3.05	2.91	2.41	4.16

3.1.3.3. The optimum "bore-sight" (Alignment) procedure

For any 3D-motion analysis system the markers or sensors must be placed at pre-determined sites and a zero reference position determined prior to data capture. The zero starting position needs to be standardised for each subject and be reproducible. Many studies claim to position the AJC in a "subtalar joint neutral position" (as defined by Root et al 1971), despite a wealth of literature to show poor inter and intra-tester reliability of determining this position (Elveru et al. 1988b; Freeman 1990; Menz 1995; Sell et al. 1994).

Firstly the principle of using the subtalar joint neutral position was explored and a study to investigate the reliability of determining the subtalar joint neutral position (STJNP) using palpation of the talar head was undertaken. In order to try and improve the reliability of determining the STJNP an alignment jig was developed to try and ensure a standardised starting position in the sagittal and transverse plane. The jig method for determining STJNP would be compared to the traditional method using talar head palpation.

The alignment jig was constructed out of Perspex, comprising of two rotating footplates connected to two vertical lower leg splints (Figure 3-9). Each footplate had a protractor dial on the front to allow alignment in the frontal plane. A central line was etched onto the footplate, running from the front edge into a heel cup situated at the back of the plate. This would enable alignment of the midline of the foot, in the transverse plane. Each splint had Velcro fastenings in order to secure the lower leg into the splint, this would ensure the lower leg was vertical and the ankle joint at 90°.

Three examiners and 5 subjects were used in the study in order to establish the reliability within and between examiners, using both methods. In this study each subject had a sensor placed on the tibia and calcaneus of the right limb and data were recorded at 60Hz. The zero-position reference point was determined once per measurement session, when the subject was in a relaxed calcaneal stance position (standing in a relaxed position, normal base of gait). Any measurement taken was relative to this position and it remained the same for each examiner. Each examiner placed the right limb in to the STJNP and data were collected for 5 seconds, this procedure was repeated 5 times for each examiner.

The subject was then positioned in the jig device, each examiner positioned and secured the foot into the jig and the footplate then was maximally inverted. The examiner then positioned the foot into the STJNP and data were recorded for 5 seconds. The subject's foot was then removed from the jig and the procedure was repeated until each examiner had taken 5 measurements for each subject. The protractor dial on the front of the jig was blanked out during the study and the order in which subjects were measured was randomized. A subsequent testing session was performed one week later.

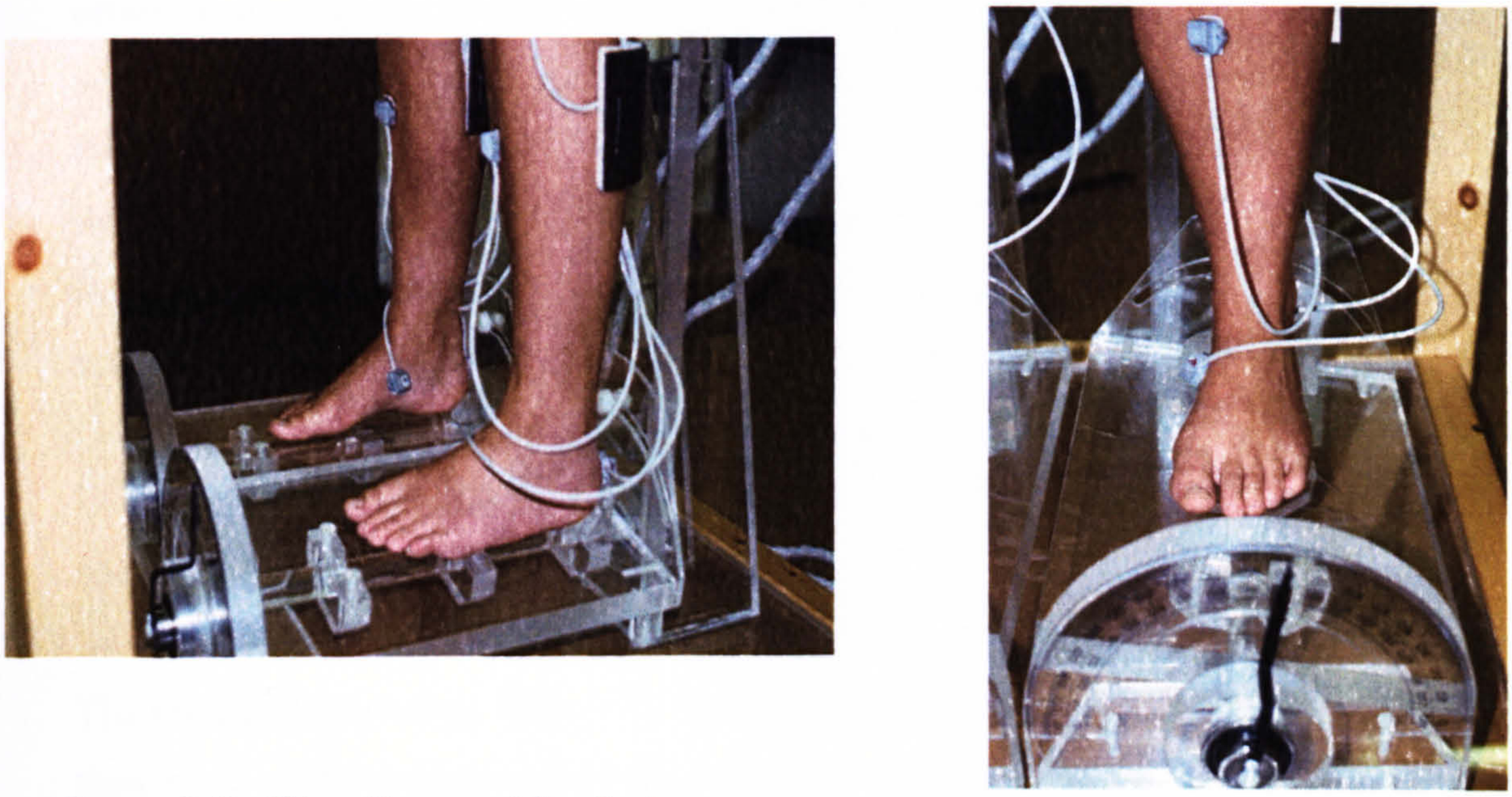


Figure 3-9: The alignment jig device

The mean position of the AJC during the five seconds was calculated, in all three-body planes for each test situation. The standard deviations were studied as a measure of internal consistency for determining the neutral position. (See Table 3-6). 1)- A balanced ANOVA showed there was a significant difference between the two different methods. 2)- The results showed that the standard deviation decreased for the second testing session with both methods, suggesting that experience decreases variability. Wide inter and intra tester variability was found in this study with both methods. The maximum range of measurements in the sagittal plane was 11.91 ° and this was using the free-standing method. The maximum range of measures in the frontal plane was 15.05° using the jig method (in view of the fact that the reported

average range of movement at the AJC in the frontal plane is approximately 30° this margin of error is unacceptable) (Root et al. 1977). In the transverse plane the range was 23.11° using the free-standing method. This study has shown that despite attempting to improve the reliability of determining the STJNP with the jig, it is susceptible to such a large margin of error (in some cases the range equated to 50% of the average total range of motion at the joint) it would not be acceptable to be used as the alignment reference position.

In light of the above findings an alternative method for alignment had to be established. The jig device offered the ideal opportunity to position the foot in a standardized position in all three planes of motion. A study was undertaken, to find the intra tester reliability of determining a pre-set position using the jig. A protocol was developed for positioning of the lower limb in the jig. The zero position was taken with the ankle at 90° in the sagittal plane, the mid-line of the 2nd toe aligned against the central line on the footplate and the rotating footplate set to zero degrees. One examiner positioned the foot in the starting position using the jig and recorded five repeated measurements on three subjects. The procedure was then repeated on one other occasion at least one day apart.

The results are shown in Table 3-7, the total range of measures in each plane is lower than either of the two previously described methods. The method was quick and easy to perform and was deemed the most suitable method to be used in the study.

Table 3-6A, The mean, standard deviation and range of measures taken of the neutral position at the ankle joint complex by all three examiners in the sagittal plane using the free-standing and jig method.

	Free standing 1			Free standing 2			Jig 1			Jig 2		
Subject:	Mean	St Dev	Range	Mean	St Dev	Range	Mean	St Dev	Range	Mean	St Dev	Range
1	-0.66	1.24	4.71	-1.47	0.73	3.14	-3.31	2.14	6.78	1.06	1.15	4.29
2	1.29	0.95	3.40	-0.65	0.89	3.38	-1.11	0.80	2.34	0.61	1.90	6.72
3	5.26	1.86	6.55	2.58	1.02	3.46	0.57	0.86	2.37	-0.50	0.97	3.51
4	2.26	2.91	10.71	3.29	2.99	11.91	0.76	0.46	1.64	1.85	1.57	4.58
5	2.76	1.11	4.52	2.67	0.70	2.24	-0.88	0.77	2.57	2.28	1.05	3.90

Table 3-6B, The mean, standard deviation and range of measures taken of the neutral position at the ankle joint complex by all three examiners in the frontal plane using the free-standing and jig method.

	Free standing 1			Free standing 2			Jig 1			Jig 2		
Subject:	Mean	St Dev	Range	Mean	St Dev	Range	Mean	St Dev	Range	Mean	St Dev	Range
1	3.47	0.81	2.78	3.05	0.84	3.81	4.79	4.34	12.39	0.94	3.45	15.05
2	5.23	0.74	2.71	4.06	1.45	4.52	3.73	1.07	3.52	4.27	1.99	8.25
3	6.17	1.38	6.04	5.65	1.31	4.01	3.21	1.45	5.04	2.18	1.70	5.34
4	6.22	1.26	5.12	3.74	1.93	6.67	1.98	1.39	4.39	0.41	2.69	12.53
5	-1.82	2.36	6.98	4.58	1.07	2.82	3.97	1.80	4.99	-3.91	1.55	6.26

Table 3-6C, , The mean, standard deviation and range of measures taken of the neutral position at the ankle joint complex by all three examiners in the transverse plane using the free-standing and jig method.

	Free standing 1			Free standing 2			Jig 1			Jig 2		
Subject:	Mean	St Dev	Range	Mean	St Dev	Range	Mean	St Dev	Range	Mean	St Dev	Range
1	2.59	1.95	6.29	1.09	2.29	9.39	8.69	3.99	11.73	3.13	4.88	18.78
2	6.27	1.35	4.66	6.38	1.69	5.10	3.37	1.16	3.72	0.82	5.17	18.04
3	6.17	2.22	7.73	7.64	2.17	7.26	1.35	1.28	4.48	3.16	2.09	6.31
4	6.38	1.83	5.51	0.52	5.38	23.11	0.64	1.93	7.50	2.58	3.08	13.11
5	3.84	5.70	16.48	5.10	1.74	7.07	-0.03	1.61	5.52	5.55	2.31	7.61

Table 3.7: The mean, standard deviation and range of measures taken for 3 subjects, when using the jig to determine a pre-defined zero position at the ankle joint complex, by one examiner in all three planes on two separate days.

	Sagittal plane						Frontal plane						Transverse plane					
	Subject 1		Subject 2		Subject 3		Subject 1		Subject 2		Subject 3		Subject 1		Subject 2		Subject 3	
	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2
	-0.02	-1.51	0.48	-1.87	0.04	2.16	-0.01	3.76	0.34	-0.19	-0.46	5.70	0.00	-2.06	1.10	-1.50	-0.46	5.79
	-2.14	-1.83	-0.22	-1.37	2.06	1.01	2.26	2.01	-0.63	-0.24	2.31	0.48	2.76	4.64	2.04	-0.97	7.55	-0.32
	-1.53	-1.58	-3.51	-4.60	5.99	4.35	0.80	0.33	1.53	-1.10	6.05	6.53	6.66	-1.18	-1.84	-4.77	9.79	10.31
	-1.52	-1.68	-0.75	-1.73	4.23	2.58	1.56	0.92	0.14	-0.08	1.12	3.13	7.53	3.89	-1.50	-4.05	4.72	6.20
	-1.15	-1.94	-1.89	-2.50	2.70	1.54	0.89	0.63	0.60	0.22	0.52	1.63	-2.37	6.08	-1.60	-2.77	1.19	1.81
Mean	**-1.27**	**-1.71**	**-1.18**	**-2.41**	**3.00**	**2.33**	**1.10**	**1.53**	**0.39**	**-0.28**	**1.91**	**3.49**	**2.92**	**2.27**	**-0.36**	**-2.81**	**4.56**	**4.76**
St. Dev	**0.78**	**0.18**	**1.57**	**1.29**	**2.25**	**1.28**	**0.85**	**1.40**	**0.78**	**0.49**	**2.53**	**2.59**	**4.24**	**3.66**	**1.80**	**1.62**	**4.27**	**4.14**
Range	**1.51**	**0.44**	**3.99**	**3.23**	**5.95**	**3.34**	**2.27**	**3.43**	**2.16**	**0.91**	**6.51**	**6.05**	**9.9**	**8.14**	**3.88**	**5.67**	**10.25**	**9.99**

3.1.3.4 The Repeatability of electromagnetic tracking at the ankle joint complex in a normal population

Quantitative methods for analysing gait, for example computer aided motion analysis and dynamic electromyography, are recognised to be valuable diagnostic and outcome measurement tools, but are not widely used in clinical practice. It has been stated that one reason for low utilisation is that the reliability of these measurements in terms of repeatability has not sufficiently been established (Vaughan et al. 1996). There is a wealth of normative data on various gait parameters, based on data taken from one testing session. It is not possible to say if the results from a single measurement session are representative of normal gait and if the data are consistent trial to trial and day to day. There have been very few published studies on the repeatability of kinematic variables, all of which have used camera-based systems. There has not been a repeatability study for kinematic data, gained from electromagnetic tracking for the AJC.

A study was performed to establish the repeatability of AJC motion parameters derived from the 6D-Research motion analysis system. In order to establish intra and inter examiner repeatability within and between days, two examiners were used and measurements taken over two sessions. Both examiners were experienced with the 6D-Research motion analysis system and the protocol for use. Five subjects and two examiners were included in the study. The first examiner placed sensors on the tibia and calcaneum, and foot switches were placed under the heel and 1st MPJ on the right limb in accordance with the protocol discussed in detail previously (Section 3.2.1). The zero reference point was taken, in the relaxed calcaneal stance position, three walking trials were recorded and all the cabling was removed. The examiner then applied the 6D-Research sensors and foot switches again, in accordance with the protocol and the zero point was re-established. Three walking trials were recorded and all the cabling removed. This procedure was repeated, until five sets of walking trials had been recorded. The second examiner performed the whole procedure. A subsequent testing session was performed one week later.

Joint co-ordinate system angles were calculated for the right AJC and motion time curves were generated using the 6D-RESEARCH software. The motion time curves

were normalised to 100 % of the stance phase using Datapac software (RUN Technologies, CA, USA). Statistical measures of similarity of waveform were quantified for repeatability within and between test days for each examiner. The similarity between waveforms was determined using the adjusted coefficient of multiple correlation, a technique previously described by Kadaba et al 1989 (Appendix 1). If the waveforms are similar the R_a^2 tends to 1, if the waveforms are dissimilar the R_a^2 tends to zero.

The adjusted coefficient of multiple correlation (CMC) data in each plane of movement for each subject and examiner is presented in Tables 3-8 to 3-11. The author is presented as examiner one. Data from subject 5 examiner 2 on the second measurement session was lost and it was not possible to repeat the testing session. All the data are presented, where data was lost, the CMC values will only be presented for four subjects.

Table 3-8: Coefficient of multiple correlation for both examiners repeated over two measurement session in the sagittal, frontal and transverse plane.

Subject	Examiner 1						Examiner 2					
	Sagittal		Frontal		Transverse		Sagittal		Frontal		Transverse	
	Day 1	Day2	Day 1	Day2	Day 1	Day2	Day 1	Day2	Day 1	Day2	Day 1	Day2
1	0.976	0.984	0.972	0.957	0.488	0.764	0.838	0.937	0.416	0.709	0.508	0.728
2	0.946	0.913	0.898	0.697	0.97	0.858	0.975	0.973	0.77	0.957	0.692	0.908
3	0.956	0.955	0.895	0.935	0.882	0.809	0.952	0.967	0.952	0.941	0.665	0.786
4	0.821	0.903	0.916	0.873	0.896	0.867	0.816	0.944	0.742	0.891	0.604	0.91
5	0.933	0.953	0.896	0.883	0.874	0.883	0.928	/	0.939	/	0.879	/
Mean	**0.926**	**0.942**	**0.915**	**0.869**	**0.822**	**0.836**	**0.902**	**0.955**	**0.764**	**0.875**	**0.670**	**0.833**
SD	**0.061**	**0.033**	**0.033**	**0.102**	**0.191**	**0.049**	**0.071**	**0.017**	**0.217**	**0.114**	**0.137**	**0.091**

Table 3-9: Inter-examiner coefficient of multiple correlation over two measurement sessions in the sagittal, frontal and transverse plane.

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Subject	Day 1			Day 2		
	Sagittal	Frontal	Transverse	Sagittal	Frontal	Transverse
1	0.907	0.59	0.374	0.945	0.84	0.8
2	0.939	0.733	0.797	0.933	0.816	0.9
3	0.952	0.919	0.79	0.951	0.941	0.798
4	0.836	0.809	0.707	0.918	0.89	0.916
5	0.928	0.914	0.883	-	-	-
Mean	**0.912**	**0.793**	**0.710**	**0.937**	**0.872**	**0.854**
SD	**0.046**	**0.137**	**0.198**	**0.015**	**0.056**	**0.063**

Table 3-10: Intra-examiner coefficient of multiple correlation repeated over two measurement sessions in the sagittal, frontal and transverse plane.

Subject	Examiner 1			Examiner 2		
	Sagittal	Frontal	Transverse	Sagittal	Frontal	Transverse
1	0.872	0.831	0.342	0.904	0.61	0.465
2	0.926	0.767	0.911	0.97	0.82	0.78
3	0.956	0.876	0.742	0.958	0.929	0.702
4	0.86	0.891	0.838	0.864	0.811	0.779
5	0.93	0.886	0.872	-	-	-
Mean	**0.909**	**0.850**	**0.741**	**0.924**	**0.7925**	**0.6815**
SD	**0.041**	**0.052**	**0.232**	**0.049**	**0.133**	**0.149**

Table 3-11: The between day inter-examiner coefficient of multiple correlation in the sagittal, frontal and transverse plane.

Subject	Sagittal	Frontal	Transverse
1	0.858	-	-
2	0.864	0.566	0.692
3	0.894	0.807	0.399
4	0.721	0.679	0.596
Mean	**0.834**	**0.684**	**0.562**
SD	**0.077**	**0.121**	**0.149**

Due to timing constraints the bore-sight (alignment) position used in the repeatability study was the relaxed calcaneal stance position. It was noted that differences in the bore-sight position would introduce a constant offset to the joint motion time curves. In other words the motion time curves are shifted up or down by a constant amount while the shape of the curve remains unchanged (similar changes have been noted with the reapplication of markers (Kadaba et al. 1989). In an attempt to eliminate the effect of a constant offset introduced by the measuring technique, Kadaba and associates calculated a relative CMC by removing the mean value of the waveforms for the particular day from each motion time curve. For this study the relative CMC were calculated by normalizing the initial contact position in all three planes to zero for all motion time curves. The relative CMC data are presented in Table 3-12 to 3-15.

Table 3-12: Relative Intra-examiner coefficient of multiple correlation repeated over two measurement sessions in the sagittal, frontal and transverse plane.

Subject	Examiner 1						Examiner 2					
	Sagittal		Frontal		Transverse		Sagittal		Frontal		Transverse	
	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2	Day 1	Day 2
1	0.98	0.99	0.98	0.97	0.71	0.91	0.98	0.99	0.96	0.97	0.81	0.90
2	0.94	0.97	0.91	0.70	0.98	0.93	0.98	0.98	0.94	0.96	0.95	0.93
3	0.99	0.96	0.83	0.89	0.83	0.71	0.98	0.97	0.95	0.95	0.24	0.83
4	0.97	0.96	0.96	0.82	0.85	0.87	0.95	0.99	0.88	0.98	0.71	0.95
5	0.94	0.97	0.91	0.89	0.77	0.91	0.96	/	0.97	/	0.96	/
Mean	**0.96**	**0.97**	**0.92**	**0.86**	**0.83**	**0.87**	**0.97**	**0.98**	**0.94**	**0.97**	**0.73**	**0.90**
SD	**0.02**	**0.01**	**0.06**	**0.10**	**0.10**	**0.09**	**0.02**	**0.01**	**0.04**	**0.01**	**0.30**	**0.05**

Table 3-13: Relative Inter-examiner coefficient of multiple correlation repeated over two measurement sessions in the sagittal, frontal and transverse plane.

Subject	Day 1			Day 2		
	Sagittal	Frontal	Transverse	Sagittal	Frontal	Transverse
1	0.968	0.966	0.768	0.984	0.971	0.47
2	0.924	0.79	0.936	0.953	0.827	0.87
3	0.979	0.901	0.846	0.948	0.911	0.809
4	0.957	0.883	0.789	0.968	0.804	0.892
5	0.947	0.904	0.863			
Mean	**0.955**	**0.889**	**0.840**	**0.963**	**0.878**	**0.760**
SD	**0.021**	**0.064**	**0.066**	**0.016**	**0.077**	**0.197**

Table 3-14: Relative Intra-examiner coefficient of multiple correlation between two measurement sessions in the sagittal, frontal and transverse plane.

Subject	Examiner 1			Examiner 2		
	Sagittal	Frontal	Transverse	Sagittal	Frontal	Transverse
1	0.98	0.966	0.766	0.981	0.97	0.824
2	0.938	0.789	0.937	0.976	0.945	0.924
3	0.972	0.872	0.776	0.971	0.934	0.534
4	0.958	0.79	0.873	0.972	0.88	0.843
5	0.938	0.87	0.841			
Mean	**0.957**	**0.857**	**0.839**	**0.975**	**0.93225**	**0.78125**
SD	**0.019**	**0.073**	**0.071**	**0.005**	**0.038**	**0.170**

Table 3-15: Relative Inter- examiner coefficient of multiple correlation repeated over two measurement sessions in the sagittal, frontal and transverse plane.

Subject	Sagittal	Frontal	Transverse
1	0.951	0.952	0.665
2	0.869	0.705	0.867
3	0.919	0.825	
4	0.92	0.655	0.744
Mean	**0.915**	**0.784**	**0.759**
SD	**0.034**	**0.133**	**0.102**

Findings from Winter and Kadaba suggest that repeatability was better within the same test day than between test days (Winter 1984), (Kadaba et al. 1989). The current study confirmed this finding; the CMC for examiner 1 within day was (0.926, 0.915 and 0.822) and between days had decreased to (0.909, 0.850 and 0.741) in the sagittal, frontal and transverse planes respectively. The intra-examiner repeatability (examiner 1) was better than the inter-examiner repeatability as demonstrated by higher CMC values in all three planes for day 1 (Intra - 0.926, 0.915, 0.822 and inter – 0.912, 0.793, 0.710 in the sagittal, frontal and transverse planes respectively). In this study sagittal plane motion exhibited the highest repeatability, followed by the frontal and then the transverse plane. This finding is in agreement with Kadaba et al 1989 and Cornwall & McPoil (Cornwall et al. 1999b). Within day, Kadaba reported CMC value of 0.975 in the sagittal plane and 0.933 in the frontal plane. Between days CMC were 0.968 and 0.881 in the sagittal and frontal planes respectively. The within day CMC values reported by Kadaba were slightly higher than in the present study, however, in the present study within day includes the effect of the re-application of markers and a new bore-sight position (the sensors were re-applied and aligned on five occasions) where as in the study by Kadaba, only one set of measurements were taken within day so the markers were not re-applied. The reported between day CMC values by Kadaba were also slightly higher, this may be due to an increased number of repeated trials in the present study. Cornwall & McPoil reported CMC based on 5 repeated trials on 153 subjects, within day with one examiner using electromagnetic tracking. The CMC values were 0.946, 0.846 and 0.846 in the sagittal, frontal and transverse plane respectively, which are comparable to the findings for each examiner within day in the present study.

The intra-subject repeatability of joint angle motion is influenced by the inherent physiological variability as well as those introduced by the measurement technique (Kadaba et al. 1989). In this study care was taken to minimise changes in the position of the sensors, however, the alignment position was taken in the relaxed calcaneal position, which could be subject to variation. In order to minimise the effect that the bore-sight position, a relative CMC was calculated and in most cases this resulted in an improvement of the CMC value. The improvement in CMC value demonstrates

that the motion time curves are strongly influenced by bore-sight position and all attempts should be made to standardise this position.

It was anticipated that most differences in joint motion repeatability data would be due to the technique and for this reason it was appropriate to test non-diabetics only. The development of methods work was performed on a distant site with no ready access to diabetic patients and the experimental procedure (60 barefoot walking trials) was deemed to not be suitable for diabetic patients who had feet at high risk of developing ulceration. Repeatability work (using the same technique) in other patient groups (those with rheumatoid arthritis) showed no significant differences in CMC data to an age matched control group (Woodburn 2000) and there was no formal hypotheis to suggest that diabetic patients would be any different. The CMC data for the first ten diabetic patients recruited into each group in the main study was calculated. These data, are not reported in the main body of the thesis as it was not a pre-determined part of the experimental procedure but can be found in Appendix 2.

The CMC calculates the level of agreement between waveforms; however, it does not allow comparison of discrete time points. It could also be valid to look at the coefficient of variation for discrete time points during the stance phase. The main study would compare groups of diabetics against non-diabetic controls. The CMC does not provide any information about minimally detectable differences between groups. For the first ten patients recruited into each study group the mean range and standard deviation across the whole stance phase was calculated for each plane of motion (Appendix 3). These data would inform the decision as to what would be determined as a minimally detectable difference.

3.2 Plantar pressure measurement

This section describes the in-shoe pressure measurement system. The measurement of plantar pressure within shoe is a well-established technique. A brief review of the literature will be presented alongside methods for calibration and data collection

3.2.1 Background and rationale

Over the past two decades developments in technology have facilitated the growth of commercially available plantar pressure systems, which are able to measure pressure within the confines of the shoe. In-shoe plantar pressure measurement has relevance for both the clinician and the researcher and is of particular relevance to the patient with diabetes, were barefoot walking should be kept to a minimum. It can be used as a diagnostic tool and has the potential to evaluate therapeutic aspects of patient care (Schaff 1993).

Earlier systems used to measure pressure within the shoe were classified as discrete devices. Individual sensors were placed at various key predefined anatomical locations. One major limitation is migration of the sensors during measurement due to shear forces between the foot / shoe interface. It has also been noted that placement of sensors under the foot may be unreliable (Laing 1999) and may act as a foreign body and alter the pressures (Cavanagh et al. 1992b).

More recently pressure mat systems have been developed which allow the measurement of pressure over the entire plantar surface of the foot. These systems are often referred to as matrix devices and consist of a large number of pressure sensing elements arranged in a grid. The PEDAR in-shoe system (PEDAR, Novel GmbH, Munich, Germany) utilises a sensor matrix in the form of a thin flexible insole. Each insole is approximately 2.7mm thick and consists of a matrix of 99 capacitive sensors

with an average sensor size of 25mm^2. Each insole is connected to an A/D conversion electronics unit, which is fixed to the subject's waist via a belt. The electronics unit is connected to a personal computer via an 8-metre cable.

The type of force sensor in the PEDAR system is based on the capacitance measurement principle. This principle is based on measurement of a change in capacitance that occurs when the distance between two conducting wires that are separated by an insulating wire is varied (Finch 1999). Increased pressure will decrease the amount of separation between the two conducting wires, which increases the capacitance. The electrical resistance is directly recorded and transformed into measurements of force and pressure.

Although the PEDAR system has advantages over discrete pressure sensors there are some inherent disadvantages. The PEDAR insole may alter the coefficient of friction at the shoe / foot interface due to the covering surface of the insole (Cavanagh et al. 1992b). The weight of the data collection pack and the thickness of the insole could potentially alter the subject's gait. Also the sensors within the insole are more susceptible to damage due to excessive repetitive loading in the same region and bending and stretching as they are placed in and taken out of the shoe or placed over an insole. The reliability of the sensors can be affected by changes in temperature and humidity associated with the in-shoe environment.

The reliability and validity of the PEDAR system under static and dynamic loading conditions has been reported (McPoil et al. 1995). For dynamic testing the intraclass correlation coefficients were found to exceed 0.95 with the exception of between session reliability, which was 0.84. The average error reported during application of a series of pressures was 16% and 0.8% at 50 and 500KPa respectively. Performance of the insole under continual pressure loading of 150Kpa was found to be 3.4% or less and the pattern of creep was linear. The authors concluded that the PEDAR insole had a linear response to applied loads with minimum error especially at high pressures (McPoil et al. 1995).

3.2.2 Calibration of Pedar system

Calibration of the PEDAR insoles was performed in the calibration unit supplied by the manufacturer prior to initial data collection and at 3 month intervals during the study. Calibration was performed as per the manufacturer's recommendations; this procedure involves inserting each pair of insoles into the calibration chamber and loading to discrete pressure levels from 0-600kPa. A calibration curve is generated and stored for each insole.

3.2.3 Description of in-shoe pressure accquisiation system

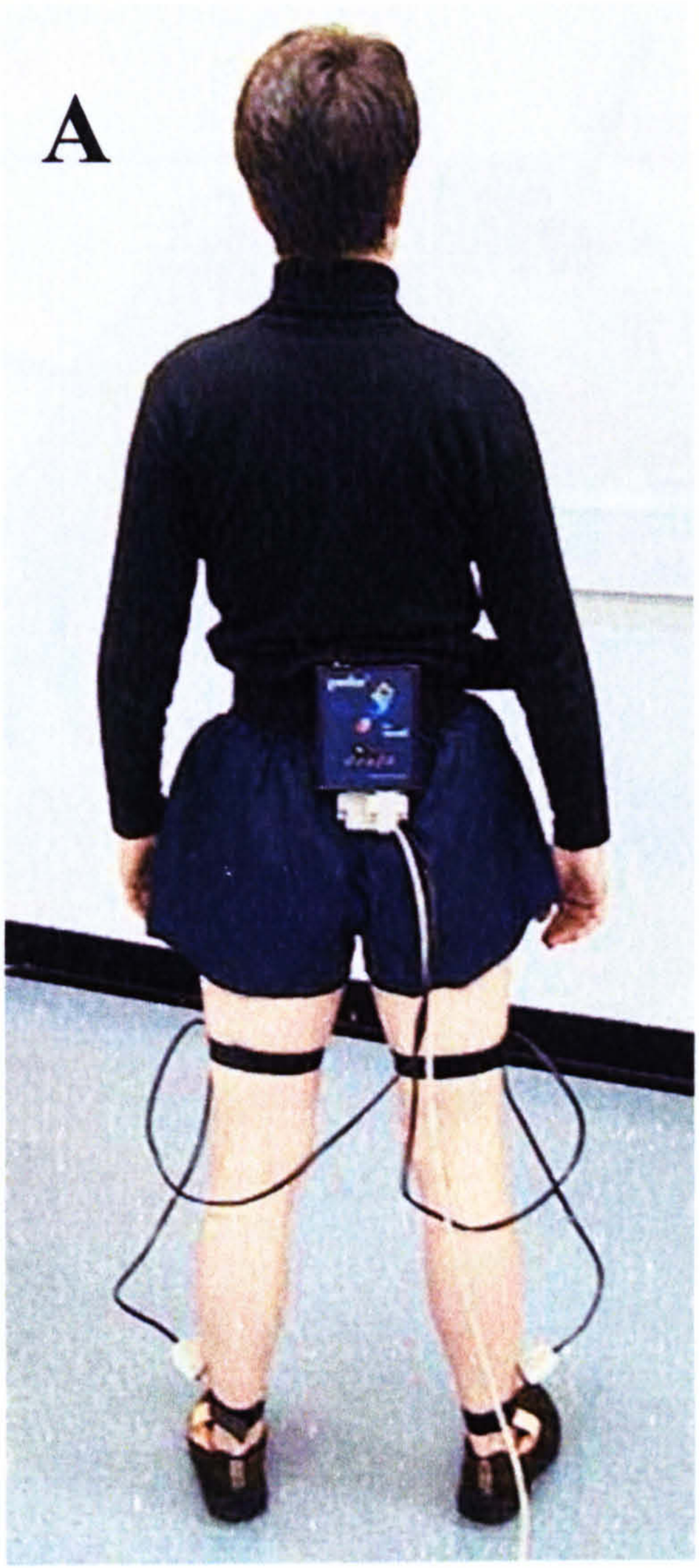
Standard test shoes previously described were used for each patient. The Velcro opening at the rear of the shoe allowed the insoles to be fitted into the shoe with minimal bending of the insole. Cables from the insoles were secured around the ankle and mid-thigh with Velcro straps, the systems electronic unit was placed on the back of a securely fitted waist belt (Figure 3-10). Patients were allowed to acclimatise to wearing the PEDAR system for a short period prior to data collection.

3.2.4 Data acquisition and analysis

During data collection subjects were instructed to walk at their normal walking speed across the walkway (previously described in section 3.2.3). Normal walking velocity was reached before any data were collected, a minimum of 5 left and right steps were recorded for each subject. Each walking trial was coded and saved for further analysis.

The pressure data were analysed using the PEDAR, NOVEL-WIN and NOVEL-ORTHO software. Five consecutive left and right steps from the middle section of the walking trial were selected and transformed into individual step files using Emed link software. Each step file was then used to create a single averaged left and right step for each subject. Ten areas were identified for specific analysis defined as medial and lateral heel, medial and lateral mid-foot, 1st metatarsal head, 2nd metatarsal head, lateral forefoot (3-5 metatarsal heads), hallux, 2nd toe, lateral toes (3-5 toes) , (Figure 3-11).

A number of pressure parameters were calculated for each mask area, including peak pressure, mean pressure, and pressure time integral. Data were saved in an ASCII format for transfer into Microsoft Excel ^{TM} and SPSS software.



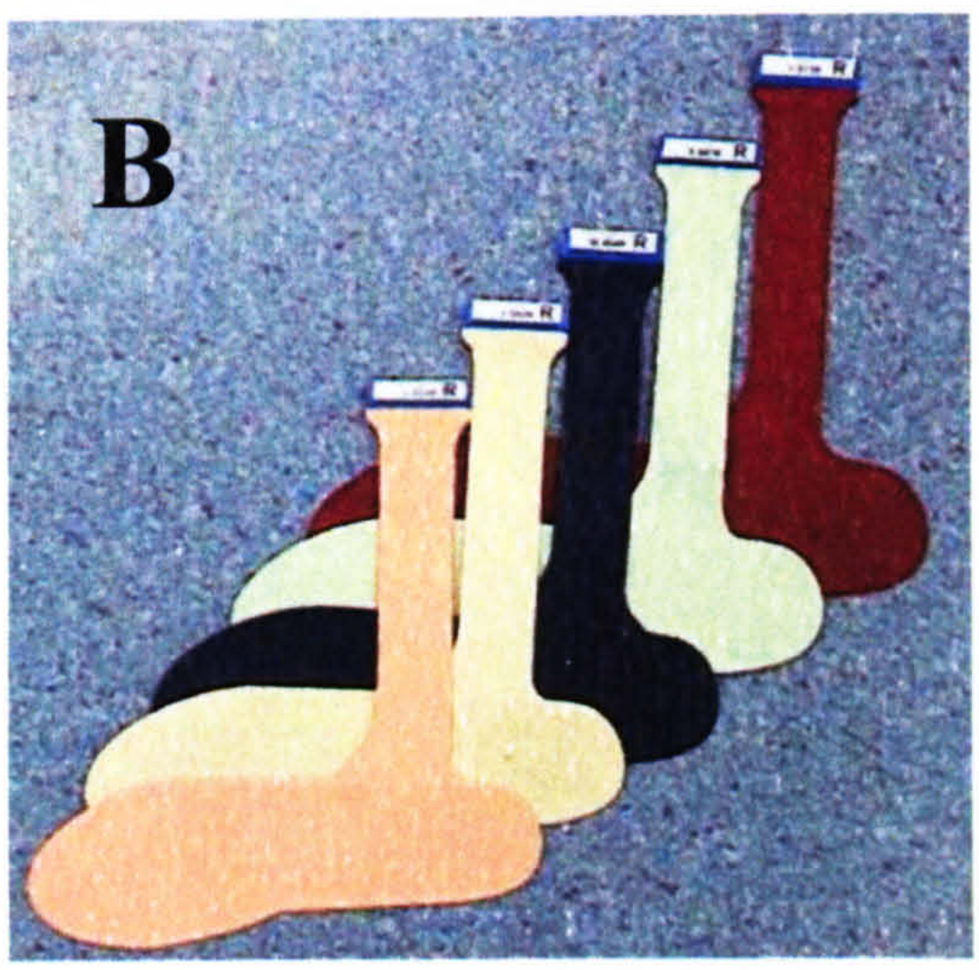


Figure 3-10: A- Person set up with Pedar in-shoe pressure measurement system.

B- Range of sizes of Pedar insoles.

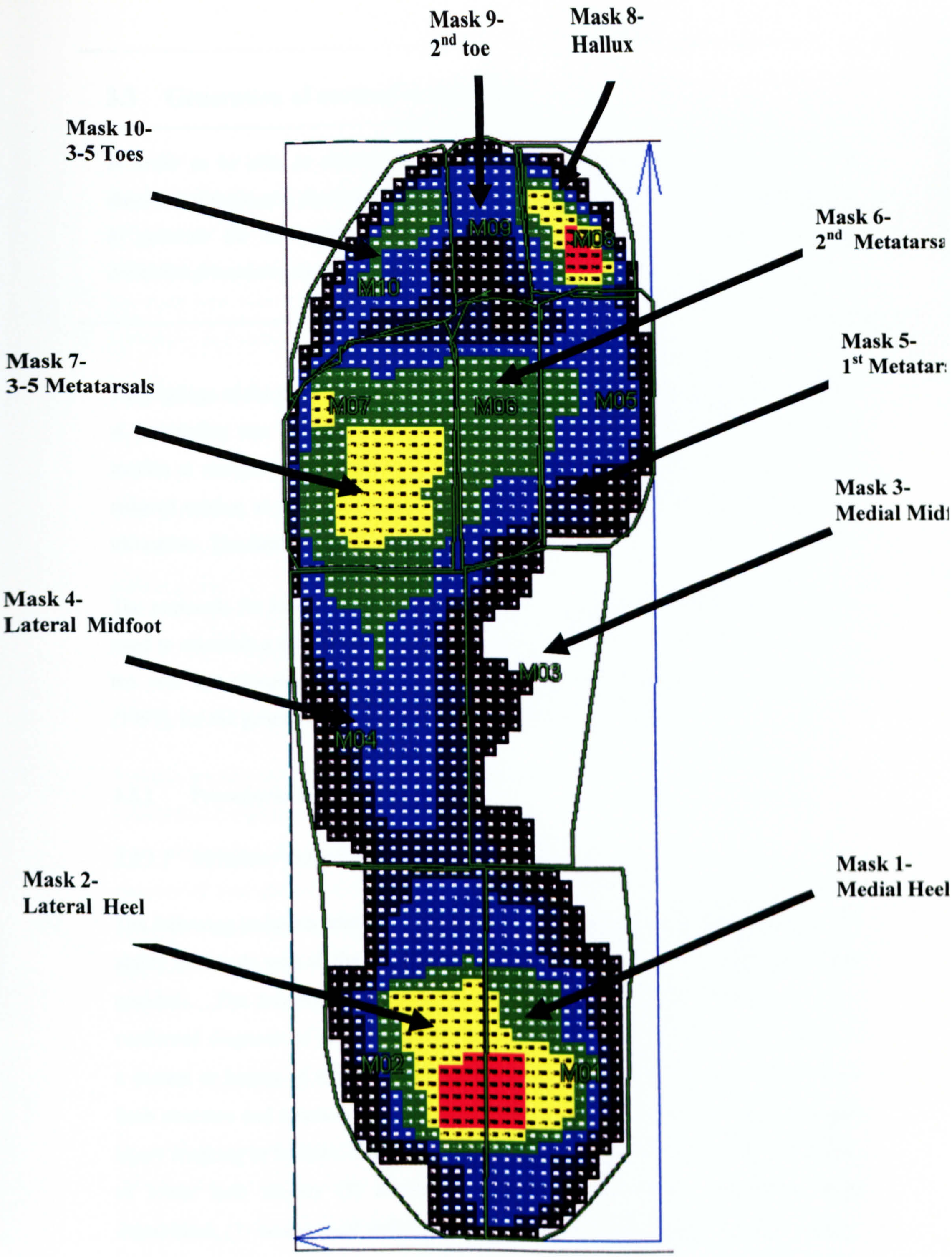


Figure 3-11: Automasks used to identify regions of the foot for further analysis.

3.3 Generation of normative database

In order to be able to answer the research questions outlined in section 2.7.1., a database of normative data had to be generated. This section outlines the methods used to generate the normative database, including recruitment, screening, and data collection procedures and data management.

The findings of the sampling rate study (showing that data could accurately be recorded at a sampling rate of 30Hz) allowed an additional two sensors to be used to study motion at another joint complex. As previously highlighted in the literature review, reduced motion at the 1st MPJ has been implicated with pathology and formation of ulceration. The database would include motion at the 1st MPJ.

The protocols for kinematic and plantar pressure measurement described above were used to establish a normative database for males and females aged 20-70, (n=100), in ten year age category groupings, based on the age groupings used by Öberg et al (1993), for the generation of reference data on a normal population.

3.3.1 Procedures for data collection

3.3.1.1 Inclusion / exclusion criteria

The following inclusion criterion was established for the normative study group, (1) the ability to comply with all the requirements of data collection (able to walk barefoot and unaided). The following exclusions were applied to volunteers in the study (1) a confirmed diagnosis of diabetes or suspected diabetes currently under investigation (2) a current or history of musculoskeletal disease or any condition likely to affect lower limb structure and function as determined by the podiatrist, (3) a history of lower limb injury resulting in fracture, dislocation or any soft tissue injury at any time, (4) a history of lower limb surgery (5) known alcoholism, (6) blindness / significant visual impairment, (7) neurological deficit, (8) a history of vascular disease, vascular surgery, (9) cognitive impairment of the patient apparent by history or during explanation of consent procedure.

3.3.1.2 Recruitment

Full ethical approval for the study was gained at the three clinical sites. Posters and email notification of the study were distributed through the University of Huddersfield and surrounding areas. Any interested participants were screened for exclusion criteria via telephone interview. Any participants who were deemed suitable for inclusion in the study were sent a study information sheet (see Appendix 4) and were invited to the University at a convenient time for data collection.

Each participant was asked to sign a consent form prior to collection of any data (see Appendix 5). The following demographic data were recorded, age (years), height (cm) and body weight (kg), Sex (male/female), race (white, Afro-Caribbean, Asian etc). Each participant underwent a thorough foot examination and the presence and location of any abnormalities were recorded (foot deformity, callus, corns, verrucae, nail pathology etc).

3.3.1.3 Screening procedures

3.3.1.3.1 Clinical examination of the foot

Patients underwent a screening process to establish vascular status; any subject with established peripheral vascular disease or a history of vascular surgery was excluded from the study. The diagnosis of peripheral vascular disease was based on the absence of foot pulses and / or symptoms of claudication or a history of vascular surgery. To be included in the study patients had to have palpable pulses, absence of rest pain, absence of intermittent claudication, no history of vascular disease or surgery, and not awaiting any vascular investigations.

3.3.1.3.2 Monofilament testing

In order to assess pressure perception, and exclude patients with neuropathy, three different sized monofilaments were used in this study, the 4.17, 5.07 and 6.10 filaments that bend at 1, 10 and 75 g of force respectively. The areas tested were the first, third and fifth plantar metatarsal heads and toes, the plantar aspect of the medial and lateral midfoot and heel. Testing was also performed on the dorsum of the foot between the first and second toes, the base of the third digit and the base of fifth metatarsal. The examiner demonstrated the monofilament sensation on one of the subject's arms. Areas

of callus, ulceration or scarring were avoided and when present the closest area to the lesion was tested. The subject was then instructed to close their eyes and to say yes each time they felt the application of the monofilament. The monofilaments were applied perpendicular to the skin's surface, with sufficient force to cause the filament to buckle, and held for approximately 1 second. Care was taken to ensure that the filament did not slide across the skin or make repetitive contact. Five trials were taken at each site starting with the 1g monofilament, if the subject could not perceive this monofilament the 10g and then the 75g monofilament were used. The order in which the test areas were tested and the time between application of the monofilaments were varied in order to try and reduce the element of guess work by the subject. As an additional test the subject was occasionally asked where they had felt the last application of the monofilament. The lowest monofilament that the subject could perceive at each test area was recorded. Five sets of monofilaments were used in the study and care was taken to ensure each monofilament was rested between testing. For the normative group any person unable to detect the 10g monofilament was excluded.

3.3.1.3.3 Vibration perception threshold

Vibration sensation was tested using the Neurothesiometer (Horwell, Nottingham UK). The Neurothesiometer was sent to the manufacturers to be calibrated prior to data collection for the study. The examiner demonstrated the sensation of vibration on the subject's ulnar process. The unit displays the applied voltage, which ranges from 0-50 volts. The Neurothesiometer was held with the tractor balanced vertically on the pulp of the great toe, the voltage was increased on the base unit until the patient could perceive the vibration. Three readings for each foot were recorded; the speed at which the voltage was altered was varied for each trial.

3.3.1.3.4 Assessment of joint movement at the AJC and 1st MPJ.

To be able to include measurement at the 1st MPJ, an expanded kinematic model was established to include 2 additional motion sensors. For this model motion sensors were placed on the tibia, calcaneus, first metatarsal and proximal phalanx of the hallux. The positions of the tibial and calcaneal sensor have previously been described (section 3.2.1). The hallux sensor was placed on the mediodorsal apect of the proximal phalanx, medial to the extensor hallucis longus tendon. The metatarsal sensor was placed on the mediodorsal aspect of the diaphysis avoiding the tendon of extensor hallucis longus and

abductor hallucis. Umberger and associates have described these sites for sensor placement for the measurement of sagittal plane motion at the first metatarsal phalangeal joint (Umberger et al 1999). In a study of reliability of skin placed electromagnetic tracking sensors they reported the reliability and validity to be high and stated that electromagnetic tracking could be confidently used for measurement of first metatarsal phalangeal joint kinematics.

The kinematic set-up procedure and method of data acquisition previously described in sections 3.2.1 and 3.2.2 were performed and an outline of the overall procedure is shown (Figure 3-12). The bore-sight position was taken using the jig method and the subject was placed into the pre-determined position previously described (section 3.1.3.2). They were then carefully guided onto a low wooden examination couch positioned in front of the transmitter units.

Passive ranges of motion at the subtalar joint and the 1st MPJ were determined with the subject in a supine position with lower part of the leg overhanging the edge of the examination couch so as not to disturb the position of the calcaneus sensor. The subject was instructed to relax and to not try and help or resist as the examiner moved the joints. The examiner moved the AJC to the end ranges of frontal plane motion three times and maximally dorsiflexed the hallux three times for each limb. The subject was then carefully guided off the examination couch making sure the position of the sensors did not alter. Five walking trials were then collected for each limb; the hallux and metatarsal sensors were then removed and secured into the waistband to allow the foot to be placed in the shoe. Five shod walking trials were then recorded for each limb. The electromagnetic tracking sensors and foot switches were removed and the in-shoe pressure data was then recorded using the method previously described in section 3.2.4.

3.3.2 Data management

Each subject was coded and all the demographic, and clinical data were recorded and stored in a Microsoft Excel spread sheet. Calculations were performed within the 6D Research software on the static and dynamic walking trials to determine the motion at the AJC and 1st MPJ. For static range of motion the total frontal plane range of motion

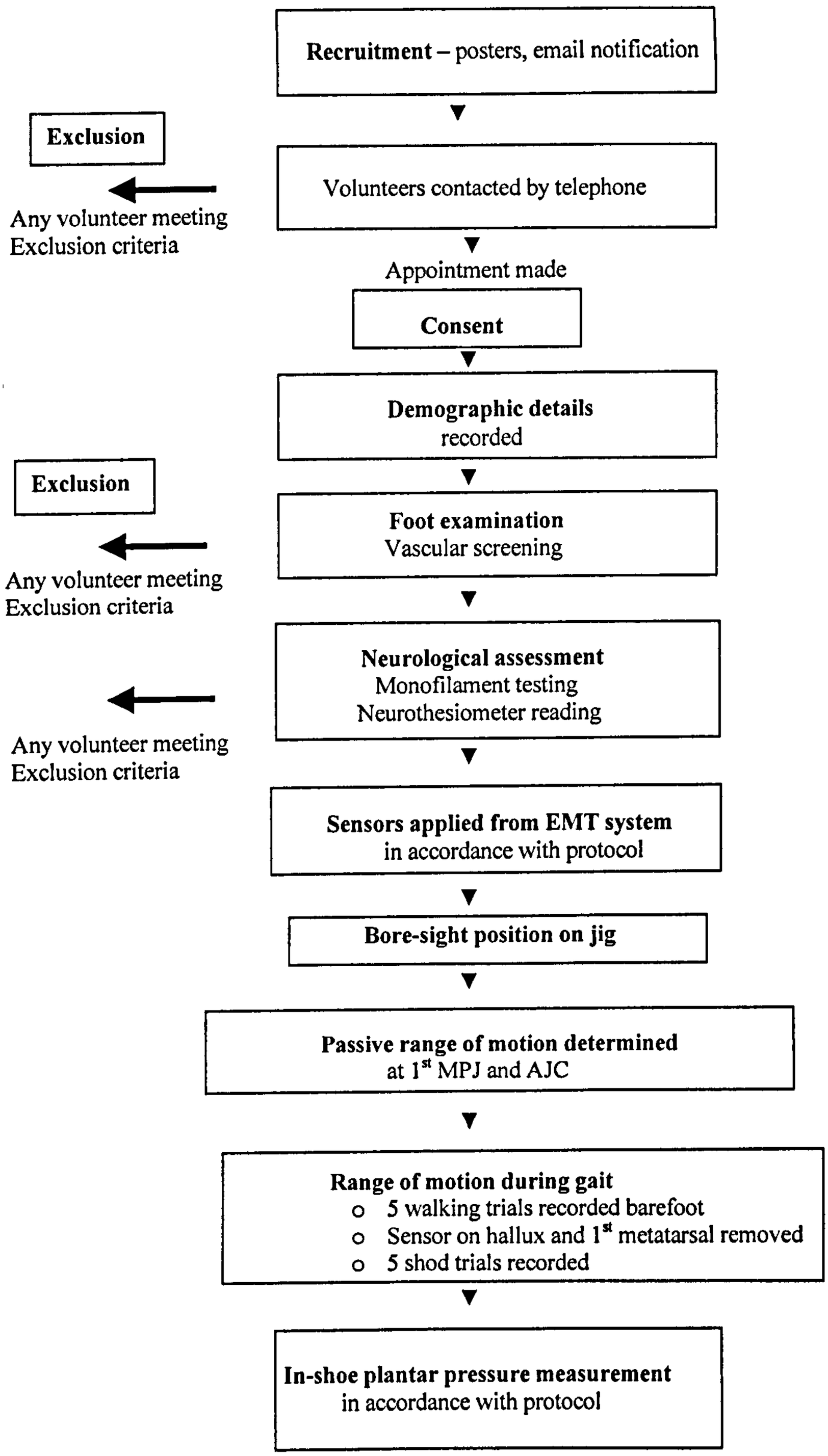


Figure 3-12. Outline of procedure for data collection on non-diabetic control group

at the AJC and the maximum dorsiflexion at the 1st MPJ were recorded. The total range of frontal plane motion at the AJC and maximum dorsiflexion at the 1st MPJ were calculated during stance phase for each walking trial (data presented for each decade can be found in Appendix 6). Each walking trial was normalised to 100% of stance phase using Datapac software and an average motion time curve for left and right limbs was generated for each subject and for each age / sex grouping. The mean motion time curve for each patient was exported into Excel spreadsheet and SPSS for Windows ™ for further analysis.

For the pressure data, the middle five steps were analysed for each subject; these were averaged using specially designed software. Masks were created to examine pressure parameters in relation to the underlying anatomical structures. The mask areas selected for analysis were the same as used by Cavanagh and Ulbrecht (1994), (anatomical regional areas of interest, developed for the diabetic foot). The automask software program was used to ensure standardised positioning of the regional masks for each subject. Calculations were performed to determine the mean peak pressure, pressure time integral and contact area in each mask area for every individual and across the whole group. The data for each subject was exported into SPSS for Windows ™ for further analysis. A table of mean pressure variables for the whole normative group can be found in Appendix 7.

3.3.3 Statistical analyses

The demographic details were prepared as mean (SD) for each of the four groups. For statistical analysis of differences between subject groups, a one-way Anova Tukey's HSD test for post-hoc multiple comparisons using a significance level of 0.05 was used.

3.4 Methods required for diabetic assessment

In order to be able generate a diabetic study group, a screening procedure had to be developed to be able to assign participants in the study to control, neuropathic or ulcerated group. This section outlines the methods used to recruit participant into the study, outlines the neurological screening assessments and the criteria used to assign participants into the appropriate study group.

3.4.1 Subject recruitment and inclusion / exclusion criteria

Participants for the study were recruited through the diabetes centre at United Leeds Teaching Hospitals Trust, Huddersfield NHS Community Trust and the Department of Podiatry at the University of Huddersfield. Ethical approval for the study was gained at all three clinical sites.

The following inclusion were established, (1) a confirmed diagnosis of diabetes, (2) the ability to comply with all the requirements of data collection (able to walk barefoot and unaided). The following exclusions were applied to participants in the study (1) a current or history of musculoskeletal disease or any condition likely to affect lower limb structure and function as determined by the podiatrist, (2) a history of lower limb injury resulting in fracture, dislocation or any soft tissue injury, (3) any known cause other than diabetes which may cause neuropathy, (4) known alcoholism, (5) blindness / significant visual impairment, (6) a history of vascular disease, vascular surgery, (7) cognitive impairment of the patient apparent by history or during explanation of consent procedure.

Those patients who satisfied the criteria and expressed a willingness to participate in the study were given a patient information sheet (see Appendix 4) and their contact details were recorded. The volunteers were later contacted to establish if they were willing to be included in the study and to arrange a mutually convenient appointment time for data collection. A consent form was then completed (Appendix 5).

3.4.2 Assessment of vascular status

Patients underwent a screening process to establish vascular status; any person with established peripheral vascular disease or a history of vascular surgery was excluded from the study, because this specifically has been shown to influence the mechanism of plantar ulcer formation (Edmonds 1996). The diagnosis of peripheral vascular disease was based on the absence of foot pulses and / or symptoms of claudication or a history of vascular surgery. To be included in the study participants had to have palpable pulses, absence of rest pain, intermittent claudication, no history of vascular disease or surgery and not awaiting any investigations related to vascular disease.

3.4.3 Demographic Data

For all patients, age (years), sex (male / female), ethnic origin, duration of diabetes (years) and type of diabetic control (diet / tablets / insulin), smoking history and alcohol consumption were recorded. The patient's height (cm) and bodyweight (kg) were recorded.

3.4.4 Screening procedure and assignment of clinical group

An optimal screening test has been defined as simple and quick to perform and yields the same results when carried out by different observers and accurately measures or predicts a clinically important condition (high validity against an independent and clinically meaningful criterion reference standard) (Smieja et al. 1999). When screening patients with diabetes for risk of foot ulceration no testing procedure has yet been demonstrated to fulfill all of these criteria, therefore, a number of different testing procedures must be undertaken.

3.4.4.1 Assessment of neurological status

Neuropathic symptoms score were assessed using a modified version of the neuropathy symptom score (NSS) (Boulton 1998). Patients were questioned about the presence or absence of muscular cramps, numbness, tingling sensations, burning pain, aching pain, abnormal hot or cold sensations and irritation from bedclothes in the feet and lower legs. If the patient did not have a given symptom, then a score of zero was assigned, if

the patient reported a symptom then a score of 1 was given, and if the patient described nocturnal exacerbation a score of 2 was assigned.

A modified version of the neuropathy disability score (NDS) was used to quantify severity of neuropathy obtained from a physical examination. The score comprises of measurement of clinical signs, including assessment of tendon reflexes, temperature, pain and vibration sensation. Pin-prick sensation using a sterile neurotip and vibration using a 128MHz tuning fork were tested at the apex of the left and right hallux. Temperature sensation was tested on the dorsum of the foot using hot and cold metal rods. A score of 1 was given if the patient could not perceive the stimulus. Achilles tendon reflexes were examined with the patient seated, a score of 0 was given if the reflex was normal, a score of 1 assigned if the reflex was present with reinforcement, a score of 2 was given if the reflex was absent. The categorization of the presence of neuropathy using the NSS and NDS are summarized in Table 3-16.

In addition to using the modified NSS and NDS to assess neurological status vibration perception and monofilament testing was also performed. Vibration perception was determined using the Neurothesiometer using the method described previously (section 3.3.1.3.3). A description of the method and sites tested with the monofilament can be found in section 3.3.1.3.2. All the screening assessments were undertaken by the lead researcher in order to eliminate inter-tester variability.

3.4.4.2 Examination of the foot

A systematic examination of feet was performed by only one examiner to minimize the possibility of missing a specific defect. Only one foot was examined at a time (right foot first) whilst the patient was non-weight-bearing. The dorsum of the foot was examined first, examining the nails, dorsum and apices of toes and finally the inter-digital area. The plantar area of the foot was examined starting with the toes then the plantar metatarsal area and finally the heel and around the borders. The presence of any foot deformity was recorded using a six-point foot deformity score (Abbott et al. 2002). The presence of small muscle wasting (wasting of the small muscle in the foot sufficient to cause "troughing" between the tendons), hammer or claw toes, bony prominences, prominent metatarsal heads, charcot arthropathy and a positive prayer sign was noted. Each deformity scored 1 when present or 0 when absent on either foot.

Table 3-16 - Categorisation of the presence of neuropathy in patients using the modified versions of the neuropathic disability score and the neuropathic symptom score. *Taken from (Young et al. 1993a).*

NSS (symptoms)

Total maximum score of 9

Score of less than 3	-	Non neuropathic
Score of 3-4	-	Mild symptoms of neuropathy
Score of 5-6	-	Moderate symptoms of neuropathy
Score of 7-9	-	Severe symptoms of neuropathy

NDS (signs)

Total maximum score of 10

Score of less than 3	-	Non- neuropathic
Score of 3-5	-	Evidence of mild neuropathy
Score of 6-8	-	Evidence of moderate neuropathy
Score of 9 or 10	-	Severe signs of neuropathy

The minimal acceptable criteria for a diagnosis of peripheral neuropathy

(1) Moderate signs with or without symptoms

(2) Mild signs with moderate symptoms

A score of 3 or more was defined as indicative of significant foot deformities (Abbott et al. 2002). Only established skin lesions were recorded, no attempt was made to classify severity of lesions but differentiation between lesion types was made. Callus was defined as any diffuse areas of relatively thickened skin easily recognized by clinical examination.

3.4.4.2.1 Active / previous ulceration

Patient's feet were examined and the presence of any active ulcer was noted. Patients were asked if they had any foot ulcer history and this was verified and documented from their medical records. An ulcer was defined as a full thickness skin defect that required more than 14 days to heal (Boyko et al. 1999).

3.4.5. Assessment of passive range of joint movement, dynamic joint movement and plantar foot pressures

The measurement protocols outlined in sections 3.1.2.1 and 3.2.4 for recording joint movement data and plantar pressure measurement were used. Any active ulcers were debrided prior to data collection and Opsite Flexigrid (Smith and Nephew Ltd, Hull, UK) was applied over the ulcer (a thin flexible dressing, which would have minimal effect on plantar pressure). Care was taken to ensure wounds were completely covered and not discharging through the Opsite Flexigrid dressing. After data collection was completed, the ulcer was cleansed with warm sterile saline and re-dressed with an appropriate dressing and the Pedar insoles were thoroughly cleaned between patients. Any patients with plantar calluses had their callus debrided prior to data collection.

3.4.6 Data management and statistical analysis

Each patient was coded and all the demographic, and clinical data were recorded and stored in an Microsoft Excel spreadsheet. Calculations were performed within the 6D Research software on the passive range of motion and dynamic walking trials to determine the motion at the AJC and 1st MPJ. For passive range of motion the total frontal plane range of motion at the AJC and the maximum dorsiflexion at the 1st MPJ were recorded. The total range of frontal plane motion at the AJC and maximum dorsiflexion at the 1st MPJ were calculated during stance phase for each walking trial. Each walking trial was normalised to 100% of stance phase using Datapac software (RUN Technologies, CA, USA) and an average motion time curve left and right limb

was generated for each subject and for each clinical grouping. The demographic details were prepared as mean (SD) for each of the four groups. For statistical analysis of differences between subject groups, a one-way Anova Tukey's HSD test for post-hoc multiple comparisons using a significance level of 0.05 was used.

CHAPTER 4

RESULTS

The results relating to demographic details are presented as means (standard deviation) for each group. The data are outlined descriptively highlighting overall trends and clinically meaningful differences. Joint motion, gait data and plantar pressure data are presented separately and then the relationship between them examined. Inferential statistics are presented; in all cases data are presented with statistical significance at the 5% level unless otherwise stated.

4.1 Descriptive summary of clinical data

4.1.1 Patient recruitment, assignment and participation flow

A sample of convenience was recruited. A total of 110 diabetic patients attending clinics at Leeds General Infirmary, The Department of Podiatry at the University of Huddersfield and the Princess Royal Clinic Huddersfield, were identified as potential subjects for the study. One hundred and four patients were screened for inclusion in the study, of these a total of twenty-one patients were excluded from the study due to poor vascular status, or inability to walk barefoot (Figure 4-1).

4.1.2 Patient demographics

A total of 83 diabetic patients were recruited into the following groups, control (n=30), neuropathic (n=28) and ulcerated (n=25). The mean age for the three groups were similar, 59.1(SD12.7) years, 62.7(SD9.3) years and 58.1(SD10.8) years in the control, neuropathic and ulcerated groups respectively. A non diabetic reference group was selected from the normative groups generated in the previous chapter, matched for age within decade. The mean age of the non diabetic reference group was 58.4 (SD9.2).

The disease duration was greater in the ulcerated and neuropathic groups compared to the diabetic control group, but the difference did not reach a level of statistical significance. One patient in the neuropathic group was Afro Caribbean origin, and one patient in the control group was Asian origin. The number of smokers in each group were similar, 8, 6 and 8 patients were smokers in the control, neuropathic and ulcerated groups respectively. The key patient demographics are presented in Table 4-1. The key difference between the groups is the higher proportion of males in the ulcerated group in comparison to the other groups. Attempts were made to try and balance the number of males and females within each group, however, during the data collection period the number of females attending the foot ulcer clinics were greatly reduced in comparison to the males. A statistically significant difference was found between body mass in the ulcerated group compared to all the other groups, the higher proportion of males in the ulcerated group could partly explain this.

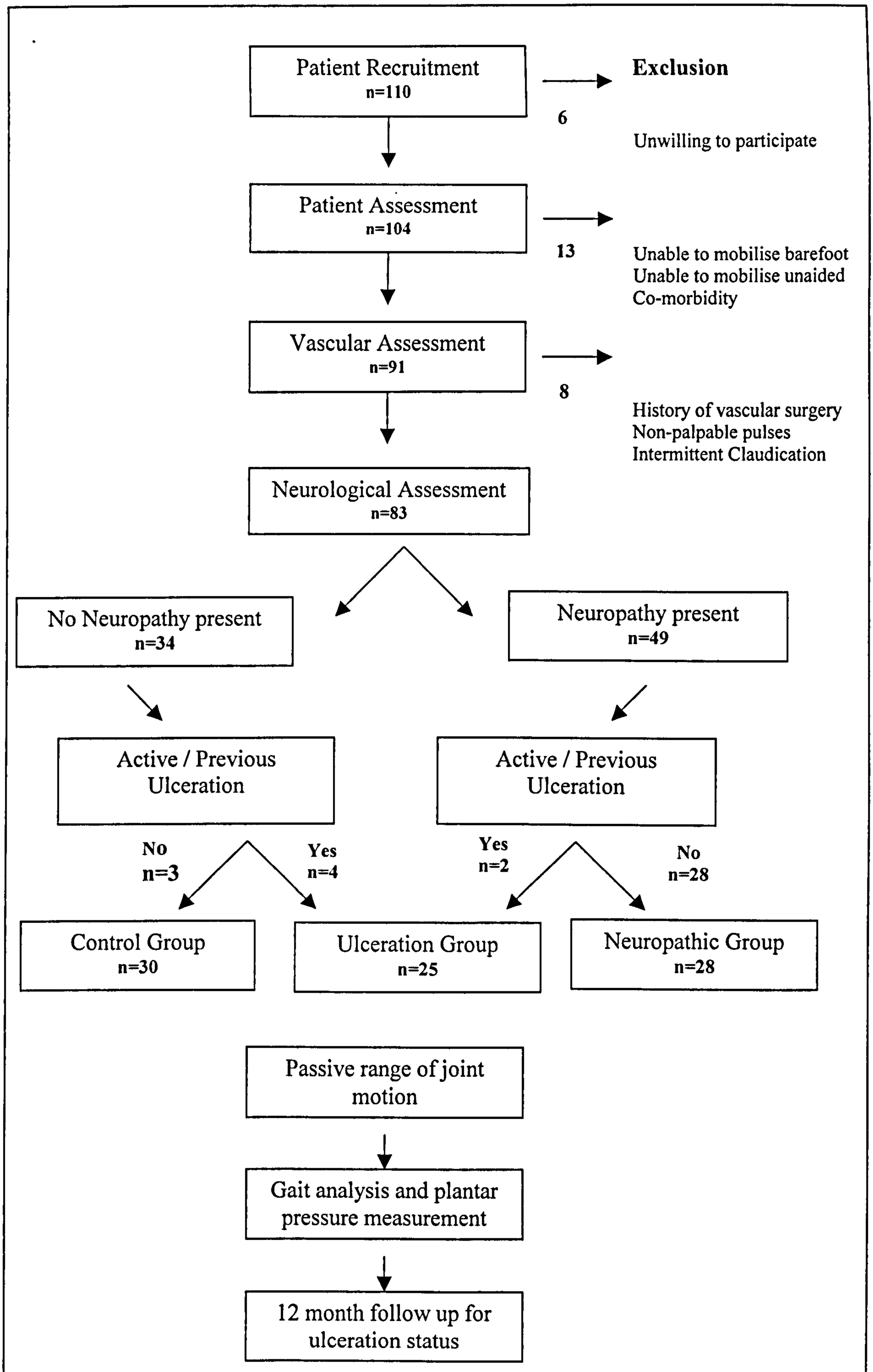


Figure 4-1. Recruitment details and participant flow

Table 4-1: Demographic and clinical details for the non diabetic reference, diabetic control, neuropathic and ulcerated groups. Values are mean (SD) unless stated.

Variable	Non diabetic Reference (n=25)	Control (n=30)	Neuropathic (n=28)	Ulcerated (n=25)
Sex (F:M)	11:14	11:19	13:15	5:20
Age (Years)	58.4 (9.2)	59.1 (12.7)	62.7 (9.3)	58.1 (10.8)
Height (cm)	172.5 (9.3)	169.0 (10.2)	171.4 (11)	175.7 (10)
Weight (kg)	79.5 (15.9)	77.7 (14.3)	80.7 (16.6)	91.7 (14.4)
Disease Duration (years)	-	10.6 (12.3)	12.7 (13.6)	16 (11.6)
Method of control (D:T:I)	-	3:19:8	2:14:12	1:9:15
NSS	-	2.47 (2.76)	5.1 (2.8)	4.6 (2.7)
NDS	-	1.1 (1.6)	5.6 (2.2)	6.7 (2.7)
Neurothesiometer (Volts)	8.5 (6.0)	10.2 (6.9)	27.5 (13.4)	35.4 (14.3)
% Unable to detect 10g MF (correct identification > 80%)	0	0	71.4	84
Positive Prayer sign (n)	-	22	24	21
FDS	-	1.9 (1)	1.3 (0.9)	2.4 (1.1)

(D:T:I), Diet: Tablets: Insulin, NSS, Neuropathic symptom score, NDS, Neuropathic disability score, MF, monofilament, FDS: Foot deformity score.

4.1.3 Location of ulceration

In the ulcerated group, 13 patients had active ulceration and the remaining 12 had a previous history of plantar ulceration. Three of the patients had ulceration present on the dorsal toe areas. Previous ulcerations were reported by patients and were then verified by the patients medical records. The distribution of plantar ulcers in this group are presented in Table 4-2.

Table 4-2: Number and location of plantar foot ulcers (active / previous) in the ulcerated group.

Anatomical Site	Active Ulceration	Previous Ulceration
1st MPJ	4	5
2nd MPJ	1	0
3-5 MPJ	2	4
Plantar hallux	2	7
Apex 2nd Toe	2	1
Apex 3-5 toes	2	1
Lateral midfoot	1	0

MPJ- metatarsophalangeal joint.

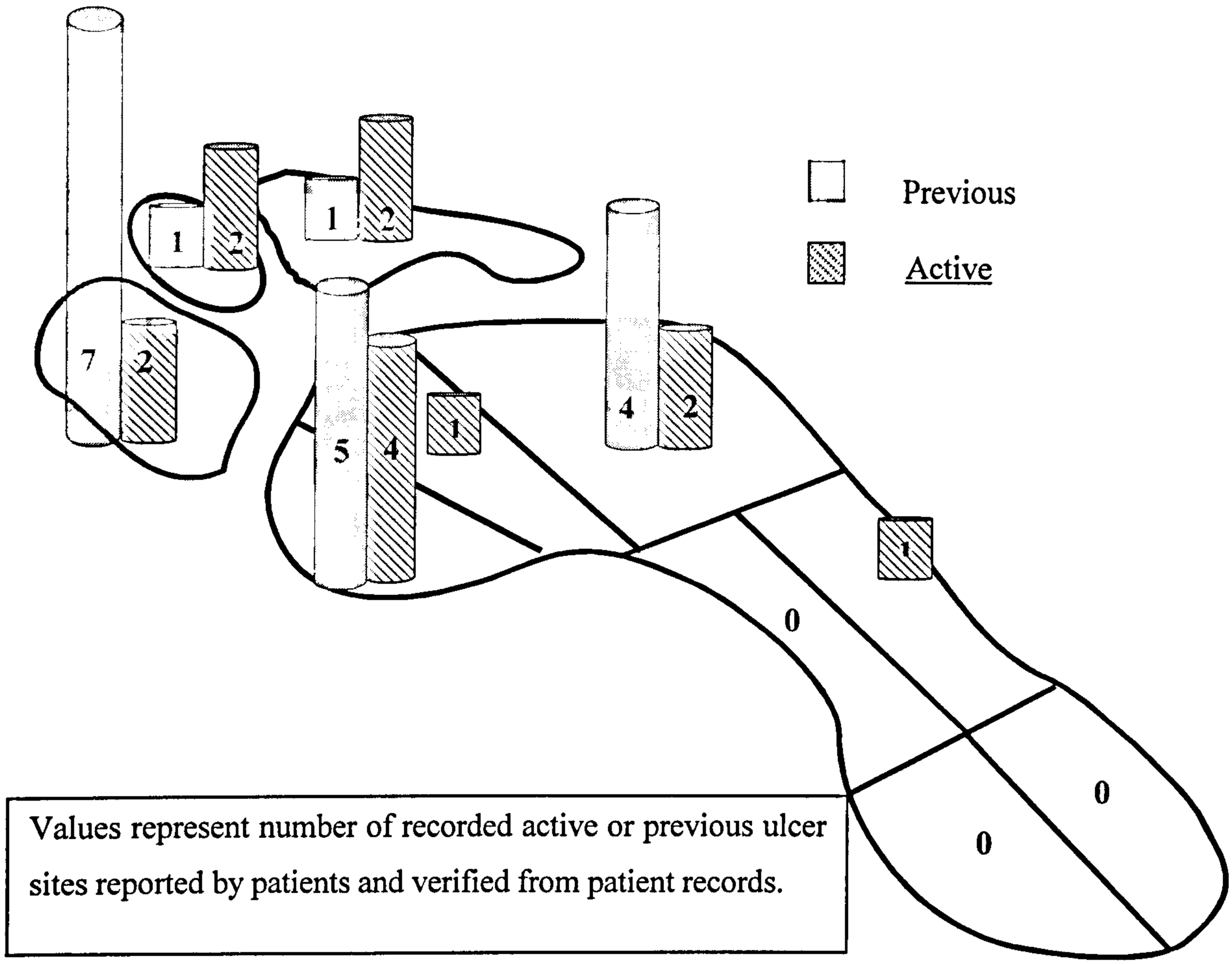
4.1.4 Location of callus

In the control, neuropathic and ulcerated groups, 11, 9 and 19 patients presented with callus formation. The most prevalent callus patterns are presented in Table 4-3.

Table 4-3: Callus patterns in the control, neuropathic and ulcerated groups

Site	Control (n=30)	Neuropathic (n=28)	Ulcerated (n=25)
1st MPJ	5	7	7
2nd MPJ	1		
3rd MPJ	2		
4th MPJ			
5th MPJ	2	2	3
1-2 MPJ			
2-3 MPJ	1	1	
3-4 MPJ			
4-5 MPJ			
2-4 MPJ	4		
1-3 MPJ	1		
1-5 MPJ	2	2	12
1st IPJ	1	3	3
Apex 2nd	2	1	
Apex 3rd	2	1	
5th Pl toe		1	
Lateral midfoot			1
Medial heel	2		
Total	**26**	**18**	**26**

MPJ, metatarsophalangeal joint, IPJ, interphalangeal joint.



Values represent number of recorded active or previous ulcer sites reported by patients and verified from patient records.

Figure 4-2: Location of active and previous ulceration.

Active or previous ulceration were recorded at ten different anatomical sites, however, three areas (the plantar hallux, plantar area of the 1st MPJ, and lateral forefoot area) predominated. These three areas represented 75% of the ulceration sites recorded during the study.

4.1.5 Foot deformity score

The mean foot deformity score was 1.87 (SD 0.8), 1.3 (SD 0.9) and 2.4 (SD 1.1) in the control, neuropathic and ulcerated groups respectively. A statistically significant difference was found between the ulcerated group and the neuropathic group $P<0.05$.

4.1.6 Neurological data

The mean neurological disability score (NDS) in the control group was 1.1 (SD 1.6) compared to 5.6 (SD 2.2) in the neuropathic group and 6.7 (SD 2.7) in the ulcerated group. The mean neuropathic symptom score (NSS) was 2.5 (SD 2.8), 5.1 (SD 3), and 4.6 (SD 2.7) in the control, neuropathic and ulcerated groups respectively. The mean Neurothesiometer reading in the control group was 10.2 (SD 6.9), in the neuropathic group 27.5 (SD 13.4), and 35.4 (SD 14.3) in the ulcerated group. The number of patients who exceeded a value of 25 volts in the neuropathic group was 16 and 17 in the ulcerated group.

All the patients in the control group could detect the 10g monofilament (based on patients giving the correct response 80% of the time). In the neuropathic group 8 patients could detect the 10g monofilament and 4 patients could detect the 10g monofilament in the ulcerated group at all sites tested.

In the identification of the 25 foot ulcer patients, the neurological screening used in this study was highly sensitive (88%) but less specific (52%) than the vibration perception threshold (sensitivity 68%, specificity 72%) and the monofilaments (84% sensitivity, 67% specificity, based on an 80% correct identification and 84% sensitivity and 60 % specificity, based on patients giving the correct response 100% of the time). When the neurological screening used in this study was combined with vibration perception or monofilament testing in order to identify the number of patients with ulceration the sensitivity increased (92% when combined with vibration perception and 100% when combined with monofilaments) but the specificity remained at 52%. Combining the monofilament testing with vibration perception in order to identify the ulcerated patients resulted in a sensitivity of 100% and specificity of 60%.

In the identification of patients at risk of ulceration the neurological screening used in this study agreed with the vibration perception threshold in 60% of cases and with the monofilaments in 84% of cases.

4.2 Joint Motion

4.2.1 Motion at the 1st MPJ

4.2.1.1 Passive range of dorsiflexion

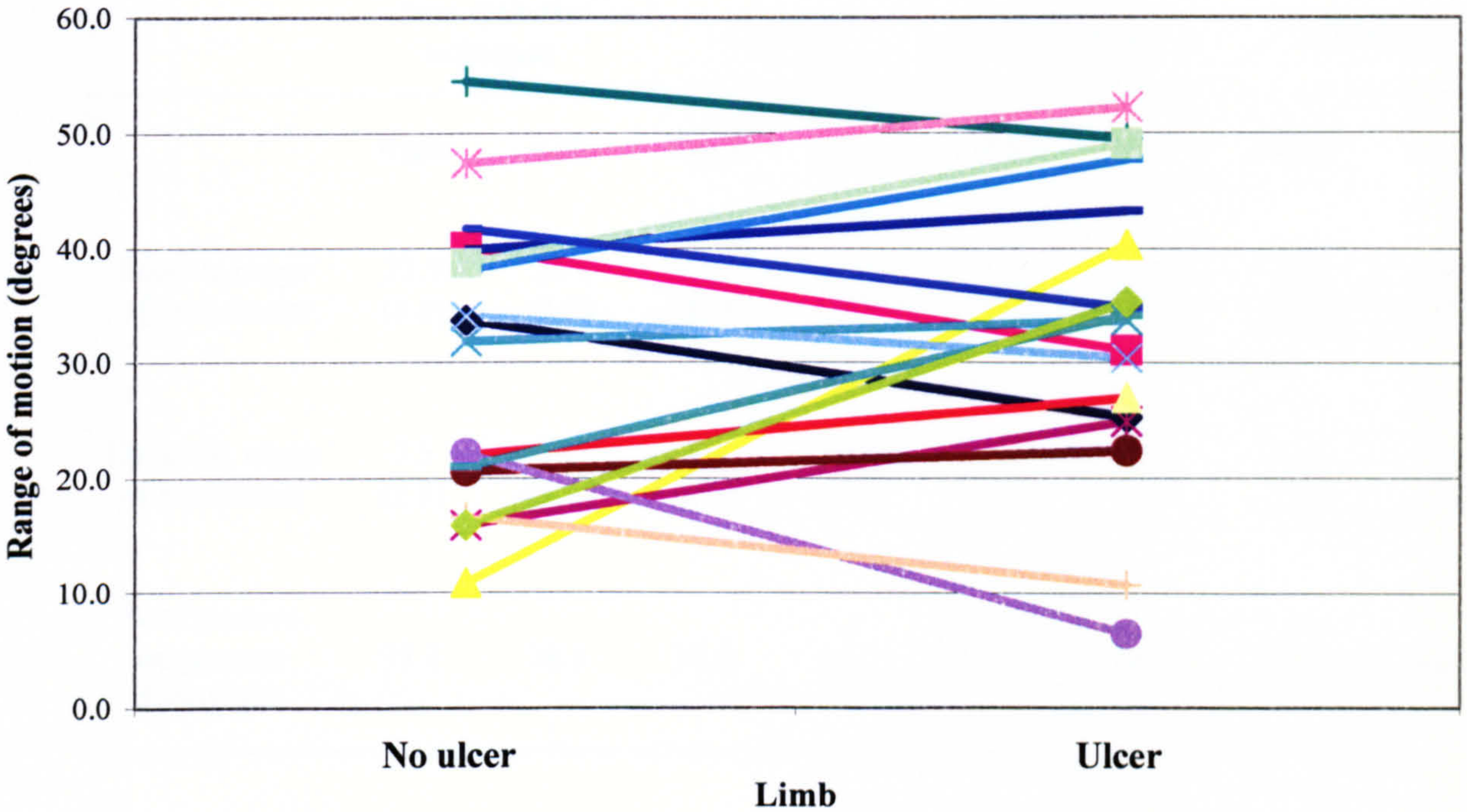
The magnitude of the passive range of dorsiflexion at the 1st MPJ was comparable in the non diabetic reference, control and neuropathic groups but was reduced in the ulcerated group (Table 4-4). A statistically significant difference was found between the non diabetic reference and control groups compared to the ulcerated group. Differences were noted between the left and right limb, in all groups the left limb had increased range of dorsiflexion, however, this did not reach statistical significance. In the ulcerated group, the joint motion in the ulcerated limb was compared to the joint motion in the contra-lateral limb (Figure 4-3). The passive range of dorsiflexion at the 1st MPJ was 31.2° (SD12.1) and 30.5° (SD10.6) in the ulcerated and contra-lateral limbs respectively. The range of dorsiflexion during the stance phase of gait was slightly increased in the ulcerated limbs 26.7° (SD6.8) compared to the contralateral non-ulcerated limb 24.3 ° (SD6.4) but did not reach a statistically significant difference. However, when the amount of dorsiflexion during the stance phase of gait, expressed as a percent of the passive range of dorsiflexion was compared between limbs, the mean percentage used in the ulcerated limb was significantly greater in the ulcerated limb compared to the contra-lateral limb (116.2% SD 106, vs 87.7 SD 41).

4.2.1.2 Dorsiflexion at the 1st MPJ during the stance phase of gait.

The mean values for range of dorsiflexion at the 1st MPJ during gait were comparable in all the diabetic groups, but were statistically significantly higher in the non diabetic reference when compared to all the diabetic groups. The ulcerated group used the highest percentage of their passive range of dorsiflexion during gait.

Table 4-4: Mean (SD) passive ranges of dorsiflexion at the 1st MPJ and dorsiflexion during the stance phase of gait (in degrees).

	Non diabetic Reference		Control		Neuropathic		Ulcerated	
	Right	Left	Right	Left	Right	Left	Right	Left
Passive Range of dorsiflexion (Degrees)	37.4 (14.1)	45.3 (13.4)	36.9 (13.9)	43.3 (13.5)	37.6 (17.2)	40.7 (16)	30.4 (12.2)	32.9 (12.5)
Dynamic Range of dorsiflexion (Degrees)	35.8 (10)	29.5 (7.1)	25.2 (7.2)	24.7 (7.2)	27.4 (7.4)	25.5 (6.3)	22.4 (7.7)	26.3 (6.5)
% of passive range of dorsiflexion used during gait	69.6	64	68.3	57	72.9	62.7	73.7	80



Each line represents an individual patient who had a unilateral ulceration

Figure 4-3: Mean passive range of motion at the 1st MPJ (in degrees) in the ulcerated and contra-lateral limb of patients in the ulcerated group.

4.2.2 Movement at the AJC

4.2.2.1 *Frontal plane movement at the AJC*

The overall trend was a reduction in the passive frontal plane range of movement at the AJC from the non diabetic reference group through to the ulcerated group (Table 4-5). The passive range of movement and movement during the stance phase of gait was similar across the diabetic groups, but significantly reduced when compared to the non diabetic reference group. When the percentage of passive range of motion used during the stance phase of gait was compared between the groups significant differences were found between the non diabetic reference group and the neuropathic and ulcerated groups *($P<0.05$)*. The ulcerated group used the highest percentage of the passive range during the stance phase of gait.

Table 4-5: Mean (SD) range of frontal plane motion at the AJC measured during passive joint assessment and recorded during the stance phase of gait.

	Non diabetic reference		Control		Neuropathic		Ulcerated	
	Right	Left	Right	Left	Right	Left	Right	Left
Passive range of movement	23.9 (5.6)	24.1 (5.7)	18.1 (6.9)	16.9 (4.9)	16.2 (5.5)	15.8 (9.7)	15.6 (4.8)	14.3 (5.3)
Dynamic range of movement	7.5 (2.5)	6.9 (2.3)	6.4 (2.2)	5.6 (3.7)	7.5 (4.8)	6.1 (2.7)	6.5 (2.6)	6.2 (2)
% of passive range used during gait	33.2	28.5	35.4	33.1	46.3	38.6	41.7	43.4

In the ulcerated group the passive range of frontal plane motion at the AJC in the limb with active / previous history of ulceration was compared to the contra-lateral non ulcerated limb (Figure 4-4). No statistical significant difference between the limbs was found. There appeared to be equal numbers of patients with an increased or decreased range of motion in the ulcerated limb compared to the contra-lateral limb.

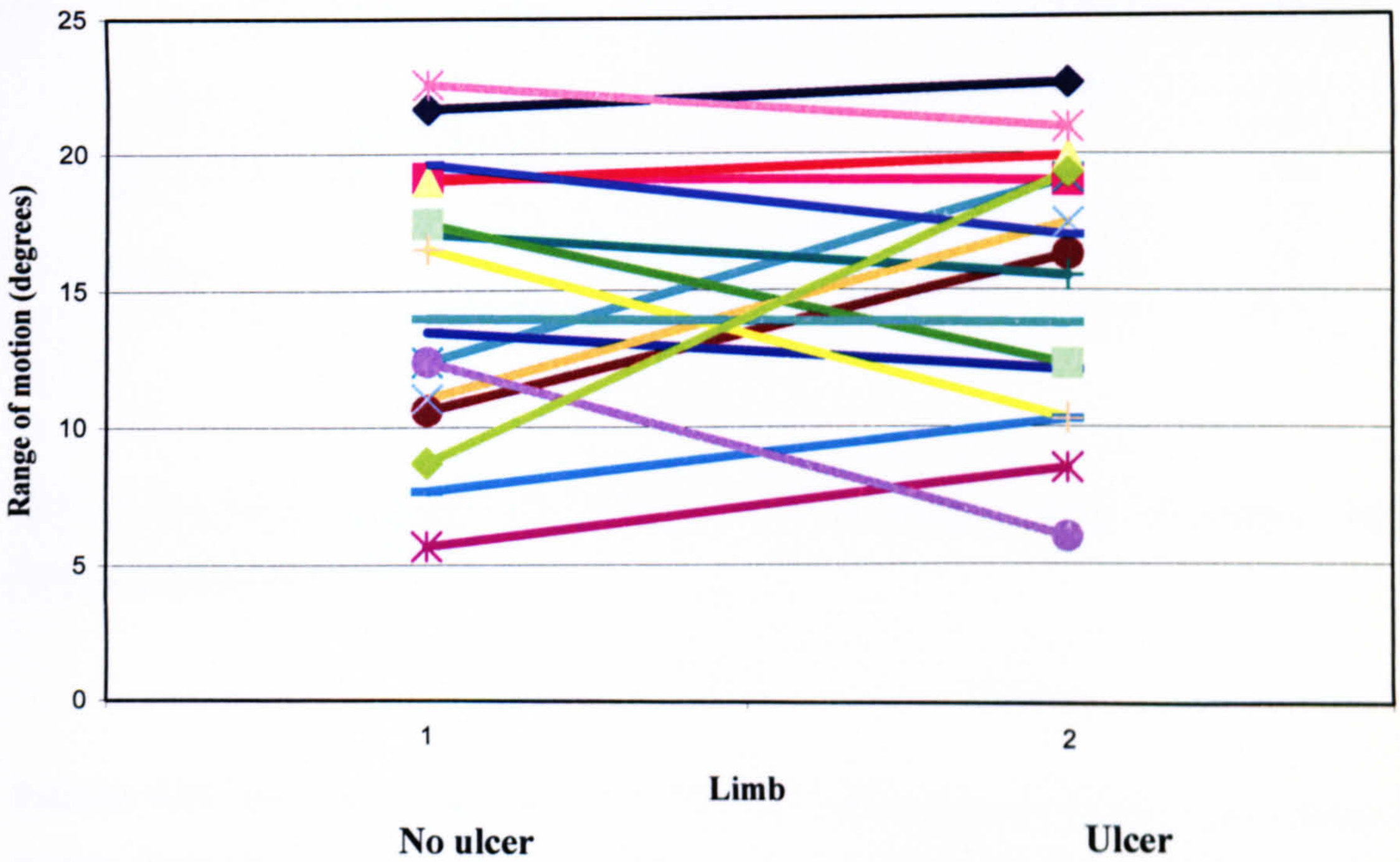


Figure 4-4: Passive range of frontal plane motion at the AJC (in degrees) in the ulcerated and contra-lateral limb in the ulcerated group.

In the ulcerated group only the patients with ulceration at the 1st MPJ, lateral forefoot area and plantar hallux were analysed in more detail (Table 4-6). These sites were chosen as they represented the most common sites for ulceration within the study group.

Table 4-6: Mean (SD) Joint range of motion (in degrees) in patients with ulceration at the hallux, lateral forefoot or under the 1st metatarsal head.

Ulcer site	AJC Dorsiflexion / plantarflexion		AJC Inversion / eversion			MPJ Dorsiflexion	
	BF	Shod	PROM	BF	Shod	PROM	BF
1st MPJ	12 (3.6)	14.7 (5)	12.5 (4.4)	7.0 (2.7)	8.3 (2.7)	29.8 (13.7)	25.4 (8.7)
MPJ 2-5	11.2 (2)	16.4 (3.9)	16.4 (3.9)	6 (4.1)	6.6 (2.6)	31.7 (11.1)	19.8 (8.4)
Hallux	14.5 (2)	16.7 (7)	16.6 (7)	7.5 1.9)	6.9 (2.4)	34.3 (15)	24 (8.5)

AJC- ankle joint complex, BF- barefoot, PROM- passive range of motion, MPJ- metatarsophalangeal joint.

Patients with ulceration under the hallux had the highest amount of ankle joint range of motion during barefoot and shod gait. Patients with ulceration under the 1st metatarsal head had the lowest passive range of motion at the 1st MPJ, however, used a higher percentage of the passive range during gait compared to the other 2 groups. Patients with ulceration under the 1st metatarsal head also had the least amount of passive range of motion at the AJC in the frontal plane, however, more movement was apparent in this group during barefoot walking compared to patients with lateral forefoot ulcers and to patients with ulcers under the hallux during shod gait.

4.2.2.2 Kinematics at the AJC during barefoot and shod walking

4.2.2.2.1 Stance phase duration

Although walking speed is known to influence joint movement and plantar pressures attempts were not made to standardise the walking speeds between the groups. Rather, the stance phase duration time was to serve as a proxy measure of walking speed to see if differences were apparent between the groups. If the stance phase duration times are increased this would represent a decrease in walking speed. The stance phase duration times recorded during the 3 barefoot walking trials were taken and a mean stance phase duration time calculated for each subject. The group mean stance phase durations are presented in Table 4-7. There was a general trend for an increase in stance phase duration in the neuropathic and ulcerated groups compared to the control and non diabetic reference groups. A statistically significant difference was found between the stance phase duration in the neuropathic group and the non-diabetic reference and diabetic control groups.

Table 4-7: Mean (SD) stance phase duration (msec) taken from barefoot walking trials.

Group	Non diabetic reference		Control		Neuropathic		Ulcerated	
	L	R	L	R	L	R	L	R
Mean stance phase duration (msec)	866	867	856	842	1120	1045	947	912
Standard deviation	157	185	234	159	335	237	188	165

4.2.2.2.2 Range of motion at the AJC during stance phase of gait

The greatest range of motion was found in the sagittal plane and the lowest in the transverse plane (Table 4-8). The range of sagittal plane motion during barefoot walking was comparable across the groups with a general trend for reduced motion in the neuropathic and ulcerated groups. When sagittal plane motion in the control, neuropathic and ulcerated groups is expressed as a percentage of the motion in the non diabetic reference group the percentages are 95.4%, 90.5% and 87.7% respectively averaged for the right and left limbs. Frontal plane motion in all groups was similar with no consistent pattern of increased or decreased motion in the groups. The range of motion in the transverse plane was the lowest range for all the groups, with a trend for reduced motion in the neuropathic and ulcerated groups in comparison to the control and reference groups.

The overall trend during shod gait was an increase in the range of motion in all planes of motion in all groups. In the sagittal plane motion increased by 31.5%, 26.8%, 38% and 32% in the non-diabetic reference, control, neuropathic and ulcerated groups respectively (averaged for left and right limbs). The increased range of motion in the frontal plane was more notable in the control group (30.6%) in comparison to the neuropathic (17.6%) and ulcerated groups (15.6%). The range of motion in the transverse plane was greatly increased in the neuropathic group (72.3%) and in the other groups the magnitude of increase was similar to that seen in the sagittal plane.

The pattern of motion at the AJC in all three planes during barefoot and shod walking is shown in all four groups (Figure 4-5 to 4-8). The angular positional data and timings expressed as percentage of stance phase duration are summarized in Appendix 6. All 4 study groups demonstrated 3 distinct phases of sagittal plane movement. In all groups the heel strike angle was plantarflexed relative to the bore-sight position, however the magnitude of plantarflexion increased in all groups during shod gait. In the frontal plane there was an initial phase of eversion followed by an inversion phase. The point of maximum eversion was reached slightly earlier in the non diabetic reference group compared to the diabetic groups. In all cases the angular position at heel strike was less everted and the maximum eversion position was reached earlier in the stance phase during shod gait. In the transverse plane the position of the AJC remained internally rotated

relative to the bore-sight position. There were 2 distinct phases of motion, internal rotation reaching a maximum at approximately 25-30% of the stance phase in all groups, then gradual eversion for the remainder of stance phase. A table of angular positional data and timings of the ankle joint complex during the stance phase of barefoot and shod gait can be found in Appendix 8.

Table 4-8: Mean range of motion at the ankle joint complex (in degrees) during barefoot and shod gait

Condition	Plane	Limb	Non diabetic reference	Control	Neuropathic	Ulcerated
Barefoot	Sagittal	Right	14.6 (4.3)	13.8 (4.7)	13.5 (5)	12.6 (4.2)
		Left	13.9 (3.6)	13.3 (3.2)	12.2 (4.4)	12.3 (3.3)
	Frontal	Right	7.5 (2.5)	5.6 (3.7)	6.1 (2.7)	6.2(2)
		Left	6.9 (2.3)	6.4 (2.2)	7.5 (4.8)	6.5 (2.6)
	Transverse	Right	5.3 (1.9)	5.2 (2.2)	4.6 (2)	4.4 (1.5)
		Left	5.1 (1.7)	5.2 (1.9)	4.7 (1.7)	4.9 (2.8)
Shod	Sagittal	Right	19.4 (4.4)	16.5 (6.6)	17.8 (6.2)	16.4 (4.1)
		Left	18.2 (3.5)	18 (4.2)	17.8 (5.3)	16.1 (5.4)
	Frontal	Right	9.7 (3)	7.4 (2.7)	8 (3.4)	7.2 (2.3)
		Left	9 (3)	8 (3.2)	7.9 (3)	7.6 (2.7)
	Transverse	Right	7.6 (3.3)	6.8 (2.6)	7.5 (3)	4.7 (5.5)
		Left	7.7 (3.1)	6.7 (2.8)	8.6 (3.2)	7.1 (3.3)

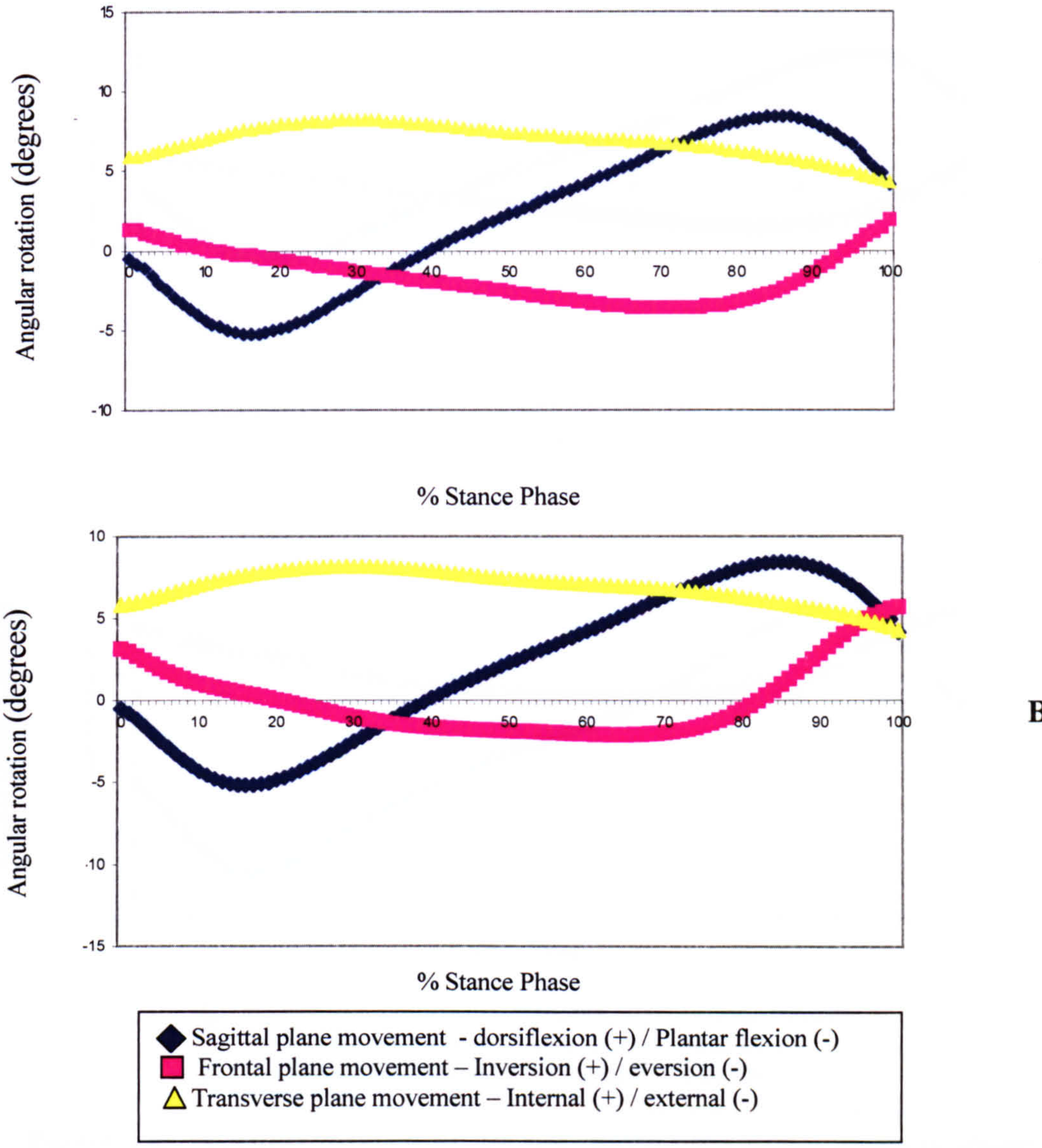


Figure 4-5: Angular rotation at the AJC during the stance phase in non-diabetic reference group during A Barefoot and B shod walking.

During barefoot walking the mean heel strike angle in the sagittal plane was –0.5° (SD4.2) dorsiflexed and the total range of movement was 14.3° (SD3.9). In the frontal plane the mean heel strike position was 1.3° inverted, reaching a maximum mean everted position of 3.6° at 71% of the stance phase. The pattern and mean angular positional data in the sagittal plane were similar between barefoot and shod walking. The mean angular position in the frontal plane during shod walking was more inverted at both heel strike and toe off and the range of motion was slightly increased when compared to barefoot gait (9.4° (SD2.9) vs 7.2° (SD2.4). The pattern and positional data of the AJC in the transverse plane was similar in both the barefoot and shod conditions, remaining in an internally rotated position relative to the bore-sight position throughout the stance phase.

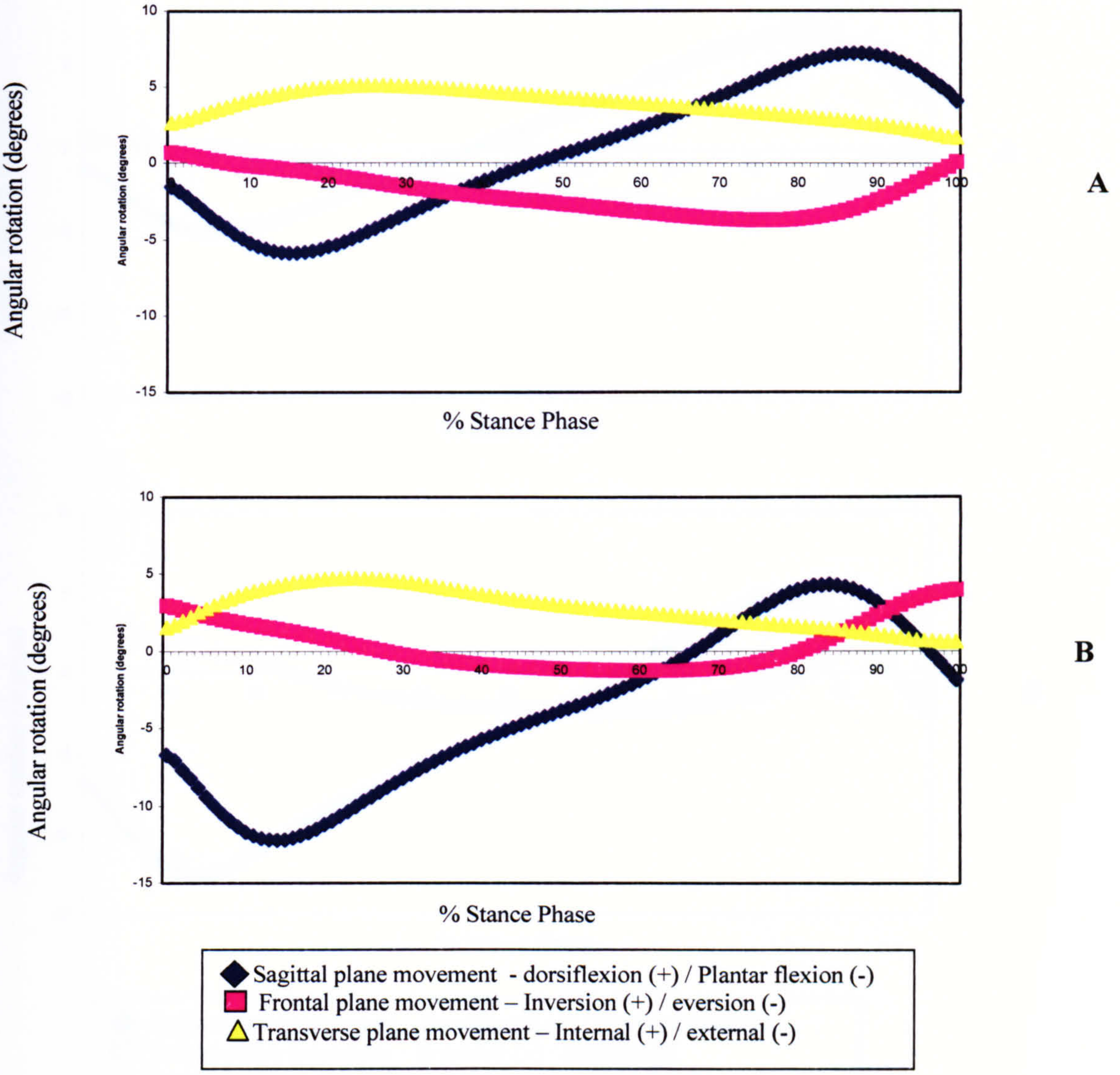


Figure 4-6: Angular rotation at the AJC during the stance phase in diabetic control group during A Barefoot and B shod walking.

The mean heel strike angle in the sagittal plane during barefoot walking was 1.5° (SD3.3) plantarflexed, the angle of plantarflexion at heel strike increasing to 6.7° during shod gait. The mean increase in total range of sagittal plane movement during shod gait was 3.3°. In the frontal plane the mean heel strike position was slightly inverted (0.7°) during barefoot stance and the position more inverted (2.9°) during shod gait. Maximum eversion was reached at 76% and 60% of the stance phase during barefoot and shod gait respectively. At heel strike the angular positional data in the transverse plane was 2.7°and 1.6° internally rotated in the barefoot and shod conditions respectively, the position of the AJC remained in an internally rotated position throughout the stance phase in both conditions.

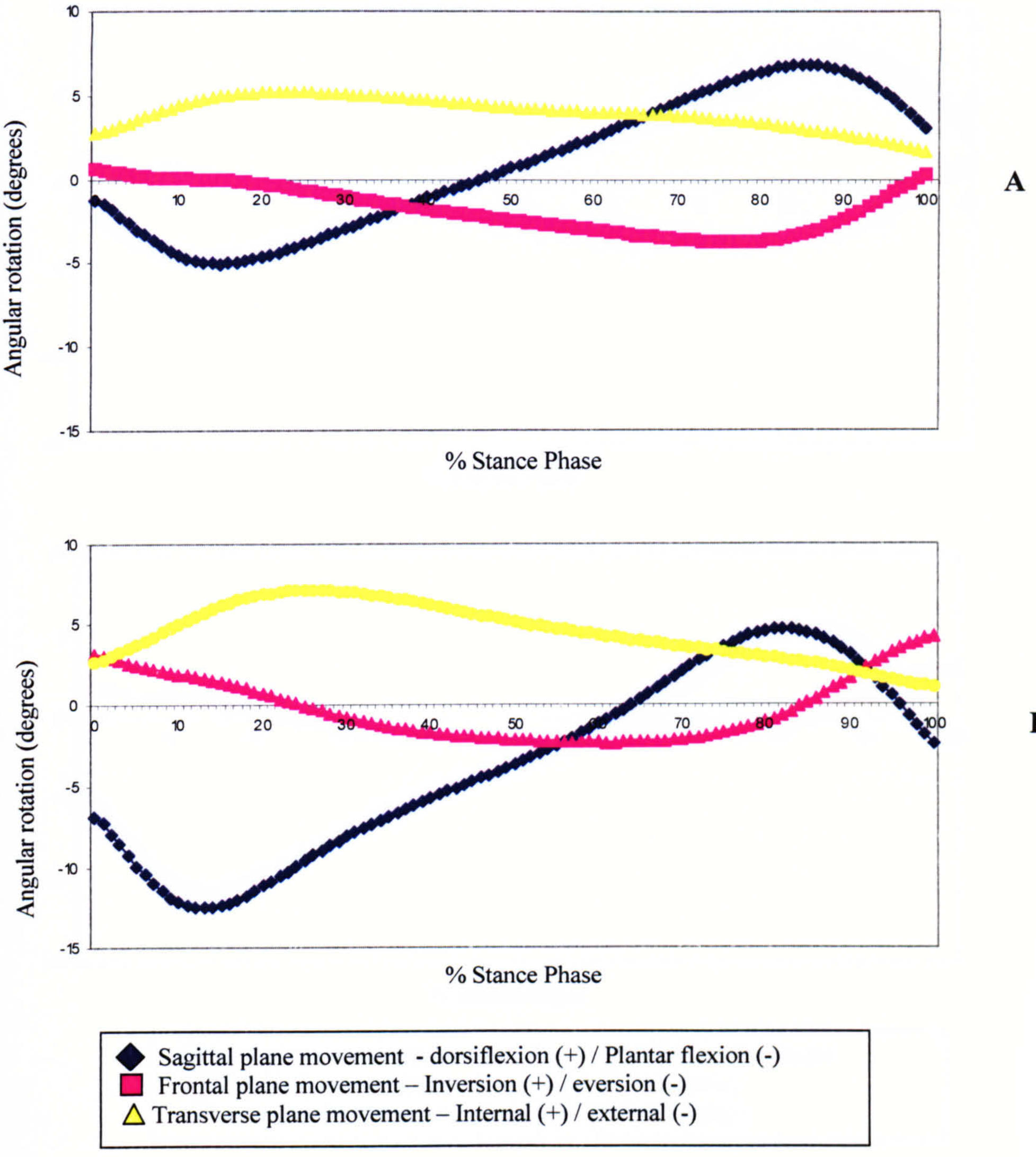


Figure 4-7: Angular rotation at the AJC during the stance phase in diabetic neuropathic group during A Barefoot and B shod walking.

In both barefoot and shod gait the heel strike position was plantarflexed and the mean range of motion was 11.9° and 16.5° during barefoot and shod gait respectively. During barefoot gait the heel strike position was slightly inverted, reaching a maximum eversion of 3.7° at 77% of the stance phase. During shod gait the heel strike position was more inverted than the barefoot position and the maximum eversion position reached earlier in the stance phase. In the transverse plane the angular positional data remained in an internally rotated in both barefoot and shod gait. The maximum internally rotated position was reached at 22% and 26% of the stance phase in the barefoot and shod condition respectively.

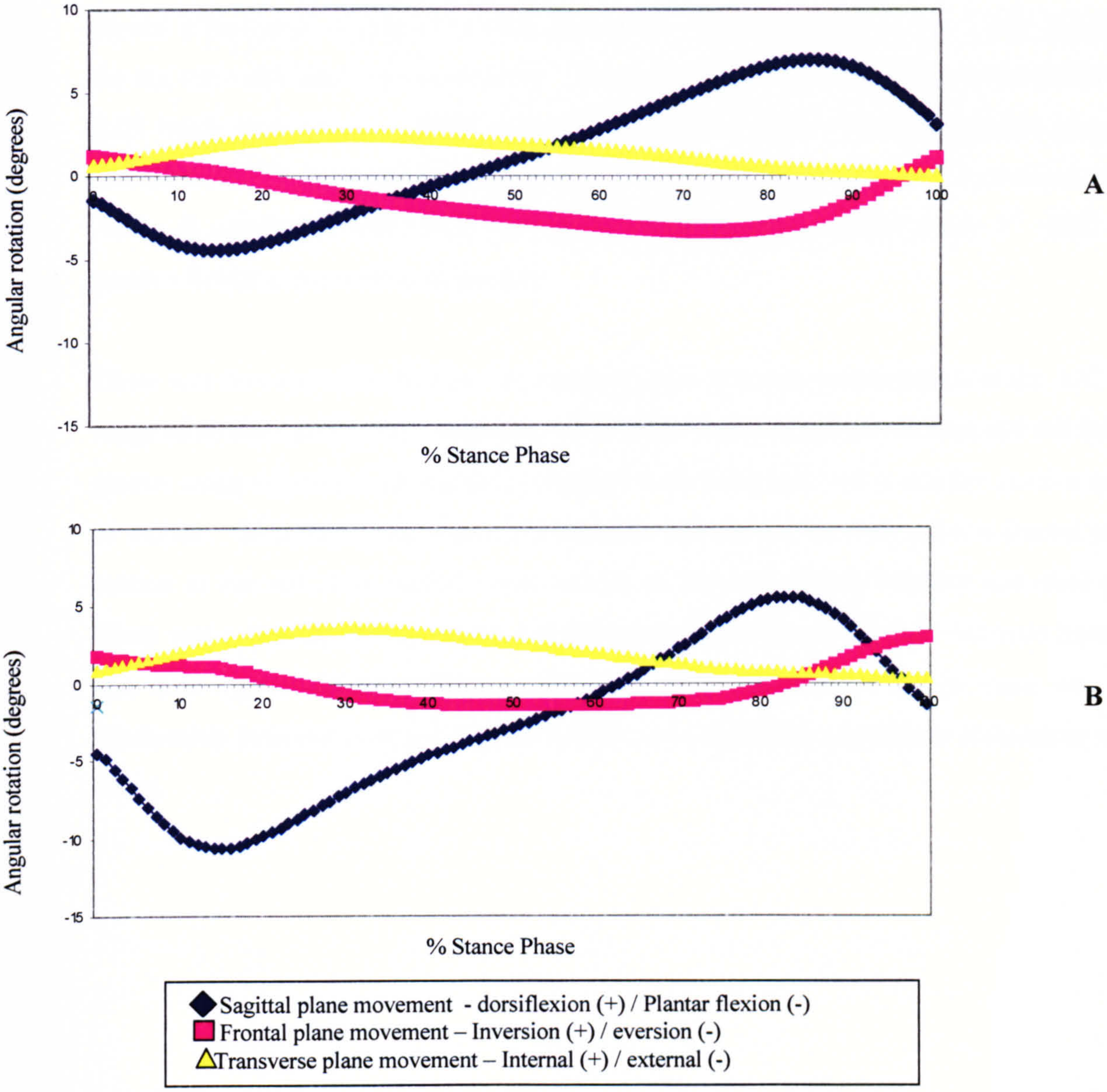


Figure 4-8: Angular rotation at the AJC during the stance phase in diabetic ulcer group during A Barefoot and B shod walking.

In the ulcerated group, the heel strike position was plantarflexed during both barefoot and shod gait. The maximum plantarflexion angle reached at 15% and 13% of the stance phase during barefoot and shod gait respectively. In the frontal plane the heel strike angle was both inverted and internally rotated. Maximum eversion was reached at 74% of the stance phase during barefoot gait and noticeably earlier (52%) during shod gait. Transverse plane motion was characterised by an initial phase of internal rotation reaching a maximum at 30% of the stance phase then a continual gradual external rotation, however, the ankle joint complex remained in an internally rotated position throughout the stance phase in both barefoot and shod gait.

4.2.2.2.3 Relationship of motion at the AJC and 1st MPJ.

There was a positive correlation between passive range of motion at the 1st MPJ and frontal plane range of passive motion at the AJC. The correlations were 0.495 and 0.283 for the right and left limb respectively. The correlations were significant at the 0.001 and 0.05 levels respectively. There were no correlations found between dynamic range of motion at the 1st MPJ and dynamic motion in any of the three planes of movement at the AJC. A correlation was found between dynamic range of motion at the 1st MPJ and passive frontal plane motion at the AJC.

There was no correlation between the range of passive frontal plane motion at the AJC and range of motion during gait. Subjects with a larger range of passive motion did not have a greater range of motion during gait compared with those who had a smaller passive range of motion. Statistically significant correlations were found between passive frontal plane motion at the AJC and sagittal plane motion at the AJC during barefoot and shod gait. There was a statistically significant correlation in the motion at the AJC between barefoot and shod gait in all three planes. The data were examined to see if there was any relationship between joint motion and gender, no statistically significant differences were found.

4.3 Plantar Pressure data

The normative database of pressure data (n=100) generated as part of the development of methods section are presented in Appendix 7 for reference. A summary of the key pressure variables in all 4 study groups can also be found in the Appendix 9. Although specific hypotheses had not been generated to compare the pressure variables between the groups, comparisons were made between the groups to allow comparison with other workers. For the purposes of highlighting the key pressure parameters in the diabetic groups clearly, data from the non-diabetic reference group has been omitted from Figures 4-9 to 4-11.

The magnitudes of peak pressures were similar across groups in most of the mask areas. Under the heel the peak pressure in the neuropathic and ulcerated group were lower than the control group. The peak pressure under the 1st metatarsal head was elevated (296kPa) in the ulcerated group in comparison with the neuropathic (254kPa) and control (253kPa) groups. The ulcerated group had slightly higher midfoot and forefoot pressures and slightly lower heel and toe pressures in comparison to the diabetic control group (Figure 4-9). The only statistically significant difference in peak pressure between the diabetic groups was found between the ulcerated and neuropathic groups in the medial heel and medial midfoot mask areas. Statistically significant differences were found between the peak pressure in the non diabetic reference group and the ulcerated group in 7 of the mask areas.

Generally the pressure: time integrals were increased in the neuropathic and ulcerated groups compared to the control group, as shown in Figure 4.9. The pressure: time integrals were statistically significant different between the non diabetic reference group and all the diabetic groups in the heel, lateral midfoot and hallux mask regions. A statistically significant difference in the pressure: time integral was found between the non diabetic reference group and the neuropathic and ulcerated groups in the forefoot region. A statistical significant difference in the pressure time integral was found between the control group and the ulcerated and neuropathic groups under the 1st metatarsal head mask region. The ulcerated group had increased integrals in all mask areas compared to the diabetic control group. The highest pressure: time integral was located under the 1st metatarsal head in the ulcerated and neuropathic group whereas the highest integral in the diabetic control group was under the heel.

There were no notable differences in the contact area in the mask areas between the diabetic groups, however, contact time within every mask area was increased in the neuropathic and ulcerated groups compared to the diabetic control group (Figure 4-10). Maximum force in the mask areas were analogous in each group, with a tendency for increased force under the heel and the 1st metatarsal head in the ulcerated group compared to the other two groups (Figure 4-11).

4.3.1 Location of peak pressure

The location of the peak pressure was recorded under both feet for each patient within the diabetic control, neuropathic and ulcerated groups (Figure 4-12). In the diabetic control group 3 areas predominated; 30% of patients in this group had their highest pressure under the heel, 30% of patients had their peak pressure under the 1st metatarsal head and 35% of patients had the peak pressure under the 2nd metatarsal head. In the neuropathic group the majority of patients had the peak pressure under their 1st or 2nd metatarsal head (37.5% of patients having the peak pressure located in each area). The most common site for peak pressure in the ulcerated group was under the 2nd metatarsal head; 44% of patients in this group had the peak pressure located at this site, with 22% of patients having peak pressure located under the 1st metatarsal head.

4.3.2 Location on maximum pressure time integral

The majority of patients in the control and neuropathic groups had the highest pressure time integral located under the heel (Figure 4-12). In the ulcerated group there were almost equal number of patients with maximum pressure time integral under the heel and under the 1st metatarsal head.

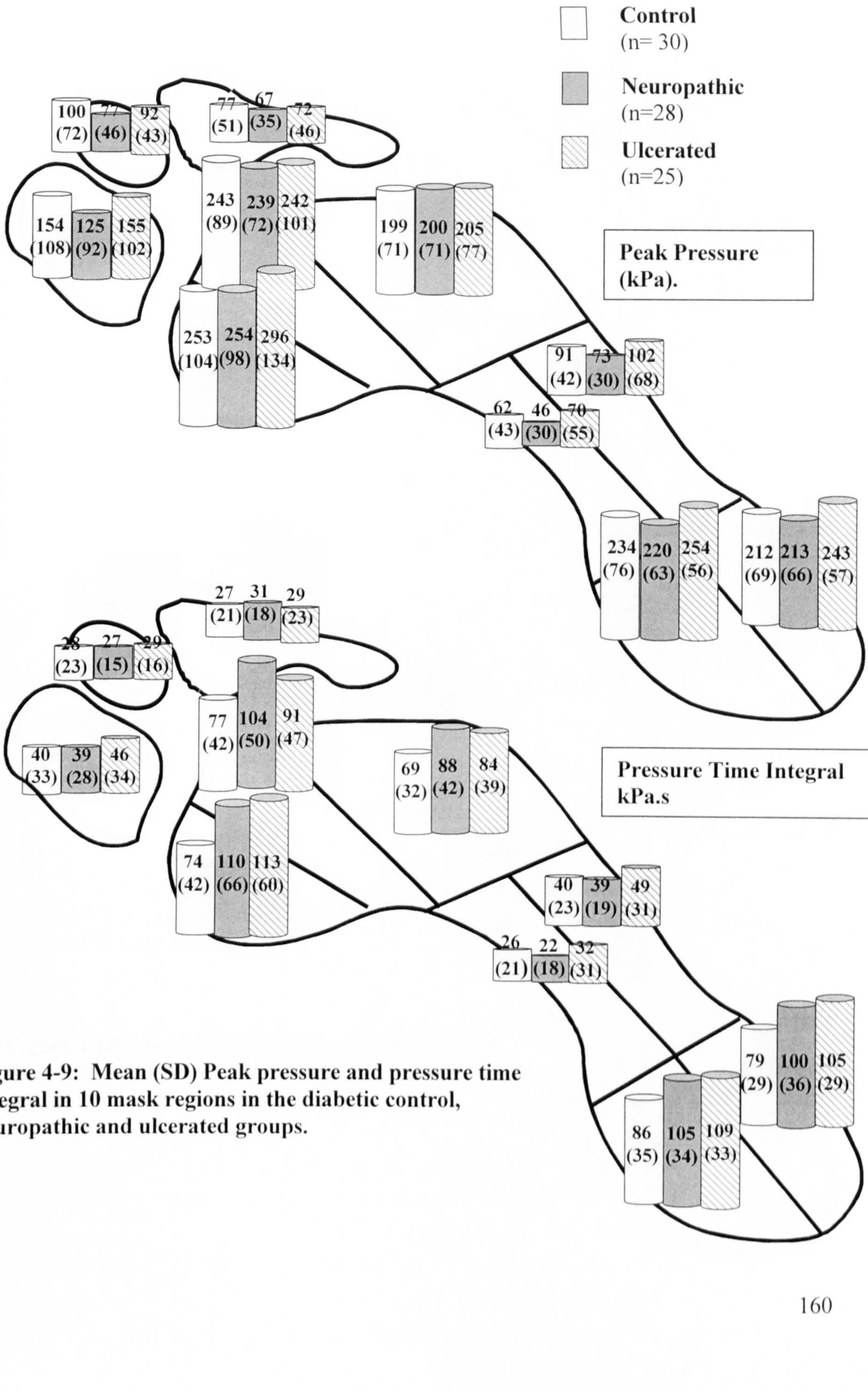


Figure 4-9: Mean (SD) Peak pressure and pressure time integral in 10 mask regions in the diabetic control, neuropathic and ulcerated groups.

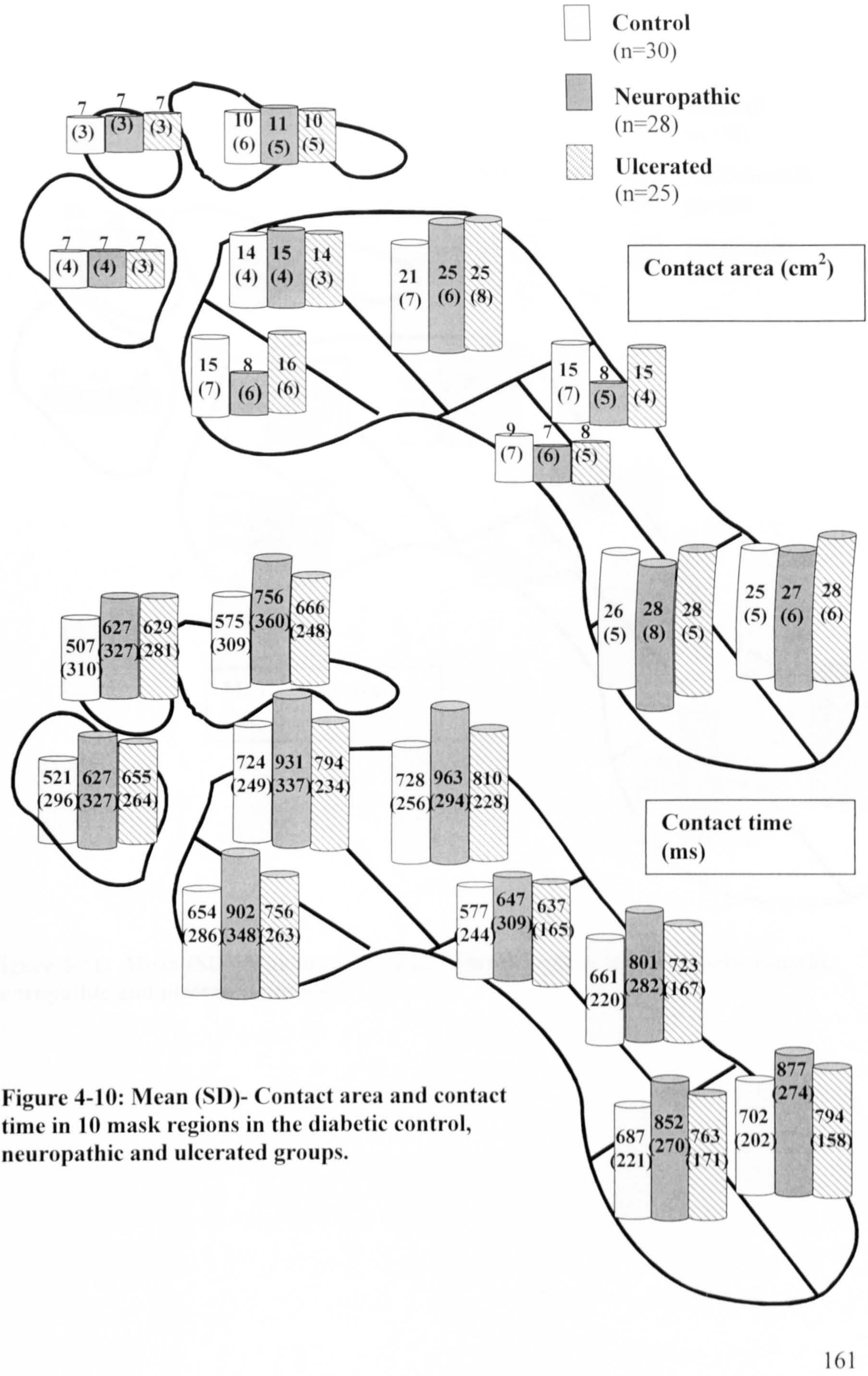


Figure 4-10: Mean (SD)- Contact area and contact time in 10 mask regions in the diabetic control, neuropathic and ulcerated groups.

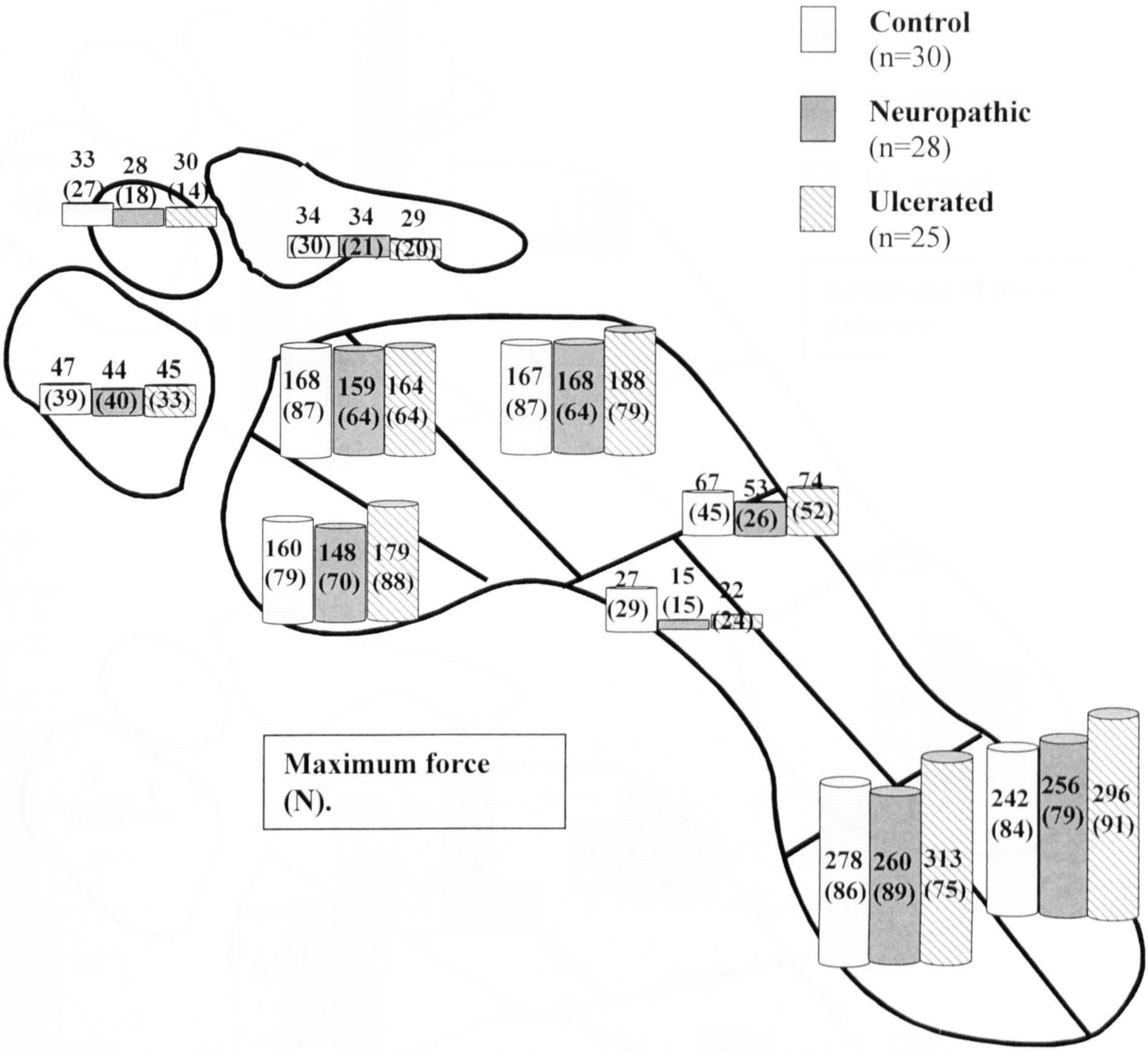


Figure 4-11: Mean (SD) Maximum force in 10 mask regions in the diabetic control, neuropathic and ulcerated groups.

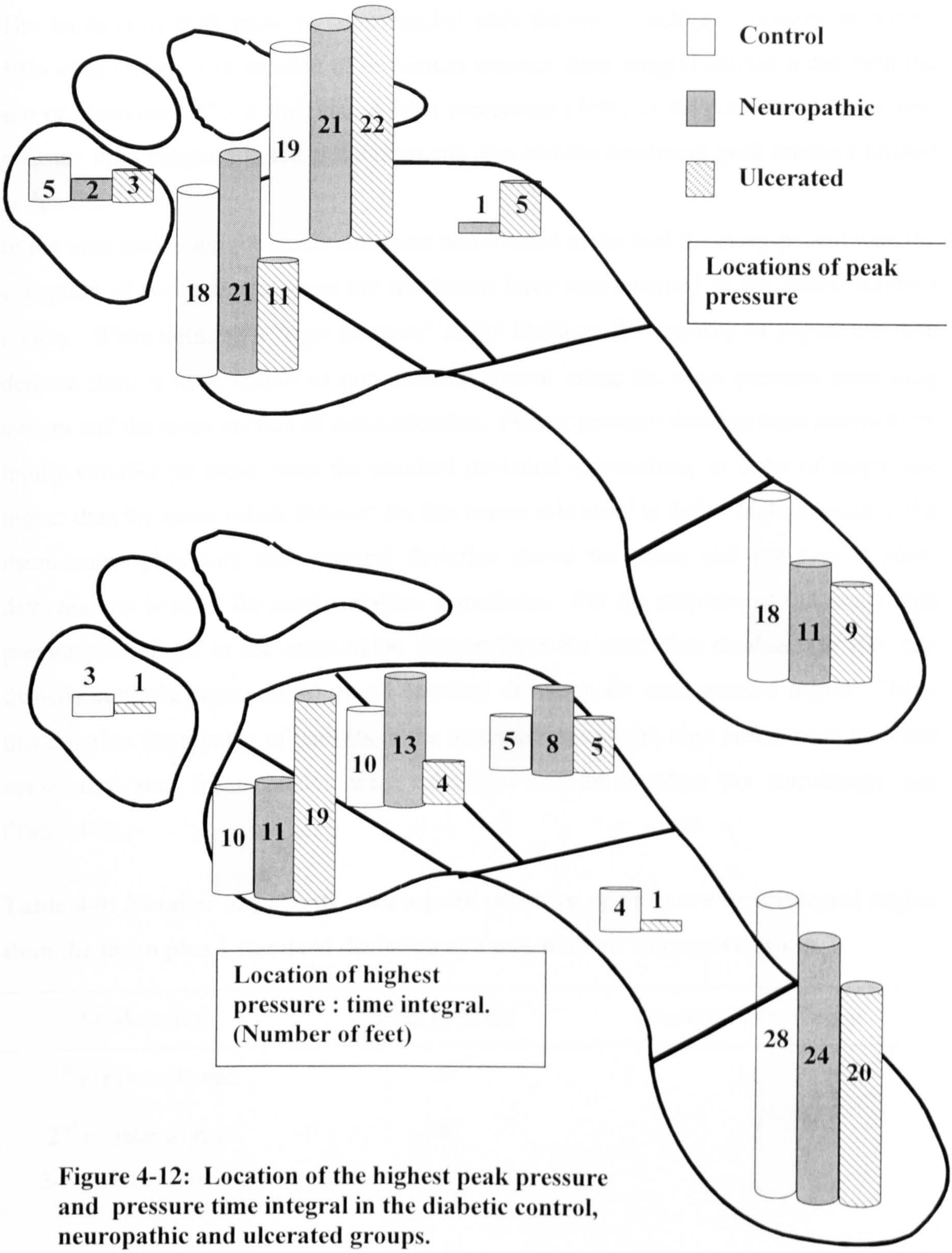


Figure 4-12: Location of the highest peak pressure and pressure time integral in the diabetic control, neuropathic and ulcerated groups.

4.3.3 Pressure and location of ulceration

The location of peak pressure corresponded with the site of active / previous ulceration 30% of the time. The location of maximum pressure time integral corresponded with the site of ulceration 21% of the time. A high percentage (71%) of the patients who had their pressure time integral located at the ulcer site also had the maximum peak pressure located at this site.

In the ulcerated group the maximum force was located in the heel for every patient with the exception of two patients where the maximum force was recorded in the lateral forefoot region. When defining a "high pressure" in the literature the majority of papers use data derived from a large cohort of non diabetic control using the same pressure measuring system and the same method of data collection. Plantar pressure data has been shown to be highly variable (in some cases the standard deviation approaching an order of magnitude higher than the mean values derived) for this reason it is usual to define high pressure as the mean value plus only one standard deviation above the mean and not two standard deviations as is usual for most variables in medicine. For the purposes of this study high pressure is defined as the mean value derived from the normative database (n=100, non diabetic controls, Appendix 6) plus 1 standard deviation for each masked region. Using this criterion the number of patients in the ulcerated group with high pressures at the three commonest sites for ulceration were calculated and the numbers are surprisingly low (Table 4-9).

Table 4-9: Number of patients with a peak pressure or pressure time integral higher than the mean plus 1 standard deviation of a non-diabetic normative group.

Mask region	Peak pressure	Pressure Time integral
1st metatarsal head	9	11
2nd metatarsal head	8	12
3-5 metatarsal heads	7	12
Hallux	1	3

In the ulcerated group patients were studied who had either an active or previous history of ulceration at the 1st metatarsal head, lateral forefoot area (metatarsal heads 2-5) or under the hallux (Table 4-9). Only data from the ulcerated limb was used for analysis. Patients with ulceration under the 1st metatarsal head had the highest maximum force at this site compared to the other 2 ulceration sites. The force under the 1st metatarsal head was approximately 60% greater when the patient has an ulcer at the site compared to an ulcer under the lateral forefoot area. The contact area under the 1st metatarsal head was increased when a patient had an ulcer at the site compared to ulceration under the lateral metatarsal heads or hallux. When the ulcer location category of the 1st metatarsal head was compared to the ulcer location category of the lateral metatarsal heads, the peak pressure under the 1st metatarsal head in the lateral forefoot ulcer group was approximately two-thirds of the magnitude of the pressure found in the 1st metatarsal head group. When a patient had an ulcer in the lateral metatarsal head region, the peak pressure at this site was higher than the peak pressures found in patients with ulceration at the other locations. In each group when a patient had an ulcer in a specific region, the pressure time integral was increased in this region compared to the other two sites. The highest contact time under the hallux, 1st metatarsal head and lateral metatarsals were found in the group which had ulcer located under the lateral metatarsal heads.

In all mask regions the peak pressure was higher in the ulcerated limb compared to the non-ulcerated limb, with the exception of the pressure under the 1st metatarsal head, hallux and lesser toes. No statistically significant differences were noted between the ulcerated and contra-lateral limb for peak pressure. In all mask regions, with the exception of the 1st metatarsal head and the hallux, the pressure time integral was increased under the ulcerated limb compared to the non-ulcerated contra-lateral limb.

Table 4-10: Mean pressure variables for patients with ulceration at the 1st metatarsal head, lateral forefoot area and hallux.

Ulcerated region	Pressure variables														
	Maximum force (N)			Contact area (Cm^{-2})			Peak pressure (Kpa)			Pressure time integral (Kpa/sec)			Contact time (ms)		
	M1	M3-5	Hallux	M1	M3-5	Hallux	M1	M3-5	Hallux	M1	M3-5	Hallux	M1	M3-5	Hallux
Metatarsal head 1	206.9	154.6	44.4	15.3	22.3	5.5	373.3	242.5	139.6	139.4	96.7	32.8	684.2	763.3	630.8
Lateral metatarsal heads	129	193.9	35.4	11.2	24.1	4.9	250	276	136	89.4	138.8	43.8	852	892	800
Hallux	99.2	205.8	31.8	11.9	27.2	5.6	239	229	191.7	70.5	60.4	43.9	685	433	568.3

M1- metatarsal head 1, M3-5- metatarsal heads 3-5

Table 4-11: Mean (SD) Peak pressure and pressure time integrals in the ulcerated group. Mean values are displayed for both limbs, the ulcerated limb only and non-ulcerated contra-lateral limb.

Mask area	Peak Pressure (kPa)			Pressure time integral (kPa.s)		
	Both limbs	Ulcerated limb	Non ulcerated	Both limbs	Ulcerated limb	Non ulcerated
Med heel	188 (79)	253 (60.9)	250 (49.2)	109 (33)	109 (35.7)	109 (34.9)
Lat heel	243 (57)	238 (62.8)	236 (41.3)	105 (29)	106 (30.4)	104 (30.6)
Med midfoot	70 (55)	76 (61.5)	67 (64.4)	32 (31)	34 (34.5)	30 (36.4)
Lat midfoot	102 (68)	123 (82.3)	90 (58.6)	49 (31)	55 (29.7)	44 (33.2)
1st MPJ	296 (134)	264 (137.6)	337 (138)	113 (60)	97 (59.9)	123 (56.3)
2nd MPJ	242 (101)	258 (105.7)	256 (115.4)	91 (47)	97 (51.3)	92 (46.6)
3-5 MPJ	205 (77)	228 (72.7)	198 (75.6)	84 (39)	91 (40)	81 (42.7)
Hallux	155 (102)	144 (101.5)	148 (91.5)	46 (34)	39 (32)	43 (34.1)
2nd toe	92 (43)	93 (49.9)	88 (40)	29 (16)	28 (16.9)	27 (15.3)
3-5 toes	72 (46)	67 (49.5)	71 (40.3)	29 (23)	28 (24.9)	27 (17.7)

Med-medial, Lat- lateral, MPJ- metatarsophalangeal joint

4.4 Relationship between motion and pressure

4.4.1 Motion at the ankle joint complex

No correlation was found between passive range of motion at the 1st MPJ, frontal plane passive range of motion at the AJC and peak plantar pressures in the mask areas. A correlation was found between passive frontal plane motion at the AJC and the pressure time integrals in the heel, midfoot and forefoot areas. No correlation was found between any of the ranges of motion at the AJC or the 1st MPJ during gait and peak pressures or pressure time integrals under the foot. Because Mueller et al (1990), indicated that patients with less than 30° frontal plane motion at the ankle joint complex have higher plantar pressures and that a range of motion less than 30° should be considered a risk factor for ulceration, passive frontal plane range of motion data for the ankle joint data were classified into limited or normal based on whether the value was less than or greater than 30°. Peak pressure and pressure time integral were calculated for the total foot and peak pressures were calculated for the forefoot area for each group. The number of feet with range of motion greater than 30° was only 20, compared to 196 with range of motion less than 30°. The pressure data for each group is presented in Table 4-12.

Table 4-12: Mean (SD) Peak pressure and pressure time integral in patients with limited or normal frontal plane motion at the AJC.

	PP Tot	PP MH1	PP MH2	PP MH3-5	Pti Tot	Pti MH1	Pti MH2	Pti MH3-5
AJC<30°	316 (91)	260 (110)	236 (86)	197 (71)	166 (57)	91 (55)	84 (43)	75 (36)
AJC>30°	282 (97)	208 (116)	218 (108)	196 (84)	146 (57)	71 (51)	79 (56)	76 (52)

PPTot- Peak pressure in total foot, PPMH1-Peak pressure in metatarsal head 1 mask, PPMH2- Peak pressure in metatarsal head 2 mask, PPMH3-5- Peak pressure in metatarsal heads 3-5 mask, PtiTotal- Peak pressure in total foot, PtiMH1- Pressure time integral in metatarsal head 1 mask, PtiMH2- Pressure time integral in metatarsal head 2 mask, PtiMH3-5- Peak pressure in metatarsal heads 3-5 mask.

4.4.2. Motion at the 1st MPJ

Because it has been stated in the literature that greater than 60° passive range of motion of dorsiflexion is needed for efficient gait and that the approximate range of dorsiflexion during gait is approximately 50°, these values were chosen to classify patients into limited or normal movement categories (Root et al 1977, Buell et al 1999). The number of patients in this study with a passive range of dorsiflexion greater than 60° was six.

If a patient had less than 50° dorsiflexion during gait one would expect higher plantar pressures in certain mask areas dependent on how the patient compensates for the limited movement at the 1st MPJ. Four loading patterns have been suggested to occur more frequently when compensating for limited movement at the 1st MPJ. The loading patterns are increased pressure under the 1st metatarsal head compared to other forefoot regions, increased pressure distally compared to other forefoot regions (under the hallux), increased pressure laterally compared to other forefoot regions (over metatarsal heads 2-5), or a combination of increased load laterally and distally. The data were categorised into those with limited or normal movement at the 1st MPJ based on 50° dorsiflexion during gait. A total of 43 feet were identified as having equal to or greater than 50° dorsiflexion and 173 feet had less than 50° dorsiflexion. Table 4-13 summarises the percentage from each group with the different patterns of loading. When expressed as a percentage of the total group the values for each loading pattern are similar in both groups, suggesting specific loading patterns are not associated with range of dorsiflexion at the 1st MPJ during gait.

Table 4-13: Strategy used for compensating for limited dorsiflexion at the 1st MPJ during gait.

Loading Pattern	Limited MPJ dorsiflexion Number of feet using strategy as percentage of limited group	Normal MPJ dorsiflexion Number of feet using strategy as percentage of normal group
MH1	54 (31%)	14 (33%)
Hallux	27 (16%)	4 (9%)
Lateral Mets	43 (25%)	15 (35%)
Combination	70 (40%)	19 (44%)

4.5 Follow up data

No patients in the diabetic control group ulcerated, one patient had an in-growing toenail that was surgically removed and total healing occurred within 6 weeks. Two patients from the neuropathic group developed an ulcer during the 12 month follow up period.

In the ulcerated group one patient was lost to follow up, one patient died of complications not related to their foot problems. There were three amputations over the 12 month follow up period, two below knee and one hallux amputation. 12 months after the measurements were taken six of the patients in the ulcer group had active ulceration.

4.6 General summary of results with reference to the hypotheses

One of the hypotheses proposed to be tested during the course of this study was that there would be a statistically significant difference between the motion time curves of diabetic groups. The overall results of this cross-sectional study indicate that there were no statistically significant differences between the mean motion time curves in the diabetic and non diabetic groups in this study. The hypothesis that there would be a difference between the motion time curves of diabetic groups was not supported.

There was a statistically significant difference between the mean passive amount of dorsiflexion at the 1st MPJ in the non diabetic control group compared to each of the three diabetic groups, but the difference between the diabetic groups did not reach a level of statistical significance. The mean range of dorsiflexion at the 1st MPJ during gait were comparable across the diabetic groups, but they were all significantly lower than the non diabetic reference group. When the ulcerated group was studied in more detail a statistically significant difference was found in the amount of the passive range at the 1st MPJ used during the stance phase between the ulcerated limb and the contra-lateral non ulcerated limb. The passive range of movement at the AJC and the range of movement during the stance phase were statistically significantly different between the non-diabetic control group and all three of the diabetic groups. When the range of motion at the AJC during gait was expressed as a percentage of passive range of motion, there was a statistical significant difference between the non-diabetic reference group and the ulcerated and neuropathic groups.

No statistically significant differences in the mean range of motion at the AJC or 1st MPJ during gait were found between the diabetic groups. The hypothesis that patients with an active or previous history of ulceration would have a lower range of motion at these joints during gait was not supported.

When the relationship between joint motion during the stance phase of gait and plantar pressure variables were examined no statistically significant correlations were found in any of the study groups. A statistically significant correlation was found between frontal passive range of motion and pressure time integrals in the forefoot area. A relationship

between joint motion at the AJC or the 1st MPJ and the location or magnitude peak pressures in the forefoot was not established.

No difference in the mean range of motion during the stance phase of gait was found in the diabetic groups; those patients with an active or previous history of ulceration did not have a significant reduction in the mean motion at the AJC or 1st MPJ. The hypothesis that patients with ulceration would have lower range of motion during gait and that this would be associated with ulceration was not supported.

CHAPTER 5

DISCUSSION

This chapter discusses the key findings of the study with reference made to related published literature. The implications of the findings are considered and placed within the context of clinical practice. The limitations of the study are considered along with discussion as to how they may impact on the results of the study. Finally suggestions for future research are made.

The pathogenesis of ulceration has been extensively studied with particular attention directed to the detection of factors that make a patient "at risk" of developing an ulcer. The causes of plantar ulceration are clearly multi-factorial but the primary aetiology is believed to be excessive repeated pressure on the insensitive foot (Mueller et al. 1994b). When neuropathy is present, more prolonged pressure needs to be exerted on the foot before the protective warning (pain) is perceived (Katoulis et al. 1996), thus making the tissue more liable to damage from otherwise normal stresses under the foot. Repeated loading of tissue (as would occur during gait) results in reactive hyperaemia and stronger cohesion of the skin cells. This decreases the rate of desquamation and subsequent callus formation occurs (Adams et al. 1989). The presence of callus and high pressures have been shown to be associated with an increased risk of ulceration in a prospective study (Murray et al. 1996).

Limited joint mobility, as determined by limited passive range of motion at the AJC and 1st MPJ, has been substantially implicated in the pathogenesis of high plantar pressure and ulceration seen in diabetics. Although high plantar pressures are generated during walking and are therefore dependent on dynamic joint motion, passive ranges of joint motion are more convenient to measure in clinical practice. The mechanism by which limited joint mobility causes high pressure has not been substantiated. A reasonable assumption is that a limited passive range of motion results in a limited dynamic range of motion and, by using biomechanical theory, can explain the generation of higher plantar pressures. Although limited joint mobility and high plantar pressures have been shown to co-exist in the presence of neuropathy, a direct causal link is speculative.

This study aimed to investigate the association between joint mobility, high plantar pressure and foot ulceration in more detail. The novel aspect of the present study is that dynamic joint mobility has been determined using three-dimensional motion analysis. It was anticipated that patients with ulceration would have a lower dynamic range of motion at the ankle joint complex in all three planes during the stance phase of gait (Fernando et al. 1991; Mueller et al. 1989; Mueller et al. 1994a). Using biomechanical models it was expected that there would be a relationship between dynamic movement at the AJC and 1st MPJ and the magnitude and location of plantar pressures (Bevans et al. 1999; Root et al. 1977). Patients in the present study were followed-up for a 12-month period following gait assessment, so that ulceration status could be monitored. It was intended to predict those patients most likely to ulcerate by using objective assessment of gait.

The discussion will examine the key findings of the study, firstly describing the recruitment procedure and study group demographics. The key results of the study will be discussed in relation to how this may impact on clinical assessment of the diabetic foot.

5.1 Recruitment

Although a recruitment strategy through local GP practices or local Diabetes UK groups may have resulted in a more pragmatic study sample, in this case this method was not used. In the present study a sample of convenience was recruited from three clinical podiatry sites. Two recruitment sites were hospital clinics (one in Leeds and one in Huddersfield) and one recruitment site was a university podiatry department clinic in Huddersfield. Recruitment from podiatry services was considered to be the best method of recruitment to facilitate the prospective element of the study and allow the patients' ulceration status to be followed up over 12 months.

It could be argued that the fact that all patients were recruited through podiatry services may have resulted in a biased sample, with a tendency for all patients to have some type of foot problem. However, many patients attending the service were referred solely for

a screening assessment and appropriate foot health advice, and did not have a current specific foot problem.

5.2 Sample demographics

Attempts were made to try and balance the demographic and clinical characteristics of the three diabetic study groups. However, no stratification was used. The major difference between the three diabetic groups was the higher proportion of males in the ulcerated group. During data collection it became apparent that more males were entering the ulcerated group, so an attempt was made to try and recruit more female patients into the ulcerated group. However, during the recruitment period there were a lower number of females attending the foot ulcer clinics. Also, many females presented with dorsal toe ulceration and not plantar ulceration and therefore could not be included in the study. Previous studies have reported consistently higher ulceration rates in males than in females (Reiber et al. 1995) and a higher amputation rate (Most et al. 1983). In a 12 month prospective study of hospital admissions related to diabetic foot disease a higher proportion of males (62%) than females were admitted in a UK city hospital (Krentz et al. 1997). Thus in the light of previous findings it was not surprising that a higher proportion of males attended the foot clinic during data collection, and it is reasonable to assume the study sample in the present study simply reflects the situation throughout the UK.

The disease duration was greater in the neuropathic and ulcerated groups than in the diabetic control group. This result was expected because there is an increase in the number of diabetes related complications, for example neuropathy and ulceration with increased disease duration (Reiber 2001b). Body mass was statistically significantly different in the ulcerated group in comparison to all the other groups. This finding is consistent with the findings of other workers (Boyko et al. 1999; Ctercteko et al. 1981) and may be partly explained by the higher proportion of males in the ulcerated group.

There was no intention to exclude ethnic minorities from the study. The lack of ethnic minorities in the podiatry clinics throughout the data collection period (approximately 6 months) was unexpected. However, there is evidence to suggest a lower prevalence of ulceration and amputation in certain ethnic groups (Clarke et al. 1998; Gujral et al.

1993) and it has been suggested that many patients of certain ethnic origin may die earlier of other complications related to diabetes, for example coronary heart disease, before developing foot ulcers (Chaturvedi et al. 2002). It is also widely acknowledged that there may be some cultural differences in behaviour, and barriers to seeking medical care in ethnic groups. The low number of ethnic minority patients in the present study is not a consequence of the geographic area as both Leeds and Huddersfield have a higher proportion of ethnic population than the UK average. Leeds is the second largest health authority in England in which South Asians comprised 5% of the local population in the 1991 census (Feltbower et al. 2003). As a consequence of the low numbers of patients of ethnic origin in the present study the results may only be applicable to the North European population.

5.3 Clinical data

In the present study neurological screening procedures were used to classify the patients into the study groups. Although electrophysiological techniques are regarded to be the gold standard for neurological assessment, these methods are never used routinely for diabetic foot screening. The methods chosen were the modified Neuropathic Disability Score and the Neuropathic Symptom Score, as previously described by Young and associates (Young et al. 1993a). Although this scoring method has not been properly validated, the modified neuropathy disability score has been shown to be associated with foot ulceration (Kumar et al. 1994), and recent studies have stated that Neuropathic Disability Score was the best predictor of foot ulceration (Calle-Pascual et al. 2001; Meijer et al. 2000). In the present study monofilament testing and vibration perception were also recorded for each patient to allow comparison with other papers.

Patients in the ulcerated group had a higher Neuropathic Disability Score but not a higher Neuropathic Symptom Score when compared to the neuropathic group (Table 4-1). This finding is not surprising as prediction of polyneuropathy from neuropathic symptoms alone is not advocated and, it has previously been reported symptoms may not always indicate underlying neuropathy (Feldman et al. 1994). It is interesting to note that the neurological screening used in this study did not identify two patients in the ulcerated group. These two patients were also not identified by the vibration perception threshold or the monofilament testing procedures. In these two cases it is

likely that neuropathy was not a major factor in the aetiology of their ulceration and that altered foot function played a greater role.

As part of the diabetic screening procedure a full assessment of the foot was undertaken and presence and location of foot pathologies were recorded for each patient. In the present study a foot deformity score, as described by Murray, was recorded for each patient (Murray et al. 1996) and the mean for each of the three diabetic groups calculated. The mean foot deformity score for the control group was higher than the mean for the neuropathic group. This result was unexpected because the literature suggests neuropathy is associated with a higher prevalence of certain foot deformities, for example lesser toe retraction. However, it must be noted that the reported higher prevalence of lesser toe deformity in diabetics is based on anecdotal clinical evidence and has not been substantiated.

In the present study the mean foot deformity score (Table 4-1) was statistically significantly higher in the ulcerated group compared to the non-ulcerated group. This finding is consistent with the finding of Murray (1996), who reported a significant difference in the presence of foot deformity in an ulcerated group compared to a neuropathic patient group and found that 95% of the ulcerated group were found to have some degree of foot deformity (Murray et al. 1996). In the present study, 100% of the ulcerated group had some degree of foot deformity determined as a foot deformity score greater or equal to one. Several papers have shown an association between presence of certain foot deformities and ulceration. A significant relationship between the presence of hammer and claw toe deformities and foot ulceration has been reported (Holewski et al. 1989; Lavery et al. 1998). It is therefore not surprising that the prevalence of foot deformity in ulcerated group in the present study was so high.

In the present study the impact that specific foot deformities had on dynamic joint mobility and plantar pressure was not investigated. If gait analysis was carried out, subdividing patients on the basis of the presence or absence of lesser toe deformity and hallux valgus this may show that patients with certain deformities have similar compensation strategies and may enable different gait styles to be identified for each group.

It has also been reported that foot deformities are associated with formation of callus (Mueller et al. 1990). In the present study the ulcerated group who had a higher mean foot deformity score also had a higher prevalence of callus than the other two diabetic groups. Overall 39.8% of the diabetic group had callus with a higher proportion of patients in the ulcerated group (56%) presenting with plantar callus compared to the non-ulcerated groups (33%). This finding is in agreement with Murray who found the overall incidence of callus for the ulcerated and non ulcerated group increased approximately two fold (Murray et al. 1996). The higher prevalence of callus in the ulcerated group is not surprising because it has been shown previously in a prospective study that callus is associated with a higher risk of ulceration (Murray et al. 1996).

The most common sites for plantar ulceration identified in the present study are consistent with the findings of other workers (Stacpoole-Shea et al. 1999; Murray et al. 1996). In contrast to previous work, the present study did not find that the location of peak pressure, maximum vertical force or maximum pressure time integral corresponded well to the site of ulceration (Ctercteko et al. 1981). These pressure parameters do not fully represent the trauma that soft tissues in the foot experience. The findings from the present study show that high peak pressure may not be the most important factor in the pathogenesis of ulceration. It would be logical to assume that other factors not investigated in the present study for example high shear forces and soft tissue function may be more important. Previous work has suggested that ulceration also occurs at sites of maximum shear (Cavanagh et al. 1991b), however, this has not been fully investigated due to technical limitations associated with measuring shear at discrete parts of the foot. Recent literature suggests that decreased thickness of soft tissue under the forefoot may be an important risk factor for ulceration (Morag et al. 1999).

Although the sample of patients in the present study was a sample of convenience, the general demographic trends in the present study sample, (higher proportion of males, increased disease duration and higher prevalence of foot deformity and callus in the ulcerated group) are consistent with the general trends highlighted in the literature.
It is therefore reasonable to assume that the recruitment procedure used in the present study resulted in a sample that reflects the diabetic population attending clinics at the three recruitment sites. The screening procedure used to classify the neurological status of patients in this study had a good level of agreement with both vibration

perception and monofilament testing, as demonstrated by similar levels of sensitivity and specificity at identifying the ulcerated group of patients. This provides confidence that the screening methods used were appropriate for the purposes of the study.

5.4 Joint mobility

A normal range of motion at the AJC and 1st MPJ is thought to be crucial to the normal biomechanics of the foot. However, most biomechanical theory is not based on research evidence. Work in the general field of biomechanics has shown that some fundamental principles of biomechanical foot function are not valid (Hamill et al. 1989; McPoil et al. 1994a; McPoil et al. 1996b). Greater mobility at the AJC has been considered to be beneficial as it enables better shock absorption at initial contact, thereby decreasing plantar pressures. It has been stated that approximately 65° dorsiflexion is needed at the 1st MPJ for efficient propulsion during gait (Root et al. 1977). The application of gait analysis to validate these inferences has not been undertaken.

In the present study all passive ranges of motion at the AJC and 1st MPJ were reduced in the diabetic groups in comparison to the non-diabetic reference group. The present study found a lower range of motion at both the ankle joint complex and 1st MPJ in all three diabetic groups when compared to the ranges reported by other workers (Delbridge et al. 1987; Fernando et al. 1991). In the present study the mean disease duration was higher in the ulcerated and neuropathic group compared to results of other workers. Because there is an increase in the prevalence of diabetic complications, such as limited joint mobility, with increased disease duration, this could account for some of the difference. A more likely explanation for the difference in passive range of motion in the present study is the difference in technique used to measure joint movement. In previous studies passive range of motion was measured with handheld goniometers, the reliability of which has recently been questioned (Menz 1995). When measuring passive range of motion at the AJC with hand held goniometers there are several areas where errors can occur. The technique is dependent on the drawing of a bisection line, which is not standardised and can be very subjective. Inaccuracy can then occur when the gonimeter is placed over the bisection line and a reading taken.

When taking passive joint measurements with the electromagnetic tracking system many of the problems associated with traditional goniometry are overcome. With this method there is no reliance on bisection lines and the data are directly transferred into computer software so there is no opportunity for subjective interpretation of the results. The person taking the results is effectively blinded to the results until after the measurements have been taken and processed so there is no opportunity to manipulate subsequent readings.

A review of the methods sections of previous papers that have used goniometry to measure passive range of motion showed that only two papers reported intraclass correlation coefficients for the examiners on the study sample being investigated (Mueller et al. 1989; Mueller et al. 1994a). Most papers outlined their methods and referred to the method originally described by Delbridge and associates (Fernando et al. 1991; Veves et al. 1995). In the paper by Delbridge, the coefficient of variation presented was based on data from a preliminary study with one examiner on a non-diabetic control patient in a single session (Delbridge et al. 1987). Although in most papers, only one examiner performed all the passive range of motion joint measurements, it cannot be assumed that different examiners would have the same level of reliability in taking these measurements. Literature elsewhere states goniometric reliability is very much dependent on the examiner's experience, and reports poor intertester reliability of passive range of motion for the ankle joint complex (Elveru et al. 1988a). Because the previous papers have not reported any reliability data for passive range of motion it is difficult to ascertain how reliable the measurements are.

In essence, based on the assumption that there is less opportunity for error to occur when using the electromagnetic tracking system this method should be considered to be superior to standard goniometry. However, the only way to substantiate this assumption would be to make direct comparison between the two methods. Due to the constraints incurred by using the electromagnetic tracking system, (sensors placed on the posterior calcanues, proximal phalanx of the hallux and 1st metatarsal) direct comparison of the passive range of motion measurements taken simultaneously using both methods could not be performed. Time constraints during the data collection period of the study (limited time access to equipment and rooming) meant that it was not possible to make passive range of motion measurements using both techniques to allow comparison. Further work exploring the relationship between measurements

taken with both techniques may explain the lower passive range of motion found in the present study.

In the present study the passive range of motion at the 1st MPJ was measured with the patient in a non-weight-bearing position. Other measurements can be taken for example measurement of weight-bearing range or the range during a heel-rise (Nawoczenski et al. 1999). Although the range of motion determined during a heel rise or weight-bearing has been shown to be more closely associated with the range of motion during gait (Nawoczenski et al. 1999), the non-weightbearing method was chosen for the present study. This was because most podiatrists will assess motion at the 1st MPJ with the patients in a non weight-bearing position and many podiatry textbooks describe the non weight-bearing method for assessment of the joint. The non-weightbearing method to assess motion at the 1st MPJ was chosen by the present study as it was thought to be more reflective of method used during routine podiatric screening.

In the present study the passive range of dorsiflexion at the 1st MPJ was found to be was reduced in the ulcerated group compared to all the other groups. The passive range of frontal plane motion at the AJC was also reduced in the ulcerated group in comparison to the other groups. These finding are consistent with previous findings (Delbridge et al. 1987). Delbridge and associates (1987) also found significant differences in the range of motion at the subtalar joint in the ulcerated limb compared to the non-ulcerated contra-lateral limb. This finding was not supported by the present study. Although no differences in the passive range of dorsiflexion were found in the ulcerated group, when comparing the ulcerated limb to the contra-lateral limb. However, when the dynamic range of dorsiflexion was expressed, as a percentage of the passive range, the mean percentage used in the ulcerated limb was significantly greater compared to the contra-lateral limb.

In some patients the dynamic range of motion at the 1st MPJ actually exceeded their passive range. This suggests the passive range of motion recorded in the present study did not represent their actual true end range of motion. It is possible that in the ulcerated group, where joint stiffness may be more prevalent, more torque would be needed to move the joint to its end of range. In future studies attempts should be made to standardise torque. A general trend in higher percentage of passive range been used

during gait occurred from the diabetic control group to the ulcerated group at both the AJC and the 1st MPJ. As the degree of neuropathy increases, it appears so does the percentage of passive range being used during gait. Measurements of proprioception were not taken in the present study. Simoneau and associates found that diabetic subjects with cutaneous sensory neuropathy demonstrated a significant loss of ankle joint movement perception (Simoneau et al. 1996). It can be speculated that patients with neuropathy may use a greater range of their passive range than those without neuropathy due to loss in ankle joint movement perception. However, this speculation would need to be validated.

A poor correlation between passive range of motion and range of motion during gait has been reported previously in patients with diabetes (Van Schie 2000) and in other clinical groups (Hamill et al. 1989). This finding was confirmed in the present study. In the literature the reported normal ranges of motion at the AJC and 1st MPJ vary considerably which hampers the clinical usefulness of this measure. It has been suggested that people with diabetes who have less than 30° frontal plane passive range of motion at the AJC are at risk of developing ulceration (Mueller et al. 1990). In the present study only a small number of patients had more than 30° and these patients did not have a higher range of motion during gait or lower plantar pressures when compared to those individuals with less than 30° motion. In the present study no relationship was found between the passive range of dorsiflexion at the 1st MPJ or frontal plane motion at the AJC and the dynamic range of motion. Patients with a larger passive range of motion will not necessarily exhibit a larger range of motion during gait. The poor relationship between passive and dynamic range of motion does question the usefulness of these measurement and the continuation of these measurements in clinical practice. It may be preferable to classify if patients have limited joint mobility in the lower limb based on their dynamic range of motion.

Both neuropathy and limited passive joint mobility have been shown to be associated with increased plantar pressures. It has been suggested that when protective sensation is intact, patients are able to compensate and that the plantar pressures may not necessarily be elevated in the presence of limited joint mobility at the AJC (Fernando et al. 1991). In this study both the neuropathic and ulcerated group had established neuropathy and used a higher percentage of their passive range of motion during gait in comparison to the non diabetic and diabetic control groups, yet this only reached statistical

significance between the ulcerated group compared to the non diabetic control group. Expressing the percentage of passive range of motion used during gait could be an alternative method to examine the impact that joint mobility has on overall foot function and aetiology of ulceration. If during gait some patients, due to neuropathy, are consistently approaching their end range of movement at the AJC and 1st MPJ, this may be detrimental and, it could be speculated that this may lead to joint damage and arthritic changes. However, there are no current epidemiological data to support this speculation.

This study has shown there is a poor relationship between the passive and dynamic range of motion at the AJC and 1st MPJ. The ultimate would be to assess joint mobility based on dynamic measurements in all patients, however, this aim is unachievable due to financial and time constraints in diabetic screening programs. Therefore, further work to improve the reliability of taking passive joint ranges of movement and to understand why there is a poor relationship between passive measures and dynamic function is warranted.

Different walking patterns associated with diabetes have been identified in the literature and have been attributed to changes in walking speed (Mueller et al. 1994a). It is widely accepted that patients with neuropathy tend walk slower. In the present study the stance phase duration was longer in the neuropathic and ulcerated groups compared to the control groups, suggesting a slower walking speed. A review of the literature has shown that there are very few papers that have described the joint movement patterns in patients with diabetes and many have concentrated on the larger proximal joints reporting data only in the sagittal plane. No published data describing three-dimensional motion at the AJC during stance phase or reported values for dorsiflexion at the 1st MPJ during gait could be found. By performing gait analysis this study generated the mean motion time curve for the AJC in all three planes for each of the four study groups. Based on the existing literature it was hypothesised that patients with ulceration would show different joint movement profile at the AJC in all three planes of motion compared to the non-ulcerated groups. This hypothesis was not supported by the present study.

The overall patterns of motion at theAJC, in each study group during the stance phase of gait followed the motion patterns described previously by other workers on non

diabetic patients using the same and different measurement technique (Cornwall et al. 1999b; Mannon et al. 1997; Mosely et al. 1996). The greatest range of motion was found in the sagittal plane, followed by the frontal plane then the transverse plane. This corresponds with literature for other clinical groups (Woodburn et al. 2002). Mueller and associates have reported reduced sagittal plane motion in diabetic patients with neuropathy during gait compared to non-diabetic controls. (Mueller et al. 1994a). This finding was supported by the present study.

On the basis of the high accuracy of the EMT system (more than 10 papers reported less than 1° error in angular rotation within the accurate field) and the high CMC data for the diabetic groups (Appendix 2) a minimal between groups detectable difference of 4° was needed. The differences between the diabetic groups were not greater than the minimally detectable difference. Kautolis and associates studied the gait style in patients with history of ulceration, non neuropathic diabetic patients and non diabetic controls (Katoulis et al. 1997b). They found that whilst no statistical differences were apparent between the groups, subtle changes in gait style were present. However, many of these differences could be related to the differences in walking speed found between the groups.

Holewski and associates (1989) found that patients with a history of ulceration had a higher prevalence of limited dorsiflexion at the ankle joint. In the present study measurement of passive ankle joint dorsiflexion was not undertaken since the measurement is dependent on determining the subtalar joint neutral position which has been reported in the literature (Freeman 1990; Pierrynowski et al. 1996), and confirmed in the development of methods section in the present study to be unreliable. As a replacement for passive ankle joint dorsiflexion, measurement of dorsiflexion during gait was taken. Contrary to the findings of Holewski and associates, a statistical difference in the amount of dorsiflexion between patients in the ulcerated group compared to the non-ulcerated groups was not found in the present study. To enable the measurement of motion at the AJC in the shod state standardised shoes were used. Looking at the kinematics at the ankle joint complex during gait in the barefoot and shod state, differences were noted in all the study groups. The magnitude of the increase in joint range of motion at the AJC in the shod state was notable in all three planes. This may have been partly due to changes in walking speed; many of the

diabetic patients were not used to walking barefoot (advice typically given to diabetic patients is not to walk around without shoes) and tended to walk a little faster in shoes.

Changes in angular position between the shod and barefoot state were noted; in the sagittal plane the heel strike position was more plantarflexed with shoes. It is possible that when the shoe made initial contact with the ground the AJC started to plantar flex and there could be a slight delay before the heel switch inside the shoe was activated. This delay in the activation of the heel switch would mean that the AJC would appear to be more plantar flexed in shoes compared to the barefoot state, where as in fact actual position at ground contact could be the similar. Locating foot switches within a shoe is problematic. The only way to accurately identify ground contact when a subject is wearing shoes is to link the motion analysis system with a force platform and use the force platform to trigger motion capture. Unfortunately a force platform was not available to be used in the present study.

In the present study during shod gait the position of the AJC was more inverted at both heel strike and at the end of the stance phase compared to barefoot walking in all of the four study groups. The mean maximum eversion position was reached earlier in the stance phase, which resulted in an increased time for the AJC to invert in preparation for propulsion. Using widely adopted biomechanical theory (Root et al. 1977), the foot appears to be functioning more efficiently during shod gait. It is widely accepted that footwear helps to protect the neuropathic foot from ulcerating by reducing plantar pressures, but the differences in kinematics demonstrated in this study could also be a major factor, which warrants further consideration.

5.5 Pressure data

Many papers have shown a statistically significant difference in the magnitude and location of peak pressures between diabetic patients who have ulceration and those who have not (Boulton et al. 1983; Fernando et al. 1991). In the present study statistically significant differences were not found between the three diabetic study groups. It is not valid to compare absolute pressure values collected using different pressure measurement systems, due to the effect that the sensor size will have on the magnitude

of pressure recorded. Generally the apparent pressure measured using a system with a smaller sensor size would be larger than the same pressure recorded with a system that had a larger sensor size. Most previous work which has documented peak pressure in diabetics have used platform systems with a much higher spatial resolution than the Pedar system used in the present study. It was therefore, expected that lower peak pressures would be recorded in the present study compared to previous work.

It is also not valid to compare absolute values of peak pressure between in-shoe and platform systems. It is generally widely acknowledged that in-shoe pressures are lower than barefoot pressures. The standard shoes in the present study had a thick sole, which would undoubtedly reduce plantar pressures. Therefore, it was expected that the pressures reported in the diabetic groups in the present study would be lower than previous reports.

Although it is not valid to extrapolate peak pressure values from one study to another when different pressure measurement systems have been used, the general trends in distribution of peak pressure should be the same irrespective of the absolute values recorded. In the present study in the diabetic control group three areas predominated, with approximately one third of patients having their highest pressure under the heel, under the 1st metatarsal head or under the 2nd metatarsal head. In the neuropathic and ulcerated group the majority of patients had the peak pressure under their 1st or 2nd metatarsal head. The transfer of peak pressure from the heel to the to the metatarsal head with increased neuropathy is well documented (Boulton et al. 1987a; Ctercteko et al. 1981; Veves et al. 1991) and it is not surprising that differences in location of peak pressures were seen between the groups. Veves and associates found that the peak pressure was located under the heel more frequently in the healthy non-diabetic control group compared to the diabetics. The present study supported this finding (Veves et al. 1991).

The peak pressure under the 1st metatarsal head was elevated in the ulcerated group in comparison with the other diabetic groups. This corresponds with the findings of Ctercteko who reported a medial shift of force in patients with neuropathic ulceration (Ctercteko et al. 1981). The ulcerated group had slightly higher forefoot pressures in comparison to the diabetic control group, although this did not reach statistical significance. The peak pressure and contact area under the hallux and toes was greater

in the non-diabetic reference group compared to the ulcerated and neuropathic group. A decrease in toe function and subsequent loading is a common reported finding with diabetes as there is a transfer of load from toes to metatarsal heads (Ctercteko et al. 1981).

In all areas with the exception of the hallux and toe masks, the pressure: time integral in the diabetic groups was higher in comparison to the non-diabetics control group, the difference being more notable in the neuropathic groups. The contact time within every mask area was increased in the neuropathic and ulcerated groups compared to the diabetic control group. The increased mean stance phase duration in the neuropathic and ulcerated group will partly explain the increase in pressure time integral and contact time within the mask areas.

Differences in the mean peak pressures between diabetic patients with foot ulceration and those without, have been documented elsewhere. However, no statistically significant differences were found in the present study. Generally the mean peak pressure in most of the foot regions was increased in the ulcerated group, compared to the other groups but did not reach statistical significance. In the present study there was either no difference in mean peak pressure between the groups or the Pedar system was unable to detect any difference due to a lower spatial resolution. It is possible that no differences were present between the diabetic groups in the present study due to a high prevalence of foot deformity in all the groups.

In the diabetic control group, 90% of the sample had the presence of foot deformity (as determined by a foot deformity score greater than equal to one). There was also a high prevalence of foot deformity in the neuropathic and ulcerated groups. In a recent paper imaging techniques were used to quantify structural aspects of the foot and were combined with pressure measurements. The presence of a hammertoe deformity (as determined from measuring the metatarsal phalangeal joint angle) was found to be the most important variable for the prediction of peak pressure in the forefoot in patients with diabetes (Mueller et al. 2003). In the diabetic control group, a high proportion of the sample had either lesser toe retraction or prominent metatarsal heads, which has been associated with increased pressures under the metatarsal heads. The prevalence of foot deformity has not been cited in many of the previous papers, where greater differences between neuropathic and ulcerated groups have been reported. It is not

possible to ascertain whether differences between the groups were not found due to the lower spatial resolution of the Pedar system or whether no differences were actually present.

Performing measurement of pressure within shoe provides the opportunity to record data from multiple bilateral steps, thus minimising the risk of developing an ulcer from prolonged barefoot walking in the high risk / ulcerated foot. Because data can be taken during normal gait, it also provides the confidence that patients have not altered their walking pattern to target a platform (a common problem when taking pressure measurements using platform systems). A limitation associated with the Pedar system is the size of the pressure sensors. Each Pedar insole contains 99 sensors, whereas the FSCAN system (a commercially available in-shoe pressure measuring system) has insoles, which contain 999 sensors. The FSCAN system has been extensively used in the study of the diabetic foot. It has a much higher spatial resolution than the Pedar, which may be beneficial. However, the reliability of the FSCAN has been questioned (McPoil et al. 1995). The Pedar system has been shown to have a high level of validity and reliability in both bench and dynamic testing (McPoil et al. 1995) and has been shown to have a low level of error, particularly at high pressures. The limitation associated with a lower spatial resolution will not jeopardise the primary results or conclusions of the study. However, unreliable data from a system with a higher spatial resolution would.

5.6 Motion and pressure data

The essence of this project was to use motion analysis in conjunction with pressure measurement. In the present study the only correlation found between joint motion and pressure parameters was between passive frontal plane motion at the AJC and the pressure time integrals in the heel, midfoot and forefoot areas. No correlation was found between passive frontal plane range or any of the ranges of motion at the AJC during gait and peak plantar pressures in the any of the mask areas. This finding was unexpected as previous literature and podiatric biomechanical theory suggests that relationships would be present (Bevans 1992; Root et al. 1977). From previous literature, one would assume that patients with a lower passive range of motion would

have a lower range of motion during gait and this would result in a higher peak pressure and increased chance of developing ulceration.

In a much smaller study that investigated the impact that ethnic origin had on joint mobility, Van Schie also found no relationship between passive range of motion and plantar pressures. Van Schie (2002) found that the Asian patients had an increased passive range of movement and lower plantar pressures. However, no difference was found in the range of movement during gait between the groups and no association was reported between any of the foot joint movement data and peak pressure. The present study also found no logical association between any dynamic foot joint movement data and plantar pressure. The findings of the present study do not support current theories about the impact that joint mobility has on plantar foot pressures.

Although the present study did not find any relationship between joint mobility in the foot and high plantar pressures, previous work has found an association. Furthermore, limited movement has been associated with the formation of plantar forefoot ulceration. Lengthening of the Achilles tendon as a mechanism to increase movement at the ankle has been performed in patients with recurrent forefoot ulceration. Indeed this procedure has been shown to be successful for facilitating healing of plantar forefoot ulceration. The mechanism as to why this procedure is successful may be different than suspected. It is possible that limited passive joint movement in the feet is acting as a proxy measure for something else that is more important, for example the quality and quantity of the soft tissue under the forefoot area.

In support of this speculation, Morag and associates (Morag et al. 1999), only found four gait related parameters entered statistical models for prediction of pressure under the 1st metatarsal head and hallux (major sites of ulceration in patients with diabetes). Structural aspects including the amount of soft tissue under the metatarsal head were found to be more important for the prediction of pressure in the forefoot. Despite previous research indicating a relationship between limited passive ankle joint dorsiflexion and ulceration, a recent paper by the same group failed to find any consistent relationship between passive range of ankle joint dorsiflexion peak pressures in diabetic patients (Mueller et al. 2003). Furthermore, the measurement of soft tissue thickness under the metatarsal head was found to make a significant contribution in explaining the variance of plantar pressure.

The patients in the present study were followed for 12 months following gait assessment and their ulceration status was monitored. It was hoped that from the motion analysis data it might be possible to predict those patients who went on to develop ulceration. Although the percentage of patients who went on to develop ulceration in the 12 month follow up period was consistent with the literature, because the overall sample size of the study was small, the number of patients ulcerating in the 12 month follow up period within the groups would not allow for any inferences to be made. In the present study the percentage of the neuropathic group that ulcerated during the 12-month follow up period was 7 %. This level of ulceration is consistent with the findings from a much larger study, where 7% of patients ulcerated in a 2 year postal follow up (Abbott et al. 1998). Murray and associates documented a slightly higher ulceration rate; they found 10% of patients developed ulceration in a mean follow up period of 15 months. To be able to substantiate any inferences about who may ulcerate based on their gait profile a much larger sample size would be needed.

In the present study no association was found between foot joint movement data and plantar pressure. This suggests that other factors for example structural aspects of the foot may be more important factors in the generation of high plantar foot pressures in diabetes. The findings of the present study fail to support many of the current theories about the impact that joint mobility has on the formation of plantar ulceration. After review of the current literature it was hypothesised that patients with ulceration would have limited movement at the AJC and 1st MPJ during gait and would have a different motion time curves at the ankle joint complex. No significant differences in joint motion time curves were found between the study groups. No relationship was found between the passive range of motion at either the AJC or 1st MPJ and the range of motion during gait. No relationship was found between foot joint movement data and plantar pressures.

5.7 Limitations of the present study

In the present study the pressure measurements were taken in a standard shoe, as opposed to the patient's own shoes. Literature has shown that different types footwear can have a major influence on plantar pressures and it was felt that controlling for

differences in footwear was valid (Kastenbauer et al. 1998; Lavery et al. 1997). However, when studying patients with plantar intrinsic foot ulceration it is useful to have a measure of cumulative load throughout an average day (combination of magnitude and duration of pressure), as it is likely that these pressures have caused the ulceration. Overall the plantar pressure values recorded in the present study may not be representative of the actual absolute pressure found under the patient's foot in their own footwear. This study failed to take into account the activity levels of patient (cumulative loading). This factor may be very important, when determining risk for ulceration. Asking patients to document activity levels can be very unreliable, however, technical advances can now overcome many of these issues. Armstrong and associates are currently using Global Positioning Sytems (GPS) integrated with pedometers to document patient activity levels. This method could be used with telemetry in-shoe pressure measurement systems to gain an accurate measure of cumulative pressures and examine the effect on plantar ulceration.

The Pedar system used in this study has a relatively low spatial resolution when compared to other methods, for example the optical pedobaragraph. As a result between group differences in the magnitude of peak pressures may have been lost. If a pressure system with a higher spatial resolution had been used, some differences in pressure parameters may have been identified between groups, however, based on the fact that no correlation was found between joint motion and pressure, the main conclusions of the study would remain. Furthermore, the EMT system used to measure joint movement has been shown to be highly accurate (less than 1° error in angular rotation) and repeatable and we can be confident that there were no differences in joint motion between the diabetic groups.

Although, the present study did not find clinically meaningful differences in gait parameters between the diabetic groups, working on an individual basis, gait analysis can be used as a mechanism by which to increase our understanding of the pathoaetiology of foot ulceration. This is highlighted by examining two cases in the study (Patient A and B, full clinical details in Appendix 10). Based on clinical data and screening for risk of ulceration, patient A would be classified as having low risk of developing ulceration, whereas patient B would be classified as having high risk for ulceration. Contrary to the screening predictions, patient A presented with an ulcer to the right 1st metatarsal head and hallux yet patient B had never developed an ulcer. By

examining the gait related parameters of each patient it becomes apparent why patient B may have been protected against developing ulceration. Patient B has a very slow walking speed and has very little ankle joint movement (perhaps through use of the "hip strategy" as described by Mueller and associates) (Mueller et al 1994a). Patient A has a normal walking speed and is probably continuing to use the "ankle strategy" during gait, with accompanying severe restriction at the right 1st MPJ has resulted in increased focal stresses under the 1st metatarsal head and hallux (ulceration site).

One potential limitation with the electromagnetic tracking system used in this study is the cabling associated system; it is possible that patients may feel constrained by the wires and may not walk naturally. In the development of methods section walking speed and double limb support times were used as a measure of overall lower limb function. If patients felt constrained by the wires it was assumed that they would walk slower and spend a greater proportion of the gait cycle in double limb support. Differences in walking speed or double limb support times were found not to alter with the addition of the motion analysis equipment. However, it must be noted that this applied to a normal population. It is possible that patients with diabetes and neuropathy may have reacted differently to the addition of the motion analysis system. The electromagnetic tracking system can now be purchased in a telemetry form, overcoming the problems associated with the tethering from the sensors to the motion capture units, this system would be preferable for use in future studies.

The pressure measurement system used in this study was an in-shoe system. It was not possible to take measurements of the motion at the 1st MPJ within the shoe. This resulted in barefoot joint movement data being compared to pressure data that was taken in-shoe. The range of dorsiflexion during barefoot gait may not be representative of the range of dorsiflexion in shoe because factors such as rigidity of the sole and upper of the shoe may have influenced the amount of dorsiflexion at the 1st MPJ. An improvement to the study design would be to use a pressure measuring platform system, which would enable simultaneous measurements to be taken barefoot of joint motion data at the 1st MPJ and plantar pressure data.

5.8 Further work

Despite the rapid expansion of motion analysis technology and its incorporation in to other clinical areas, such as assessment and management of patients with cerebral palsy, at present there are very few studies describing the gait characteristics in patients with diabetes. This study has shown that motion analysis can be performed within the confines of clinical practice and has the potential to support much larger scale studies.

Changes in loading patterns of pressure have been shown to occur in relatively short periods of time (Veves et al. 1992a), suggesting that changes in gait may also occur in a relatively short time period. This study did not find major differences in movement at the AJC between the diabetic and the non-diabetic reference group after long disease duration.

However, it must be noted that the present study did not investigate motion patterns at the more proximal joints and did not take into account joint moments. It would be valuable to take repeated gait measurements (including proximal joint) in patients who have diabetes, to see if changes do occur and to document the magnitude of these changes. As there is currently no data on how gait changes with the progression of diabetes, a large prospective study collecting both clinical and gait related data is needed. If diabetic patients were followed from initial diagnosis (within weeks / months) and had annual gait assessment for the next ten years, this would provide objective data about how gait changes during the disease process. It may also allow prognostic indicators of subsequent diabetes related foot disease, for example development of foot deformity and ulceration to be identified.

The changes in kinematics at the AJC whilst wearing standardised shoes reported in this study warrant further investigation. Many papers have described the effects of specific interventions / modification to footwear or orthoses in terms of changes in plantar pressures. However, how these interventions alter foot function, in terms of joint kinematics remains unanswered.

Limited passive range of motion at the 1st MPJ has been linked with the generation of high plantar foot pressures and the formation of ulceration in the diabetic neuropathic

foot. The findings of this study show that there is no relationship between the passive range of motion and the range of motion during gait. Motion patterns at the 1st MPJ need to be investigated in a much larger sample size taking into account the structural and functional aspects of soft tissues to establish the precise mechanism by which ulceration occurs. Development of motion analysis techniques to allow accurate measurement of the 1st MPJ within the confines of a shoe may further enhance understanding why certain treatment modalities are successful in preventing / healing ulceration and others are not.

Developments in pressure measuring hardware and software have been made in recent years, making data collection and analysis easier and quicker to perform. Many pressure systems now allow for a wide range of pressure variables to be studied, including measures of foot geometry and the velocity of the centre of pressure. The value of these parameters has not been fully investigated and the value that these parameters may have if any, in the prediction of ulceration warrants a prospective study.

5.9 Overall synthesis

This study has for the first time documented three-dimensional motion at the AJC during gait and dynamic dorsiflexion at the 1st MPJ. The data showed no relationship between any joint movement data at the AJC and peak pressure or pressure time integrals in any of the four study groups. From a review of the literature it was expected to find a difference in the motion time curves for the AJC between diabetic patients with and without previous foot ulcer, yet differences in the motion time curves at the ankle joint complex were not found between any of the study groups.

The findings of the present study show that there is no basis for the current theory that people with a limited passive range of movement will have a limited range of motion during gait. The results of this study suggest that there is not a causal relationship between limited joint mobility and high pressures. Previous cross sectional reports have shown that limited joint mobility is associated with diabetic foot ulceration, however, the present study did not support this. Limited joint mobility has been cited as a risk factor for ulceration; however, it is possible that limitation in joint movement occurred after ulceration (following prolonged periods of non-weight bearing).

Diabetic foot ulceration is clearly multi-factorial and gait related parameters (plantar pressures and joint movement) represent only a small number of factors, which may be important in the formation of plantar ulceration. Although between group differences in gait parameters were not found in this study, assessment of gait may be useful on an individual basis to increase our understanding why some patients develop ulceration. Further technological advances that allow cumulative pressure, shear measurements and joint function to be taken alongside imaging techniques to measure the behaviour of soft tissue during gait may facilitate a much greater understanding in the pathogenesis of diabetic foot ulceration.

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Appendix 1:

The formula for calculating the adjusted coefficient of multiple correlation

The adjusted coefficient of multiple determination for evaluating repeatability of waveforms is given by

where R_a^2 is given by,

$$R_a^2 = 1 - \frac{\sum_{i=1}^{M}\sum_{j=1}^{N}\sum_{t=1}^{T}\frac{(Y_{ijt} - \overline{Y}_t)^2}{T(MN-1)}}{\sum_{i=1}^{M}\sum_{j=1}^{N}\sum_{t=1}^{T}\frac{(Y_{ijt} - \overline{Y})^2}{(MNT-1)}} \qquad (1)$$

where $\overline{Y}_t$ is the average at time point t over NM gait cycles,

$$\overline{Y}_t = \frac{1}{MN}\sum_{i=1}^{M}\sum_{j=1}^{N}Y_{ijt} \qquad (2)$$

and $\overline{Y}$ is the grand mean over time and is given by,

$$\overline{Y} = \frac{1}{MNT}\sum_{i=1}^{M}\sum_{j=1}^{N}\sum_{t=1}^{T}Y_{ijt} \qquad (3)$$

The coefficient of multiple determination is used for evaluating the repeatability of waveforms. In the expression the numerator in expression (1) represents variance about the mean at time point t over all test days. The denominator of the ratio represents the total variability about the grand mean of all test days

Appendix 2:

CMC data for the first ten diabetic patients recruited into each group.

Group	Subject	Sagittal	Frontal	Transverse
Control	1	0.948	0.958	0.908
	2	0.878	0.77	-
	3	0.975	0.922	0.77
	4	0.982	0.917	0.533
	5	0.982	0.915	0.887
	6	0.995	0.829	0.781
	7	0.990	0.803	0.720
	8	0.991	0.957	0.855
	9	0.991	0.784	0.920
	10	0.992	0.983	0.835
	Mean	0.972	0.884	0.801
	SD	0.036	0.079	0.121
Neuropathic	1	0.994	0.980	0.859
	2	0.971	0.810	0.942
	3	0.978	0.952	0.869
	4	0.994	0.880	0.962
	5	0.987	0.904	-
	6	0.989	0.924	0.924
	7	0.978	0.949	0.830
	8	0.964	0.898	0.564
	9	0.966	0.824	0.546
	10	0.960	0.864	0.688
	Mean	0.978	0.889	0.798
	SD	0.013	0.055	0.160
Ulcerated	1	0.960	0.902	0.692
	2	0.977	0.975	0.840
	3	0.988	0.992	0.938
	4	0.986	0.876	0.748
	5	0.994	0.920	0.688
	6	0.960	0.902	0.692
	7	0.939	0.679	0.898
	8	0.962	0.958	0.887
	9	0.958	0.790	0.412
	10	0.997	0.896	0.742
	Mean	0.972	0.889	0.754
	SD	0.019	0.093	0.152

Appendix 3:

Range and standard deviation of angular positional data for the ankle joint complex in all three planes of motion, for the first ten diabetic patients recruited into each group.

GROUP	SUBJECT	MEAN RANGE S	SD	MEAN RANGE F	SD	MEAN RANGE T	SD
	1	0.68	0.54	0.61	0.40	0.36	0.31
	2	1.06	0.59	0.69	0.25	2.02	1.55
	3	4.26	1.97	3.27	2.87	8.08	5.21
	4	0.98	0.61	1.13	0.97	1.36	1.10
CONTROL	5	2.47	0.89	1.91	0.71	5.54	2.18
	6	1.85	0.47	2.21	1.60	4.30	3.42
	7	1.37	0.46	1.58	0.70	1.26	0.49
	8	1.49	0.53	0.83	0.54	0.90	0.35
	9	1.03	0.49	1.00	0.41	2.15	0.83
	10	1.88	0.94	2.44	0.71	1.26	1.00
Mean		1.71	0.75	1.57	0.92	2.72	1.64
SD		1.04	0.46	0.88	0.78	2.47	1.58
	1	2.19	1.26	1.75	0.40	3.42	0.85
	2	2.18	0.72	2.34	1.19	2.32	1.04
	3	1.85	1.36	1.54	0.80	1.57	0.76
	4	2.24	0.73	2.73	0.91	2.55	0.68
NEUROPATHIC	5	0.98	0.33	1.74	0.78	2.48	0.81
	6	4.83	2.83	3.27	1.01	2.62	0.75
	7	0.92	0.51	0.76	0.26	1.94	1.43
	8	1.89	0.96	2.18	0.58	1.97	0.97
	9	2.50	0.96	2.10	0.73	4.27	2.14
	10	1.45	0.84	0.62	0.36	2.79	1.40
Mean		2.10	1.05	1.90	0.70	2.59	1.08
SD		1.10	0.70	0.81	0.30	0.78	0.46
	1	1.65	0.66	2.28	0.87	5.10	1.48
	2	0.64	0.58	0.85	0.56	2.20	1.68
	3	1.22	0.75	0.31	0.31	0.69	0.39
	4	1.77	1.24	4.66	2.53	4.83	2.31
ULCERATED	5	0.99	0.64	1.05	0.39	0.95	0.41
	6	1.65	0.66	2.28	0.87	5.10	1.48
	7	1.25	0.42	1.32	0.61	1.25	0.44
	8	2.47	1.11	1.34	0.48	1.38	0.49
	9	1.27	0.58	1.88	0.53	2.24	0.89
	10	0.70	0.35	0.87	0.50	2.19	1.12
Mean		1.36	0.70	1.68	0.76	2.59	1.07
SD		0.55	0.28	1.22	0.65	1.75	0.66

Appendix 4:

PATIENT INFORMATION SHEET

Study Title: A study of motion and foot pressures during walking in a diabetic and non-diabetic population

Name of Researchers: Miss D Turner, Dr P Helliwell, Dr B Bodansky, Mrs C Widdows

You are invited to take part in this research study. As a volunteer you will not benefit directly from taking part in the study. However, the findings will lead to a greater understanding of how the foot functions in patients with diabetes and hopefully will lead to an improvement of care for patients with diabetes related foot problems.

Background information: Diabetes can cause many problems in the foot including loss of feeling, loss of movement at joints and foot deformity which results in high pressures underfoot and possible symptoms. It is felt that a good range of movement at joints is needed for the foot to function properly. There is little information which tells us how the foot works and especially in those individuals who have established diabetes related foot problems.

Study aims: This study aims to determine the ranges of motion at the ankle joint and pressure distribution underfoot in a sample of non diabetic volunteers aged 18-80. These findings will then be compared to a sample of volunteers with diabetes, so any differences can be highlighted. As a volunteer you will not benefit directly from taking part in the study. However, the findings will lead to a greater understanding of how the foot functions in patients with diabetes and hopefully will lead to an improvement of care for patients with diabetes related foot problems.

What the study will entail for you: We will measure the way you walk (motion analysis and foot pressure measurement). This will involve sensors been taped to the lower leg and heel with hypoallergenic tape and wearing pressure measuring insoles in your shoes The measurements will take about 30 minutes. You will need to change into a pair of shorts, which will be provided, or you could bring a pair of your own. Changing facilities are available.

If you have diabetes, you will need to be assessed prior to the measurements been taken. This will involve testing your ability to determine vibration using a tuning fork and your ability to differentiate between hot and cold temperatures and sharp and blunt objects. An assessment will be made to determine if you have any joint changes, which are related to your diabetes. You will also be asked some questions about any symptoms you have related to your diabetes. Any information you give will remain strictly confidential. If you decide to volunteer, you can withdraw at any time without having to give a reason and this will not affect your future care.

Questions:

If you are unsure about any part of this study please ask the researcher now.

IMPORTANT

If you have a pacemaker please tell the researcher now

Consent: If you are happy to take part in this study please read and complete the attached consent form.

Contact: Should you require any further advice please contact Miss D. Turner (PhD student) at The Spinal Research Unit, University of Huddersfield, Queensgate, Huddersfield, HD1 3DH, Telephone (01484) 472657, in office hours.

Appendix 5:

University of Huddersfield

PATIENT CONSENT FORM		
A study of motion and foot pressures during walking in a diabetic and non-diabetic population		
Name of Researcher: Dr/Mr/Mrs/Miss/Ms		
The patient should complete the whole of this sheet himself/herself	Please circle as necessary	
Have you read the patient information sheet?	**YES**	**NO**
Have you been given a copy to keep?	**YES**	**NO**
Have you had the opportunity to ask questions and discuss this study?	**YES**	**NO**
Have you received satisfactory answers to all of your questions?	**YES**	**NO**
Have you received enough information about the study?	**YES**	**NO**
Whom have you spoken to? ……..		
***Do you understand that you are free to withdraw from the study*:** - **At any time** - **Without having to give a reason for withdrawing** - **Without affecting your future care** **YES / NO**		
Do you agree to take part in the study? YES / NO		
Signed: …….. Date: …… **Name (block capital) ……..** **Signature of person talking consent …… Date: ……** **Name (block capitals) ……..**		

Version 01 (01/05/1999)

Appendix 6: Mean (SD) Ranges of motion at the ankle joint complex and 1st metatarsophalangeal joint in non-diabetic control groups, data presented by decade.

			Ankle joint complex Frontal plane		1st metatarsophalangeal joint Sagittal plane	
			Mean	**SD**	**Mean**	**SD**
20-29	Male	Left	31.01	(3.46)	56	(16.47)
		Right	31.37	(7.26)	54.89	(15.2)
	Female	Left	26.74	(5.17)	57.5	(14.13)
		Right	24.4	(6.04)	53.33	(10.46)
30-39	Male	Left	27.15	(2.4)	48.69	(18.4)
		Right	23.01	(3.11)	47.18	(14.7)
	Female	Left	27.03	(4.27)	58.87	(15.1)
		Right	24.41	(5.59)	52.87	(14.7)
40-49	Male	Left	24.26	(3.67)	44.68	(7.78)
		Right	23.91	(4.12)	41.87	(8.84)
	Female	Left	26.76	(6.89)	49.3	(14.63)
		Right	26.35	(4.85)	52.48	(11.61)
50-59	Male	Left	24.55	(8.75)	37.58	(6.79)
		Right	21.48	(4.76)	37.3	(16.1)
	Female	Left	25.04	(4.59)	54.77	(13.01)
		Right	24.19	(5.53)	50.06	(14.17)
60-69	Male	Left	20.05	(3.25)	36.01	(20.26)
		Right	21.92	(6.25)	39.68	(14.6)
	Female	Left	23.6	(7.31)	56.1	(12.79)
		Right	23.37	(7.1)	56.73	(20.5)

Appendix 7: Plantar pressure from non-diabetic control group (n=100). Mean (SD) and lower and upper limits based on mean ± 1 SD.

	Total	Medial heel	Lateral heel	Medial midfoot	Lateral midfoot	Metatarsal head 1	Metatarsal head 2	Metatarsal heads 3-5	Hallux	Toe 2	Toes 3-5
Peak pressure (kPa)	323.11 (91.46)	233.5 (52.3)	229.6 (54)	43.5 (25.2)	71.3 (29.8)	249 (91.6)	231.2 (82.6)	193.9 (67.2)	233.7 (120.2)	132.4 (60.1)	101.5 (42.5)
Lower	231.65	181.2	175.6	18.3	42	157.4	148.6	126.4	113.5	72.3	59
Upper	414.57	285.8	283.6	68.7	101.1	340.6	313.8	261.1	353.9	192.5	144
PTI (kPa*s)	143.4 (40.3)	72.4 (22.4)	72.5 (23.3)	15.3 (11.1)	28.7 (14)	75.9 (35.5)	71.2 (26.9)	65.3 (24.8)	59.6 (33.9)	34.9 (18.3)	32.9 (15.6)
Lower	103.1	50	49.2	4.2	14.7	40.4	44.3	40.5	25.7	16.6	17.3
Upper	183.7	94.8	95.8	26.4	42.7	111.4	98.1	90.1	93.5	53.1	48.5
Max force (N)	731.96 (187.66)	284 (79.76)	259.93 (78.44)	12.56 (12.99)	54.39 (29.36)	169.41 (80.78)	155.4 (57.5)	187.2 (76.55)	87.02 (54.03)	47.38 (25.41)	52.65 (31.93)
Lower	544.3	204.24	181.49	-0.43	25.03	88.63	97.9	110.65	32.99	21.97	20.72
Upper	919.62	363.76	338.37	25.55	83.75	250.19	212.9	212.9	141.05	72.79	84.58
Contact area (Cm^2)	159.68 (28)	26.63 (6.11)	26 (4.96)	5.94 (4.36)	14.39 (3.92)	14.82 (5.35)	14.56 (3.72)	25.43 (6.35)	8.92 (4.37)	8.41 (2.44)	12.79 (4.9)
Lower	131.68	20.52	21.04	1.58	10.47	9.47	10.84	19.08	4.55	5.97	7.89
Upper	187.68	32.74	30.96	10.3	18.31	20.17	18.28	31.78	13.29	10.85	17.69
Contact time (ms)	821.5 (182.8)	631.25 (193.8)	653.5 (189.35)	490.13 (224.88)	612.88 (191.48)	655.75 (244.76)	668.5 (233.4)	715.25 (206.14)	592.38 (266.9)	571.63 (243)	629 (207.94)
Lower	638.7	437.45	464.15	265.25	421.4	410.99	435.1	509.11	325.48	328.63	421.1
Upper	1004.3	825.05	842.85	755.38	804.36	900.51	901.9	921.39	859.28	814.63	836.9

Appendix- 8: Angular positional data and timings of the ankle joint complex during the stance phase of barefoot and shod gait.

Motion			Non diabetic reference		Diabetic control		Neuropathic		Ulcerated	
			AP	%SP	AP	%SP	AP	%SP	AP	%SP
Barefoot	Sagittal	HS	-0.5	0%	-1.5	0%	-1.3	0%	-1.5	0%
		Max PF	-5.2	15%	-5.9	15%	-5	15%	-4.4	13
		Max DF	8.4	85%	7.3	87	6.9	85%	7	85%
		T Off	4.2	100%	4.1	100%	3.1	100%	3.1	100%
	Frontal	HS	1.3	0%	0.7	0%	-0.1	0%	1.2	0%
		Max Eve	-3.6	71%	-3.7	76%	-3.7	77%	-3.4	74%
		T Off	1.9	100%	0.1	100%	0.3	100%	1.1	100%
	Transverse	HS	5.86	0%	2.7	0%	2.8	0%	0.7	0%
		Max Int	8.1	27%	5.1	25%	5.2	22%	2.4	30%
		T Off	4.2	100%	1.7	100%	1.6	100%	0	100%
Shod	Sagittal	HS	-0.5	0%	-6.7	0%	-6.9	0%	-4.5	0%
		Max PF	-5.2	16%	-12.2	14%	-12.5	13%	-10.5	15%
		Max DF	8.4	85%	4.3	84%	4.7	82%	5.5	85%
		T Off	4.2	100%	-1.8	100%	-2.4	100%	-1.4	100%
	Frontal	HS	3.1	0%	2.9	0%	3.2	0%	1.8	0%
		Max Eve	-2.1	65%	-1.3	60%	-2.3	62%	-1.5	52%
		T Off	5.6	100%	3.9	100%	4.2	100%	2.9	100%
	Transverse	HS	5.8	0%	1.6	0%	2.8	0%	0.9	0%
		Max Int	8.1	29%	5.1	25%	7.1	26%	3.6	30%
		T Off	4.2	100%	0.6	100%	1.1	100%	0.26	100%

AP- Angular position, %SP- % of stance phase, HS – Heel strike, Max PF- maximum plantarflexion, Max DF-maximum dorsiflexion, T Off- Toe off angle.

Appendix -9: Mean (SD) pressure data in the 10 mask areas in the non-diabetic reference, diabetic control, neuropathic and ulcerated groups.

		Non diabetic reference	Control	Neuropathic	Ulcerated
Peak Pressure (Kpa)	Medial heel	212(54)	234(35)	254(63)	254(56)
	Lateral heel	214(57)	212(69)	213(66)	243(57)
	Medial midfoot	40(34)	62(43)	46(30)	70(55)
	Lateral midfoot	69(31)	91(42)	73(30)	102(68)
	1st Metatarsal head	226(86)	253(104)	254(98)	296(134)
	2nd Metatarsal head	210(74)	243(89)	239(72)	242(101)
	3-5 Metatarsal heads	184(63)	199(71)	200(71)	205(77)
	Hallux	225(112)	154(108)	125(92)	155(102)
	2nd Toe	123(55)	100(72)	77(46)	92(43)
	3-5 Toes	100(52)	77(51)	67(35)	72(46)
Pressure Time Integral (Kpa.sec)	Medial heel	72(19)	86(35)	105(34)	109(33)
	Lateral heel	74(20)	79(29)	100(36)	105(29)
	Medial midfoot	14(13)	26(21)	22(18)	32(31)
	Lateral midfoot	29(17)	40(23)	39(19)	49(31)
	1st Metatarsal head	72(31)	74(42)	110(66)	113(60)
	2nd Metatarsal head	67(27)	77(42)	104(50)	91(47)
	3-5 Metatarsal heads	64(27)	69(32)	88(42)	84(39)
	Hallux	66(40)	40(33)	39(28)	46(34)
	2nd Toe	33(15)	28(23)	27(15)	29(16)
	3-5 Toes	35(20)	27(21)	31(18)	29(23)
Contact Area (Cm^{-2})	Medial heel	27 (5)	26(5)	28(8)	28(5)
	Lateral heel	27 (5)	25(5)	27(6)	28(6)
	Medial midfoot	6(5)	9(7)	7(6)	8(5)
	Lateral midfoot	14 (5)	15(8)	8(5)	15(4)
	1st Metatarsal head	14 (6)	15(7)	8(6)	16(6)
	2nd Metatarsal head	14 (4)	14(4)	15(4)	14(3)
	3-5 Metatarsal heads	25(6)	21(7)	25(6)	25(8)
	Hallux	10(6)	7(4)	7(4)	7(3)
	2nd Toe	8(2)	7(3)	7(3)	7(3)
	3-5 Toes	12(6)	10(6)	11(5)	10(5)
Contact Time (ms)	Medial heel	692(178)	687(221)	852(270)	763(171)
	Lateral heel	711(177)	702(202)	877(274)	794(158)
	Medial midfoot	474(274)	577(244)	647(309)	637(165)
	Lateral midfoot	615(225)	661(220)	801(282)	723(167)
	1st Metatarsal head	680(235)	654(286)	902(348)	756(263)
	2nd Metatarsal head	706(217)	724(249)	931(337)	794(234)
	3-5 Metatarsal heads	734(210)	728(256)	963(294)	810(228)
	Hallux	650(251)	521(296)	627(327)	655(264)
	2nd Toe	607(225)	507(310)	756(360)	629(281)
	3-5 Toes	652(207)	575(309)	756(360)	666(248)
Maximum force (N)	Medial heel	267(82)	278(86)	260(89)	313(75)
	Lateral heel	259(102)	242(84)	256(79)	296(91)
	Medial midfoot	13(14)	27(29)	15(15)	22(24)
	Lateral midfoot	51(33)	67(45)	53(26)	74(52)
	1st Metatarsal head	157(89)	160(79)	148(70)	179(88)
	2nd Metatarsal head	144(58)	168(87)	159(64)	164(64)
	3-5 Metatarsal heads	182(85)	167(87)	168(64)	188(79)
	Hallux	86(52)	47(39)	44(40)	45(33)
	2nd Toe	46(27)	33(27)	28(18)	30(14)
	3-5 Toes	55(43)	34(30)	34(21)	29(20)

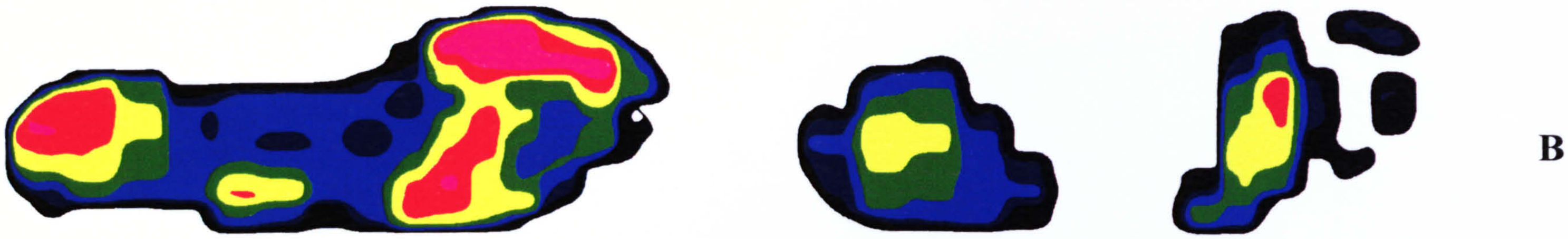
Appendix -10:

Outlined below are two clinical cases from the study, which highlight the usefulness of gait analysis techniques. In these two cases, details of patients gait are paramount in explaining why patient A has developed ulceration and patient B has never ulcerated.

		Patient A			Patient B		
Clinical details	**Gender**	Female			Male		
	Age(Yrs)	33			55		
	Duration of diabetes (Yrs)	18			30		
	Body mass (Kg)	63.5			127		
	Diabetic complications	None			Retinopathy, Renal damage		
Neurological status	**NDS**	5/9			4/9		
	NSS	4/10			8/10		
	Vib ppt (Volts)	R 5.5 L6.5			R&L >50		
	Monofilaments	Full detection of 1g monofilament			No detection of 75g monofilament		
Foot examination	**Ulceration**	Active ulceration R 1st IPJ and MPJ			No current or previous ulceration		
	Range of motion AJC	R5° L 7°			R27° L 28°		
	Range of DF 1st MPJ	R27° L 75°			R34° L 34°		
Gait	**Stance phase duration**	740ms			1540ms		
		Sag	**Frontal**	**Trans**	**Sag**	**Frontal**	**Trans**
	Min	-9.3	-1.5	-6.0	-3.7	-3.2	-0.2
	Max	7.4	8.8	0.8	9.6	1.2	1.5
	Range	16.7	10.3	6.8	13.3	4.4	1.7
Pressure Profile	**Plantar region**	MF (N)	PP(kPa)	Pti(kPa.sec)	MF (N)	PP(kPa)	Pti(kPa.sec)
	Medial heel	169	230	66	208	130	123
	Lateral heel	36	40	11	12	60	48
	Medial midffot	87	130	43	9	40	33
	Lateral midfoot	254	350	79	63	100	92
	1st MPJ	116	190	36	74	120	113
	2nd MPJ	199	200	51	82	100	99
	MPJ 3-5	73	200	29	12	40	31
	Hallux	35	140	18	8	40	40

Peak pressure profiles

A

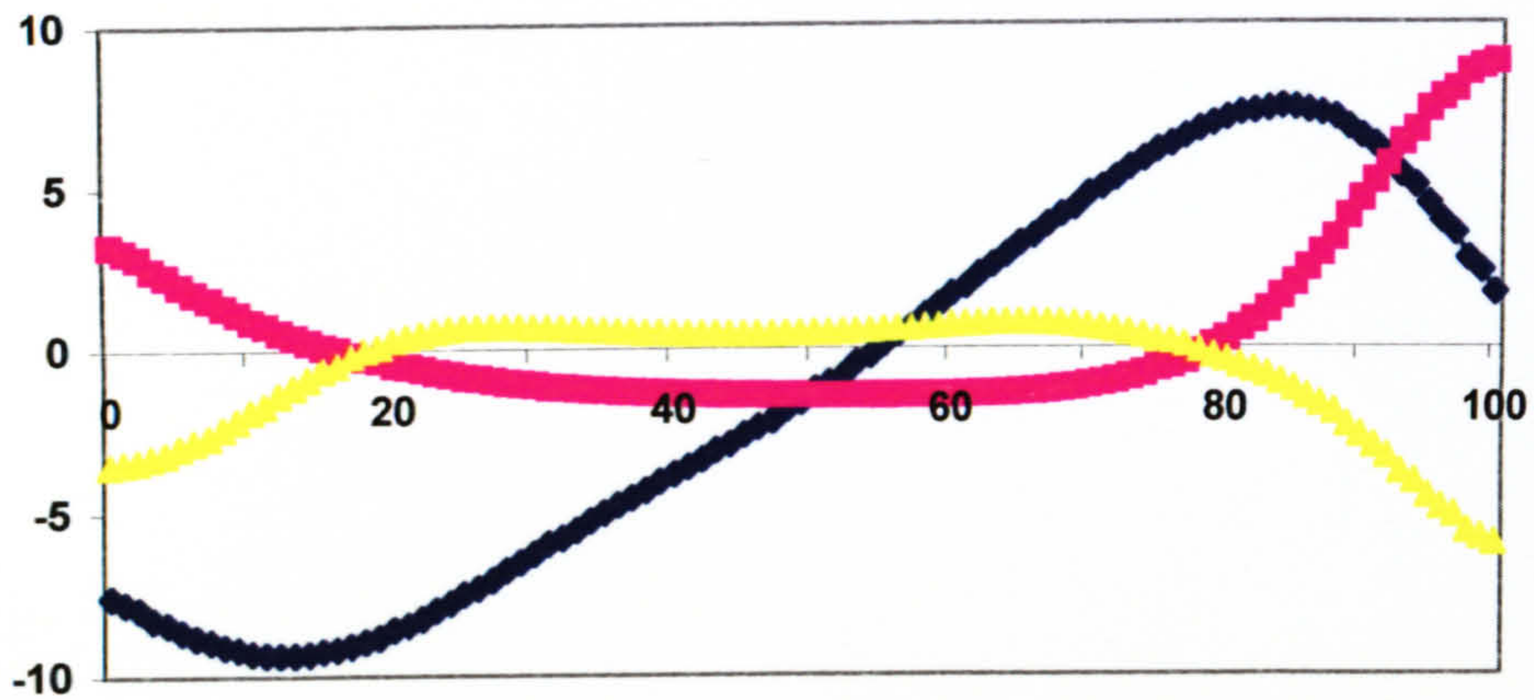


B

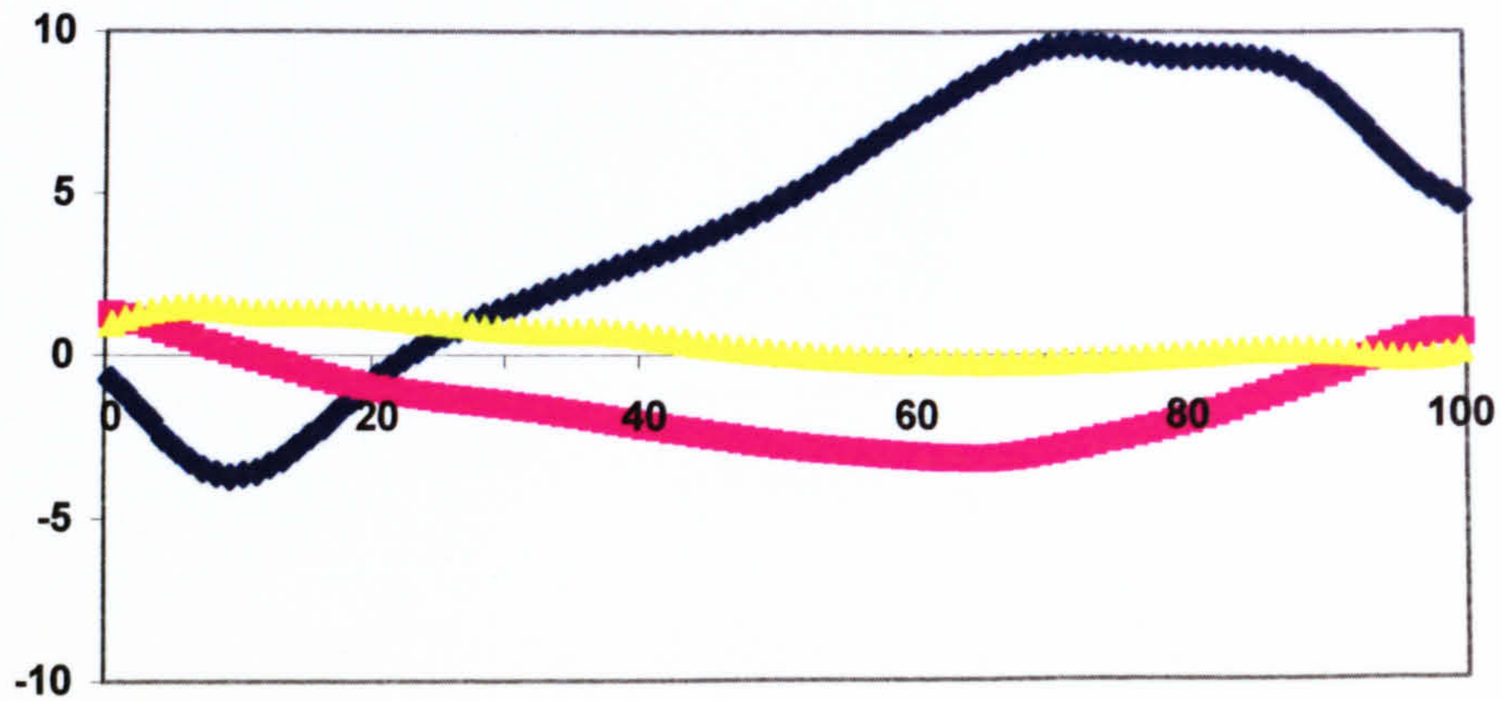
Patient A, Peak pressure over hallux and 1st metarsal head, 140kPa and 190kPa respectively. Patient B, Peak pressure over hallux and 1st metarsal head, 40kPa and 120kPa respectively.

Motion Graphs

A



B



◆ Sagittal plane movement - dorsiflexion (+) / Plantar flexion (-)
■ Frontal plane movement – Inversion (+) / eversion (-)
△ Transverse plane movement – Internal (+) / external (-)

Note the decreased range of motion in all three planes of motion in Patient B. In the sagittal plane there is loss of plantar flexion at terminal stance, indicating that the patient is using a hip strategy instead of ankle strategy during gait. Patient A has a normal range of motion in all three planes and continues to use the ankle strategy during gait.