Investigating the Effect of Simulated Diabetic Claw Toe Deformity on the Gait Patterns of Healthy Adults

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This thesis is the result of the author's original research. It has been composed by the author and has not been previously submitted for examination which has led to the award of a degree.'

Signed: Date:

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ABSTRACT

Background: In the UK, approximately 2.9 million people currently have diabetes mellitus (DM), with that number expected to reach 5 million by 2025. DM amputation and ulceration greatly impact NHS budgets and individuals' quality of life. Neuropathy, ischemia and high plantar pressures are reported as the main risk factors in development of ulceration, with the latter being increased in the presence of structural foot abnormalities, such as claw toe deformity (CTD). Areas of high pressure have been shown to correlate with common ulcer sites including MTPJs and hallux; however, little research has been carried out to identify the links between foot deformity, gait changes and ulcer development.

Research Rational: Numerous studies report the effect of DM on normal gait patterns. However, the multi-factorial aetiology of DM gait modifications makes the investigation of single factors problematic. By simulating CTD in healthy participants, gait changes owing to the deformity may be studied in isolation by removing the influence of concomitant DM complications such as neuropathy. Findings may inform future practice by enabling clinicians to better understand the pathways linking CTD, gait, and plantar pressures; hence assisting them to both assess and reduce ulcer risk in DM individuals, and to identify areas requiring further investigation.

Aims: To infer whether significant differences in spatiotemporal, kinetic and kinematic gait parameters occur in DM individuals with CTD when compared to healthy adults.

Methods: Design and verification (against current literature sources) of a device to simulate diabetic CTD was completed. Gait data was obtained at self selected walking speed using a Vicon 3D motion analysis system and Kistler force platform for 12 healthy participants wearing a CTD device. Participants acted as their own controls (by removing device). Statistical analysis of spatiotemporal, kinetic and kinematic gait parameter data collected from both groups was carried out.

ABBREVIATIONS

The following abbreviations are provided for terms which are used frequently within this thesis.

General Abbreviations:

AP	Anterio-posterior
CTD	Claw Toe Deformity
CTDSD	Claw Toe Deformity Simulating Device
DM	Diabetes Mellitus (Type 1 or Type 2)
DMN	Diabetes Mellitus with Neuropathy Present
DMU	Diabetes Mellitus with History of Ulceration
GRF	Ground Reaction Force
GRF_X	Anterio-posterior Component of the Ground Reaction Force
GRF_Z	Vertical Component of the Ground Reaction Force
IPP	Increased Plantar Pressure
PPP	Peak Plantar Pressure
MTH	Metatarsal head
MTPJ	Metatarsophalangeal joint
ROM	Range Of Motion

Gait Parameter Abbreviations:

FX1, FX2	Peak and Min. GRF_X
FZ1, FZ2	First and Second GRF_Z Peaks
AAX1, AAX2	Peak and Min. Ankle Angles
HAX1, HAX2	Peak and Min. Hip Angles
KAX1, KAX2	Peak and Min. Knee Angles
AMX1, AMX2	Peak and Min. Ankle Moments
HMX1, HMX2	Peak and Min. Hip moments
KMX1, KMX2	Peak and Min. Knee Moments

GLOSSARY

The list of definitions below is intended to assist the reader in understanding terms that may not be familiar to them (particularly regarding biological and medical terms), or the meaning of which may be ambiguous in current literature sources.

Anhydrosis – Abnormal dryness of the skin (often observed in DM individuals with neuropathy).

Atherosclerosis – A plaque which develops on the wall of a blood vessel so narrowing it and hence restricting the flow of blood

Cerebrovascular accident (CVA) – Also known as 'stroke'. Results from brain tissue damage due to a bleed or blocked vessel (e.g. thrombus, emboli) in an area of the brain.

Cardiovascular Disease (CVD) – Any pathology affecting the heart or blood vessels.

Concomitant Disease – A condition which exists alongside another in the same person, e.g. someone with diabetes may additionally have neuropathy, kidney and/or heart problems.

Contralateral – in terms of the lower limb, refers to the opposite limb, e.g. during single support, when one limb is in stance the contralateral limb is in swing.

Erythrocytes – The red blood cells, which bind to oxygen in the lungs before delivering it to tissues via the bloodstream

Foot Joints:

Metatarsophalangeal Joints (MTPJs) – The joints of the foot which connect the metatarsal and phalanx bones (Figure 0-1).

Interphalangeal Joints (IPJs) – The joints of the foot which connect the phalanges. N.B. There are two phalanges (proximal and distal) in the great toe and hence only one IPJs, while there are three phalanges in the remaining digits (proximal, intermediate and distal) and hence two IPJs (proximal and distal IPJs) (Figure 0-1).

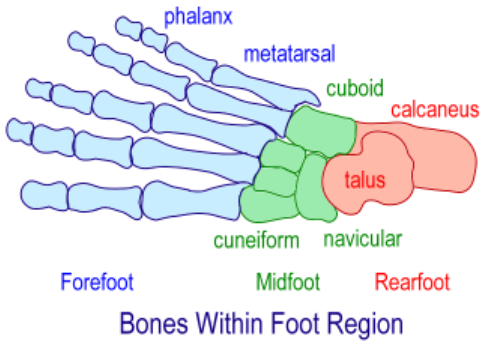

Figure 0-1 Bones constituting MTPJs and IPJs of the foot

Insensate foot – Where neuropathy is present in the lower limb causing the individual to lose their normal response to pain stimuli.

Intrinsic foot muscles – Muscles which have both their origin and insertion within the foot, This is opposed to large muscles such as gastrocnemius (calf muscle) which, although attaching to the calcaneus (see above diagram) via the Achilles tendon, has its origin in the femur (making it an **extrinsic foot muscle).**

Ischemia – Occurs when tissues are starved of oxygen, often due to a proximal blockage (atherosclerosis, thrombus, emboli) of the vessels which supply that tissue.

Ischaemic Heart Disease (IHD) – Most often caused by atherosclerosis of the vessels of the heart, and can lead to damage to the heart muscle wall (myocardial infarction) and angina.

Muscle Atrophy (Muscle Wasting) – Occurs when the muscles lose mass, due to e.g. nerve abnormality or lack of use, and therefore are weaker.

Neuropathy – Nerve damage leading to loss of nerve signals, which in diabetes is most commonly noted in the feet and hands. May cause loss of pain/hot-cold/sharp sensation (**sensory**); **motor** function (i.e. musculoskeletal control); or **autonomic** control (i.e. automatic responses such as sweating or control of blood vessel diameter).

Nephropathy – Damage to the functional segments of the kidney due to altered blood supply.

Orthostatic Intolerance - Dizziness, light-headedness etc. brought on by a sudden drop in blood pressure when standing from seated or inclined position.

Osteolysis – Degradation of bone tissue.

Peak Pressure - The highest pressure value recorded by a single sensor (e.g. F-scan sensel) during the stance phase of gait for a specific anatomical region of the foot.

Pedal – In relation to the foot e.g. pedal pulses.

Peripheral Vascular Disease (PVD) – Pathology occurring in blood vessels, other than those of the heart, and usually initially detected in the extremities such as feet and hands.

Plantar – The lower surface of the foot, i.e. surface which contacts the ground during gait.

Plantar Pressure – The amount of pressure experienced by the plantar surface which is dependent upon the force due to body mass, gravity, and acceleration of the centre of mass (i.e. Force = mass x gravity + mass x acceleration), and the area of the foot which is in contact with the ground (i.e. Pressure = Force/Area).

Retinopathy – Damage to tissue within the back of the eye due to changes in circulation.

Three Functional 'Rockers' of Gait – During stance phase sagittal plane progression can be attributed to three 'rocker' phases: the first rocker occurs after initial contact as the foot rotates about the heel into foot flat; the next phase (second rocker) describes the mid stance period where the body centre of mass (COM) moves over the straight stance leg by pivoting about the ankle; with the third rocker taking place due to forward momentum of the COM coupled with contraction of the calf and foot intrinsic muscles, resulting in heel lift about an MTH fulcrum, accompanied by tibial rotation (Perry 1992).

Triplanar motion – Motion which occurs in three planes, these being: *the **sagittal plane**, which divides the body in two (left and right) halves; the **frontal plane**, which divides the body into anterior*

*and posterior portions; and the **transverse plane**, which splits the body into upper and lower segment:.*

Abduction/adduction – Movement in the transverse plane, e.g. out-toeing or in-toeing of the foot.

Inversion/eversion - Movement in the frontal plane, e.g. moving the foot so that the plantar surface faces towards or away from the other limb.

Flexion/extension and dorsiflexion/plantarflexion – Movement in the sagittal plane e.g. bending or straightening the knee or hip (in the ankle only, dorsiflexion refers to the top of the foot being pulled closer to the shin, while plantarflexion is the opposite).

Ulcer – Failure of a damaged tissue to heal within six weeks. In terms of DM foot disease this is usually a wound on the foot caused by skin trauma from e.g. footwear, increased plantar pressure or poor circulation.

Vasomotor Changes - inappropriate blood vessel dilation or constriction.

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CHAPTER 1 INTRODUCTION

The number of individuals with diabetes mellitus (DM) is increasing rapidly, largely due to factors such as obesity, lack of exercise and poor diet. As the prevalence of DM escalates so do the psychological, financial and healthcare demands placed on the patient and their care providers, including the NHS (Diabetes UK, 2006).

Poor glucose control and failure to make recommended life changes (e.g. smoking cessation and diet habits), often in combination with concomitant disease, can lead to DM progression and ultimately complications. The risk of developing neuropathy, cardiovascular disease, kidney dysfunction, eye disease, ulceration, infection and amputation are all increased in individuals with DM.

Of all DM complications, ulceration and amputation probably have the greatest impact on both a person's quality of life, and the level of subsequent care they require; with the cost of the latter being borne mainly by the tax-payer. In personal terms, a significant number of patients with ulceration will go on to require amputation, and within a short period, the inevitable outcome for many of these individuals will be an early death. In addition to the physical difficulties faced by people who develop ulceration or experience amputation, the psychological impact is often even more striking, with depression and isolation being common features (Diabetes UK, 2006).

Possible pathways to ulceration have been frequently investigated (Figure 2-1); however, aetiology in relation to DM appears to be multi-factorial, making prevention, treatment and risk reduction complex. Foot ulcer formation has been previously associated with circulatory deficiency, neuropathy (nerve damage leading to e.g. loss of sensation), callus formation, poor footwear, changes in the material properties of the skin, reduced immune function and increased plantar pressure, to name but a few (Boulton 2006).

In the DM population, precursors of ulceration are often difficult to isolate due to the presence of a number of coexisting complications, which may all, to differing degrees, have an impact on ulcer development. For this reason determining the risk associated with each complication, and hence selecting the best treatment route, becomes much more challenging. If assessment of individual factors could be achieved then practitioners would be better equipped to target resources and time and would ultimately be more effective in lowering ulceration rate.

Each year around 10% of the annual NHS budget is spent on DM care, including treatment of ulcers and vascular complications relating to DM (Diabetes UK 2011). Although figures have been reported (Hex, et al. 2012) regarding 2010/11 expenditure on complications as diverse as erectile dysfunction (over £ 13mn), kidney failure (around £ 514mn), and ulceration/amputation (over £985.6mn), no funds to address deficits in terms of off-loading, biomechanics or gait pattern were listed. This seems illogical since one of the major pathways to ulceration frequently considered within the literature involves increased plantar pressures (Birk, et al. 1991; Boulton 2006).

Studies suggest that higher pressures, over a period of time, may lead to soft-tissue damage and eventually skin breakdown (Armstrong, et al. 1998). A number of causes have been suggested for the increased plantar pressures (IPPs) observed in people with DM (Bus, et al. 2005; Gefen 2003). Presence of foot deformity has been found to correlate with both IPP and sites of ulceration (Bus, et al. 2005). However, further investigation is required to determine the specific role of foot deformity in altering peak plantar pressure (PPP) patterns in DM patients.

In addition to influencing plantar pressure (Bryant, et al. 1999; Mickle, et al. 2011), hyperkeratotic lesion development (Spink, et al. 2009) and ulceration, foot deformity has also been investigated in relation to altered gait patterns in both older people (Mickle, et al. 2011) and individuals with rheumatoid arthritis (Turner and Woodburn 2008). Gait modifications, including slower gait and shorter stride length (Allet, et al. 2010) have been attributed to various factors, and a number of

studies have considered the pathological processes underlying these (e.g. muscle dysfunction) (Savelberg 2010). However, to the author's knowledge there has been no research thus far which has examined gait modifications as a direct result of foot deformity in DM individuals. This is a highly relevant area for research since -as previously alluded to- altered foot biomechanics, PPP and gait have been frequently associated with ulcer formation. Consequently, establishing direct associations between foot deformity and gait pattern changes may provide further information regarding the complex pathways from DM foot disease to the development of potentially life threatening ulceration.

Previous studies in DM individuals have failed to separate the effects of specific factors from those of concurrent complications. For example, in the DM foot, assessment of the effects of foot deformity may be undertaken where a combination of neuropathy, altered range of motion (ROM), fibro-fatty pad migration or callus, to name but a few, may also be present. In such a situation it is nearly impossible to quantify the contribution from individual factors, and as a consequence any treatment or assessment decision based on these results is not a fully informed one.

The scope of current DM research is vast, and although many studies have explored a variety of complications, significant gaps in the literature still remain. Much is understood about the aetiology of diabetic neuropathy (DMN), vascular pathology and diabetic ulceration (DMU) (Tesfaye 2006); however, the inter-related nature of the underlying pathological processes, make it extremely difficult to draw firm conclusions from studies involving DM participants. Although the effects of numerous complications have been evidenced to some degree, in many cases a direct correlation between cause and effect is difficult to prove conclusively.

The main purpose of this thesis is therefore to use a claw toe deformity simulation device (CTDSD) in healthy adults to infer whether the presence of anatomical claw toe deformity (CTD) is likely to result in any significant changes in spatiotemporal, kinematic and kinetic gait parameters in DM individuals.

It is not within the scope of this thesis to describe and evidence the relationships which interlink the vast range of DM disease processes, and indeed many of these are not yet fully understood by the wider scientific community. However, this thesis does attempt to explain, in simple terms, some of the pathways which are thought to lead to the formation of ulcers (Figure 2-1) and explores the role that foot deformity may play in both increasing PPP and bringing about gait pattern alterations.

The layout is structured as follows: the background and literature review (Chapter 2) will set the scene in terms of the effects which DM has on the lower limb (complications, IPP, gait changes) and the relevance which these have to ulcer risk. This Chapter also introduces claw toe deformity and current research into its aetiology and effects. Project aims will be briefly stated In Chapter 3, while Chapter 4 discusses the experimental design. The studies themselves are then described in the two subsequent Chapters (preliminary validation, Chapter 5; main gait parameter study, Chapter 6) which will further detail the methodology, results, discussion and limitations relating to each of the two studies. Conclusions will then be provided in Chapter 7, along with a discussion of areas recommended for future research.

CHAPTER 2 BACKGROUND AND LITERATURE REVIEW

This Chapter is intended to provide an introduction to diabetes mellitus (DM) and its complications, with particular reference to lower limb pathologies arising due to DM that may result in abnormal gait patterns, or increased plantar pressures (IPPs). The importance of DM within the context of ulcer formation and its subsequent effects on both the individual, and health care providers such as the NHS, is highlighted. A number of relevant foot and lower limb abnormalities attributed to DM are then examined, and their aetiologies are briefly explained.

The introductory section is followed by a discussion of the specific effects of DM on both gait and plantar pressure, as reported in current literature. A number of theories are proposed for the gait/pressure pattern alterations observed in individuals with DM, while current gaps in the literature are identified.

Finally, claw toe deformity (CTD) is defined, and suggestions for its aetiology are provided followed by a summary of the findings of recent research papers investigating the influence of CTD on gait patterns and plantar pressure.

2.1 CLINICAL BACKGROUND

2.1.1 Introduction to Diabetes: Prevalence, Aetiology and Complications

Diabetes mellitus (DM) is an accelerating problem worldwide, and is one of the greatest health care challenges for the NHS today. In the UK, approximately 2.9 million people currently have the condition, with that number expected to reach 5 million by 2025. These figures correspond to around 17 new diagnoses every hour (Diabetes UK 2011).

DM is a metabolic condition in which the production of insulin (a hormone which controls blood glucose) is either reduced (type 2) or prevented (type 1). Most new diagnoses are in type 2 individuals, and are largely due to lifestyle choices and

obesity. The risk of developing DM is related to a variety of factors including genetics, family history, ethnicity and bodyweight. Individuals from deprived backgrounds are much more likely to develop DM due to e.g. poor diet, smoking, reduced physical exercise, higher blood pressure and obesity (Diabetes UK 2011).

A number of complications are commonly observed in people with DM. The prevalence and impact of these vary greatly between individuals, and will depend upon factors such as glucose control, lifestyle and DM duration. Frequently reported pathologies observed in DM include micro-vascular (retinopathy, nephropathy) and macro-vascular complications (cerebrovascular accident (CVA), peripheral vascular disease (PVD), coronary artery disease (CAD)), and neurological disorders (autonomic, motor, sensory dysfunction) (Tesfaye 2006). Neuropathy and cardiovascular disease (CVD) will be discussed in more detail in later sections (2.1.3.1 and 2.1.3.2); however, a number of further, lesser-known deficiencies are also known to occur, which, although of a more minor nature, can still have a noteworthy effect on the feet and lower limbs, and may therefore influence gait and plantar pressure. A number of these are discussed below.

2.1.1.1 Tissue Alterations

Soft-tissue

Soft-tissue modifications, brought about by collagen cross-linking, are a commonly occurring consequence of DM, and may result in various changes including skin and tendon stiffening, reduced ROM and callus formation (Gefen 2003; Stanley and Collier 2008).

Callus, or Hyperkeratosis, describes a hardened plaque of skin, most often occurring in areas of high loading such as the plantar surface of the foot, or more distally on the digits where shear may take place at the skin-shoe interface. Although ulceration may be observed at sites of previous callus formation this is reported to be unlikely to happen in individuals who have normal pedal sensation (Minor, et al. 1990).

Bone

Studies regarding the influence of DM on tissue material properties have largely concentrated on connective and deep plantar tissues. Although osseous changes have been investigated in a number of animal studies, there exists limited quantitative evidence regarding alterations as a result of DM in humans. A report by Fleiscli, et al. (1998), which investigated the properties of metatarsal bone, demonstrated that changes occurring as a result of DM were comparable to the effects of normal bone ageing.

It has been suggested that demineralisation (possibly caused by metabolic dysfunction or altered calcium levels), increased glucose concentration, and decreased collagen synthesis, may also contribute to osteolysis and therefore impede fracture healing in DM patients (Fleiscli, et al. 1998). In a study exploring plantar pressure in diabetic individuals with neuropathy (DMN) and either lateral or medial foot deformity, it was noted that calcaneal bone density was reduced in this group when compared to healthy controls, and that mineral density in the medial column of the foot was lower than that found laterally (Sinacore, et al. 2008).

2.1.1.2 Blood Component Alterations

In addition to tissue alterations, a number of abnormalities attributed to the presence of DM, may also be observed in blood components. With regard to erythrocytes, these include reduced oxygen release brought about by abnormal red blood cell deformation, and changes in blood flow dynamics, particularly in small diameter vessels. A decrease in the capability of white blood cells (e.g. neutrophils or monocytes) to carry out phagocytosis will also dampen the bodies normal, non-specific defence mechanisms against invading organisms. In addition, abnormal platelet function may impede post-injury clotting (Lorimer, et al. 2002; Campbell 1993).

2.1.1.3 Microcirculation

Further to the abnormalities which arise in blood components, changes in the vessels which carry the blood are also commonly noted in DM, with small diameter vessels being particularly vulnerable to damage.

Ocular

Both cataract formation and retinopathy may occur in DM, with the latter resulting from damage to the small vessels of the eye, and the former probably via repeated osmotic damage to the lens (Lorimer, et al. 2002). Within the working age population, DM is the greatest cause of loss of vision in the UK (Diabetes UK 2011). Not only do these DM complications greatly affect an individual's quality of life, but they also significantly reduce a person's ability to monitor, and hence care for, their own feet.

Renal

Nephropathy occurs when the capillary network that delivers molecules from the blood to the kidney's functional unit (the nephron) is damaged, and progression to renal failure may result if the damage is such that filtration of blood is significantly affected (Campbell 1993; Tortora 2004). DM is the most common cause of end stage renal failure, with approximately one in every three people diagnosed with type 2 DM developing kidney disease (Diabetes UK 2011). Kidney dysfunction may result in oedema, a greater risk of blood vessel calcification, and necrosis of tissue at the extremities (e.g. the digits); particularly where a kidney transplant has been performed (Lorimer, et al. 2002).

2.1.1.4 Immunological and Wound Healing Function

As discussed above, pathological changes due to DM have been observed in soft-tissues (including skin), microvasculature and blood constituents. The altered function of these tissue components will obviously have adverse consequences for

both the immune and healing response, since each is closely involved in the body's non-specific and specific lines of defence against foreign materials (Campbell 1993).

A reduction in the oxygen and nutrients available to tissues, and changes in vasomotor control due to nerve pathology, in addition to the glycosylation of tissue proteins, together make the skin more susceptible to breakdown and hence reduce its capacity for healing (Lorimer, et al. 2002). Furthermore, once the skin has been breached, people with DM are more at risk of developing infection, since poor tissue perfusion restricts the delivery of repair and immune components to the site of injury, and the removal of pathogens is hindered by inefficient white blood cell phagocytosis.

2.1.1.5 Section Summary

The abovementioned DM pathologies and modifications provide some indication of the scope of problems faced when treating, and attempting to limit, the advancement of DM complications. Although some of these changes may seem relatively minor, when acting in combination they can produce complex patterns of dysfunction, the specific pathophysiology of which is poorly understood.

The diverse spectrum of complications observed in DM, and the relationship of each to ulcer formation is outlined in Figure 2-1. Linkages will be revisited frequently in later sections (particularly with respect to the influence of various lower limb pathologies on gait patterns and plantar pressure) and hence it is recommended that the reader regularly review this flowchart as they navigate through the thesis. By doing this it is hoped they will gain a better understanding of the combined contribution of individual, often minor, alterations, to the overall, medically significant, patterns of change that can arise due to DM.

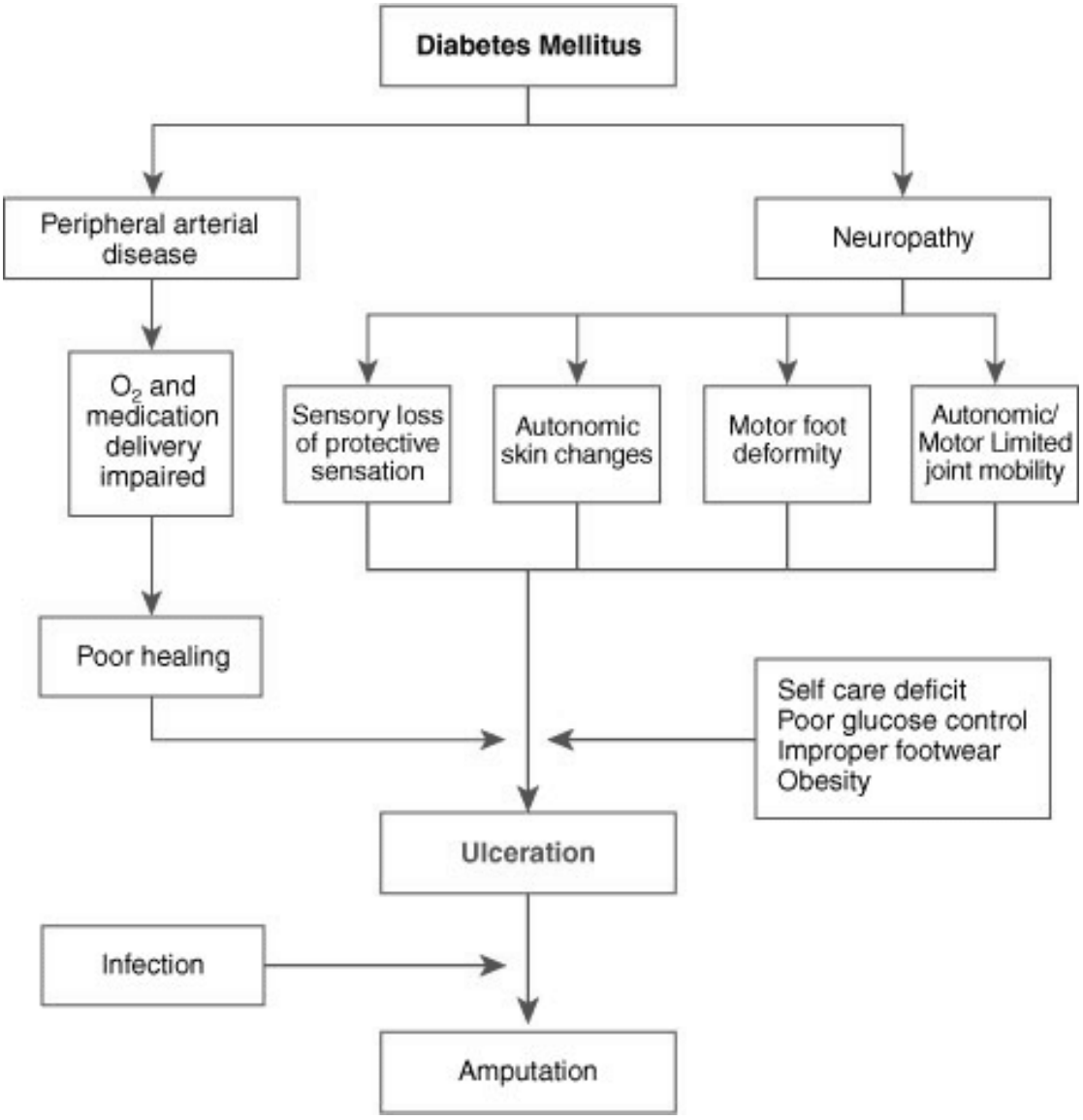


Figure 2-1 Linkages between the variety of complications frequently observed in individuals with diabetes and possible pathways to ulceration and amputation in the diabetic foot.

2.1.2 Ulceration and Amputation

Of all the DM complications observed, amputation and ulceration will have the greatest long-term impact on both health care providers (such as the NHS) and the quality of life of the individuals who ultimately develop them.

Annually, around 5% of people with DM will develop ulceration, with at least 10% of all ulcers resulting in a foot or lower limb amputation. DM is recognised as the main predisposing factor in lower limb amputation and, on an international scale, its presence increases the risk of amputation by around 15% (Diabetes UK 2011).

In addition to the obvious physical restrictions experienced by individuals with ulceration and amputation, the psychological factors are often just as debilitating. Twice as many people with DM will face depression when compared to their non-DM counterparts (Diabetes UK 2011). And in 2010/11 over £33mn was spent on treating depression in the DM population (Hex, et al. 2012). Odour, pain, shoe fitting difficulties, loss of mobility and the stigma associated with ulceration may prevent individuals from taking part in normal daily and social activities. Whilst amputation may reduce a person's ability to ambulate or function normally in certain environments, and hence exclude them from continuing with previous hobbies or occupations, so significantly reducing their quality of life and feelings of societal worth.

In addition to the considerable personal impact of ulceration and amputation, costs to the taxpayer in terms of factors such as staff working hours (treatment, monitoring, rehabilitation) and training; resources (prosthetics, wound dressings, mobility aides); management of complications; surgical procedures; and long-term care arrangements (hospital, community care, care homes), are vast, and will continue to increase in line with rising DM prevalence.

The cost of diagnosis alone exceeded £9.6mn in 2010/11 (Hex et al., 2012). And in England in 2010 around £713mn worth of prescriptions were filled in relation to DM (Diabetes UK, 2011). It has been reported that around 10% of the annual NHS budget is spent on DM care (Diabetes UK, 2011). Findings of the Diabetes Impact report (Hex et al., 2012), which investigated DM expenditure in the UK for the year 2010/2011, estimated that direct patient care costs were £9.8bn, with indirect costs such as death and sickness absence being in excess of £13bn. Reported costs involved in the treatment of DM complications included £4mn for gestational DM;

£509.6mn on ischaemic heart disease; £514mn regarding kidney failure; £309.6mn relating to neuropathy; and £985.6mn for the management of amputation and ulceration.

Striking, was the absence of any reference to biomechanical assessment or offloading intervention, or even to foot deformity as a complication. Although these factors may have been included elsewhere under an umbrella heading (e.g. 'foot ulcer and amputation'), the fact that they have not been documented in their own right highlights the failure of current healthcare professionals to recognise the important roles that musculoskeletal abnormalities and foot deformities play in altering plantar pressure and gait patterns, and ultimately in the formation of ulcers.

2.1.3 Pathways to Ulceration

Foot and leg ulcers are chronic wounds occurring below knee level which have failed to heal within 6 weeks. They result where there is a disruption to the healing pathway, which prevents progression through the normal phases of wound healing (Bradley, et al. 2007).

There are a great number of factors reported to increase the risk of developing DM foot ulceration, which include diabetic neuropathy (DMN) (sensory-motor, motor and autonomic neuropathy), peripheral vascular disease (PVD), reduced joint range of motion (ROM), increased plantar pressure (IPP), poor glycaemic control, age, DM duration, ethnicity, gender, presence of foot callus or deformity, and previous history of ulceration or amputation (Caselli, et al. 2002; Birk, et al. 1991; Bofelli, et al. 2002; Cowley, et al. 2008; Laing 1998; Ledoux, et al. 2005; Boulton 2006; Merza and Tesfaye 2003). However, DMN, PVD and IPP are generally cited as the most influential causative processes (Ledoux 2008).

The pathogenesis of ulceration involves the complex interplay between a number of underlying processes; however, authors generally agree that skin breakdown results from a combination of the aforementioned risk factors, with any single cause

being inadequate to bring about ulcer formation (Boulton 2006). For example, one pathway to ulceration could include DMN in combination with either a minor wound (e.g. a stone in the shoe causing skin abrasion) or the presence of deformity resulting in IPP, which over time, may result in skin damage. In addition, the presence of PVD will interfere with normal healing so predisposing the individual to chronic ulceration.

The three most commonly reported risk factors; namely, DMN, PVD and IPP, are discussed in more detail below:

2.1.3.1 Neuropathy

According to Tortora and Grabowski (2004) neuropathy is "any disorder that affects the nervous system, but particularly a disorder of a cranial or spinal nerve." In relation to DMN it generally refers to a combination of nerve syndromes which affect the peripheral nerves of the hands and feet (so called stocking and glove distribution), and which are greatly influenced by blood glucose levels (Tesfaye 2006). Nerve damage may lead to a number of impairments including reduced vibration sensation, loss of sensitivity to touch or temperature, or decreased awareness of the body's position (proprioception) or motion (kinaesthesia) in space (Yavuzer, et al. 2006).

DMN is a frequently reported symptom of DM, with prevalence suggested to be around 66% and 59% in type 1 and 2 DM respectively (Low, et al. 2004), or 20-50% of the total DM population (Yavuzer, et al. 2006). DMN generally begins in the sensory nerves, before progressing to the motor nerves where it may result in muscle atrophy and subsequent loss of function (Yavuzer, et al. 2006). Neuropathy may be classified according to the type of nerve damage, and may include: acute painful neuropathies, the more chronic symmetrical sensory and autonomic neuropathies, mononeuropathies and impingement neuropathies such as tarsal tunnel entrapment (Lorimer, et al. 2002).

The commonest form (90%) of all DMN is distal sensorimotor symmetrical neuropathy, the symptoms of which can be more simply broken down into autonomic, sensory and motor effects.

Autonomic Neuropathy

The autonomic nervous system is the part of the nervous system that is under subconscious control, and which manages the function of structures such as sebaceous glands and blood vessel walls. It therefore follows that the autonomic symptoms most commonly affecting the feet and lower limbs include anhydrosis and poor vasomotor control (Lorimer, et al. 2002).

In a study by Low, et al. (2004) the severity of autonomic symptoms was found to be significantly higher in DM participants when compared to age matched controls; with a prevalence of 54% in type 1 and 73% in type 2 DM. Symptoms included: orthostatic intolerance; gastrointestinal, urinary and sleep disorders; erectile dysfunction and vasomotor changes.

Sensory neuropathy

Sensation incorporates proprioception as well as perception of temperature, light touch, vibration, pain, and two-point discrimination (Merriman 2002). Assessment of sensory neuropathy in four groups of white males (control, DM, DMN, and DM with a history of ulceration (DMU)) demonstrated significantly higher dysfunction in C-fibres (temperature sensation) and Aβ-fibres (cutaneous vibration/ pressure perception) in DMN subjects when compared with DM and control participants (van Schie, et al. 2004). Reduced stimulus perception can leave DMN patients vulnerable to skin breakdown, since an absent pain response may mean they fail to react to an object, friction or pressure occurring e.g. within footwear.

Presence of sensory neuropathy can also produce a wide range of additional symptoms such as paraesthesia, burning, shooting pains, numbness or “walking on stones”, depending upon the nerve fibres affected (Tesfaye 2006).

Motor neuropathy

The main symptom of motor neuropathy in DM individuals is atrophy of intrinsic muscles in the hands and feet. A number of previous studies have suggested that foot deformity may arise from motor effects on muscles, although some recent reports have questioned a direct link. These areas will be discussed in more detail in section 2.4 in relation to CTD.

Diabetic neuropathic osteoarthropathy (Charcot foot)

Charcot foot is a condition affecting the bones and joints of the foot, and may result in fracture, dislocation or permanent deformity. Its aetiopathogenesis is not fully understood although a number of theories currently exist including nerve dysfunction leading to reduced bone nutrition or repetitive osseous trauma in the presence of an insensate foot (Tesfaye 2006). If offloading is not initiated quickly in the disease process, Charcot foot can result in severe, permanent midfoot deformity, which will have a major impact on both plantar forces and ambulation.

Although DMN is the primary risk factor for ulceration (Boulton 2006) it does not generally work in isolation. Indeed, alone it is unlikely to cause ulceration; however, what it may do is mask the symptoms of underlying tissue damage, and therefore delay treatment that would otherwise prevent the development of more chronic disease processes. Cardiovascular disease (CVD) is one of the key pathological processes which act in conjunction with DMN to bring about tissue damage, and it is explored further below.

2.1.3.2 Peripheral Vascular Disease

Cardiovascular disease (including stroke (CVA), ischaemic heart disease (IHD), atherosclerosis and peripheral vascular disease (PVD)) is a major DM complication, which results in 52% of all deaths in people with type 2 DM (Diabetes UK 2011).

Microvascular changes observed in DM result largely from the effects of hyperglycaemia on the vessel walls, which acts to limit the effective exchange of

oxygen and other molecules (e.g. nutrients) between the blood and tissues. This in turn leads to tissue and muscular changes (atrophy, necrosis), nerve damage (i.e. DMN), and a reduced capacity for healing (Stanley and Collier 2008).

DM has also been reported to increase the susceptibility of vessels to atherosclerosis; particularly in the peripheral circulation (Lorimer, et al. 2002). Historically it was suggested that the proliferation of endothelial cells observed in DM resulted in microcirculation occlusion, and therefore played a part in ulcer formation, even where pulses appeared normal. More recently the haemodynamic hypothesis has proposed that significant increases in capillary blood flow and pressure (noted in the first stages of DM) may result in a damage response in endothelial cells that brings about the formation of sclerotic plaques and leads to basement membrane thickening (microangiopathy) within vessel walls. These blood pressure dependent changes are thought to impair vasodilation and other vascular functions in the lower limb and foot (Krishnan and Rayman 2006).

Further exploration into the impact of DM on the cardiovascular system has indicated that vascular pathophysiology involves a number of microcirculation modifications, which may include: an impaired vasodilation response (i.e. to heat or injury), altered arteriovenous shunting (resulting in reduced nutrition), and loss of the normal vasoconstriction reaction to postural increases in blood pressure. The aforementioned alterations are all due to sympathetic denervation; however, a reduction in the normal hyperaemia response following vessel occlusion (resulting in lowered tissue oxygen perfusion and diminished capillary and arteriolar dilatory response to injury) may also play a part (Krishnan and Rayman 2006).

The abovementioned changes may therefore contribute not only to a reduction in the ability of vessels to provide oxygen and nutrients to tissues, but also to the individuals capacity for wound healing, and hence their susceptibility to ulceration. Although PVD has been reported as a major risk factor for ulceration, generally its effects only become significant when a higher demand for oxygen (e.g. as a result of

injury or infection) means that vessels can no longer provide the levels required for healing (Krishnan and Rayman 2006).

The processes discussed above highlight the importance of minimising any tissue stresses which may subsequently lead to initial skin breakdown. For that reason it is essential to consider factors that may bring about increased plantar foot pressures, in order that the effects of these can be limited.

2.1.3.3 Abnormal Plantar Pressures

Increased plantar pressure (IPP) in DM individuals has previously been attributed to factors such as structural variation (Morag and Cavanagh 1999); DMN (Caselli, et al. 2002); biomechanical abnormalities and reduced joint ROM (Birk, et al. 1991); plantar tissue thickness and presence of hyperkeratosis (Abouaesha, et al. 2001); body mass and foot deformity (Menz, et al. 2007) and altered gait dynamics, including walking speed (Cavanagh, et al. 1997). Since foot deformity and gait pattern modifications are the main focus of this thesis they will accordingly be discussed in some detail in sections 2.2 and 2.4.

In DM, normal activity in the presence of DMN may result in a foot which is subjected to IPP and which is therefore susceptible to ulcer formation (Armstrong, et al. 1998). Whereas healthy individuals are likely to respond to foot pressure or pain by removing the pain stimulus, changing footwear, or altering their foot position or gait pattern, DM individuals may lack the facility to perceive pain, and will therefore fail to respond appropriately. However, sensory neuropathy in itself is not an absolute precursor to ulceration; a fact that is supported by the absence of foot pathology in many individuals who experience non-diabetic loss of foot sensation (Pinzur 2008). It seems likely then, that the motor and autonomic changes also associated with DMN must have some influence on skin breakdown. As such, it should be no surprise that both hyperkeratotic lesions and foot deformities (reported to be brought about by autonomic and motor nerve

pathology respectively) have been shown to correlate with both IPP and ulcer formation (Menz, et al. 2007; Bus, et al. 2005).

Tissue stress resulting in ulceration may occur by three separate methods. IPP continuously acting over a few hours duration may damage tissues by preventing normal perfusion through superficial capillaries. Alternatively, acute focussed pressure, resulting from e.g. a drawing pin or stone, will bring about an instantaneous tissue injury that may not be immediately noticed. In contrast, frequent repetitive loading at levels that would otherwise be non-pathological may, over a period of days or months, provoke an inflammatory reaction that eventually will lead to ulceration (Pinzur 2008). The latter process is the most probable cause of ulcer formation in the insensate DMN foot and has been previously investigated by applying repeated pressure to the footpads of rats. Animal subjects developed ulceration within 10 days under conditions that simulated a daily 7 mile walk, with ulcers arising more quickly when the feet were denervated (Laing 1998).

Even in a normal static standing position it has been calculated that around 399kN/m^2 of pressure may be transferred through the metatarsophalangeal joints (MTPJs) via ground reaction forces (GRFs) (Stanley and Collier 2008). Although skin is very strong and can cope with pressures of around 9.81MN/m^2 before rupturing (Laing 1998), plantar pressures which exceed normal values have been found to strongly correlate with common ulcer sites, including the first metatarsal head (MTH), lesser MTHs and the hallux (Armstrong, et al. 1998). The cut-off magnitude of pressure deemed by Armstrong and colleagues (1998) to best estimate ulcer risk was calculated to be about 700kN/m^2.

Kanade and associates (2006) found that the severity of foot complications (DMN, DMU, partial foot amputation or trans-tibial amputation) present in DM subjects correlated with an escalation in maximum peak pressure (MPP). Whole foot MPPs were found to be significantly higher in partial foot amputation, DMU and DMU contralateral foot groups when compared to DMN only participants. Studies have also shown IPP to be present in individuals with DMN. For example, Ledoux and

colleagues (2005) observed IPPs in the first and third MTPJs and the hallux, while Armstrong, and associates (1998(b)) found plantar pressures to be highest in the lesser MTPJs (i.e. first to fourth) and the hallux. Caselli, et al. (2002) carried out a prospective study in 248 DM individuals with varying severity of DMN. Dynamic PPPs were recorded for the rearfoot and forefoot regions, and both forefoot PPPs and forefoot/rearfoot ratios were found to relate to risk of ulcer development.

It seems logical to conclude that in order to reduce the incidence of ulceration within the DM population it is essential to accomplish a fuller understanding of the factors which may lead to increased foot pressure. These include the specific effects of CTD in relation to gait parameter alterations, which will be discussed in more detail in section 2.4.4.

2.1.4 Clinical Background Section Summary

A varied combination of pathological processes may be observed in DM, all of which can play some part in heightening the risk of ulcer formation. For example, motor nerve dysfunction leading to toe deformity can create sites of IPP, and may ultimately lead to an increased risk of skin trauma. This may be further aggravated by poor skin nutrition (and hence ineffective healing), autonomic and structural tissues changes, and inappropriate footwear made tighter by the presence of oedema (Stanley and Collier 2008).

The vast number of pathological processes occurring within the DM foot cannot be covered within this report; however, the following sections (2.2, 2.3 and 2.4) will discuss the specific effects of DM with reference to plantar pressure and gait, and will further clarify the relationship of CTD, both to the aforementioned parameters, and to ulcer pathogenesis.

2.2 EFFECT OF DIABETES ON GAIT PATTERN

2.2.1 Introduction

Reported DM gait pattern alterations include reduced cadence, shorter step length, increased base of gait, and a longer period spent in stance and double support (Brach, et al. 2008). These modifications have been attributed to a range of abnormalities such as neurological dysfunction (Brach, et al. 2008), muscular weakness (Savelberg 2010), modified muscle activity (Sawacha, et al. 2011), foot complications (Kanade, et al. 2006), and loss of balance due to altered proprioception (Allet, et al. 2010).

Gait pattern modifications considered in this Chapter will be divided into the following areas: spatiotemporal factors, which refer to gait parameters that alter over space and time (e.g. gait velocity, stride length or support duration); kinetic parameters, which pertain to measures such as the moments, forces and inertial forces, that are involved in producing movement (e.g. GRF, joint moments) and kinematic measures that describe movement without reference to the causative forces (e.g. joint angles). The final segment will explore some of the underlying pathological processes that have been proposed to play a part in producing the gait modifications observed in people with DM.

2.2.2 Spatiotemporal Parameters

Spatiotemporal gait parameters have been widely studied in the DM population, although, experimental procedures and subject characteristics are wide ranging, making it difficult to identify conclusive patterns of change. DMN participants were shown by Courtemanche and colleagues (1996) to exhibit a more cautious and stable gait pattern compared to control subjects, including a longer double support phase and reduced gait cycle amplitude and velocity. Results were attributed to loss of sensory control of gait (specifically proprioception), which consequently required a higher cognitive effort by subjects. Authors suggested that this may lead to an increased fall risk in DMN individuals, particularly on uneven terrain. Similar

observations were obtained by Richardson and associates (2004) who revealed that DMN gait was slower, step time and step width-step length ratio were increased, and step length was reduced when compared to gait in healthy controls. In subsequent trials on uneven surfaces in low lighting conditions, it was noted that gait character modifications were more exaggerated in controls than in DMN subjects (with the exception of gait velocity). A study by Shaw, et al. (1998) explored varying degrees of DMN, and discovered that as severity increased, individuals walked at a correspondingly slower pace. Brach and colleagues (2008) suggested that the change in step width observed in DM patients was due to either motor neuropathy or the systemic effects of DM. These findings appear to confirm that physiological changes associated with DMN are closely linked to the resulting altered gait patterns in this group.

However, gait modifications have also been reported to occur in DM individuals without symptomatic DMN. Petrofsky, et al. (2005(a)) conducted a study which compared gait characteristics in non-neuropathic DM subjects and healthy adults. The DM group were found to exhibit slower gait, wider swing width (defined as the furthest combined distance that the feet are displaced laterally from the spine during swing), smaller stride length, and wider stance, for both straight walks and during turns. And in a recent investigation by Sawacha and colleagues (2009), changes in gait were again observed in the absence of neuropathic symptoms. Authors measured gait velocity, stride length and stance duration and noted that these varied significantly between controls and both DM and DMN groups.

Contrastingly, Yavuzer, et al. (2006) found that although gait speed and step length were reduced in DMN individuals, no significant changes were noted in either control or DM participants. Other spatiotemporal gait characteristics have been found to differ significantly between DMN and control groups. For example, single (right foot) and double support periods were both observed to be higher in DMN subjects; however, as with the previous study, no significant differences were observed in DM versus control groups (Sacco and Amadio 2000).

A further study by Petrofsky, et al. (2005(b)) demonstrated that gait pattern modifications (in this case reduced velocity, wider stance and conservative gait pattern) may be reversed by administration of *rosiglitazone* - a drug that increases an individual's responsiveness to insulin. This would appear to further substantiate a direct link between the physiological and functional effects of DM, and changes in spatiotemporal parameters.

2.2.3 Kinematic Parameters

2.2.3.1 Joint Angles

Savelberg and associates (2010) observed, in DM participants walking at an imposed velocity, that the ROM of the knee was reduced during stance phase, knee joint flexion angle was decreased and, prior to toe-off, the ankle was more plantarflexed, when compared to a self selected gait speed. No differences were noted in control participants when velocity was altered. In a gait deviation study by Yavuzer, et al. (2006), a significant reduction in sagittal plane knee and ankle mobility was observed in DMN (but not DM) subjects when compared to controls.

Sawacha and colleagues (2009) noted changes in gait in the absence of clinical symptoms of DMN. Authors investigated foot morphology (foot type, deformity and lesions) and gait characteristics during walking, in control, DM and DMN individuals. A number of alterations in joint rotation angle were noted between the two DM groups and controls during each phase of gait. Phases were described as: initial contact (IC), loading response (LR), midstance (MS), terminal stance (TS), pre-swing (PSW), initial swing (ISW), midswing (MSW) and terminal swing (TSW), and joints investigated were the trunk, hip, knee and ankle. The most significant differences ($p<0.01$) included trunk abduction-adduction at MS and PSW for the DM group; trunk internal-external rotation at LR, MSW and TSW for DMN subjects; hip adduction-abduction at LR, MS, ISW for DM individuals; hip internal-external rotation for both DM groups during IC, MSW and TSW; and ankle internal-external rotation between MS and PSW for both groups.

An investigation by Liu and colleagues (2010) reported that during object clearance trials DM subjects displayed a greater pelvic tilt, an increased dorsiflexion angle at the ankle, increased eversion, and reduced external tibial torsion in the stance leg when compared to controls. Conversely, in the leading limb, hip external rotation was increased, hip adduction reduced, and ankle inversion and internal tibial rotation were decreased. In light of the extent of joint angle changes which have been reported to occur in DM individuals during object clearance, it seems likely that kinematic parameter modifications would also take place during normal gait.

Petrofsky and colleagues (2005(b)) used an accelerometry analysis system to investigate joint excursions, and reported that these were greater in both the sagittal and frontal plane for DMN participants than for healthy controls. The increase was attributed by the authors, at least in part, to signal tremor; probably as a result of asymptomatic neurological changes.

2.2.4 Kinetic Parameters

2.2.4.1 Ground Reaction Forces

Yavuzer and colleagues (2006) failed to find a relationship between DMN and the magnitude of the vertical GRF component (GRFz) recorded at heel strike and toe-off. In a further study by Akashi, et al. (2008), a control group and two DMN groups (one with a history of ulceration, i.e. DMU), were compared in relation to GRF parameters. When normalised for bodyweight, a significantly lower second GRFz peak was observed in both experimental groups, which authors suggested was caused by inefficient propulsion due to late lateral gastrocnemius activation. Although activation of vastus lateralis was similarly delayed, this was not found to affect the magnitude of the first GRFz. GRF measures obtained by Sacco and Amadio (2000) included a significantly reduced GRFz first peak in DM participants (left foot only), and a significantly greater minimum force between peaks in DMN individuals (right foot only). In addition, the latter group demonstrated lower first

and second peak growth rates (i.e. gradient of GRFz peak curves) in relation to controls.

An earlier study (Shaw, et al. 1998) investigated the effect of varying levels of peripheral neuropathy (control (C), DM, DMN, DMU and DM with Charcot neuroarthropathy) on both the anterio-posterior GRF component (GRFx) and vertical GRF (GRFz) peaks. Higher peak GRFz (normalised for bodyweight) were observed in all DM groups, but particularly in DMN individuals. A decrease in peak GRFz, as observed with lower walking speeds, was found to be less noticable in DMN subjects than in controls. Medio-lateral GRF component (GRF_Y) peaks were greater in all DMN participants, and force-time patterns were altered in the same groups when compared to both DM subjects and matched controls. For example, in the majority of footsteps, the GRFz maximum peak occurred during heel strike rather than toe-off. Only in Charcot individuals was the peak GRFz at toe off significantly higher than in controls. Authors ascribed the altered timing of peak GRFz to the increased initial contact forces, rather than to lower propulsion forces.

In considering the above results, it must be noted that measured GRFz parameters (e.g. peak to peak period, total step duration, or area under the force/ time graph) may be affected by both the force analysis system used and the type of footwear worn, and therefore compilation of findings for comparison must be viewed in light of the specific experimental conditions (Barnett, et al. 2000).

2.2.4.2 Moments and Centre of Pressure

Yavuzer and colleagues (2006) reported that hip and knee moments measured in DM subjects did not alter significantly when compared to controls. Centre of pressure excursion was explored by Sawacha, et al. (2009), and was shown to be significantly reduced in both sagittal and frontal planes in people with DM and DMN.

2.2.5 Additional Factors Affecting Gait in Diabetes

2.2.5.1 Neuropathy and Muscular Weakness

Neurological changes (as previously discussed in section 2.1.3.1) may lead to motor nerve dysfunction with resulting muscle weakness, autonomic skin changes such as anhydrosis and loss of sensation (including reduced proprioception). More obvious signs of DMN may also include loss of balance and coordination, gait abnormalities and foot drop (Mueller, et al. 2006). It has been proposed that changes in the quantity and quality of the neurological impulses which control motor function may produce both postural and dynamic instability in DM individuals as a result of reduced muscle strength (Akashi, et al. 2008).

The presence of motor function abnormalities are reported to be significant in chronic DMN, and are thought to lead to an increasing loss of leg muscle power. The pathway to loss of function is thought to begin with motor axon damage in conjunction with inadequate reinervation, progressing to muscle fibre denervation and weakness, and eventually leading to reduced muscle volume (Mueller, et al. 2006; Andreassen, et al. 2009). In an investigation by Sacco and Amadio (2000) all pedal nerves tested in DM participants were found to require a longer period of electrical stimulation before bringing about a sensory or motor response (i.e. increased 'sensitive cronaxie'), with a more noticable increase in DMN subjects.

People with DMN have been shown to experience changes in the mass and isokinetic strength of muscles in the foot and lower limb. In an MRI follow-up study (Andreassen, et al. 2009) results indicated that in DM individuals, atrophy of the foot muscles begins in the early stages of DMN, and that the degree of muscle loss corresponds to the level of nerve dysfunction present. Assessment of knee extensors and flexors, and ankle dorsiflexors and plantarflexors, showed an annual decrease in strength in all muscles tested, with volume loss and atrophy observed in the ankle muscles. All alterations were significantly higher in the DMN compared to DM and control groups. However, atrophy was found to be present in DM

individuals suggesting that muscle loss may occur even in the presence of sub-clinical nerve changes.

Akashi and colleagues (2008) found no significant differences in the results of dynamic electromyography trials when comparing control subjects with either DMN or DMU individuals. This was the case for both normalised peak magnitude and tibialis anterior activation time. For the lateral gastrocnemius a seven percent delay in peak muscle activation was observed in the DMU group compared to the remaining groups, and a four percent delay was noted for the vastus lateralis in the DMU group when compared to controls. Yavuzer and associates (2006) assessed peripheral nerve function using electromyography in three participant groups (C, DM, DMN). F-wave latency (a measure of nerve conduction velocity) for both peroneal and tibial nerves correlated with a number of gait parameters, including: gait speed, step length, double support duration, ankle plantarflexion moment and power, and sagittal plane ankle excursion.

2.2.5.2 Muscle Activity

Any disturbance in sensation or muscle activity may lead to gait changes in people with DMN. It has been proposed that prolonged activation of muscles may result from reduced muscle strength leading to decreased braking capabilities, therefore requiring muscles to work over a longer period in an attempt to overcome forward progression of the body during gait (Savelberg 2010).

The role of muscle activation in gait pattern was explored by Sawacha, et al. (2011). Authors observed that the timing of activation in number of muscles was altered in both DM and DMN patients, suggesting that neurological changes may affect gait even before symptoms of DMN are present. Savelberg, et al. (2010) noted that in contrast to controls, muscle activity during an imposed gait velocity was prolonged in the ankle dorsiflexors (DMN) and the knee extensors (DM, DMN) in DM groups. These findings therefore correlate with the theory stated in the aforementioned report by Sawacha and associates (2011).

2.2.5.3 Foot Complications

Number of daily steps and gait velocity were both found to decrease with increasing severity of foot complications (DMN, DMU, foot amputation and trans-tibial amputation) in DM subjects (Kanade, et al. 2006). Although authors failed to provide an explanatory theory for this relationship, findings were supported by a previous study (Shaw, et al. 1998), which also demonstrated a link between DMN severity and reduction in gait velocity.

2.2.6 Influencing Factors Not Related to Diabetes

Research has previously demonstrated that gait velocity (Kirtley 2006), distance travelled (Najafi, et al. 2009) and type of ground surface or terrain (Allet, et al. 2008), each have an influence on gait parameter magnitude. It has been shown that the reliability of results improved over longer distances, and that gait speed increased on tarred surfaces when compared to both grass and cobblestones. In addition, changes in some gait parameters, including gait speed, have been partially attributed to participant characteristics such as health status and lower limb muscle strength (Brach, et al. 2008). Authors have also accepted that conclusions drawn from gait study outcomes must be considered carefully in light of the experimental procedures and patient groups reported. For example, Shaw and colleagues (1998) acknowledged that DMN may indirectly bring about GRF alterations via it effects on gait speed.

2.2.7 Effect of Diabetes on Gait: Section Summary

As highlighted in this section, gait changes observed in DM individuals are complex, wide ranging, and often interlinked. Therefore, isolating the underlying causes leading to altered gait pattern is extremely difficult, and as such, results reported in the current literature are frequently contradictory or inconclusive. It is hoped that by simulating CTD in healthy individuals, the effects of anatomical CTD on gait (including spatiotemporal, kinetic and kinematic parameters) expected to occur in

DM individuals may be inferred. Previous studies regarding gait alterations brought about CTD are sparse but will be discussed in snection 2.4.2.

2.3 EFFECT OF DIABETES ON PLANTAR PRESSURE

2.3.1 Introduction

Factors reported to contribute to IPP in the feet of people with DM include: age, DM duration and reduced joint ROM (Caselli, et al. 2002); soft/connective tissue changes (including fatty pad atrophy, hyperkeratosis and changes in plantar skin thickness and durability)(Menze, et al. 2007; Abouaesha, et al. 2001; Gefen 2003); sensory peripheral neuropathy, which may result in loss of protective sensation and hence prevent the normal pain response (Caselli, et al. 2002; Kanade, et al. 2006); foot structure, including measures of calcaneal angle, first and fifth metatarsal angle and sesamoid/calcaneal height (Cavanagh, et al. 1997); foot deformity, such as Charcot foot and digital deformity (including claw, mallet and hammer toe)(Bus, et al. 2005; Yu, et al. 2011); and altered lower limb musculoskeletal function as a result of one or more of the above structural changes.

Plantar pressure changes due to DMN, muscle weakness, soft tissue changes and reduced ROM are discussed in more detail in this section. These pathologies are all commonly associated with DM and may, to differing degrees, produce physiological changes which bring about structural foot abnormalities. The influence of both normal variations in foot structure (foot type), and pathology (including foot deformity), are also discussed in relation to their effects on PPPs. Lastly, the effectiveness of footwear and offloading devices currently employed in the clinical environment to redistribute pressure from vulnerable areas of the foot (e.g. previous site of ulceration) are explored with a view to identifying possible options for early intervention in DM individuals who are assessed as being at high risk of developing ulceration.

2.3.2 Factors which Influence Plantar Pressure in Diabetes

2.3.2.1 Neuropathy

A diverse range of physiological alterations observed in the DM foot have been discussed in earlier sections. Peak whole foot plantar pressures have been shown to correlate with DM severity (Kanade, et al. 2006), and increased forefoot and rearfoot pressures have been found in DMN patients when compared to controls (Caselli, et al. 2002). Neurological effects in relation to pressure can be divided into sensory, autonomic and motor effects (Tesfaye 2006), which are each considered in detail below.

Sensory Effects: Reduced Pain Threshold

Minor skin damage would usually be insignificant in a non-DM individual, but may result in infection or even ulceration in a person with DM. Where DMN is present, lack of sensation means that skin damage resulting from repetitive trauma, caused e.g. through poorly fitting footwear or standing on a sharp object, may go undetected for some time (Tesfaye 2006). Reduced healing and increased susceptibility to infection transform these seemingly trivial events into a significant risk for people with DM (Laing 1998).

Motor Effects

Muscle Activation

Previous studies have suggested that neuromuscular dysfunction in DM individuals alters the timing of muscle action and therefore exposes the foot to higher pressures (Merza and Tesfaye 2003). In an investigation by Abboud, et al. (2000), results indicated that activation of tibialis anterior was delayed in DM individuals, resulting in quicker progression to foot flat and hence higher plantar forefoot pressures when compared to controls.

Muscle Weakness

Compared to their healthy counterparts, people with DM have been found to exhibit a greater reduction in muscle mass in the plantar- and dorsiflexors of the ankle. In addition, the level of atrophy has been found to correlate with the degree of DMN present (Andreassen, et al. 2009). Atrophy of intrinsic foot muscles has been linked to the development of digital deformities, with corresponding IPPs. However, the relationship between muscle volume and foot deformity has been disputed in recent studies (Bus, et al. 2002; Bus, et al. 2009). The role of muscle strength in relation to the development of foot deformity is discussed more fully in section 2.4.2.

Muscle Contracture

Caselli, et al. (2002) suggested that an increase in the forefoot-rearfoot peak plantar pressure (PPP) ratio, which they observed exclusively in individuals with severe DMN, may provide evidence that the development of ankle equinus (i.e. tightening of the gastrocnemius and soleus muscles of the calf) is more likely with advancing DMN. In subjects with less severe DMN, PPPs were found to increase across both the forefoot and rear foot. Equinus may also occur as a result of Achilles tendon tightening (see section 2.3.2.2 below).

Autonomic Effects

The anhydrotic and vasomotor effects of autonomic neuropathy were previously discussed in section 2.1.3.1. Autonomic reduction in sweat production will also contribute to IPP via its influence on the formation of hyperkeratotic lesions which are discussed alongside other tissue changes below (section 2.3.2.2).

2.3.2.2 Tissue Changes and Reduced ROM

Although tissue modifications (e.g. plantar fat pad atrophy) may occur as a normal function of ageing (Merriman 2002) a number of tissue properties appear to be more prominent in DM feet.

Raised glucose levels are thought to lead to a number of structural changes in collagen in relation to fibril size, shape, organisation and cross-linking. Coupled with glycosylation of keratin, these alterations have been reported to result in stiffening, loss of elasticity and hyperkeratosis (callus formation) in plantar tissues, which may significantly affect their capabilities with regard to both cushioning, and redistribution of pressure across the foot surface (Gefen 2003).

Callus has been previously linked to ulceration due to its external effects on the skin (Merza and Tesfaye 2003). Menz and associates (2007) reported that plantar pressures were significantly increased at hyperkeratotic sites on the plantar surface of the foot. Authors suggested that IPPs brought about the development of callus via their effects on normal epidermal cell proliferation. Reduced plantar tissue thickness, which frequently occurs in the presence of callus, has also been found to closely correlate with IPPs at all MTPJs (Abouaesha, et al. 2001).

Computer models have been used to deduce the influence of plantar tissue thickness on pressure. A finite analysis model which simulated DM and non-DM feet was used by Gefen, et al. (2003) to investigate the abovementioned relationship. Stresses under the first and second MTHs were found to be four and eight times greater respectively, in simulated DM feet, when compared to the maximum pressures observed in the non-DM model. Increased tissue stiffness correlated with a further rise in PPPs. Authors suggested that damage was likely to begin in deep tissues, and that the most susceptible areas were those plantar to the MTHs.

Collagen cross-linking may also cause tendon tightening (e.g. Achilles), and connective tissue stiffness, resulting in decreased joint ROM. Decreased joint mobility subsequently prevents effective accommodation of uneven terrain by the foot, and hence brings about higher loading with a consequent increase in plantar pressures (Merza and Tesfaye 2003; Stanley and Collier 2008; Mueller, et al. 1990).

2.3.2.3 Fibro-Fatty-Pad Migration

In addition to structural modifications of the fibro-fatty pad, its displacement may also influence plantar foot pressures. The fibro-fatty pad is normally positioned in such a way as to cushion the MTPJs, an area of the foot which in DM individuals has been reported to experience IPPs (Gefen 2003). When foot biomechanics are altered (e.g. via the development of foot deformity), plantar tissues have been observed to migrate distally, possibly in response to the initiation of the windlass effect (Figure 2-3). It has been suggested that altered fat pad placement subsequently reduces forefoot cushioning, hence leading to IPPs at the MTPJs (Bus, Maas, et al. 2005; Mueller, et al. 1990; Merriman 2002). This is further supported by an investigation by Abouaesha, et al. (2001) who found a strong inverse association between plantar tissue thickness and PPPs.

2.3.2.4 Foot Structure and Deformity

As suggested above, higher loading may result from the migration of plantar tissues as a direct result of deformity. Although the exact pathophysiology of deformity and its effects on pressure in the DM foot are unclear, current research has implied a link between foot deformity and the development of ulceration (Bofelli, et al. 2002; Mueller, et al. 1990; Ledoux, et al. 2005).

Deformities, including digital deformity and those resulting from Charcot foot, have been found to produce abnormal plantar pressure patterns and lead to callus formation (Merza and Tesfaye 2003). Charcot neuroarthropathy specifically targets the midfoot (midtarsal joint and MTPJs), and where deformity occurs, it may result in a 'rocker bottom' foot that concentrates plantar pressures, and further exacerbates fracture formation and joint damage (Mueller, et al. 1990).

Foot type abnormalities may be congenital or acquired and may occur in the rearfoot or forefoot. Varus and valgus deformities are observed in the frontal plane and can be either functional or may arise from improper bony development. Root, Orion and Weed (1977) proposed theories both for the pathogenesis of these foot

abnormalities, and for the pattern of callus likely to be observed in response to these. Conjecture was based largely on clinical experience rather than scientifically proven evidence; however, more recent studies have supported the hypothesis that sites of callus/ulceration (i.e. indicating IPPs) correspond to specific types of foot deformity (Mueller, et al. 1990; Cavanagh, et al. 1997). Foot type has also been linked to the presence of foot deformity (Cowley, et al. 2008).

Structural abnormalities such as claw and hammer toe deformity, hallux abductovalgus ('bunion'), and pes cavus (high arch) are commonly observed in DM feet (Bus, et al. 2009; van Schie, et al. 2004) and have been shown to play a significant part in altering PPPs (Bus, et al. 2005; Gefen 2003). A discussion of CTD and its effects on PPP will be explored in more detail in section 2.4.5 below.

2.3.2.5 Effects of Clinical Intervention

Peak plantar MTPJ pressures and pressure-time integrals (force multiplied by the time over which it acts) have been previously assessed in relation to offloading devices commonly used in the clinical environment (Mueller, et al. 2006). Authors noted that total contact insoles reduced MTPJ loading by redistributing load more evenly across the plantar surface, whilst metatarsal padding invoked its effects through concentrating pressure proximal to the MTPJs, so reducing the magnitude of pressure on the joints themselves. Bus and colleagues (2004) similarly found a significant decrease in PPPs and pressure-time integrals in both the heel and first MTPJ when using custom made, compared to flat, insoles. They did however note that the influence of insoles varied greatly between participants.

Comparative investigation of various footwear types (Lavery, et al. 1997) has also demonstrated that 'comfort' shoes provide significant reductions in the peak pressure measured at sites of previous ulceration, and that viscoelastic insoles, in all shoe types, provided an additional decrease in loading.

The above studies demonstrate that intervention -either via footwear education or through the prescription of a cushioning or functional insole- may reduce the

pressures exerted on plantar foot structures. Although robust evidence in relation to the effectiveness of orthotic devices is limited, offloading techniques have been successful in reducing pressure related pathology in the clinical situation for a number of years. Evidence based knowledge pertaining to the causes and effects of abnormal foot pressures would enable existing pressure relieving methods and devices to be improved and would hence assist in limiting ulcer formation in DM patients.

2.3.3 Effect of Diabetes on Plantar Pressure: Section Summary

The relationship between plantar pressure and gait parameters has been used recently to deduce the body centre of mass (COM) from measured kinematic data (Winiarski and Rutkowska-Kucharska, 2009). Links between gait and GRFs established in this study indicate that in DM patients, changes in PPP will influence gait parameters and vice versa. It is therefore reasonable to presume that through a fuller understanding of the correlation between these factors researchers should be better equipped to prescribe interventions (e.g. functional offloading devices and muscle strengthening exercises) which act to both improve dynamic musculoskeletal function and minimise plantar pressures. Only by highlighting the major influences of both gait and foot pressure on plantar tissue integrity, can we hope to reduce the long-term incidence of ulceration in the DM population.

As stated in previous sections, a great number of factors have been associated with modifications in both gait and pressure. This thesis concentrates on investigating the impact of a single complication, namely claw toe deformity (CTD), that is observed in around 40% of DM individuals (Farndon 2000). An outline of the causes and specific effects of CTD, as currently reported in the literature, are provided in the following section.

2.4 CLAW TOE DEFORMITY

2.4.1 Introduction

'Claw toe deformity' is often used interchangeably with terms such as: digital deformity, retraction, hammer toe or mallet toe. Anatomically, CTD is classified as hyperextension of the MTPJs, coupled with hyperflexion of the proximal and distal IPJs (Figure 2-2).

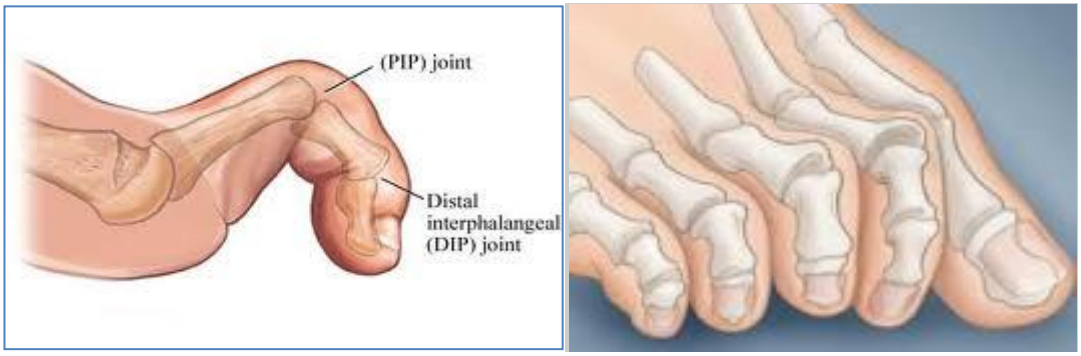


Figure 2-2 Joint involvement in claw toe deformity (left), and common pattern of deformity observed in the digits (right).

CTD produces a phenomenon called the 'windlass' effect (Figure 2-3)(Michaud 1997), whereby the hyperextended first MTPJ position results in tightening of the plantar fascia as it is pulled around this joint, so bringing about heightening of the medial longitudinal arch. In addition to altered arch height and MTPJs prominence, CTD has also been associated with migration of the fibro-fatty pad and changes in the distribution of weight across the plantar foot surface, which may result in an increased risk of callus/ulcer formation at corresponding sites of IPP (Bus, et al. 2005; Mueller, et al. 1990; Merriman 2002).

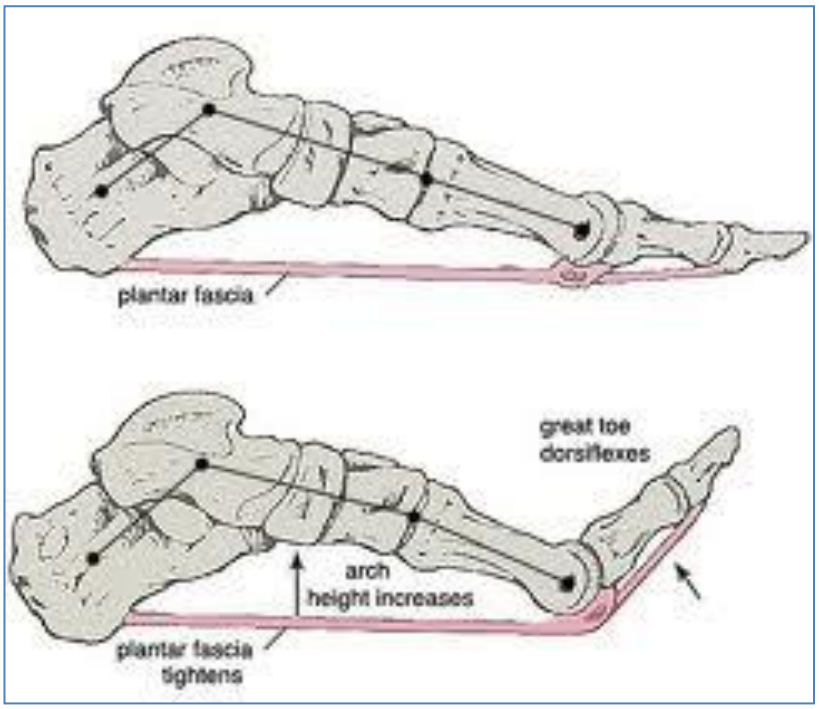


Figure 2-3 The windlass effect. In a normal foot (top), the full length of the digits are in contact with the ground surface, and the plantar fascia assists in maintaining the medial longitudinal arch of the foot. Where CTD is present (bottom), hyperextension of the first MTPJ results in functional shortening of the plantar fascia with an associated increase in the height of the medial longitudinal arch.

2.4.2 **Aetiology**

The incidence of CTD observed in both DM and non-DM individuals attending a podiatry clinic was reported by Farndon (2000) to be around 40%. However, in the former group this appeared to be associated with DMN, while the latter group had more restricted joint mobility, suggesting that the mechanism of development differed for each group. Further research has demonstrated that foot type (e.g. pes cavus, valgus or varus rearfoot/ forefoot) and toe deformity (e.g. hammer toe, CTD) show a significant association with the presence of both DM and DMN (van Schie, et al. 2004). In addition, the correlation between the incidence of CTD/hammer toe deformity, and pathologies such as retinopathy, microalbuminia and PVD, provides evidence of a link between DM complications and the development of foot deformity (Sawacha et al., 2009). Despite the numerous theories proposed in

relation to the aetiolgy of DM foot deformity (including CTD), current research has thus far failed to provide consistent, rigorous substantiating evidence.

Until recently the most widespread hypothesis regarding the aetiology of foot deformity in DM individuals concerned the effects of intrinsic foot muscle weakness, brought about by neurological abnormalities. A study using magnetic resonance imaging concluded that in DMN individuals, age related progression of muscle atrophy was higher for all intrinsic foot muscles, when compared to a control group (Bus, et al. 2002). These findings were supported by Andreassen (2009) who further stated that the degree of atrophy was directly related to the extent of nerve damage.

van Schie, et al. (2004) reported a connection between foot deformity, muscle strength and the efficacy of nerve conduction in control, DM, DMN and DMU participants. Loss of muscle strength was associated with reduced conduction velocity in peroneal and tibial nerves, and with the presence of foot deformity in both DMN and DMU groups. However, authors recognised that findings did not provide conclusive evidence that deformity occurred as a direct result of the muscle changes arising from nerve dysfunction. And in an MRI study, Bus and associates (2002) found no significant relationship between muscle weakness and the presence of foot deformity, and no difference in MTP joint extension (i.e. indicating the presence of CTD) between DMN and control groups. The same author later demonstrated that neither muscle atrophy nor muscle imbalance were significantly higher in DM patients with CTD than in individuals with normal digital alignment (Bus, et al. 2009). In addition, the severity of any deformity present bore no relation to the extent of muscle atrophy.

Further work is required in order to better understand the pathomechanics of CTD; however, it is likely that several factors (e.g. inappropriate footwear, ligamentous laxity, tissue glycosylation and functional changes) play a part in the development of DM foot deformity. The role of biomechanical factors was explored by Michaud (1997), who proposed that changes in foot alignment lead to CTD and hammer toe

deformities due to the altered function of various foot structures. For example, he suggested that rigid forefoot valgus produced a high medial arch, forcing the proximal phalanges into a more dorsiflexed position so altering the pull of the intrinsic foot muscles, which provided the extrinsic extensors with a mechanical advantage. Keenan, et al. (1991) theorised that valgus rear foot deformity in rheumatoid patients arose from both muscle weakness and attempts by the participant to avoid pain, leading to heightened pronatory forces, and eventually causing structural foot changes.

A full discussion of potential aetiological processes is beyond the remit of this thesis. Instead it will explore the role of CTD in altering gait parameters and plantar pressures, and will discuss how these may be involved in influencing ulcer development (see sections 2.4.3 - 2.4.5).

2.4.3 Effect of Claw Toe Deformity on Ulceration

Previous studies (Ledoux, et al. 2005; Mueller, et al. 1990) have related the presence of foot deformity (including hammer and CTD) to both foot type and ulcer occurrence. And of all the pathways to ulcer formation, it has been reported that more than two thirds of these involve foot deformity in combination with DMN and a minor foot injury (Ledoux 2008).

Mueller, et al. (1990) investigated the association between ulcer location and the classification of foot deformity (Charcot foot, compensated forefoot varus or uncompensated forefoot varus/ valgus) in DMN participants. Assumptions regarding the sites at which IPP, and subsequent ulceration, would occur were made on the basis of existing clinical theory regarding the biomechanical compensation which takes place as a result of the aforementioned foot abnormalities. A significant association was identified, which suggests that in the DMN foot, changes in foot structure produce a major risk for the development of ulceration.

CTD was considered, along with other foot deformities, in a study by Ledoux and colleagues (2005). Authors explored the relationship between foot deformity (hallux valgus, hallux limitus and hammer/ CTD), foot type (pes cavus, pes planus and normal arch height) and the incidence of ulceration. Presence of hammer/ CTD was found to be strongly related to both foot type and risk of ulcer development; although, no direct correlation was noted between foot type and ulceration. Vulnerability to ulceration was also investigated by Bokan (2010), who found that patients with sensory DMN in conjunction with hammer toe deformity were more likely to experience skin breakdown and foot ulceration. Results reported by Cowley, et al. (2008) further corroborated a link between the presence of deformity/ foot type and ulceration; however authors failed to establish a connection between the category of foot deformity and ulcer location.

2.4.4 Effect of Claw Toe Deformity on Gait Patterns

Few reports discuss the direct influence of deformity on gait patterns in DM patients. It has been proposed that DM motor neuropathy may lead to both foot deformity (through muscle atrophy) and reduced joint ROM; therefore, affecting both PPPs and gait characteristics by altering the pattern of foot loading during stance (Sawacha, et al. 2009). However, Yavuzer and colleagues (2006) observed significant gait changes in DM participants, which were absent in both DMN and control groups. This suggests that factors unrelated to DMN may also influence gait; however it may also be the case that neurological changes in the lower limb occur prior to clinical signs. In an investigation exploring gait changes in DM individuals, a significantly greater number of subjects presented with digital deformity in both DMN (CTD and hammer toe deformity), and DM (hammer toe deformity only) groups, when compared to controls (Sawacha, et al. 2009). It was further reported that both diabetic groups displayed reduced dynamic joint ROM, joint mobility and gait velocity when compared to healthy subjects.

Gait modifications have also been examined in non-DM individuals with foot deformity. Mickle and associates (2011) suggested that in older adults, altered

biomechanical function (as a result of digital deformity) may lead to reduced dynamic stability; although, in their study, spatiotemporal gait characteristic were not found to change in the presence of toe deformity. People with rheumatoid arthritis (RhA) have been reported to exhibit a delayed heel rise, and a reduction in velocity, stride length and single support duration (Keenan, et al. 1991). Turner and Woodburn (2008) noted that in RhA subjects with forefoot deformity, gait velocity was slower, and both ankle plantarflexion and hallux dorsiflexion were decreased at terminal stance, while double support duration and midstance forefoot inversion were increased.

Indirect links between foot deformity and gait alterations may also be inferred in DM individuals since various abnormalities (e.g. nerve dysfunction, muscular weakness, callus, and ulceration) that are frequently noted in DM have been previously associated with both aforementioned factors. For example, evidence of a link between lower extremity strength and gait changes in DM subjects was reported by Brach and colleagues (2008). While reduced muscle strength was shown by van Schie, et al. (2004) to correlate with the presence of foot deformity. Together these suggest an indirect link between foot deformity and gait alterations may be present.

2.4.5 Effect of Claw Toe Deformity on Plantar Pressure

Authors have previously described the changes in foot biomechanics and the subsequent pattern of IPP and callus formation which are likely to be observed in individuals with different foot types (Root, et al. 1977; Michaud 1997). However, these reports have been largely reliant on clinical experience and observation rather than evidence based research.

More recent studies (Bus, et al. 2005; Spink, et al. 2009) have explored the effects of CTD/ hammer toe deformity on PPPs and pressure-time integrals. In an investigation by Bus and associates (2005), loading in individuals with deformity was significantly higher across the central MTPJs than was observed in control subjects,

and was related to the severity of deformity. While Spink and colleagues (2009) found that in older people, plantar hyperkeratotic lesions (known to occur at sites of increased plantar pressure) were associated with the presence of both hallux valgus and digital deformity, the latter of which corresponded to lesions at the MTPJs of the affected toes.

Yu, et al. (2011) explored the distribution of plantar pressure across different foot regions in DM individuals with CTD and/ or hammer toe deformities. Subjects in the experimental group demonstrated a significant increase in PPPs at the hallux and MTPJs, and a significant reduction in rearfoot PPPs, when compared to healthy controls. A strong correlation was also noted by Sinacore and associates (2008), between observed loading patterns and the region (lateral or medial) of foot deformity. Authors found that maximum GRFz, PPPs, and force/ pressure-time integrals were significantly higher in DMN patients with foot deformity than in controls, despite the latter group exhibiting faster gait.

2.4.6 Claw Toe Deformity Section Summary

CTD and other foot deformities have been explored in relation to the presence of a variety of pathological changes in the DM lower limb, including ulceration, callus, reduced muscle function and plantar pad migration. Other studies have examined the links between foot deformity and gait in older people, and in individuals with RhA; however, little evidence exists regarding the relationship between CTD and gait in the DM population.

Further exploration is therefore required if a better understanding of the influence of DM foot deformity on both plantar forces and gait patterns is to be achieved. Evidence of any significant correlation would enable a more comprehensive assessment of ulcer risk, and would therefore allow early intervention aimed towards improving dynamic musculoskeletal function and reducing PPPs. It would be hoped that these combined actions would assist individuals to maintain their mobility and independence, and to reduce their risk of developing ulceration.

2.5 LITERATURE REVIEW SUMMARY

In reviewing the literature it is evident that DM has significant systemic effects on the tissues, nerves and circulatory system of the lower limb (Stanley & Collier, 2008). These physiological and structural changes may act together in DM individuals to alter musculoskeletal function, hence leading to IPPs (Abboud, et al. 2000; Caselli, et al. 2002; Abouesha, et al. 2001; Menz, et al. 2007) and gait character modifications (Shaw, et al. 1998; Liu, et al. 2010; Yavuzer, et al. 2006; Allet, et al. 2010). Over time, these functional modifications may also increase the risk of developing ulceration and ultimately the requirement for amputation (Armstrong, et al. 1998; Laing 1998).

Specific links between foot deformity and IPPs (Bus, et al. 2005; Sinacore, et al. 2008; Yu, et al. 2011), gait modifications (Keenan, et al. 1991; Turner and Woodburn 2008; Sawacha, et al. 2009; Mickle, et al. 2011), and ulcer development (Mueller, et al. 1990; Ledoux, et al. 2005; Cowley, et al. 2008; Bokan 2010) have also been highlighted in the literature. Recent studies have demonstrated that despite noteworthy progress in understanding the underlying pathophysiological changes which occur in the DM lower limb, the exact nature of many of the important pathways to altered function and disease in this population remains unclear. This is largely due to the diverse range of DM complications which often arise simultaneously (but to differing degrees) in the feet and lower limbs (Figure 2-1). As a result, accurately attributing modifications in lower limb characteristics (such as PPPs and gait patterns) to a specific factor (such as CTD) is problematic. However, if this were possible, it could potentially inform and improve clinical treatment modalities, as well as providing invaluable information about the pathways that lead from DM foot pathology, to functional changes, and ultimately to the emergence of chronic ulceration and amputation.

This aim of this thesis is therefore to make inferences about the specific gait parameters alterations that are likely to be observed in DM individuals as a result of anatomical CTD, and in doing so, to provide some indication of the possible links

between CTD, gait, PPPs and ulcer risk. Aims are discussed in more detail below (Chapter 3).

CHAPTER 3 PROJECT AIM AND HYPOTHESIS

As highlighted in the previous Chapter the multi-factorial nature of DM makes investigation of single pathological facets (e.g. CTD) difficult. However, by simulating CTD in healthy adults its effects on gait character can then be studied without interference from any other co-existing DM complications.

As such the general aim of this thesis is to identify any significant differences in gait parameters that occur in healthy adults wearing the CTDSD, and as such to infer which (if any) gait modifications are likely to take place in DM patients with anatomical CTD.

The specific objectives of this thesis are to:

1) Validate the effectiveness of a device designed to simulate the effects of CTD in healthy individuals.
2) Specify and quantify any kinetic, kinematic and spatiotemporal gait modifications occurring as a result of the CTDSD, by comparison with a control group (i.e. the same participant with the CTDSD removed).
3) Analyse data statistically to determine whether any significant gait parameter changes occur as a result of the CTDSD.
4) Make inferences about the gait parameter alterations that are likely to be observed in DM individuals due to anatomical CTD, and further, to make assumptions regarding the influence that these changes may have on the individuals' risk of developing ulceration.
5) Suggest appropriate recommendations for improved clinical practice in relation to any gait modifications ascertained in (4).
6) Identify areas which warrant further research.

CHAPTER 4 EXPERIMENTAL DESIGN INTRODUCTION

Previous Chapters have explored the impact of DM complications on the lower limb, and have discussed the findings of recent literature regarding the effects of DM on gait, plantar pressure and ulceration. However, it is evident that there are significant gaps within the current literature in terms of the specific effects of isolated factors, such as CTD.

As such, the initial part of this study involved the validation of a CTDSD, while the latter part attempted to make inferences about the gait pattern alterations that may be expected to occur in DM individuals with anatomical CTD. This was achieved by comparing gait parameter (kinetic, kinematic and spatiotemporal measures) and GRF component data obtained from the same healthy participants, before and after the CTDSD was applied to one foot.

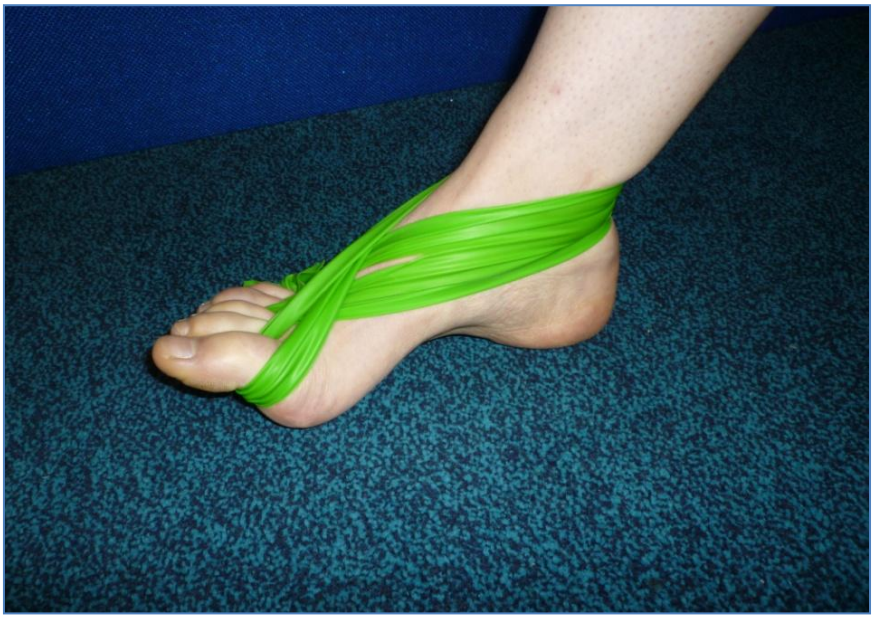
The following Chapters provide detailed methodology, results and discussion sections in relation to both the preliminary validation study (Chapter 5) and the main gait parameter study (Chapter 6).

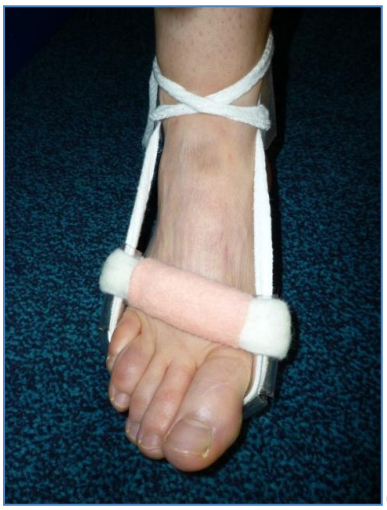
CHAPTER 5 PRELIMINARY STUDY: VALIDATION OF CLAW TOE DEFORMITY SIMULATION DEVICE

5.1 MATERIALS AND METHODS

5.1.1 Design and Selection of Claw Toe Deformity Simulation Device

A range of initial devices designed to replicate CTD in healthy participants were conceived. Mock-up CTDSDs were manufactured for the three most suitable designs (Figure 5-1), and were rudimentarily assessed by the researcher in relation to general comfort, fit, ease and consistency of application, and ability to hold the digits effectively in a clawed position. Pre-requisite features of the preliminary device included adjustability for foot size, ability to permit a degree of joint flexibility during gait, and consistency in control across all digits. Design criteria required that the device should create an extensor moment at the MTPJs that produced a significant MTPJ extension angle and hence result in prominent MTHs.


(A)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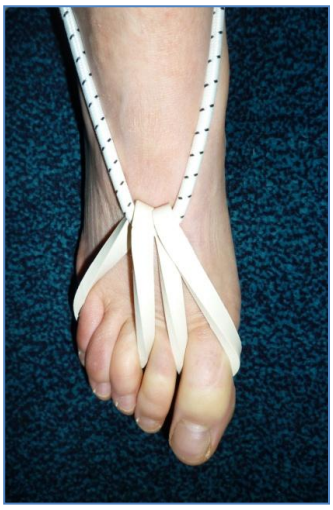

(C)

Figure 5-1 Three Simulated CTD Device Designs. Latex rubber sheet device (selected device) (A), device using plastic tubing which loops under all five digits (B), and rubber loop device which is worn over hallux and third to fifth digits (C).

Following initial testing, three F-scan walking trials were carried out by the researcher on herself whilst wearing each of the three potential devices. Desired trial outcomes were to assess physically (i.e. through wearing device) and visually (by reviewing F-scan 2D and 3D contour graphic movies for each trial) whether any obvious problems existed in terms of using the CTDSD in conjunction with the F-scan system and sensors. After assessment the researcher selected the CTDSD which was deemed to most closely fulfil the criteria discussed above.

The selected device was produced from a thin sheet of natural rubber latex approximately 100mm x 430mm. Three equidistant, longitudinal cuts, approximately 50mm in length, were made at one end (Figure 5.2A) and the resulting projections were tied to form two toe loops (Figure 5-2B). To apply the device to the right foot the toe loops were placed over the third and fifth digits (Figure 5-3A) and then, under tension, the band was passed medially across the dorsum then posterio-laterally around the distal calf (Figure 5-3B). The free end was then looped around the hallux, from lateral to medial, and secured under itself on the dorsum. Figure 5-4 shows the affixed CTDSD from various angles. The

effectiveness of this device in simulating CTD was validated in the preliminary study using a range of methods.

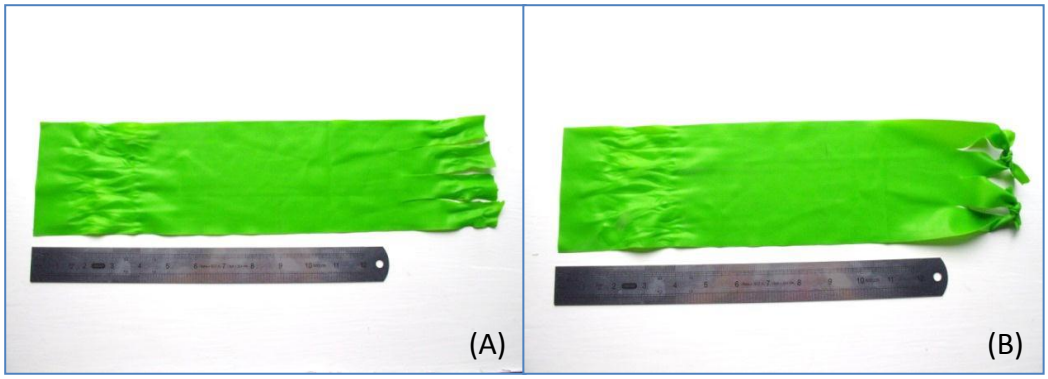


Figure 5-2 Selected device design. A thin sheet of latex rubber is cut at one end (A), and the tabs created are tied together to form two toe loops (B).

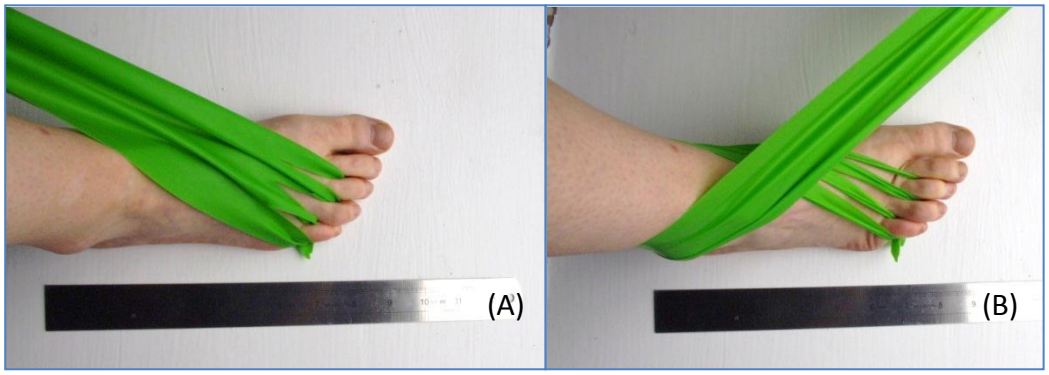


Figure 5-3 Selected device design. Toe loops are placed over 3rd and 5th digits (A) and the free end is passed medially across the dorsum and then around the ankle (B) before being looped under the hallux.

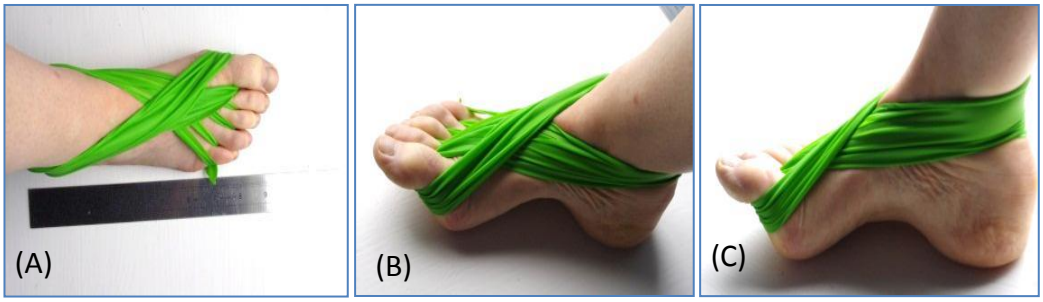


Figure 5-4 various views of the CTDSD following application to the right foot: dorsal (A), dorsal-medial (B) and medial (C).

5.1.2 Study Design

Substantiation of the effectiveness of the CTDSD was obtained by comparing PPP data from current literature, with values recorded using the F-scan in-shoe pressure system during shod walking trials involving healthy participants with and without the CTDSD placed over one foot. Research literature selected for comparison described PPP patterns measured in both DM patients (Yu, et al. 2011) and older people (Mickle, et al. 2011) with anatomical CTD.

In addition, visual observation of the effects of the CTDSD on both static and dynamic foot position was conducted by a qualified, HPC registered podiatrist, experienced in conducting clinical assessment for diagnosis of foot and lower limb pathologies.

5.1.3 Participants

Participants comprised of three healthy adults (two females, one male) aged between 23 and 36. Exclusion and inclusion criteria were identical to that used in the main study (see section 6.1.2); however, assessment was via visual examination and verbal questioning only, i.e. with no physical assessment or health questionnaire being carried out. Informed consent was obtained prior to patient participation and ethical approval was provided by the Bioengineering Department Ethics Committee, University of Strathclyde. Full details of the recruitment, consent and assessment procedures are described in the next Chapter.

5.1.4 Plantar Pressure Measurement

The F-scan in-shoe plantar pressure analysis system (*F-scan®, Tekscan Inc., Boston MA, USA*) was used to measure PPPs during level walking trials with and without the CTDSD applied. F-scan is an analysis system which uses thin (0.15mm), high resolution sensors that are placed in the shoe to measure pressures at the foot-shoe interface. Each sensor can be trimmed to fit the individual, and consists of 960 resistive sensing elements (variable depending on the shoe size that the sensor is

cut to fit), which are located in a grid system over the sensor surface (sensel density 4/cm^2) (Rosebaum and Becker 1997). Sensor area is approximately 107mm x 305mm (pre-trimmed), and sensitivity to dynamic pressures fall within the range of 345-517kPa (maximum pressures of up to 862kPa may be detected). Data can be displayed visually as coloured 2D or 3D 'movies' and/or in a quantitative format (e.g. outputs may include contact pressure, peak pressure and force). Various limitations have been noted regarding the F-scan system, including significant creep and hysteresis, questionable calibration accuracy, output variation/ repeatability problems, and limited sensor longevity (Woodburn and Helliwell 1996). However, the F-scan system has been used in a number of previous plantar pressure studies (Luo, et al. 1998; Sacco and Amadio 2000; Yu, et al. 2011) and the capabilities of the system were deemed suitable by the researcher for the purposes of preliminary study data collection; namely, to ensure that that PPP data obtained for participants wearing the CTDSD provided an accurate reflexion of those likely to be observed in DM participants with anatomical CTD.

Previous investigations have supported the use of F-scan for collection of pressure data in conditions where contact loading occurs with a soft material (e.g. in-shoe interface), although it has been recommended that calibration be carried out under experimentally identical conditions (i.e. equivalent temperature, loading and contact surface conditions) for better accuracy (Luo, 1998).

5.1.5 Experimental Procedures

The F-scan pressure analysis system was set up and operated according to the manufacturer's instructions. Prior to data collection each participant's weight was measured using a medical scale and calibration was performed. In order to assess the influence of the device on PPP, three initial trials were carried out without the CTDSD, followed by three trials with the CTDSD placed over the right foot. In both control (without device) and experimental (with device) trials, frequency of collection was 100 frames per second (100Hz). Participants were asked to walk at a

self selected walking pace in a straight line along a level walkway approximately 6m in length.

Sensors were used in-shoe, and were secured to the interior of the shoe using a low allergy dressing retention tape (Hypafix®) to minimise sensor movement. A thin sock was worn between the sensor and skin to minimise sensor damage and for hygiene purposes. Participants wore similar, flat, comfortable shoes to reduce any inconsistencies in results due to the influence of footwear design. Data collection was carried out by the same investigator, in the same location with identical equipment each time; and therefore conditions (temperature, lighting, walkway etc.) were consistent across each trial.

Subsequent to data collection, F-scan system (Tekscan) software was used to create masks to isolate PPPs occurring within seven specific areas of the foot. These were: the hallux, first MTPJ, second MTPJ, third to fifth MTPJs, midfoot, heel and total foot area. A template (Figure 5-5) was produced and was subsequently adjusted for each trial to ensure that masks were accurately located, and therefore pressures measured were in relation to the intended region. For each trial, a PPP-time graph was produced to enable visualisation of the peak pressure values for each of the discrete foot regions. Pressure-time graphs (examples shown in Figure 5-6) were used to identify trial data that was suitable for further analysis. Data from the first and last gait cycles, plus any which differed significantly from the other cycles, was excluded. Mean PPPs were then calculated from the selected cycles (between two to four gait cycles) for each trial. Intra-participant means were calculated by combining data from the three trials recorded for each individual. Mean intra-group (i.e. control and experimental) PPP values were then obtained for each foot region, by averaging all intra-participant means. Pressure ratios (PPP recorded with the CTDSD, as a proportion of the PPP measured without the CTDSD) were also calculated for each participant.

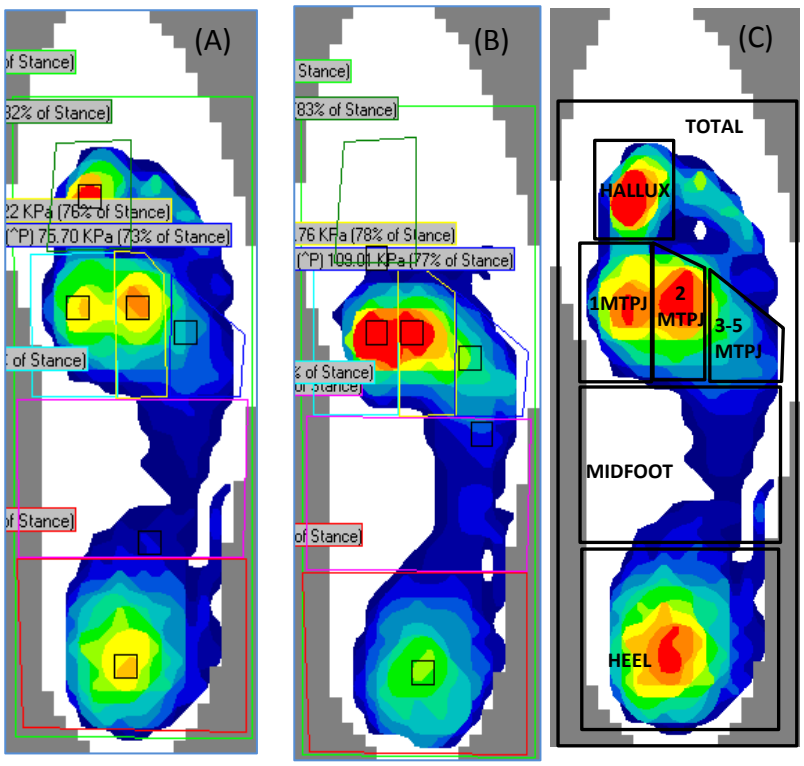


Figure 5-5 Example foot region masks for subject without (A) and with (B) CTDSD placed over foot. Accuracy maximised for each trial by overlaying the mask template onto 2D contour graphs of mean PPPs (as shown) and adjusting 'polygons' (areas demarcated by coloured lines) as required. Seven foot regions are: hallux (dark green), 1MTPJ (cyan), 2MTPJ (bright green), 3-5MTPJ (navy), midfoot (pink), heel (red), total foot (green). Foot regions are also highlighted in black on unmasked 2D image for clarity (C).

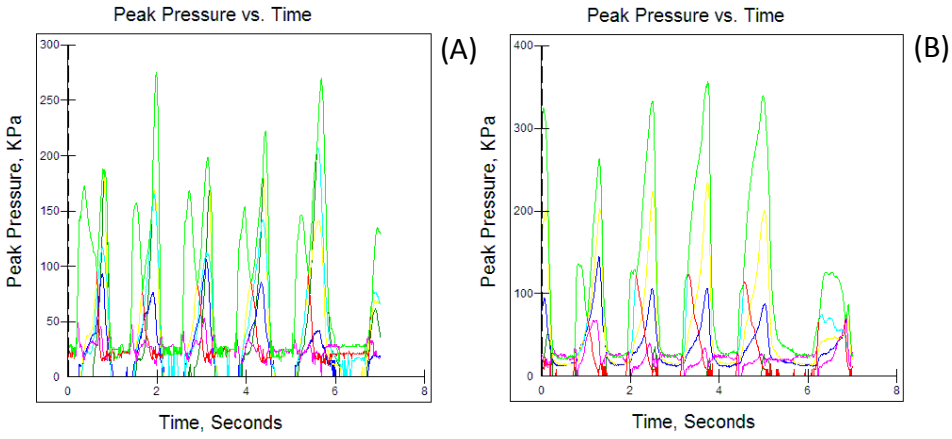


Figure 5-6 Peak plantar pressure-time graphs for no device (A) and with device (B) groups indicating the seven foot regions; hallux, 1MTPJ, 2MTPJ, 3-5MTPJ, heel, midfoot, total foot (each region is represented by a different colour). N.B. each double peak on graph represents one gait cycle. Graphs were used to identify cycles suitable for inclusion in analysis. First, last and any gait cycles with a characteristic pattern markedly different from the majority were excluded.

Due to the small number of participants and absence of matching, statistical analysis was not deemed appropriate for this part of the study.

5.2 RESULTS

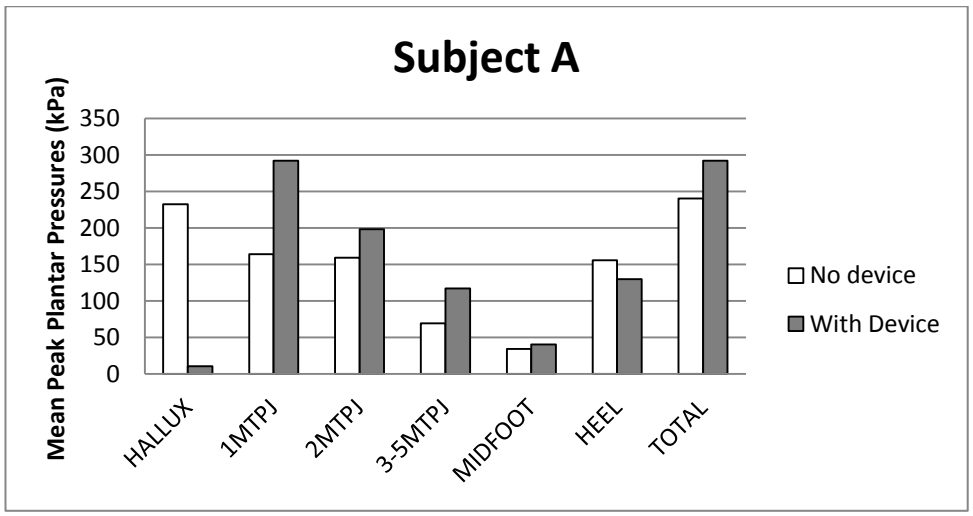
5.2.1 Explanation of terms

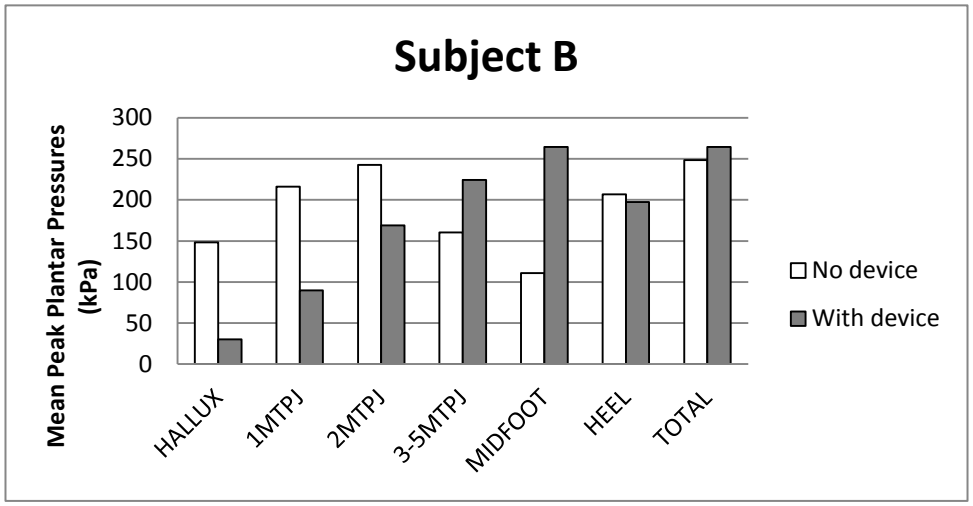
Peak plantar pressure (PPP) describes the highest measurement of pressure that occurs within a particular foot region (mask) throughout a single stance phase. The mean PPP for a given region refers to the average of all stance phase PPPs from one trial (i.e. from multiple gait cycles). Intra-participant mean PPP describes the average PPP over the three trials (control or experimental) for each participant. And lastly, the intra-group mean refers to the total average of all intra-participant means within one group.

The seven foot regions considered were: hallux, first MTPJ (1MTPJ), second MTPJ (2MTPJ), third to fifth MTPJs (3-5MTPJ), midfoot, heel and total; where 'total' PPP represents the highest pressure measured across the whole plantar surface of the foot during one stance phase.

5.2.2 Plantar Pressure Measurement Results

Peak plantar pressures (PPPs) obtained for all participant trials are shown in Appendix A, mean intra-participant PPPs shown in bold. Graphs comparing within subject mean PPPs for each foot region before and after CTDSD application, are found in Figure 5-7 (with corresponding numerical tables provided in Appendix B). For all subjects, PPPs recorded at the 3-5MTPJs and midfoot regions increased, and at the hallux and heel decreased, when comparing the CTDSD group with controls. Changes in PPP for 1 and 2MTPJs and total foot area varied between subjects.





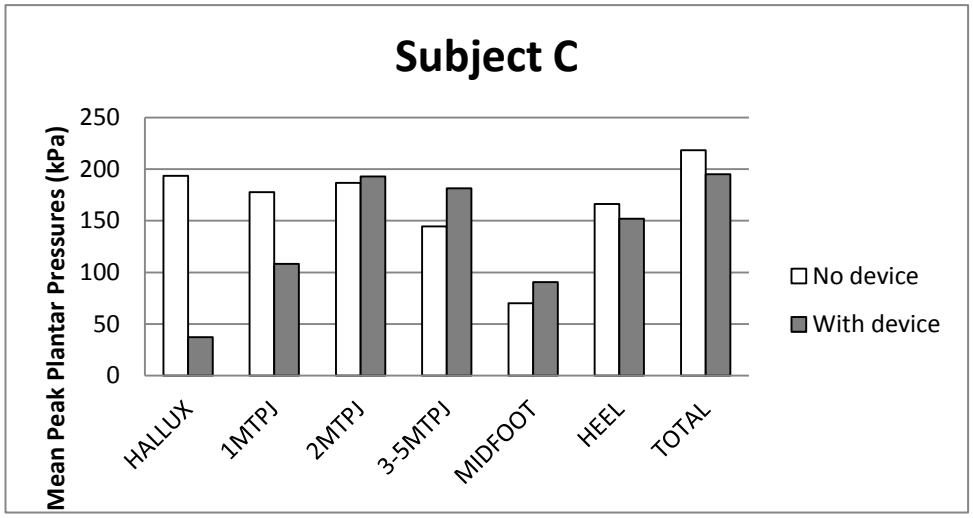
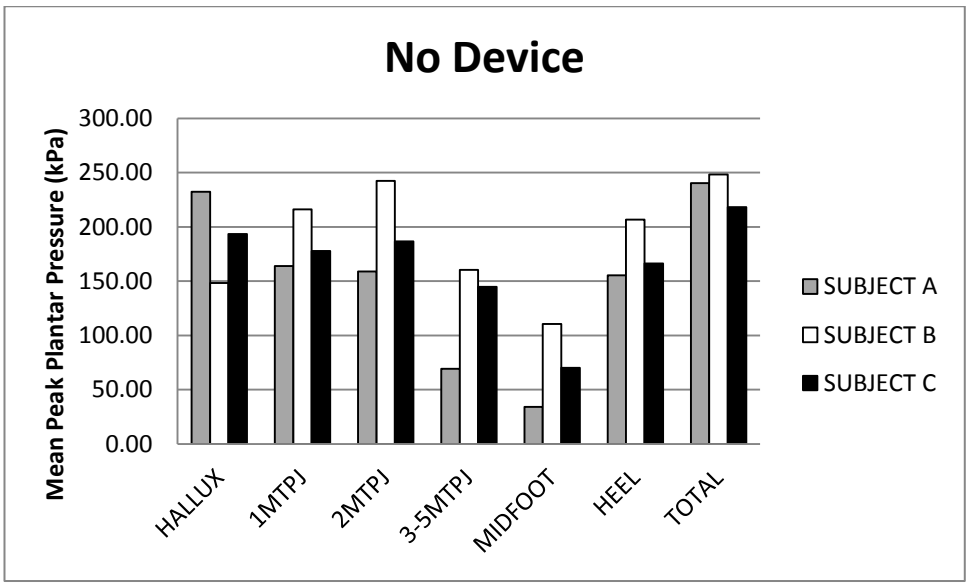


Figure 5-7 Mean within subject PPPs for each foot region before and after application of CTDSD (subject A, top: subject B, middle; subject C, bottom).



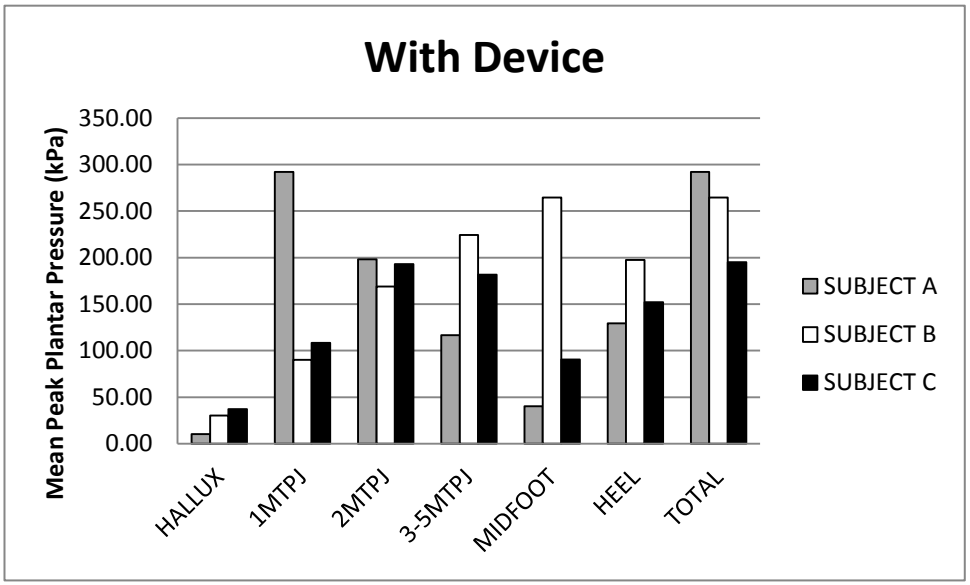


Figure 5-8 within subject mean PPPs for each foot region without (top) and with CTDSD device (bottom).

Figure 5-8 compares mean intra-participant PPPs for each foot region. It can be noted from this figure that during no device trials (Figure 5-8, top) the highest PPPs occurred at 2MTPJ, hallux and 1MTPJ (i.e. distally and medially). High pressures were also noted at the heel, while the lowest pressures were recorded in the midfoot region. Following CTDSD placement (Figure 5-8, bottom) PPPs were, in

general, displaced more proximally and laterally, with the greatest PPPs being recorded at the 2, 3-5, and 1MTPJ regions (in that order of magnitude). Conversely, PPPs measured at the heel were slightly lower, at the hallux were significantly reduced, and in the midfoot were increased (see Figure 5-8, top and bottom, and corresponding numerical tables in Appendix B). It can also be noted that in subject B, PPP at the midfoot was higher than for any other region (Figure 5-8, bottom). Total foot PPPs were smaller in one, and increased in two subjects following CTDSD application (Figure 5-8, bottom), with the overall intra-group mean being slightly higher.

Sample 2D (Figure 5-9) and 3D (Figure 5-10) F-scan system graphics illustrating mean PPPs for one subject before and after application of the CTDSD are shown below. These figures demonstrate the high pressures exerted on the hallux, 1MTPJ, 2MTPJ and heel during control trials (Figure 5-9A and Figure 5-10A), and the effects of applying the CTDSD (Figure 5-9B and Figure 5-10B); namely, a comparitive increase in PPPs across all MTPJs (in particular first and second), with an associated reduction in PPP at the hallux. Minimal digital contact and increased midfoot contact were also noted following device application (see Figure 5-9A and B).

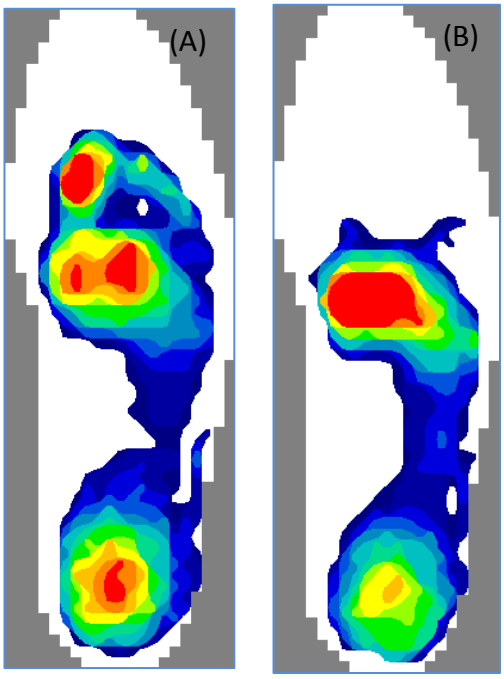


Figure 5-9 2D F-Scan Images illustrating mean stance peak plantar pressures for a subject without (A), and with (B) CTDSD worn over right foot. Note lack of digital pressure and greater pressure at first and second MTPJs.

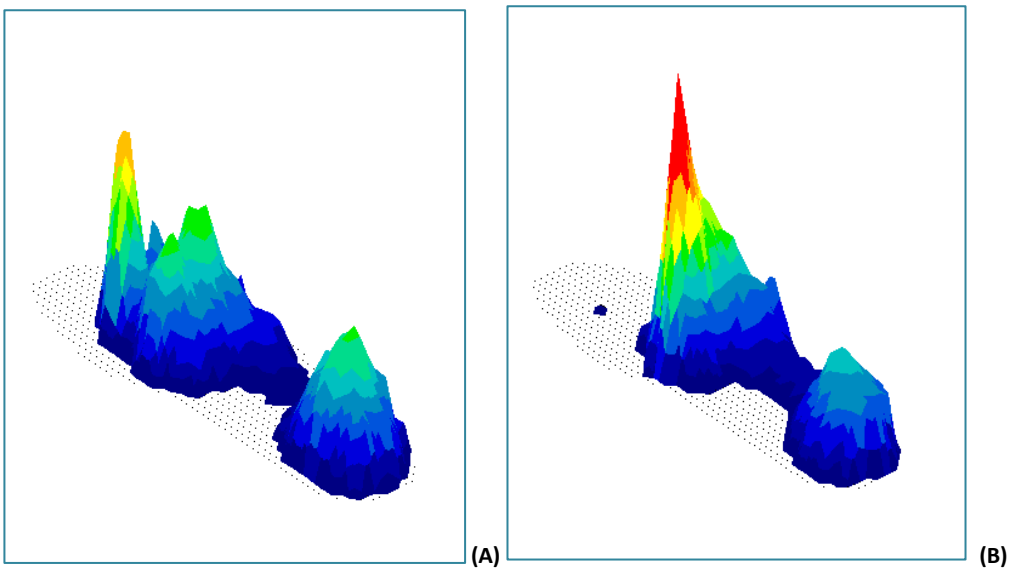


Figure 5-10 3D contour graphs showing PPP at midstance for a single subject before (A) and after (B) CTDSD applied. Note higher peak pressure at medial MTPJs and absence of pressure at digits, including hallux.

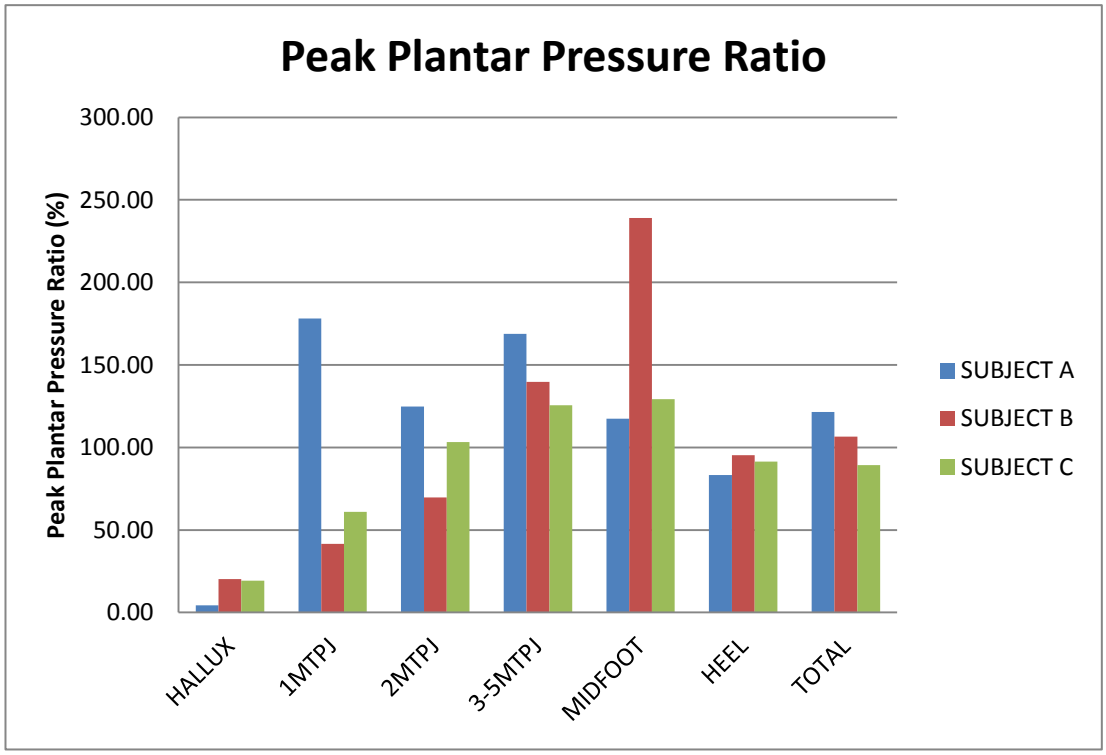


Figure 5-11 Intra-participant peak plantar pressure ratios (PPPs recorded with subject wearing CTDSD as a percentage ratio of the PPPs obtained prior to CTDSD application) for each foot region. Please note that 100% indicates no change has occurred.

Comparison of PPP ratios (intra-participant mean PPPs for experimental trials expressed as a percentage of the corresponding control trial value) was performed to clearly show the relative 'before-and-after' effects due to the CTDSD. PPPs ratios (see Figure 5-11) recorded at 3 to 5MTPJs were between 25.5% and 68.8% higher, at the heel were reduced (by between 4.6% and 16.7%), and at the hallux were notably reduced (to between 4.4% and 20.3% of the control PPP value) following CTDSD application. The mean midfoot PPP recorded for subject B during experimental trials was 238.9% of the recorded control pressure; however, the corresponding increase noted in the remaining two subjects was only between 17.5% and 29.21% higher (Figure 5-11).

5.3 DISCUSSION

A substantial degree of variation was observed between preliminary study participants in terms of foot type, foot shape, foot size and body weight etc. However, since participants acted as their own controls, it may be presumed that any PPP alterations observed in experimental trials arose as a direct result of CTDSD effects. For example, the significant increase in mean midfoot PPPs recorded for subject B when wearing the CTDSD may appear erroneous, but PPP values obtained were consistent across all three trials for this individual. Furthermore, the general no device PPP pattern obtained for subject B involved PPPs that were originally located more proximally and laterally in relation to subjects A and C. As such, any variations are likely to result from individual participants' foot type or shape. Subject B, for instance, had a low arch and therefore exhibited greater midfoot and heel pressures, as well as demonstrating more concentrated forefoot PPPs across the MTPJs (particularly first and second MTPJs) as opposed to the hallux and lesser digits. Taking these subject differences into consideration it may be noted that the general PPP pattern still moved more medially and laterally in this subject when the CTDSD was applied (compare Figure 5-8 top and bottom).

When the digits are pulled back (retracted) into a clawed position, the MTPJs become more prominent and the forefoot plantar fibro-fatty pad migrates distally,

therefore providing less cushioning over the MTPJs. Higher pressures would therefore be expected to occur at the MTPJs, along with lower digital pressures due to minimal ground contact. Findings of this study would hence support the aforementioned theory. In normal gait, contact begins at the lateral heel, and progresses along the lateral aspect of the plantar foot surface, followed by lateral to medial loading of the MTHs and digits, before toe off finally occurs at the hallux. CTD results in IPPs across the exposed MTPJs and will therefore impede normal propulsive function. Without the balancing action of the digits, lateral to medial MTPJ loading is difficult and, in addition, the windlass effect (see Figure 2-3, i.e. increased arch height and effective foot shortening occur when the hallux is dorsiflexed) (Michaud 1997) exacerbates MTH prominence and results in discomfort if toe off is attempted from the 1MTPJ and hallux. The requirement for prior plantarflexion of the first metatarsal, to enable maximal 1MTPJ dorsiflexion (required for efficient and fluid propulsion), is also prevented when CTD is present. It can be noted that although the windlass effect (and associated MTH prominence and increased arch height) occurs as a result of the CTDSD in static stance, its effects are restricted dynamically since normal roll-over onto the forefoot (which usually exaggerates the windlass effect) during terminal stance and pre-swing cannot be achieved. Therefore, the windlass effect may be noted early in stance, but cannot progress normally following heel lift and so has minimal influence in mid to late stance. Careful examination of Fig. 5-5 A and B demonstrates a functional shortening when the CTDSD is worn (note the distance between the 1MTPJ and heel in each figure), which can be attributed to the windlass effect. However, greater pressures are noted in the midfoot region with the CTDSD, which appears contradictory. This may be explained by the lack of digital support when the CTDSD is applied resulting in bodyweight be borne over a smaller area (including the midfoot). In addition, windlass may result in the plantar fascia being more prominent plantarly and therefore pressure may be transmitted to the ground through this structure.

Due to the lack of current literature describing the effects of anatomical CTD on PPPs, comparison of existing data with the findings of the preliminary study was difficult. Variation in data collection methods, pressure analysis systems, inclusion criteria, participant characteristics and experimental procedures meant that results were inconsistent and hence it was difficult to draw any definitive conclusions. In addition, the way in which data was reported in the literature meant that its comparative value was hard to assess (e.g. what do authors define peak pressure as? Did each participant perform more than one trial? How did authors select data that was suitable for further analysis? What constituted a lesser toe deformity?). Failure by authors to specify footwear type also precluded selection of identical footwear for use in this study. However, the preliminary study did provide a quantitative (PPP values) and subjective (observation) indication of the changes in PPP which arose following CTDSD application. Results therefore did offer a degree of supporting evidence for the CTDSDs effectiveness in replicating the effects of anatomical CTD. The general findings reported in current literature regarding the effects of anatomical CTD, as well as a comparison of these with the preliminary study findings are outlined below. To avoid confusion, for the remainder of this discussion, the first comparative study (Mickle, et al. 2011) will be referred to as study M; the second report by Yu, et al. will be denoted study Y, and the preliminary study, carried out as part of this thesis, will be referred to as study P.

Mickle and colleagues (2011) reported that in subjects with lesser toe deformities, PPPs were significantly higher in both the 2 and 3MTPJ regions, and the lesser toes, when compared to control subjects. Results of study P were in agreement that, in relation to the control group, PPPs increased in the 3MTPJ region following CTDSD application. However, in study P, the group mean PPP values for the 2MTPJ area were found to decrease in the experimental group, which conflicts with the reference study, M. Effects on PPP at the lesser digits could not be compared since they were not selected for measurement in study P (owing to the poor accuracy involved in placing masks over this foot region). However, it is reasonable to infer from the reduction in hallux PPP (Figure 5-8), and lack of digital contact illustrated in

both 2D (Figure 5-9) and 3D (Figure 5-10) images (representing differences in PPP between groups), that digital PPPs would be likely to decrease with the CTDSD. Again, these inferences disagree with results found in study M. The PPP pattern noted in study M would suggest that the type of toe deformities exhibited by trial participants are likely to have involved significant ground contact by some or all of the digits, e.g. as would be observed in mallet toe or CTD where no retraction of digits had taken place (i.e. hyperflexion in IPJs more extreme than hyperextension at the MTPJs). In addition, since deformity was not specified in the literature, this may have been isolated in only one or two digits and/ or may not have been severe. Due to these unknown factors the true comparative value of the data presented in study M is therefore questionable. Also, since participants were non-diabetic older adults, the effects of CTD in this group would not necessarily produce the same PPP patterns as would be observed in an age matched DM group.

Yu and colleagues (2011) reported that DM patients with hammer and/ or claw toe deformities demonstrated significantly higher PPPs in the forefoot, and lower PPPs in the heel when compared to a healthy control group. No significant changes in PPPs were noted in the midfoot. Authors further observed that within the foot deformity group, participants with ulceration (in each case occurring at the 2MTPJ) demonstrated significantly higher PPPs when compared to those with no ulceration. Findings of study Y agreed with those of study P, in terms of reduced heel PPPs and increased PPPs at the third to fifth MTPJs when either anatomical CTD or the CTDSD was present. However, the higher levels of PPP observed at the hallux, 1MTPJ and 2MTPJ in study Y contradict the results of study P in which these were all reduced when the CTDSD device was in place. This may be explained by the fact that in study Y 'toe deformity' was used to describe both claw toe and hammer deformities. Presence of hammer toe deformity would therefore account for the higher PPP recorded in the medial forefoot. Midfoot PPPs obtained in study P were slightly higher (significantly higher in subject B) with the CTDSD compared to controls. In the case of subject B this was due probably to foot shape, while in the remaining subjects it was likely to be due to proximal displacement of weight from

the digits to the midfoot. It is unclear whether digital contact took place in any or all participants of study Y; however, by definition, hammer toe deformity causes the distal phalanx to be either plantarflexed against the surface, or in a neutral position. In addition, since hammer toe involves hyperflexion of the proximal IPJ, and patients were shod in study Y, therefore it is likely that contact took place between the dorsal surface of the proximal IPJ and the footwear, so resulting in plantarward pressure on the digit against the ground. Again, the abovementioned factors, along with the lack of information in study Y regarding the degree, nature (i.e. CTD or hammer toe deformity) and location of digital deformity, made direct comparison of PPPs difficult.

5.4 LIMITATIONS OF STUDY

The purpose of the preliminary aspect of this study was to validate the effectiveness of a device designed to simulate CTD in healthy adults, via determination of its accuracy in reproducing PPP patterns as reported in the current literature.

The scope, time frame and resources allocated to completion of this preliminary study did not allow for a thorough and rigorous validation of the CTDSD. Furthermore, gaps in the current literature prevented a meaningful comparison with the findings of this preliminary study. Only one report investigated the effects of digital deformity on PPPs in DM subjects (Yu, et al. 2011). This report involved a relatively small subject number (n=30 for each group), and authors omitted specific information about the nature of foot deformity present. In addition, healthy adults, rather than asymptomatic DM subjects, were used as controls. Therefore, findings could not be directly related to a specific pattern of deformity, and it could be further argued that any changes in PPP may have been caused by the effects of DM, rather than CTD. The second investigation, by Mickle and associates (2011), involved older people with no history of DM, and therefore findings may not have accurately reflected PPP measures experienced by the DM population. These issues prevented an ideal comparison for accurate validation of the device, and future studies would be required to provide robust evidence of its effectiveness.

Since literature regarding the effects of anatomical CTD on PPPs was found to be lacking, and therefore limited options for comparison were available, the authors felt that observational and clinical assessment of validity should also be considered in combination with experimental results.

Clinical assessment of validity was conducted by an HPC registered podiatrist, trained in lower limb assessment, who observed changes in the anatomical positions of the joints when the CTDSD was affixed to the foot. They also performed live observation, in addition to examination of 2D and 3D F-scan colour PPP images, as subjects walked along a straight, level walkway with and without the CTDSD in place. Observations were judged by the podiatrist to be consistent with those previously noted in individuals with anatomical CTD. And in the opinion of the podiatrist, the only limitation of the CTDSD was that in order to produce effective prominence of MTHs (which would be consistent with anatomical CTD) the retraction of the proximal phalanges was such that it prevented any ground contact at the distal phalanges (consistent with retraction rather than claw toe deformity specifically). From an engineering perspective, a major disadvantage of the CTDSD design was the action of the device about the ankle. Design criteria required that an extensor moment be invoked about the MTPJs; however, the device simultaneously acted to produce an unwanted dorsiflexion moment about the ankle. This was minimised by placing the device as close to the ankle joint axis as possible to minimising the moment arm. Nonetheless, the remaining moment is likely to have influenced the gait parameter measures obtained, as well as producing effects which were inconsistent with anatomical CTD.

However, considering the limited resources and time, gaps in the current literature, and taking into account the subjective observations, it was felt that enough evidence had been gathered to support the effectiveness of the CTDSD to replicate anatomical CTD for the purposes of the main gait study.

CHAPTER 6 MAIN STUDY: GAIT PARAMETERS

6.1 MATERIALS AND METHODS

6.1.1 Study Design

Inferences regarding the effects of CTD on gait parameters in DM individuals were drawn by conducting a number of walking trials in healthy adult participants. Initial data was collected from participants wearing the previously validated CTDSD on one foot. Following this phase, identical parameters were obtained from the same individuals following removal of the CTDSD. Since subjects acted as their own controls the requirement for participant matching (e.g. age, gender, health status, weight) was negated. Kinetic, kinematic and spatiotemporal characteristics were measured using a Vicon 3D motion analysis system, and GRF component values were simultaneously obtained using a Kistler force platform. Experimental and control data were statistically compared to determine any significant changes attributable to the CTDSD.

6.1.2 Participants

Healthy volunteers, of both sexes, aged between 22 and 36 (mean (±SD), 22.67 (4.44)), were recruited from the Bioengineering department within the University of Strathclyde, Glasgow. Inclusion criteria required that participants were healthy adults with good mobility and balance (i.e. participants were able to walk normally, and without assistance, up and down a level walkway). Potential participants were excluded if they had any condition which significantly affected circulation or sensation in the lower limbs or if they had undergone any previous surgery, had any condition or displayed any symptoms, which resulted in abnormal gait. Significant foot deformity, callus, or reduced ROM in the joints of the lower limb also resulted in exclusion. To assess the inclusion/ exclusion factors discussed above, both a health questionnaire and physical assessment were carried out by a qualified podiatrist prior to data collection (Appendix Ciii & Civ). Quality of joint ROM was

assessed at the hip, knee, ankle, 1MTPJ and first ray; pedal pulses (posterior tibial and dorsalis pedis) and plantar sensation (heel, first, third and fifth MTPJ and hallux) were checked; and feet were inspected with regard to pedal skin condition (including callus) and presence of foot deformity. Assessment was completed for screening and comparison purposes only, and therefore findings were not included in any statistical analysis. Of those screened, 17 candidates were deemed suitable for selection, and subject parameter measurements (body weight, height, knee and ankle width, leg length) were subsequently obtained and recorded for each participant for entry into the Vicon Nexus software platform prior to data collection.

Ethical approval (Appendix Ci) was obtained from the Bioengineering departmental ethics committee, University of Strathclyde. Informed, written consent (Appendix Cii) was obtained from each participant prior to their participation.

6.1.3 Ethical issues, Hazards and Data Protection

Potential participants were provided with a participant information sheet (Appendix Cii), as well as having the study procedures explained to them verbally. They were then provided with the opportunity to ask any questions they had about the study, and were advised that they could withdraw their participation at any time. Potential hazards (risk of a trip or fall during completion of trials, skin reaction due to marker adhesive and slight discomfort should device be too tightly applied) were explained in the participant information form. The physical assessment (Appendix Civ) involved minimal physical contact and was performed by a qualified HPC registered podiatrist. A unique identification number was assigned to each participant. The consent form contained the participant's name, while all other documentation contained only the unique ID number to ensure anonymity. Documentation was viewed only by the researcher and was stored securely in a locked filing cabinet on university premises. Participants were asked to consent to having their data presented anonymously in this thesis (Appendix Cii).

6.1.4 **Gait Parameter Measurement**

Motion analysis was carried out using a Vicon MX Giganet 3D motion capture system (Vicon Motion Systems Ltd., Oxford UK) consisting of twelve (six T160, and six T40) wall mounted cameras positioned around a 10m walkway incorporating four (three 9182C and one 9261A) Kistler central floor set force platforms (Kistler Instruments, Hampshire, UK) measuring 600 x 400mm and each incorporating four piezoelectric quartz sensors (see Figure 6-1 for 3D motion analysis and force plate system set-up). Sampling was carried out at a frequency of 100Hz. The Vicon motion capture system allowed measurement of spatiotemporal and kinematic parameters while force plates were used to obtain kinetic data. Vicon Nexus software (v 1.8.1) enabled synchronisation of motion analysis and force plate data collection.

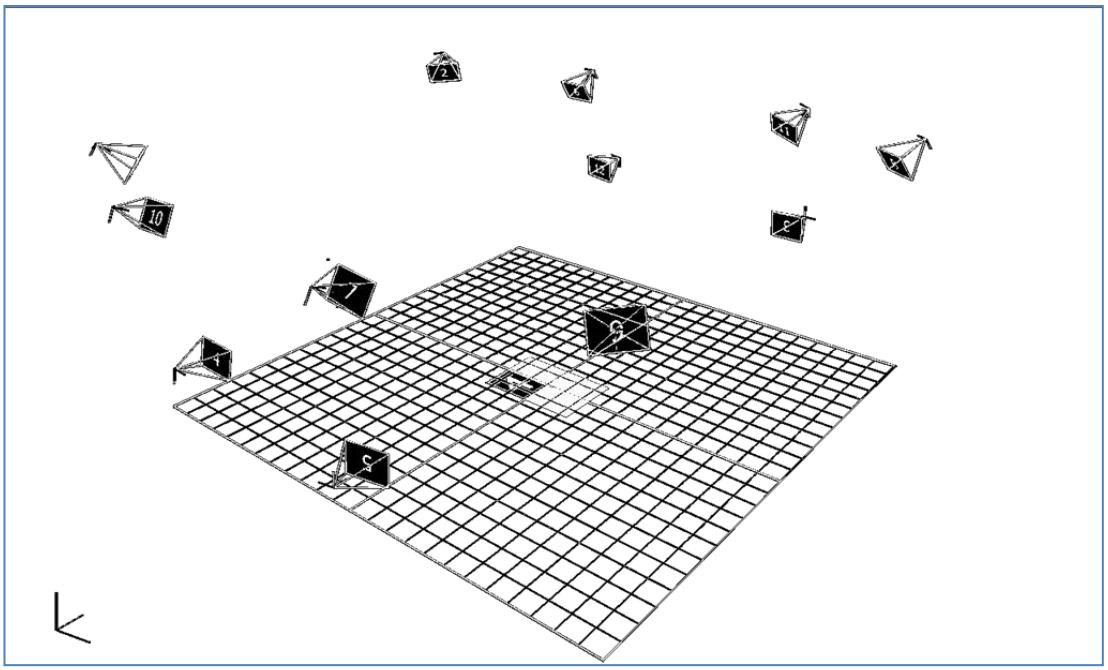


Figure 6-1 Virtual representation (Vicon nexus software screen dump) of camera and force plate set-up used for all trials.

The Vicon motion capture system has been widely used in gait labs to obtain quantitative information regarding a variety of gait parameters, and has been found

to produce good intra-class correlation and low standard error values when set-up using optimum system parameters (Meldrum, et al. 2012). Differences in intra- and inter-session kinematic measurements obtained with the Vicon motion capture system over a three year period, were attributed by Davenport and colleagues (2009) to better hardware and software, as well as changes in camera set-up and marker size. In their review investigating the reliability of motion analysis measurements, although McGinley, et al. (2009) noted clinically acceptable errors in a number of studies, in some cases they found inter-assessor and inter-trial values to be inconsistent. Authors determined that marker placement was the most influential factor regarding the accuracy and repeatability of results. Authors of a similar review (Tishya, et al. 2011) suggested that existing studies did provide evidence to support the efficacy of gait analysis systems; however, they did indicate that further randomised controlled trial studies were needed to confirm its use at the higher levels of efficacy. In general the aforementioned studies promote the view that correct system set-up and marker placement should enable accurate collection of intra-participant and intra-group gait parameter data with a Vicon motion capture system.

6.1.5 Experimental Procedures

Prior to data capture, a full set of 16 reflective markers were adhered to anatomical landmarks on the lower body of participants (Figure 6-2) as per Vicon Plug-In Gait model specifications (Vicon Motion Systems Limited 2010). 3D kinetic, kinematic and spatiotemporal data were captured simultaneously using a Vicon motion capture system and a Kistler force plate embedded within the trial walkway, both at a sampling frequency of 100Hz.

Before data collection, Kistler force plates were reset and Vicon system calibration and set-up were performed according to the manufacturer's instructions. Full calibration was completed, both dynamically and statically, prior to each trial set using a t-bar of known dimensions with markers placed at set positions on its surface. Subject calibration was achieved by performing a static data capture with

the participant standing in the centre of the capture volume with the full set of makers visible. Anthropometric participant parameters (previously measured by the researcher) were entered into Vicon Nexus software prior to data capture. Synchronised data capture, for both 3D motion and GRF measures, was conducted for each participant during completion of three normal walking trials along a straight, level walkway at a self-selected speed. Following initial control trials, the same participant was asked to apply the CTDSD to one foot (the right foot for all but one participant who, for hygiene purposes, wore the device on the left foot due to having a verruca on the right plantar foot surface). Participants were asked to self regulate the tension used when applying the device, thereby determining the degree of digital retraction produced. For comparative purposes retraction angle was measured at the 1MTPJ using a basic clinical tractograph. After CTDSD application three further (experimental) trials were similarly obtained. Both sets of trials were repeated until a total of three clean force plate strikes (for the desired foot) were captured for each.

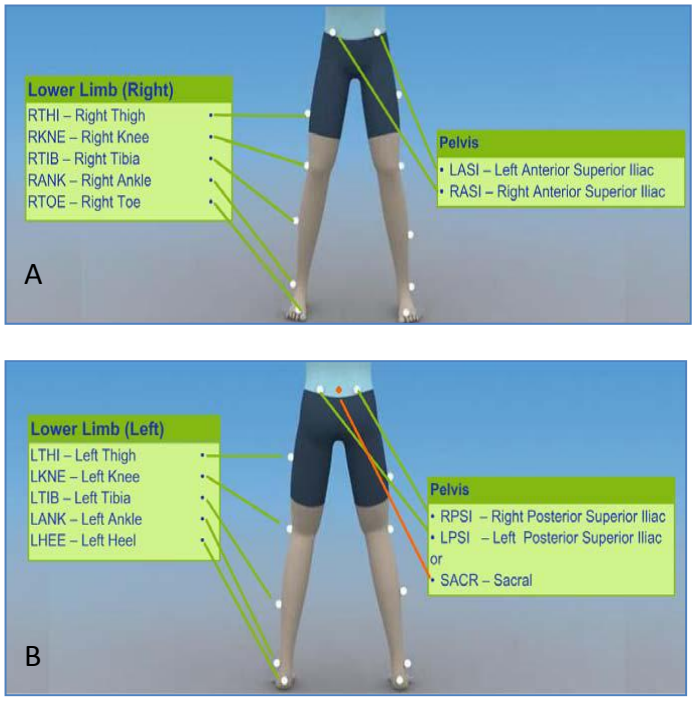
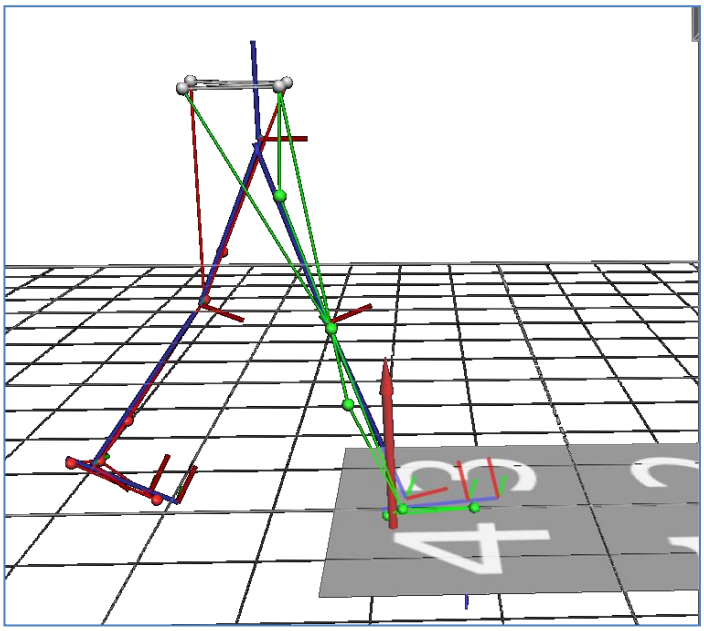
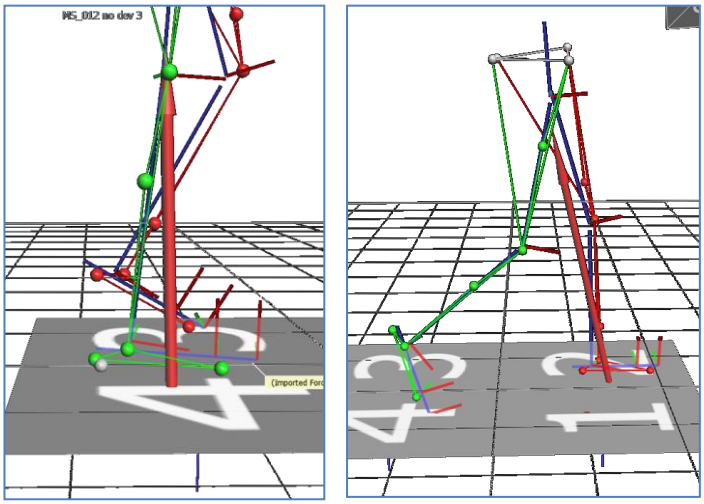


Figure 6-2 Plug-in Gait marker placement



(A)



(B), (C)

Figure 6-3 Vicon Nexus screen dumps showing labelled and reconstructed markers joined to form lower limb segments. GRF vector (red arrow) following initial contact. Clean force plate strike deemed as entire foot falling within force plate boundaries with no contact on force plate by contralateral foot (A). Midstance, i.e. point during stance at which heels are vertically aligned (B). Toe off in experimental limb (C).

Physical assessment, marker placement and trial data capture were performed in identical lab conditions, using the same equipment by a single researcher.

Following capture, all data was processed using Vicon Nexus software (Figure 6-4) prior to the relevant parameters being exported to MS Excel for analysis. Vicon

software was used to determine the locations of the ankle, knee and hip centres of rotation. Markers were reconstructed and manually labelled for all participant calibration files, allowing subsequent auto-reconstruction of trajectories for dynamic trials using the Plug-In Gait model (Figure 6-3). Gait events (Figure 6-3A, B and C) were identified and any gaps were filled. Data was then cropped to a single gait cycle (heel strike to heel strike), which included a clean force plate strike (Figure 6-3A) by the relevant foot.

Participant/ trial data was excluded if marker reconstruction and/ or gap filling for all markers was not possible; if labelling was inaccurate and could not be rectified (e.g. where calibration data incomplete) or if significant force plate data was absent. Following processing, data from twelve of the seventeen subjects tested was deemed to be suitable for analysis. Where data for one trial was incomplete, or appeared inaccurate, the intra-participant mean was obtained using data from the two remaining trials only (see Figure 6-4 for detailed processing method).

6.1.5.1 Parameter Measures

Gait characteristics exported for investigation included spatiotemporal, kinetic and kinematic parameters. Spatiotemporal measures consisted of walking speed, stride and step length, stride and step time, single (SSD) and double (DSD) support duration, and gait symmetry (SSD and DSD ratios). In terms of kinetic parameters (sagittal plane only), values were recorded for maximum and minimum hip (HMX1, HMX2), knee (KMX1, KMX2) and ankle (AMX1, AMX2) moments; vertical ground reaction force component (GRFz) first (FZ1) and second (FZ2) peaks, and anterior-posterior (AP) ground reaction component (GRFx) maximum (FX1) and minimum (FX2) values. Kinematic parameters obtained were: hip (HAX1), knee (KAX1) and ankle (AAX1) minimum, and maximum (HAX2, KAX2 and AAX2 respectively) joint angles (in the sagittal plane).

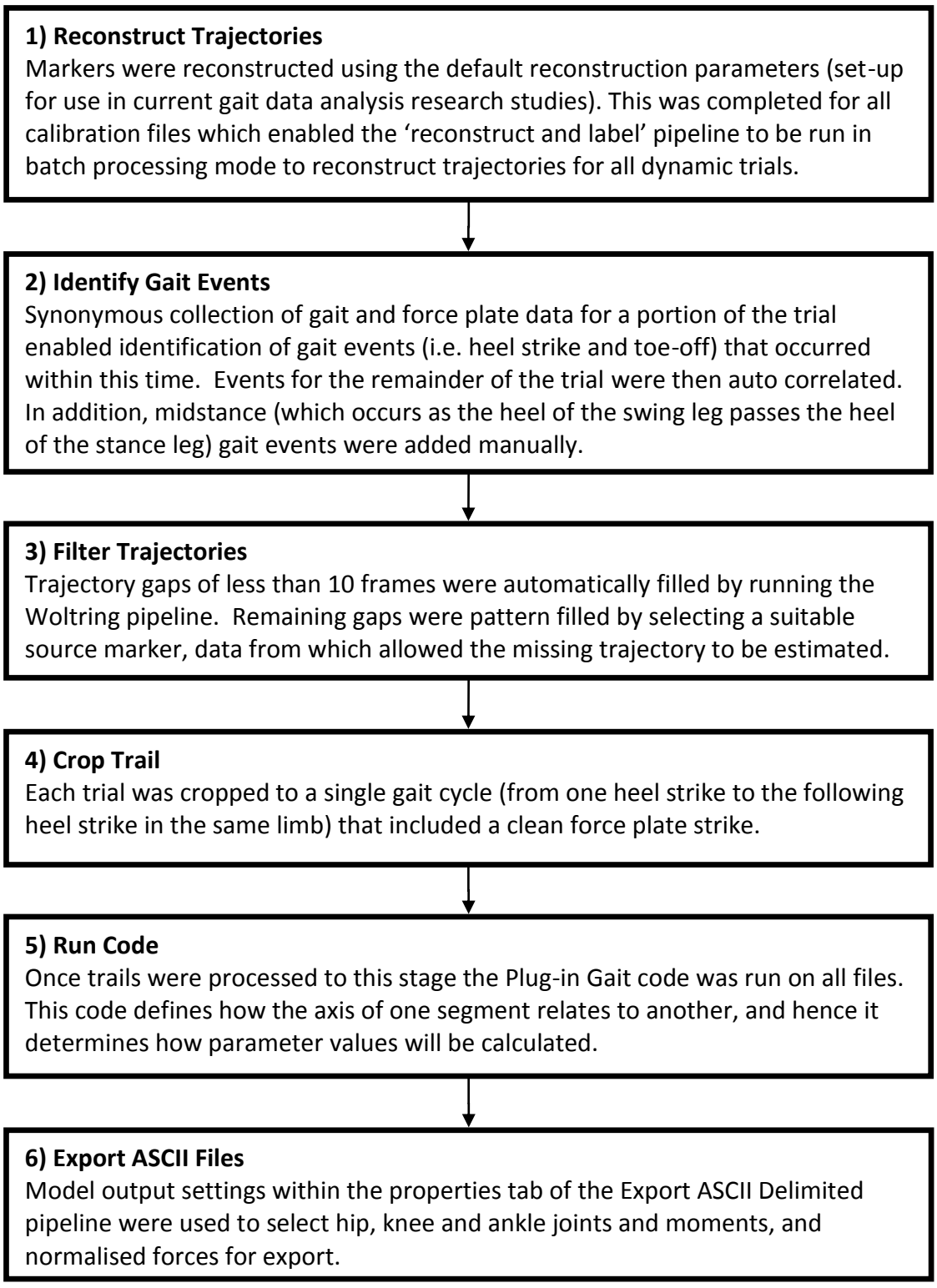


Figure 6-4 Processes involved in extracting the desired parameter measurements from raw data using Vicon Nexus software.

Stride length was defined as the distance for one gait cycle (e.g. between one heel strike (HS) and the following HS of the same limb). The distance between consecutive HSs in opposite limbs (i.e. between left and right HSs) was taken to be the step length. Single (SSD) and double (DSD) support duration represented the periods during which one, or both feet, respectively, were in ground contact. The SSD ratio was calculated by dividing the SSD measurement for one limb by the measurement for the contralateral limb in such a way that the value obtained was less than 1.00. The DSD ratio was similarly calculated.

The significant gait events referred to within this thesis are described in Figure 6-5 below. A combination of classic and new terms are used in this thesis to more clearly describe gait events. Abbreviations relating to the relevant gait events are as follows: new terms - initial contact, IC; loading response, LR; mid stance, MS; terminal stance, TS; preswing, PSW; initial swing, ISW; midswing, MSW; terminal swing, TSW. Classic terms – heel strike, HS; foot flat, FF; heel off, HO; toe off, TO.

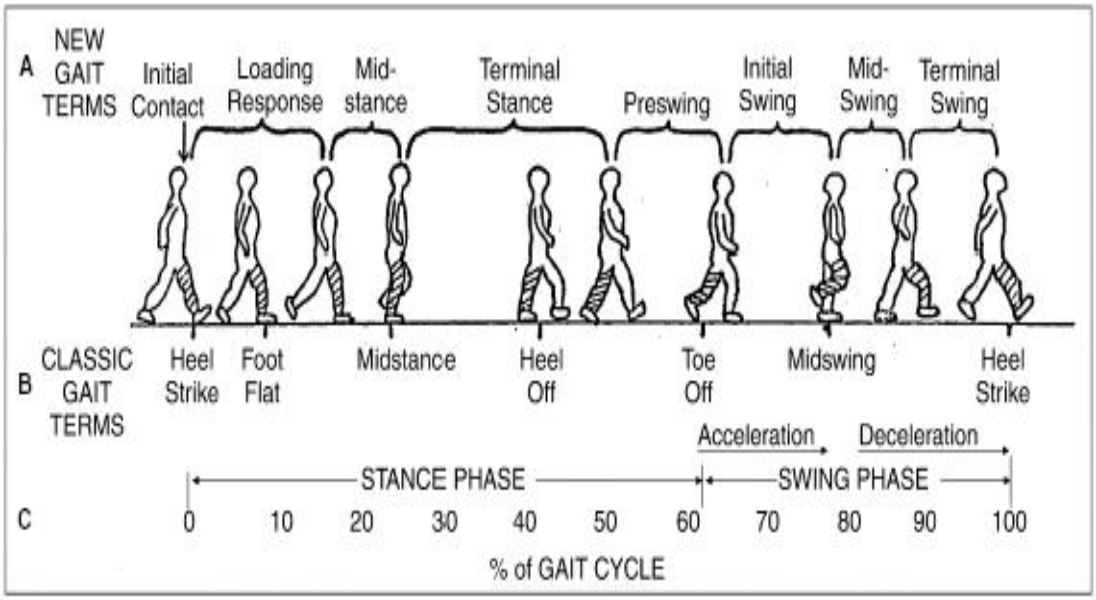


Figure 6-5 Events occurring during gait cycle swing and stance phases. New gait event terms (A), classic gait event terms (B), and timing of gait cycle events (C).

6.1.5.2 Statistical Analysis

Anthropometric data were expressed in terms of mean and standard deviation values. Both intra-participant (i.e. average data from the three trials completed for each subject) and intra-group (i.e. average of all intra-participant trial means for the relevant group) mean and standard deviation were calculated in Excel for each gait parameter. For each parameter Minitab was used to obtain descriptive statistics (mean, SE mean, SD, minimum, maximum) and to carry out paired t-tests for comparison of no device and with device mean values (i.e. before and after device application) in order to determine whether any differences were significant (i.e. $p<0.05$) at the 95% confidence level.

6.2 RESULTS

Demographic and anthropometric participant measures are shown in Table 1.

Table 1 Demographic and anthropometric data including mean and SD values. ND=no data.

PARTIC-IPANT	SEX	AGE (yr)	BODY WEIGHT (kg)	HEIGHT (mm)	LEG LENGTH (mm)	KNEE WIDTH (mm)	ANKLE WIDTH (mm)	1ST MTPJ ANGLE (°)
MS/001	M	22	70	1850	940	103	80	134
MS/002	M	23	89	1880	1000	105	81	124
MS/003	M	28	71	1725	910	95	68	102
MS/005	F	27	56	1655	880	94	64	129
MS/006	M	25	61	1720	930	97	76	132
MS/007	F	25	99	1685	970	125	68	148
MS/008	M	23	65	1755	940	98	70	166
MS/012	F	23	79	1800	1000	104	72	138
MS/014	M	22	62	1680	925	92	64	130
MS/015	F	36	54	1680	880	97	59	ND
MS/016	F	22	62	1810	970	98	69	102
MS/017	M	32	60	1720	910	99	75	ND
Mean		**25.667**	**69.000**	**1746.667**	**937.917**	**100.583**	**70.500**	**130.500**
SD		**4.438**	**13.678**	**72.843**	**40.758**	**8.649**	**6.640**	**19.167**

Participants each acted as their own controls, hence inter-group variations should have been associated purely with the effects of the CTDSD. Intra-group means, standard deviations, and p-values are shown in Table 2 for each spatiotemporal, kinematic and kinetic parameter. Additionally, intra-participant means for all gait parameters are shown in separate tables (i.e. divided into spatiotemporal, kinetic and kinematic parameters) in Appendix G, with the intra-group mean and standard deviation highlighted in bold.

Altered gait measures were noted between the no device and with device groups for all spatiotemporal, kinetic and kinematic parameters; however not all differences were significant. Paired t-tests were completed to identify any inter-group gait parameter differences, with significant p-values highlighted in Table 2 (*$p<0.05$, **$p<0.01$).

Descriptive statistics (count, mean, standard error, standard deviation, minimum, maximum) and paired t-test results for each gait parameter are provided in Appendix E (NB. with device group headings are denoted '_1'), while mean, interquartile values (including median), and outliers for all paired groups are displayed as box and whisker plots in Appendix F. Comparative bar charts showing mean intra-group measures for those parameters which demonstrated significant alterations when the CTDSD was applied are shown in Figure 6-8.

Gait parameter results have been divided into spatiotemporal, kinetic and kinematic parameters for further discussion below.

6.2.1 Spatiotemporal Parameters

Table 2 shows quantitative spatiotemporal parameter data for both the experimental and control groups. It can be noted, that following application of the device, participants experienced a reduction in walking velocity, as well as ipsilateral stride and step length, coupled by an increase in both stride and step time. Whilst both single (SSD) and double (DSD) support duration were increased in the contralateral limb (left limb for all participants except MS_017 where this was the

right limb) and reduced in the experimental limb (right limb for all participants except MS_017 where this was the left limb). In addition, gait symmetry was assessed in terms of a decrease in both single and double support duration ratios. Nonetheless, the only spatiotemporal gait parameter found to demonstrate a significant alteration was walking speed (Figure 6-8) which decreased from 1.28m/s in the control group, to 1.207m/s in the experimental group ($p<0.032$).

6.2.2 Kinetic Parameters

Comparative graphs displaying vertical (GRF_Z) and anterio-posterior (GRFx) ground reaction force (GRF) components are provided in Appendix D for each participant. Both experimental and control group values are displayed on the same graph to aid comparison (GRFx, lowermost; GRFz, uppermost; no device, dotted line; with device, solid line). Figure 6-6 provides one such graph (from subject MS_003) which illustrates GRFx and GRFz components before and after device application, and also indicates the relevant data points (FX1, FX2, FZ1, and FZ2) selected for comparison in this study. Similarly, sample graphs (subject MS_003) for each joint angle parameter considered are given in Figure 6-7. All sample graphs are provided to provide a clear visual representation of the specific points chosen for analysis, and any inter-group parameter changes measured; however, it must be noted that the data displayed may vary between participants, and is not necessarily representative of normal values.

In general, kinetic parameters (i.e. GRF components and joint moments) recorded in the experimental group exhibited shallower troughs and peaks than those in the control group. However, in terms of significant changes, these were noted only for FX1 ($p<0.002$), FZ2 ($p<0.004$), AMX1 ($p<0.015$) and KMX2 ($p<0.013$) measures (Figure 6-8). When the CTDSD was applied, FZ2 and FX1 decreased from 113.87% body weight (bw) to 108.19%bw, and from 21.30%bw to 17.82%bw respectively. Whilst, for joint moments, a reduction in both the AMX1 peak (from 1.54 to 1.34 Nm/kg), and the KMX2 trough (from -0.49 to -0.43 Nm/kg, i.e. values moved closer to zero) were observed when compared to the control group.

6.2.3 Kinematic Parameters

Sample graphs (subject MS_003) are further provided for each joint moment parameter (Figure 6-7) to enable visualisation of inter-group differences. As for previous sample graphs, relevant peak and minimum points measured for inter-group comparison of parameters have been identified.

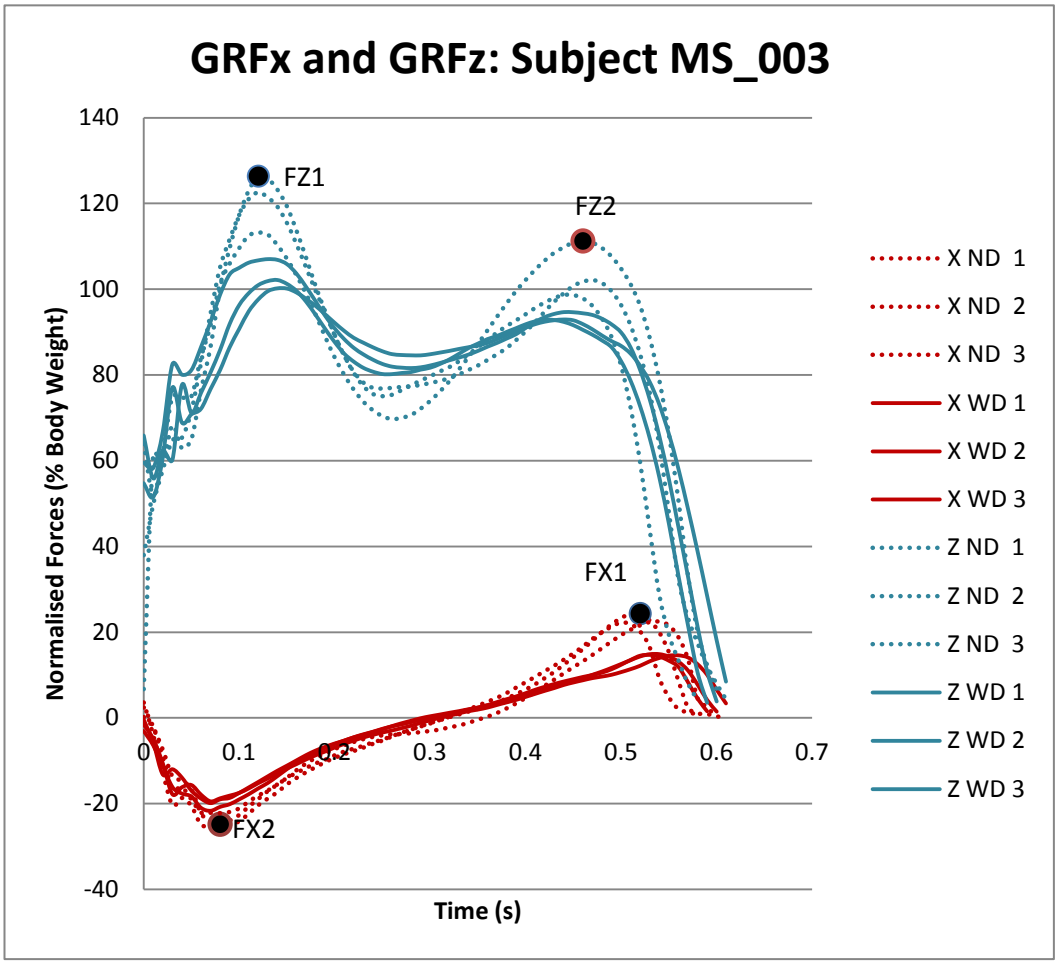
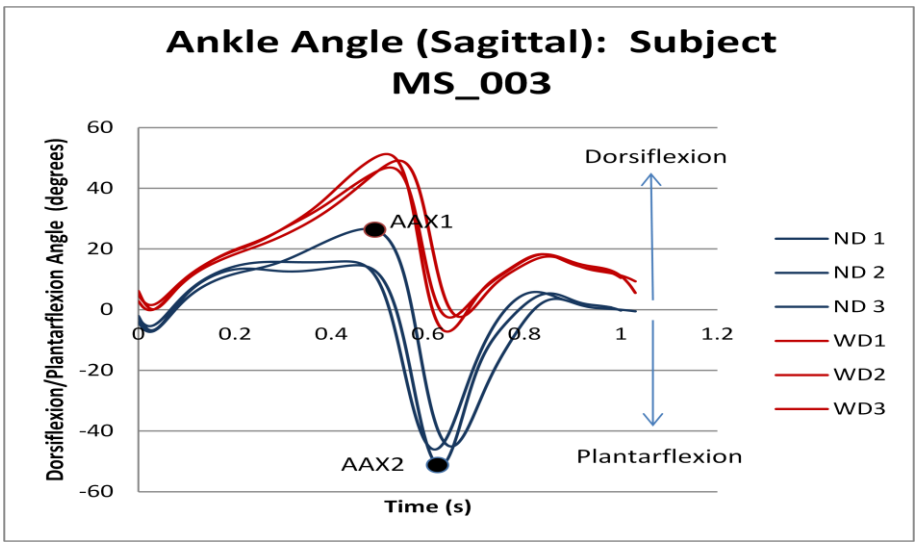
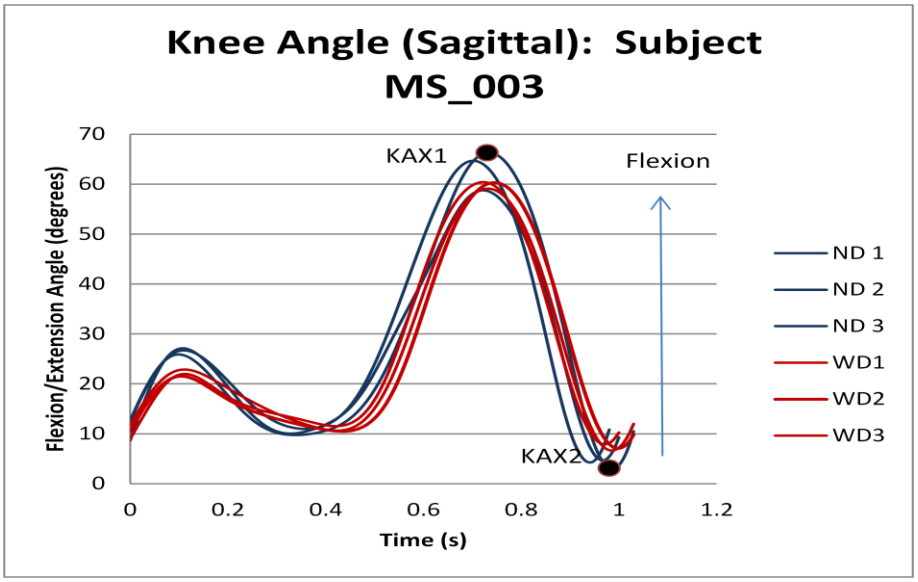
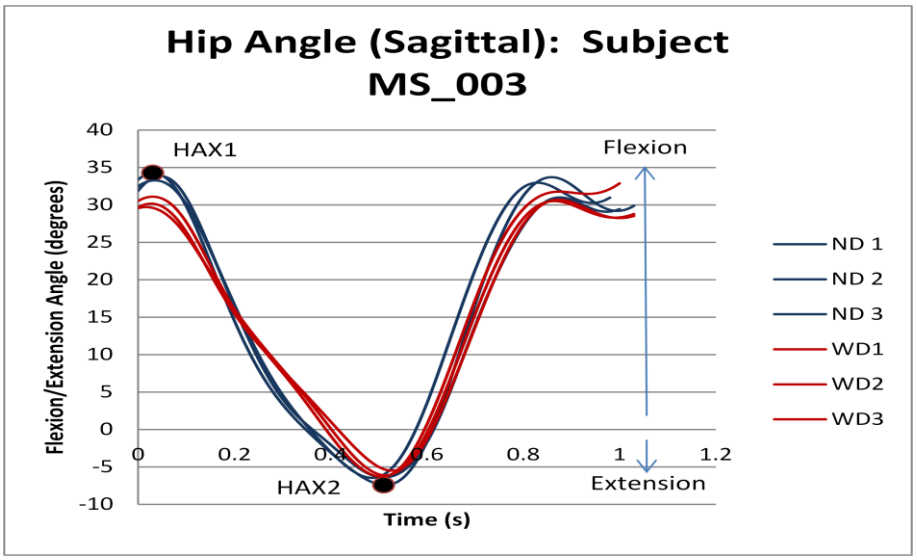
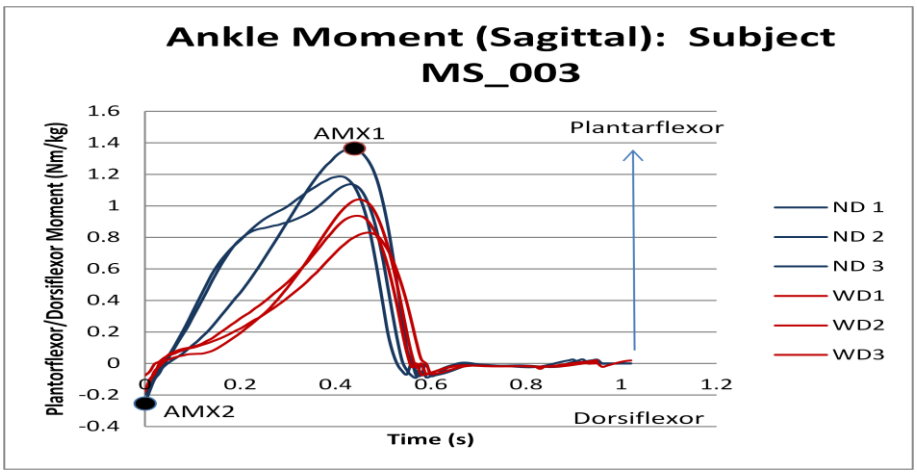


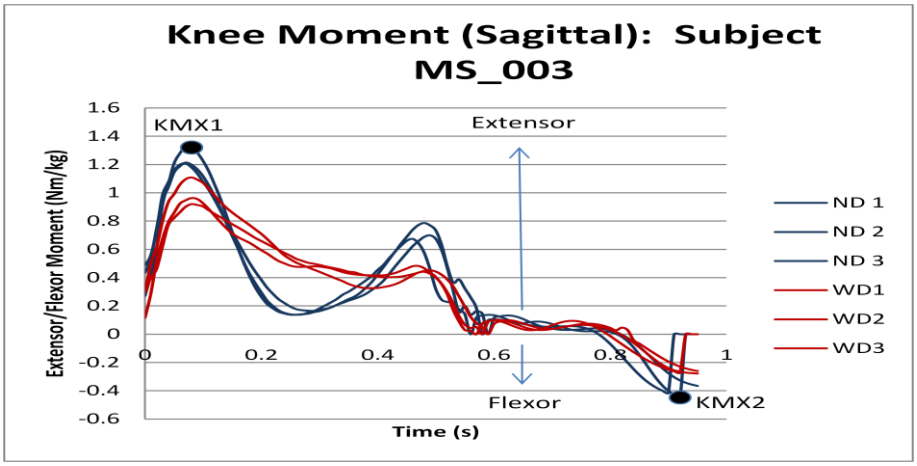
Figure 6-6 Sample vertical (Z, blue) and AP (X, red) GRF graphs for one participant (MS_003) before (dotted line) and after (solid line) CTDSD application. Kinematic parameters measured in relation to GRF were: GRFz first (FZ1) and second (FZ2) peaks, and GRFx peak (FX1) and minimum (FX2) values (shown as black dots).


Ankle Angle (Sagittal): Subject MS_003
Dorsiflexion/Plantarflexion Angle (degrees)
60
40
20
0
-20
-40
-60
0
0.2
0.4
0.6
0.8
1
1.2
Time (s)
AAX1
AAX2
Dorsiflexion
Plantarflexion
ND 1
ND 2
ND 3
WD1
WD2
WD3


Knee Angle (Sagittal): Subject MS_003
Flexion/Extension Angle (degrees)
70
60
50
40
30
20
10
0
0
0.2
0.4
0.6
0.8
1
1.2
Time (s)
KAX1
KAX2
Flexion
ND 1
ND 2
ND 3
WD1
WD2
WD3


Hip Angle (Sagittal): Subject MS_003
Flexion/Extension Angle (degrees)
40
35
30
25
20
15
10
5
0
-5
-10
0
0.2
0.4
0.6
0.8
1
1.2
Time (s)
HAX1
HAX2
Flexion
Extension
ND 1
ND 2
ND 3
WD1
WD2
WD3





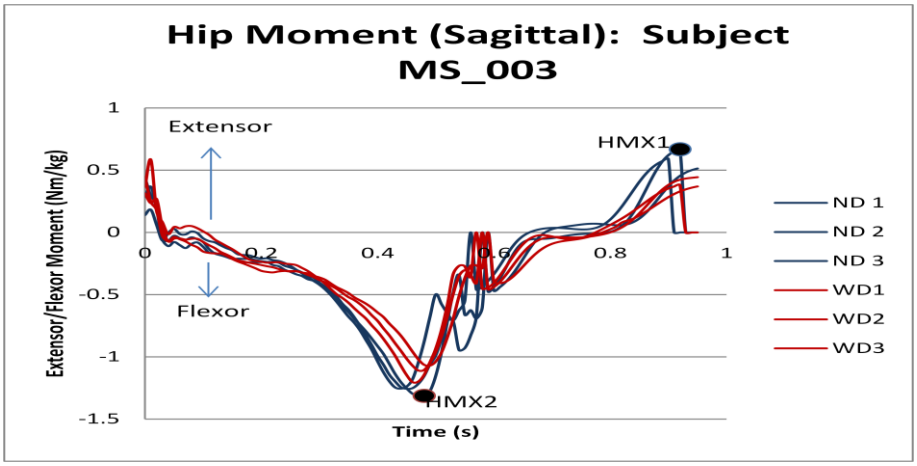
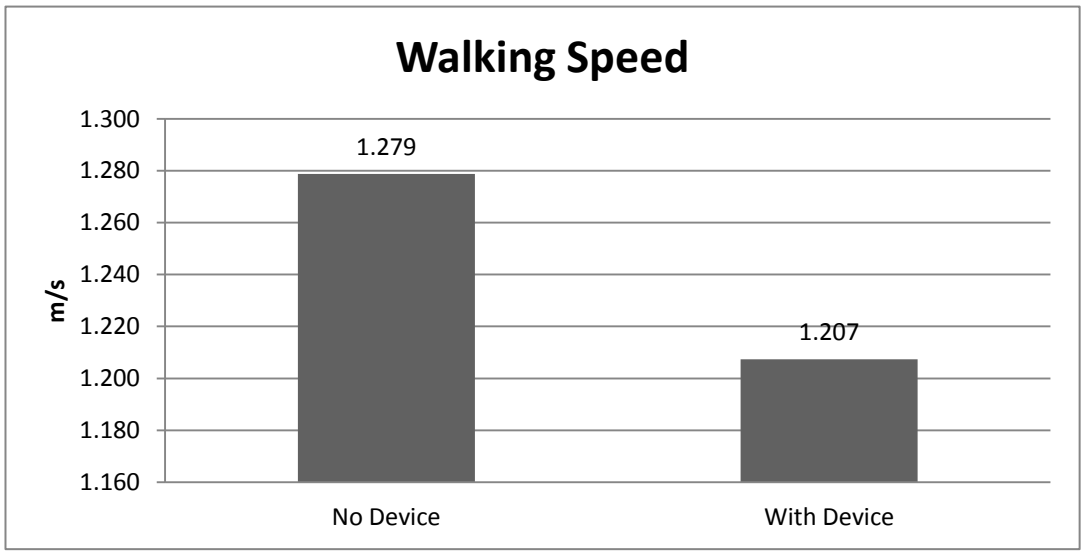
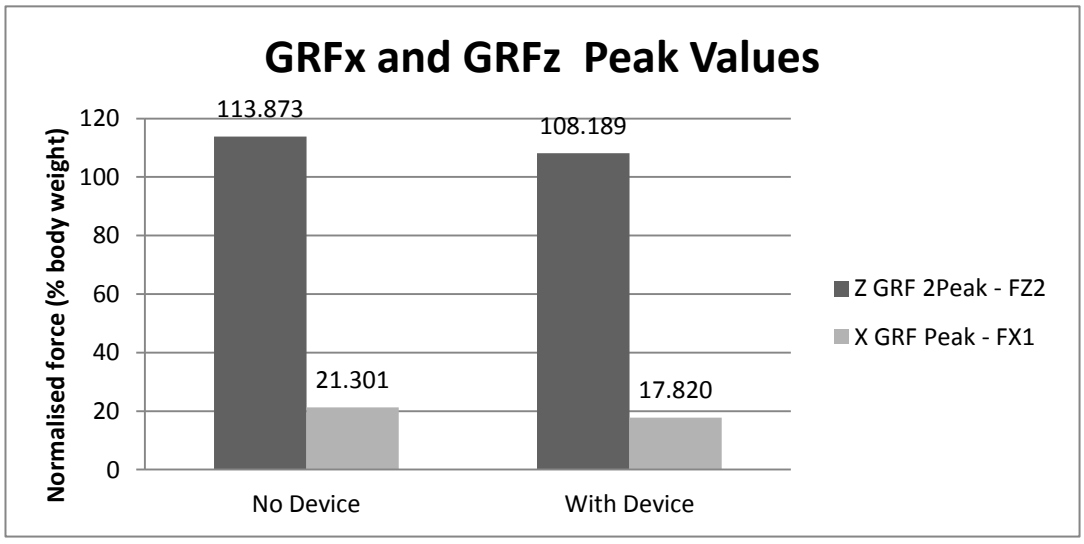
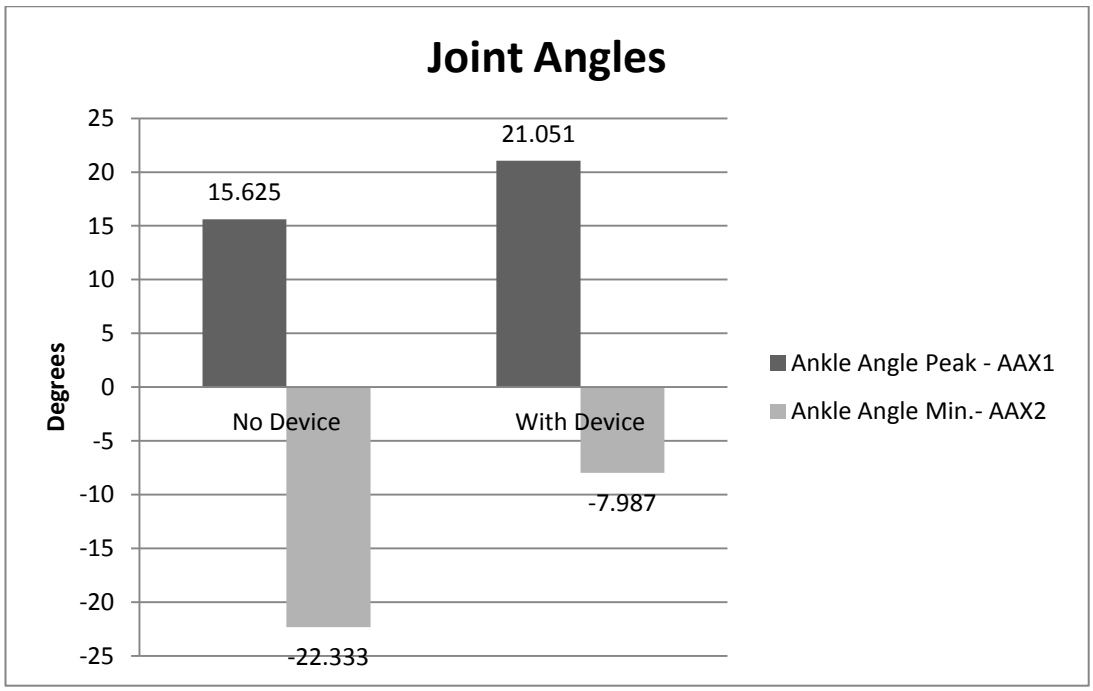


Figure 6-7 Sample (participant MS_003) gait parameter (joint angles and joint moments) graphs showing measures for three normal trials (blue line) and three CTDSD trials (red line). Peak and minimum values measured for comparison in this study are shown on each graph (black dots). Note that for joint angles flexion/dorsiflexion angles are positive, whilst for joint moments, extensor/plantarflexor moments are positive.

A similar pattern of change to that noted for kinetic measures was also observed in relation to kinematic parameters; namely, that the magnitude of parameter peaks and troughs was reduced in participants wearing the CTDSD. Significant changes were noted for AAX1, which increased to 21.05 degrees from 15.63 degrees ($p<0.042$), and AAX2 which decreased from -22.33 to -7.99 degrees ($p<0.001$) (see Figure 6-8).







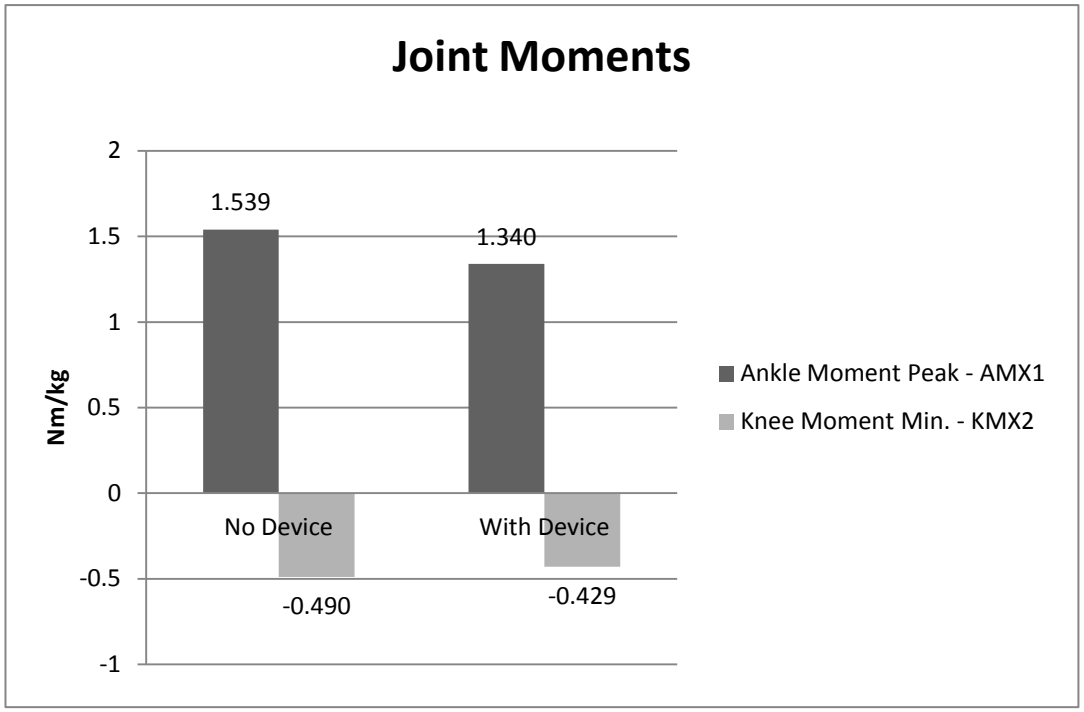


Figure 6-8 Bar charts comparing no device and with device group means for each gait parameter that demonstrated a significant change (i.e. where p<0.05).

Table 2 Intra-group gait parameter values (mean, SD, SE of mean) for no device and with device groups (*= p < 0.05, **= p < 0.01)

PARAMETER	TRIAL TYPE						T-TEST
	No Device			With Device			
	Mean	SD	SE	Mean	SD	SE	P-Value
Spatiotemporal							
Walking Speed (m/s)	1.28	0.15	0.04	1.21	0.18	0.05	0.032*
Stride Length (m)	1.40	0.13	0.04	1.33	0.16	0.05	0.06
Step Length (m)	0.70	0.06	0.02	0.67	0.08	0.02	0.06
Stride Time (s)	1.10	0.08	0.02	1.11	0.10	0.03	0.17
Step Time (s)	0.55	0.04	0.01	0.56	0.05	0.01	0.27
Left SSD (s)	0.44	0.03	0.01	0.46	0.04	0.01	0.13
Right SSD (s)	0.44	0.03	0.01	0.43	0.06	0.02	0.20
Left DSD (s)	0.21	0.04	0.01	0.22	0.04	0.01	0.23
Right DSD (s)	0.21	0.04	0.01	0.23	0.04	0.01	0.09
SSD Symmetry (ratio)	0.96	0.03	0.01	0.92	0.07	0.02	0.16
DSD Symmetry (ratio)	0.95	0.03	0.01	0.94	0.03	0.01	0.80
Kinetic							
GRFz Peak 1 (FZ1)(%bw)	112.28	10.21	2.11	108.35	10.05	1.82	0.40
GRFz Peak 2 (FZ2)(%bw)	113.87	6.24	14.30	108.19	8.33	9.30	0.004**
GRFx Peak (FX1)(%bw)	21.30	4.57	4.27	17.82	3.91	0.08	0.002**
GRFx Min (FX2)(%bw)	-20.48	5.01	13.90	-17.71	4.24	13.50	0.43
Ankle Moment Peak (AMX1)(Nm/kg)	1.54	0.19	0.54	1.34	0.27	0.08	0.015*
Ankle Moment Min (AMX2)(Nm/kg)	-0.30	0.33	0.10	-0.24	0.16	0.05	0.53
Hip Moment Peak (HMX1)(Nm/kg)	0.81	0.25	0.07	0.77	0.33	0.09	0.73
Hip Moment Min (HMX2)(Nm/kg)	-1.09	0.48	0.14	-0.87	0.36	0.11	0.10
Knee Moment Peak(KMX1)(Nm/kg)	0.85	0.49	0.14	0.74	0.35	0.10	0.31
Knee Moment Min (KMX2)(Nm/kg)	-0.49	0.17	0.50	-0.43	0.16	0.45	0.013*
Kinematic							
Ankle Angles Peak (AAX1)(degrees)	15.62	3.63	1.09	21.05	9.71	2.93	0.042*
Ankle Angles Min (AAX2)(degrees)	-22.33	9.61	2.90	-7.99	7.40	2.23	0.001**
Hip Angles Peak (HAX1)(degrees)	27.92	9.69	2.92	33.93	7.56	2.28	0.16
Hip Angles Min (HAX2)(degrees)	-9.11	8.71	2.63	-10.40	9.75	2.94	0.41
Knee Angle Peak (KAX1)(degrees)	62.19	5.72	1.72	59.50	6.05	1.82	0.23
Knee Angle Min (KAX2)(degrees)	-0.61	10.90	3.29	1.09	11.76	3.55	0.06

6.3 DISCUSSION

The main aim of this thesis was to determine whether CTD results in significant changes to the gait patterns of DM patients. In order to isolate the effects of CTD from other co-existing DM complications, CTD was simulated in healthy participants by applying a device to one foot. A range of spatiotemporal, kinetic and kinematic gait parameters were investigated (Table 2, Appendix G). Please refer to Figure 6-5 in relation to the gait events (and abbreviations) used throughout this discussion.

In general, participants displayed changes in all gait parameters tested when comparing control and experimental trial data; however, relatively few of these differences were found to be significant. With regard to spatiotemporal parameters only walking speed was significantly affected, while kinematic gait alterations included AAX1 and AAX2 joint angle measures. Statistically significant kinetic changes included FZ2 and FX1 ground reaction force component measures, and AMX1 and KMX2 joint moment values (Figure 6-8).

6.3.1 Spatiotemporal Gait Parameter Changes

Evidence of decreased gait velocity in the CTDSD group, is supported by findings in a number of studies investigating gait changes in DM individuals (Sawacha, et al. 2009; Allet, et al. 2008; Brach, et al. 2008). Contrastingly, Mickle and associates (2011), who compared older people with toe deformities (hallux valgus and claw toe) with a healthy control group, failed to find differences in any spatiotemporal parameters including walking speed. Since authors did not specify the extent or severity of deformity in their report it is difficult to draw firm conclusions; however, findings of this thesis would suggest that when CTD is simulated across all digits, this results in a significant reduction in walking speed. Since healthy participants were used it can also be inferred that the effects of deformity on gait velocity are independent of any other co-existing pathologies that may be present in DM individuals. This reduction in gait speed may be attributed to discomfort experienced as a result of MTH prominence (i.e. due to the retracted position of the

digits). Slower speed may be adopted to reduce the inertial forces (i.e. due to COM acceleration) which contribute to the GRF vector magnitude, thereby minimising pain and discomfort. Studies in DMN and DM individuals with CTD would provide evidence regarding any compensatory gait changes employed to avoid discomfort (since compensation would not be expected in an insensate foot).

Previous research has shown that changes in walking speed may have significant effects on a variety of other gait parameters (Kirtley 2006), including joint angles (Savelberg 2010) and GRFs (Shaw, et al. 1998). It must therefore be taken into consideration whilst reading the following discussion below that any significant inter-group modifications observed in the kinetic or kinematic parameters may be attributable, at least in part, to the effects of reduced gait velocity.

6.3.2 Kinetic Gait Parameter Changes

It can be noted from the sample (MS_003) GRFx/ GRFz graph (Figure 6-6), that the general pattern of change observed following CTDSD application, for both vertical and AP GRF components, involved a relative reduction in peak and minimum values (i.e. smaller peaks and troughs).

6.3.2.1 Vertical Ground Reaction Force Component (GRFz)

Although example subject (MS_003) data exhibited a decrease in both first (FZ1) and second (FZ2) GRFz peaks, as well as a shallower between peak trough (not investigated in this thesis), the only significant change in inter-group values involved the FZ2 mean. Figure 6-6 illustrates that the FZ2 peak normally exceeds bodyweight (bw) (no device, FZ2 max. = 111% bw); however a maximum of only around 95% bw was evident in the experimental group. In general terms the GRFz peaks and trough occur as a result of changes in the vertical acceleration of the body centre of mass (COM) during stance (Winiarski and Rutkowska-Kucharska 2009), which will increase or decrease the GRFz above or below bw (i.e. $F = mg \pm ma$). Since it has already been demonstrated that the experimental group exhibited slower gait velocity, it is also likely that the gait pattern was generally more conservative, which could

account for the lower COM acceleration produced during PSW. If the force caused by the forefoot pushing against the ground at TO were to decrease, a reduction in FZ2 would be expected (asnoted in this study). It could therefore be the case that GRF component changes may occur simply as a result of reduced gait velocity.

Altered GRF measures would also occur if altered digital alignment, due to the CTDSD, were to restrict TO via exaggeration of the windlass effect (Perry 1992). In functional terms the windlass effect produces digital dorsiflexion, which causes approximation of the rearfoot and forefoot, resulting in heightening of the arch and MTH prominence (all of which are further exaggerated following HO). In the presence of CTD, the digits (which usually transmit a significant proportion of bw following HO) are unable to achieve ground contact and therefore will not contribute to vertical propulsion at PSW (including TO at the hallux). In addition, since body weight cannot be effectively transferred from the rearfoot to the forefoot, the ankle is unable to achieve a full range of plantarflexion. These factors act to reduce the force transmitted by the forefoot to the ground (and therefore the GRFz) at TO and will therefore dampen the FZ2 peak. This theory is also supported by the preliminary study results, which demonstrated that digital PPPs were notably reduced during CTDSD trials (Figure 5-9, Figure 5-10).

6.3.2.2 Anterior-posterior Ground Reaction force Component (GRFx)

With regard to the GRFx (Figure 6-6 and Appendix D), changes in peak PSW values (FX2) were found to be significant. GRFx describes the GRF component that occurs horizontally and parallel to the line of progression (i.e. due to friction). By convention, GRFx forces that are directed forwards are positive, and backwards are negative. Figure 6-6 illustrates the approximate GRFx pattern that would be expected during normal gait (no device; red, dotted line). An initial trough (FX1) occurs as body weight is transmitted through the heel at HS producing a posteriorly directed/ negative GRFx (i.e. AP braking force). During PSW a peak (FX2) is again observed as a result of rapid plantarflexion (employed to drive the leg forward into

swing), which causes an anteriorly directed, positive GRFx (i.e. AP propulsive force). As such, in relation to the GRFx, a decrease in min./ peak values may occur with a slower walking pace since this requires both smaller propulsive forces, and results in lower IC forces (due to reduced swing leg inertial forces). In normal gait, following HS, weight is transferred along the lateral aspect of the foot and at HO is medially displaced across the MTPJs until finally being transmitted through the hallux at TO. In a healthy foot the digits therefore play an important role in AP propulsion, by accelerating the COM forwards. However, if CTD is present, it is proposed that MTPJ prominence will lead to discomfort when plantarflexion (controlled by the superficial calf muscles: gastrocnemius, soleus, plantaris) is triggered to initiate HO, since this shifts bodyweight forward onto the forefoot, increasing the proportion of forces transmitted through the MTHs. Additionally, the deep calf muscles (tibialis posterior, flexor digitorum longus and flexor hallucis longus) are prevented from facilitating toe off, since their forces may only be exerted if the digits are stabilised against the ground. These factors combine to limit the AP forces transmitted via the foot to the ground at TS, and hence the GRFxs which oppose these (Figure 6-6).

It is further theorised that in order to reduce discomfort, the three functional rockers (see glossary) (Perry 1992), normally utilised during stance to propel the body forwards, may be avoided in CTDSD individuals, through adoption of a steppage type motion. Steppage gait is usually associated with reduced tibialis anterior, or abnormal peroneal, function (e.g. as observed in poliomyelitis) resulting in a requirement for a greater vertical hip and/or knee excursion to enable foot clearance (Merriman 2002). A steppage pattern also contributes to a reduction in GRFx by maintaining the trunk (and therefore COM) in a more posterior position. This limits forward momentum, and hence allows the swing leg to be lowered to the ground in a controlled manner (i.e. lower shear braking forces), via hip and knee extension in the swing leg with simultaneous knee flexion and ankle dorsiflexion in the stance leg (Figure 6-9). The abovementioned theory also supports the remaining kinematic gait alterations identified in this study.

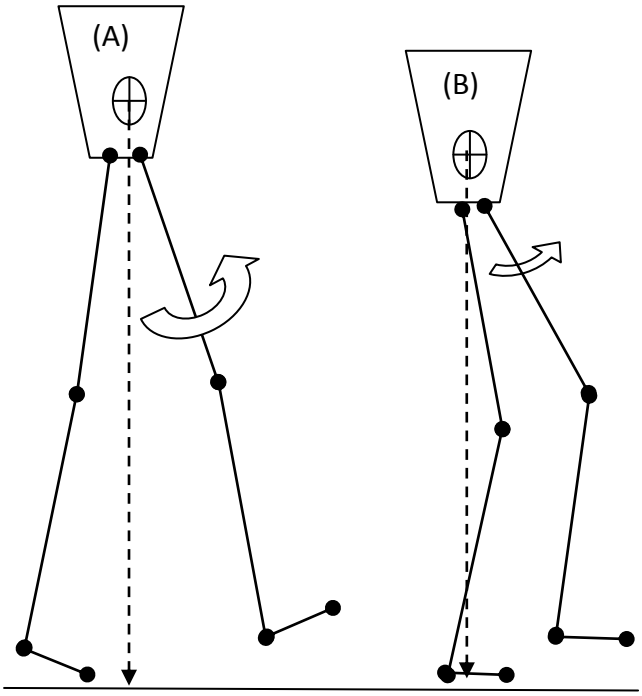


Figure 6-9 Effect of trunk (and hence COM) position. When COM (crossed circle) is forward of the stance leg this will produce forward displacement of the tibia due to the effects of gravity (dotted line) on the COM, and will require a plantarflexor moment (soleus) to control dorsiflexion. Continued tibial translation and deep calf muscle control of dorsiflexion, with associated knee flexion by the gastrocnemius muscle will eventually lead to heel lift. The swing leg, initially in a hyperextended position prior to toe off, will be propelled forward once the limb leaves the ground due to momentum arising from the anterior position of the COM (A). When COM is more posteriorly placed (e.g. steppage gait) gravitational effects are minimised and hence tibial acceleration and dorsiflexor moment are reduced. The swing leg also has less momentum and can be 'lifted' and placed into initial contact by flexing the stance knee and hip and dorsiflexing the ankle (B).

6.3.2.3 Sagittal Plane Joint Moments

Significant alterations in joint moment parameters were noted for the AMX1 peak and KMX2 minimum only (Table 2). The AMX1 moment refers to the internal plantarflexor moment that is required increasingly throughout stance phase to control the external dorsiflexor moment brought about by tibial forward progression. Tibial displacement is caused by gravity acting on the body's COM,

particularly once it has moved forward of the stance foot (which further influences the GRFz). CTDSD intra-group measures demonstrated a reduced AMX1 peak when compared with control group data (see relevant charts in Figure 6-7 and Figure 6-8). This suggests that the plantarflexor forces usually generated (by the gastrocnemius and soleus muscles) to counter the external dorsiflexor moment were reduced when the CTDSD was worn. One explanation for the modified AMX1 moment again involves the previous steppage gait theory. If a steppage type gait were employed, this would result in a more posterior trunk position with the knee rarely passing anterior to the forefoot of the stance limb. Consequently, forward displacement of the COM (and hence the tibia) would decrease, leading to a reduction in the external dorsiflexor moment, and therefore requiring smaller plantarflexor forces to counter this (Figure 6-9). Alternatively, the shallower ankle moment trough may simply correspond to reduced plantarflexor muscle activity in an attempt to delay HO and hence painful forefoot load bearing during the PSW phase of stance. One thing to note is that we would have expected an increased internal plantarflexor moment to counter the dorsiflexor moment occurring as a result of the action of the CTDSD (i.e. due to tension generated by the device between the digits and posterior ankle). This was not observed, and in fact the plantarflexor moment was reduced with the device. It must however be considered that in an improved CTDSD design (i.e. one which does not induce a moment about the ankle) the muscular moments measured at the ankle may differ from those recorded in this study.

Minimum knee moment (KMX2) was the final kinetic parameter to demonstrate a significant difference (trough shallower in CTDSD group) (see relevant charts in Figure 6-7 and Figure 6-8). KMX2 may be observed as the negative peak in Figure 6-7 (see knee moment graph) which corresponds to the internal (i.e. muscle or knee ligament) flexor moment, and which acts about the knee joint axis to counter any external (i.e. GRF, inertial force) extensor moments. Again, the change noted in this parameter corroborates the steppage gait theory. During swing, external knee extensor moments in the swing leg are usually high due to both inertial forces developed around TO, and the simultaneous action of the hip flexors. Therefore, in

TSW, deceleration of the tibia (and thigh) must occur prior to IC, which is achieved through an internal flexor moment generated at the knee by the hamstrings and posterior ligaments. Employment of steppage type gait produces a posteriorly located COM, and has the combined effect of dampening swing leg inertial forces and enabling a gait pattern that involves 'stepping' rather than 'swinging' the leg (Figure 6-9). Therefore, the knee flexor moment required to decelerate TSW knee extension will be reduced, which corresponds to the findings of this study.

6.3.3 Kinematic Gait Parameter Changes

6.3.3.1 Sagittal Plane Joint Angles

A significant increase was noted in the AAX1 peak (Figure 6-7 and Figure 6-8), which occurs in TS just prior to HO. The increase in AAX1 may be explained simply by the action of the CTDSD to induce a dorsiflexor moment about the ankle joint. This would account for the increased dorsiflexion angle noted throughout the gait cycle. However, inter-group differences may also be attributable to increased ankle dorsiflexion (and knee flexion) required in the stance limb to lower the swing foot into ground contact in an individual with a steppage type gait pattern (see Figure 6-9). In addition, increased dorsiflexion in the stance limb would keep the COM low thereby improving balance. Similarly, a delay in the transference of weight between limbs during LR would increase stability and would also require increased dorsiflexion. A further alternative explanation would be that in order to reduce the discomfort associated with transferring weight onto the forefoot (and prominent MTPJs), the foot spends a longer period in the foot flat position, delaying HO, and so requiring a greater range of dorsiflexion to maintain heel contact as the proximal tibia moves forward over the dorsum of the foot.

Reduction in the AAX2 minimum (i.e. plantarflexion angle) (Figure 6-8) may also be accounted for due to the effects of the CTDSD in terms of creating a dorsiflexor moment about the ankle, thereby reducing the degree of plantarflexion achievable. Alternatively, changes may be explained again by a steppage gait pattern, since this

would involve the stance foot being lifted into swing, rather than utilising the third rocker mechanism (see glossary for definition, Perry 1992; and Figure 6-9) to propel the limb forward and upwards. As such the foot would remain in a more neutral or dorsiflexed position at PSW. Finally, the decrease in AAX2 may be explained by the fact that plantarflexion will act to shift weight from the heel towards the digits. Therefore, as previously discussed (see sections 6.3.2.1 and 6.3.2.2), individuals may simply be trying to minimise plantarflexion to avoid the instability and pain associated with distal transfer of body weight. Figure 6-7 illustrates that in the CTDSD group, ankle angle values were more positive (i.e. plantarflexion was reduced) over the whole period of the gait cycle, thereby supporting the aforementioned hypotheses.

6.3.1 General Issues

Although differences existed between all parameter group means, only seven were significant. It has been shown that both the reduction in GRFx and GRFz, noted in this study during late TS and PSW, and the decrease in the ankle plantarflexor and knee flexor moments all support the steppage gait theory proposed. Similarly joint angle changes (i.e. increased ankle angle peak and reduced ankle angle minimum) give some credence to this theory. However, if we presume that the steppage gait theory proposed is factual, in addition to the aforementioned findings we would also have expected to detect significant alterations in a greater number of gait parameters. For example, a reduced hip extensor moment at TSW may occur due to reduced inertial forces on the swing leg; an increased knee extensor moment at late TS may be caused since the posteriorly positioned COM would result in a GRF vector posterior to the knee joint axis, resulting in a knee flexor moment; increased hip flexion may occur, both in the swing leg to enable adequate foot clearance, and in the stance leg to allow controlled lowering of the swing leg into ground contact; and a decreased step length would be expected due to reduced inertial forces acting on the swing limb. None of the aforementioned changes were observed in this study. These absences may be due to a range of factors including: imprecise

inter-participant marker placement; inaccurate calculation of joint centres by Vicon Nexus software, producing corresponding inaccuracies in joint parameters; variation in CTDSD placement and tightness, resulting in inconsistent inter-participant digital retraction; failure of CTDSD to simulate anatomical CTD; small study sample and limited parameter analysis (e.g. only max. and min. values measured). Some of these factors will be discussed in more detail in the following study limitations section (6.4).

This study demonstrated that in healthy adults, the CTDSD did bring about significant changes in gait pattern, including alterations in walking speed, GRFs, joint moments and joint angles. This may have been as a result of gait adaptations employed in order to both avoid pain, and maintain balance and stability, in the presence of altered foot function, particularly during TS and PSW. Regardless of the precise causes which underlie these gait modifications, each is likely to result in changes in the distribution of pressure over the plantar surface of the foot, and hence may ultimately influence a person's risk of developing ulceration. The increasing prevalence of DM, in conjunction with the serious consequences associated with DMU, make understanding the linking pathways between gait changes, plantar pressure, and ulcer development increasingly important; particularly if clinicians are to be successful in reducing the incidence of ulceration and amputation in the future. Therefore, research that can attribute specific gait modifications to individual complications of DM, such as CTD, may enable clinicians to identify those individuals who are vulnerable to abnormal PPPs, to provide early interventions such as offloading, and ultimately, to reduce the risk of ulcer formation and amputation.

6.4 LIMITATIONS OF STUDY

Due to the time and resource restrictions associated with this study there are a number of issues which may have influenced results, and which could have been minimised or avoided.

Firstly, the small study sample limited the validity of findings, since intra-group variability may have produced results which were skewed as a result of participants who demonstrated specific attributes that had a significant influence on gait characters (e.g. high arched or flat foot). In addition, participants were all healthy young adults and, since in DM individuals severe deformity would not usually occur at such a young age, an older group may have provided a more representative sample. Division of participants into cohorts based on characters such as foot structure, BMI, age etc. would therefore have minimised the impact of inter-participant variations, and would have provided further information about how additional factors may, in conjunction with CTD, influence gait.

As part of pre-trial preparation it would have been beneficial to provide participants with a greater opportunity to ambulate whilst wearing the CTD device, which was not possible in this study due to a tight lab schedule. This may have allowed participants to adopt compensatory gait pattern characteristics which more closely resembled those developed by individuals with anatomical CTD. Another CTDSD issue involved ethical considerations that resulted in variations in device placement and tightness, and therefore inconsistent degrees of inter-participant digital retraction, and thus wide ranging effects on gait. A device which could be adjusted in a stepwise fashion, or which provided a quantitative measure of tightness would allow a more consistent degree of digital retraction, and would enable placement of participants into different cohorts based on retraction angle/ tension etc. In terms of engineering criteria, the production of an external dorsiflexor moment about the ankle was a significant disadvantage of the CTDSD and may have greatly influenced the study findings. Therefore, an improved design bringing about an extensor moment at the MTPJs with minimal or no effects at the ankle would provide a much better approximation of the gait parameter changes that are likely to occur in individuals with anatomical CTD.

Pre-trial marker placement was essential to the accurate calculation of joint centres using Vicon Nexus software, and hence any inconsistencies would have affected all

subsequent measures. Even with mm accuracy in terms of marker placement, calculations automatically performed as part of the Vicon Nexus software package were not under the control of the researcher, and therefore the specific method utilised to calculate gait parameters could not be altered. For example, the Plug-in Gait code used defines the specific axes assigned to each segment and determines how each segment relates to each of the others. Since the convention used to calculate trajectories is not adjustable it must be taken for granted that this method is effective, and provides a close approximation to the true values. Nonetheless, the Plug-in Gait model is used extensively in gait labs all over the world by experts in their field and therefore it would be expected that any serious issues with this model would have been addressed previously.

Both time restrictions, and availability of/ familiarity with software (Minitab, MS Excel), meant that analysis of data was limited. For example, with regard to GRFz measures, it would have been preferable to compare not only the two peak values, but also the inter-peak minimum and the between peak duration. In addition to changes in peak values, it may also be expected that with the CTDSD events would occur earlier or later and hence calculation of the timing of events would have enabled better inter-group comparison. It was originally intended to normalise all data so that the time axis unit of measurement was '% of total gait cycle' rather than 'seconds', allowing events to be compared both quantitatively and chronologically. However, lack of expertise and available software precluded temporal analysis. Other factors influencing data quality included aspects of processing and analysis. For example, manual cropping of data files and reliance on the accuracy of the Nexus platform to label gait events meant that data included for comparison may have varied between trials. Use of Matlab would have also allowed quicker processing across a wider range of parameters.

Although the above factors may have had an influence on the gait parameter measures obtained, since participants acted as their own controls (hence negating the need for patient matching), any gait alterations noted should have been

attributable to the CTDSD rather than external factors (which would have remained consistent throughout both experimental and control trials).

CHAPTER 7 CONCLUSIONS AND FUTURE WORK

7.1 CURRENT LITERATURE

This thesis has outlined the physiological effects that diabetes can have on the cells, tissues, neurological and circulatory systems of the body, and has related these to functional and gross changes that may be observed in the DM population, and which are collectively termed 'diabetic complications'. Among the many effects which commonly occur due to DM, the local and systemic changes that result in abnormal plantar pressures and gait patterns, and which act to increase a person's risk of ulceration, were more thoroughly investigated. CTD, which is a frequently observed complication of DM (Farndon 2000), was discussed with respect to both its aetiology and specific involvement in ulceration risk, and to its effects on plantar pressure and gait pattern.

Current literature has identified a strong relationship between DM and increased plantar pressure, gait parameter modifications and occurrence of ulceration. Diabetes may produce pathological levels of pressure due, for example, to changes in joint ROM (Caselli, et al. 2002), soft/connective tissues (Menz, et al. 2007; Abouaesha, et al. 2001), peripheral neuropathy (Kanade, et al. 2006), or via the effects of foot structural changes (Cavanagh, et al. 1997; Yu, et al. 2011). Similarly gait alterations may be brought about as a result of neurological dysfunction (Brach, et al. 2008), musclular abnormalities (Savelberg 2010; Sawacha, et al. 2011), structural changes (Kanade, et al. 2006) or reduced stability (Allet, Armand and de Bie, et al. 2010). These changes may combine to contribute to increased stress on the tissues of the foot, therefore ultimately leading to loss of skin integrity.

Ulceration has been associated with a number of internal (age, diabetes duration, ethnicity) and external (presence of callus or deformity, reduced ROM) influences; however, the three main risk factors reported for diabetic individuals are DMN, PVD and IPP (Bokan 2010; Boulton 2006; Ledoux, et al. 2005; Ledoux 2008; Merza and Tesfaye 2003; Pinzur 2008).

Literature investigating the relationship between CTD and plantar pressure, gait and ulceration was scarce. Conflicting views were noted regarding CTD aetiology (van Schie, et al. 2004; Bus, et al. 2002; Bus, et al. 2009) and, although a number of studies identified a relationship between general foot deformity and ulcer development (Ledoux, et al. 2005; Ledoux 2008; Mueller, et al. 1990; Cowley, et al. 2008; Bokan 2010), specific evidence relating to CTD alone was not found by the researcher. Links between deformity and pressure have also been reported in the literature (Spink et al. 2009; Sinacore, et al. 2008; Yu et al. 2011). General findings included a significant increase in pressure across the MTPJs in DM subjects, with correlation between the site of ulceration and type/location of deformity. However, none of these studies explored the influence of CTD in isolation. Current research was also lacking with regard to gait pattern changes caused by CTD. Results from studies investigating the effects of CTD in non-diabetic individuals (Keenan, et al. 1991; Mickle, et al. 2011; Turner and Woodburn 2008) were variable, and gait changes in rheumatoid feet may have be influenced by the specific disease processes involved with this condition.

The primary aim of this study was to make inferences about the specific gait pattern modifications that are likely to be observed in DM individuals with anatomical CTD. This was achieved via design of a device to simulate CTD in healthy adults, which was subsequently worn by participants during completion of 3D motion analysis trials. Gait data was captured using a Vicon MX system and Kistler force plates whilst participants performed walking trials along a straight, level walkway at a self-selected speed. Participants acted as their own controls by completing further trials following removal of the device. Following data capture, a number of relevant gait parameters were selected for further statistical analysis, including spatiotemporal, kinetic and kinematic parameters. As such, the specific objectives were to: validate the CTDSD; identify which, if any, gait alterations occurred as a result of the CTDSD; analyse the data to determine which differences were significant; make inferences from the findings regarding the gait modifications which would be expected in DM individuals with anatomical CTD; propose possible changes in clinical practice to

improve longer-term outcomes for diabetic individuals and finally, to identify areas that would benefit from future research.

Specific conclusions drawn in light of the study findings are provided below.

7.2 CONCLUSIONS

1) Validation of the CTDSD: The scarcity of current literature in relation to plantar pressure measures in DM individuals, in addition to lack of detail regarding the nature of deformity present in study participants, meant that comparison between reported findings and values recorded in this study was difficult. However, by reviewing the literature in conjunction with observation by a HPC registered podiatrist, and taking into account both time and resource constraints, it was deemed that the device provided suitable simulation of anatomical CTD for use in the main study.

2) Gait parameters investigated using 3D motion analysis and force plate data included spatiotemporal, kinetic and kinematic parameters. Significant gait pattern alterations (*$p<0.05$, **$p<0.01$) were noted in the following parameters when comparing controls to the experimental group:

    a. Spatiotemporal
        i. Gait velocity was decreased*
    b. Kinetic
        i. Vertical ground reaction force component second peak was reduced**
        ii. Anterio-posterior ground reaction force component peak was decreased**
        iii. Ankle moment peak was reduced*
        iv. Knee moment minimum was decreased (i.e. value moved closer to zero)*
    c. Kinematic
        i. Ankle angle peak was increased*
        ii. Ankle angle minimum was reduced (i.e. value moved closer to zero)**

3) Adoption of a steppage gait pattern would account for the abovementioned alterations (see Figure 6-9).
4) It is further theorised that in order to reduce discomfort, the three functional rockers (see glossary), normally utilised during stance to propel the body forwards, may be avoided in CTDSD individuals, through adoption of a steppage type motion. Steppage gait is usually associated with reduced tibialis anterior, or abnormal peroneal, function (e.g. as observed in poliomyelitis) resulting in a requirement for a greater vertical hip and/or

knee excursion to enable foot clearance . A steppage pattern also contributes to a reduction in GRFx by maintaining the trunk (and therefore COM) in a more posterior position. This limits forward momentum, and hence allows the swing leg to be lowered to the ground in a controlled manner (i.e. lower shear braking forces), via hip and knee extension in the swing leg with simultaneous knee flexion and ankle dorsiflexion in the stance leg (Figure 6-9). The abovementioned theory also supports the remaining kinematic gait alterations identified in this study.

Figure 6-9
This gait style involves a more posteriorly placed COM, a decrease in the inertial forces acting on the swing leg, and a reduced reliance on the three 'functional rockers' (Perry 1992) to create forward progression. The outcomes of these functional changes include:

a. Controlled lifting and lowering of the relevant leg rather than normal 'roll-over'.
b. Decreased momentum of the swing leg with an associated reduction in the braking shear occurring at HS.
c. Decreased action by the ankle plantarflexor muscles, and hence reduced propulsive forces at TS and PSW (with an associated reduction in inertial forces on the swing leg).
d. Reduced requirement for knee flexor muscles to decelerate forward progression of tibia at TSW.

Other alterations expected if a steppage gait pattern were to be adopted were absent. A number of suggestions as to why additional gait changes were not observed included the limited study population, suitability and variation in application of the CTDSD, generation of an ankle dorsiflexor moment by the CTDSD, accuracy of parameter calculations from raw data (i.e. using Vicon Nexus software and Plug-in Gait model code), and data analysis issues (i.e. using MS Excel and Minitab).

5) It is acknowledged that in order to improve validity of the data set that some modifications in the CTDSD and experimental procedures would be required; however, it may be inferred from the results of this study that a number of gait parameter alterations would be expected to be observed in diabetic individuals with anatomical CTD, and therefore further more rigorous research would be warranted in this area.

6) As a result of the preliminary findings obtained in this study it would be recommended that a number of changes in clinical practice take place:

a. Clinicians should be aware of the links which exist between gait pattern changes, abnormal plantar pressures, and risk of ulcer development.

b. Early and regular assessment of diabetic individuals should take place, and assessment should include an analysis of plantar pressures and/ or gait parameters in order to provide early identification* of individuals who may be at risk of developing ulceration.

   (*It must be noted that in much of the literature, alterations in gait and/ or plantar pressure were observed prior to the presence of clinical symptoms).

c. Where changes in gait pattern or plantar pressures are detected in diabetic individuals, early intervention should be adopted. This may involve education, footwear advice, and provision of orthotic, or other offloading and/ or cushioning devices, in order to reduce gait deviations, improve normal foot function and redistribute pressure over the plantar surface of the foot. The main outcome of clinical intervention should be to minimise the likelihood of ulceration, and to maximise the patients' comfort, confidence, function, mobility, independence and stability.

7) Areas related to the work carried out in this study, and which may benefit from further research, are discussed below. Increased knowledge leading to a better understanding of the pathways to ulcer development may provide improved longer-term outcomes for diabetic individuals, as well as having a beneficial impact in relation to the demands placed upon caring partnerships, including families, carers and the NHS.

7.3 RECOMMENDATIONS FOR FUTURE WORK

As previously highlighted (in Section 7.1) a number of significant gaps currently exist in the literature. The importance of understanding the pathophysiological processes which underlie the development of diabetic complications, and the complex interlinking relationships between these (Figure 2-1), is essential if the incidence of severe and life-threatening DM complications such as ulceration and amputation are to be reduced.

This study validated the effectiveness of a device which simulates CTD, and has enabled inferences to be drawn regarding the spatiotemporal, kinetic and kinematic gait parameter alterations which are likely to be exhibited by diabetic individuals with anatomical CTD. A number of areas of further research are recommended in light of the findings of both the validation and gait parameter studies.

Gaps in the literature relating to the 'normal' plantar pressures expected in DM individuals with CTD meant that validation of the CTDSD was difficult. More robust validation of the CTDSD is therefore recommended to ensure that effects observed in participants wearing the device closely reflect those which would be seen in the diabetic population. It is suggested that validation evidence would be best obtained by a direct comparison of the pressures recorded in healthy individuals wearing the CTDSD, with results from three DM groups: DM plus CTD; DMN plus CTD and DMU history plus CTD. In this way the influence of DMN and DMU could also be assessed. Ideally, these groups would also be matched for age, gender, BMI etc.

No literature was identified by the researcher in relation to the specific effects of CTD on gait parameters in a DM population. Due to the time and resource limitations involved in this study it would be beneficial to carry out a similar, more robust investigation following the basic procedural framework, but using a significantly larger study sample, and preferably using cohorts to accommodate for inconsistencies in inter-participant characteristics (e.g. BMI, age, foot type, joint ROM, gait velocity). Ideally the current device would be adjusted to enable tension and/ or retraction to be easily measured, and hence allow post data capture analysis to account for any inter-participant differences in the degree of retraction produced. The CTDSD itself would also require modification prior to further validation tesing to negate any external effects on the ankle (i.e. to prevent it from inducing a dorsiflexor moment).

Use of the CTDSD allows inferences to be made about its effect on gait in people with DM, without interference from co-existing complications. However; it is recommended that further investigation also takes place using DM subjects with

CTD, the results of which could then be compared with CTDSD study findings. Failings in previous literature include use of multivariate groups (e.g. healthy control group compared to an experimental group with both DM and CTD). It is therefore suggested that future studies using DM individuals should be restricted to one variable (e.g. two DM groups; one with CTD, one without) or should expand the number of experimental groups to allow a more detailed analysis of effects (e.g. group 1; healthy control, group 2; healthy control with CTDSD, group 3; DM wearing CTDSD, group 4; DM with anatomical CTD). To ensure a meaningful comparison between experimental and control groups, it would be preferable to use DM subjects in both the experimental and control groups, and to limit the former group to those who have deformity of a similar severity and location.

Recent investigations involving forefoot/ digital deformity have failed to specify the site, severity and exact nature of deformities present in the experimental group (s). And it appears from a number of these reports that the participants used often displayed varying combinations of coexisting deformities. Although it is not realistic to expect that anatomical foot deformity will be uniform, it would be recommended that future investigations (either in relation to CTDSD validation, or gait analysis using DM individuals with CTD), should use cohort groups relating to severity, site, and specific type of digital deformity in order to better identify the influence of these factors on either pressure (CTDSD validation) or gait.

It is further suggested that future work is carried out to increase the breadth of gait parameters studied (e.g. include other planes of motion; carry out temporal comparison of events by normalising to % of gait cycle; investigate a number of points in the gait cycle rather than just max. and min.). While investigation regarding consequences in the contralateral leg would also be helpful (e.g. if vertical propulsive forces are reduced in the experimental limb, is there a corresponding increase in the contralateral limb, and similarly, if stance duration is increased in the CTDSD limb, will this be reduced in the other limb).

Finally, recent literature suggests that factors such as activity (including obstacle clearance), gait speed, and terrain will all affect gait, therefore it would be beneficial if, following level walking studies as described above, work could be completed to investigate the effects of CTD (anatomical and/ or CTDSD) on dynamic parameters during completion of various activities of daily living, such as stair and incline climbing, sit to stand, walking on different surfaces, and clearing obstacles. This would provide a more holistic view of the effects of CTD on pressure and dynamic parameters (e.g. spatiotemporal parameters, joint angles, moments and GRF vector components), and would therefore allow a better 'real-life' risk assessment with regards to ulcer development.

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APPENDIX A

All PPP trial data for each participant (A, B & C) both without and with the CTDSD. Mean values shown in bold.

Participant A - No Device

PRESSURE (kPa)	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
TRIAL 1	224.26	150.95	168.22	75.7	38.89	155.54	231.88
TRIAL 2	299.63	226.43	136.58	40.44	29.6	151.71	299.63
TRIAL 3	173.01	114.69	171.81	91.31	33.97	158.84	189.57
MEAN	**232.3**	**164.02**	**158.87**	**69.15**	**34.15**	**155.36**	**240.36**

Participant B - No Device

PRESSURE (kPa)	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
TRIAL 1	166.77	233.82	245.98	156.52	102.16	211.31	255.13
TRIAL 2	140.78	217.35	240.09	153.83	102.08	196.64	246.39
TRIAL 3	136.98	197.18	241.23	171.03	127.83	212.56	243.22
MEAN	**148.18**	**216.12**	**242.43**	**160.46**	**110.69**	**206.84**	**248.25**

Participant C - No Device

PRESSURE (kPa)	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
TRIAL 1	246.03	213.17	163.9	125.56	56.82	180.6	246.03
TRIAL 2	202.54	167.7	189.62	155.25	64.48	149.2	202.54
TRIAL 3	131.55	152.52	206.27	153.08	88.79	168.72	206.27
MEAN	**193.37**	**177.87**	**186.67**	**144.63**	**70.03**	**166.17**	**218.28**

Participant A - Shoe With Device

PRESSURE (kPa)	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
TRIAL 1	22.43	331.81	209.69	97.32	31.71	121.78	331.81
TRIAL 2	1.77	253.55	187.17	129.93	50.66	134.26	253.55
TRIAL 3	6.47	290.91	198	123.02	38.04	132.41	290.91
MEAN	**10.22**	**292.09**	**198.29**	**116.76**	**40.14**	**129.48**	**292.09**

Participant B - Shoe With Device

PRESSURE (kPa)	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
TRIAL 1	25.64	86.31	173.1	225.6	260.55	205.55	260.55
TRIAL 2	34.83	93.97	172.18	223.07	257.55	196.99	257.55
TRIAL 3	29.95	89.59	161.37	223.99	275.36	189.38	275.36
MEAN	**30.14**	**89.96**	**168.88**	**224.22**	**264.49**	**197.31**	**264.49**

Participant C - Shoe With Device

PRESSURE (kPa)	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
TRIAL 1	24.36	100.15	182.06	176.26	99.57	163.43	183.53
TRIAL 2	59.85	106.68	210.4	179.49	93.39	157.35	210.4
TRIAL 3	27.52	118.4	186.01	188.7	78.5	134.83	191.4
MEAN	**37.24**	**108.41**	**192.82**	**181.48**	**90.49**	**151.87**	**195.11**

APPENDIX B

Within subject mean PPPs for each foot region measured in participants without (A) and with (B) the CTDSD worn over the right foot. Mean PPP values for three subjects and corresponding standard deviations are also given

No Device

SUBJECT	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
A	232.30	164.02	158.87	69.15	34.15	155.36	240.36
B	148.18	216.12	242.43	160.46	110.69	206.84	248.25
C	193.37	177.80	186.60	144.63	70.03	166.17	218.28
MEAN	**191.28**	**185.98**	**195.97**	**124.75**	**71.62**	**176.12**	**235.63**
SD	**42.10**	**26.99**	**42.56**	**48.79**	**38.29**	**27.14**	**15.53**

With Device

SUBJECT	HALLUX	1MTPJ	2MTPJ	3-5MTPJ	MIDFOOT	HEEL	TOTAL
A	10.22	292.09	198.29	116.76	40.14	129.48	292.09
B	30.14	89.96	168.88	224.22	264.49	197.31	264.49
C	37.24	108.41	192.82	181.48	90.49	151.87	195.11
MEAN	**25.87**	**163.49**	**186.66**	**174.15**	**131.70**	**159.55**	**250.56**
SD	**14.01**	**111.76**	**15.64**	**54.11**	**117.72**	**34.56**	**49.97**

APPENDIX C

Ethical and Participant documentation comprising of:

i. Application for Departmental Ethical Approval
ii. Participant Information Sheet (Incorporating Consent Form)
iii. Health Information Sheet (Incorporating Health Questionnaire)
iv. Participant Physical Assessment Form

NB. Original documentation was produced on headed paper carrying University of Strathclyde logo and address.

Ethics Form

Please answer all questions

1. Title of the investigation
Investigating the effect of simulated diabetic claw toe deformity on the gait patterns of healthy adults

2. Chief Investigator (Ordinance 16 member of staff only)
Name: Philip Rowe Status: ☒ Professor ☐ Reader ☐ Senior Lecturer ☐ Lecturer Department: Bioengineering Telephone: 0141 548 3032 E-mail: philip.rowe@strath.ac.uk

3a. Other Strathclyde investigator(s)
Name: Bruce Carse Status (e.g. lecturer, post-/undergraduate): Research Fellow Department: Bioengineering Telephone: 0141 548 3028 E-mail: bruce.carse@strath.ac.uk

3b. Other Strathclyde investigator(s)
Name: Beverley Caie Status (e.g. lecturer, post-/undergraduate): MSc Bioengineering Postgraduate Student Department: Bioengineering Telephone: 07812131893 E-mail: beverley.caie@strath.ac.uk

4. Non-Strathclyde collaborating investigator(s)
Name: N/A Status (e.g. lecturer, post-/undergraduate): Department/Institution: If student(s), name of supervisor: Telephone: E-mail: Please provide details for all investigators involved in the study:

5. Overseas Supervisor(s)

Name(s): N/A
Status:
Department/Institution:
Telephone:
Email:

I can confirm that the local supervisor has obtained a copy of the Code of Practice: Yes ☐ No ☐
Please provide details for all supervisors involved in the study:

6. Where will the investigation be conducted

Bioengineering Department, University of Strathclyde, 106 Rottenrow, Glasgow

7. Duration of the investigation

Duration(years/months) : 6 months

Start date (expected): 10 / 06 / 2012 Completion date (expected): 10 / 12 / 2012

8. Sponsor (please refer to Section C and Annex 3 of the Code of Practice):

University of Strathclyde

9. Funding body (if applicable)

Name of funding body: N/A
Status of proposal – if seeking funding (please click appropriate box):
☐ In preparation
☐ Submitted
☐ Accepted
Date of Submission of proposal: / /
Date of start of funding: / /

10. Objectives of investigation (including the academic rationale and justification for the investigation)

Diabetes is an increasingly prevalent condition (2.9 million presently in UK) that accounts for around 10% of the annual NHS budget and has a great impact on quality of life. High plantar pressure in the presence of neuropathy is reported as a major risk factor in the development of ulceration, and is increased in individuals with foot deformity, such as claw toe. Although many studies exist regarding gait and foot pressure changes in diabetic participants the multi-factorial nature of diabetes makes investigation of single factors (e.g. foot deformity) problematic. By simulating claw toe deformity in healthy adults this allows the effect of deformity on gait parameters and foot pressure to be studied in isolation.

Therefore the general aim of this study is to determine if there are significant differences in

the gait parameters of patients with claw toe deformity when compared to participants lacking deformity.

The specific objectives of this study are to:

7) Validate the effectiveness of a simulated claw toe deformity in mimicking the effects of foot deformity in diabetic individuals.
8) Specify and quantify any kinetic, kinematic and spatiotemporal gait modifications by use of a device that simulates claw toe deformity in healthy adults.
9) Analyse data statistically to determine whether significant changes occur as a specific result of deformity by comparing to control data.
10) Make inferences about the relationship between gait and pressure changes due to chronic foot deformity, and the development of ulceration.

11. Nature of the participants
Please note that investigations governed by the Code of Practice that involve any of the types of projects listed in B1(b) must be submitted to the University Ethics Committee for prior approval

Are any of the categories mentioned in Section B1(b) (participant considerations) applicable in this investigation?
☐ Yes
☒ No

Please detail nature of participants: Healthy adults
Number: 12 Age (range): 16-60

Please also include information on: recruitment methods (see section B4 of the Code of Practice); inclusion/exclusion criteria; and any further screening procedure to be used

Participants will be selected from fellow Bioengineering students and other healthy adults.

Potential participants will be approached regarding taking part in the study and will initially be provided with verbal information about the study and will be given a participant information sheet. It will be made clear that there will be no pressure for individuals to take part and they will be given 24 hours to consider whether they wish to take part or not, and to ask the researcher any questions they might have. After the 24 hour period the researcher will re-approach them and ask if they are willing to participate. If they do wish to participate the researcher will ask them to read and sign the consent form. They will then be invited to attend an initial screening session where a short health questionnaire and physical examination (see Health Questionnaire and Physical Assessment sheet attached) will be completed. A copy of the Health Questionnaire and Physical Assessment sheet will be given to the participant when they have given consent and they will be asked to familiarise themselves with the questions before they attend the physical assessment. The physical assessment will assess joint range of motion, pedal pulses and pedal touch sensation to ensure there are no existing foot/lower limb pathologies which would adversely affect gait.

Inclusion criteria:
- Healthy adult
- Good mobility and balance (i.e. participants are able to walk without assistance along a walkway)
- Free from foot pain or pathology

Exclusion criteria:
- Diabetes, rheumatoid arthritis (or other forms of arthritis), neurological

abnormalities or other conditions which significantly affect the circulation or nerve function in the feet and/or legs
- Previous foot/leg surgery or any medical condition which significantly alters the way participants walk
- Significant foot deformity or callus
- Poor movement in the joints of the legs and/or feet
- Any pre-existing medical conditions that are currently being treated

12. What consents will be sought and how?
Please note that the information sheets and consent forms to be used should be attached to this form

Potential participants will be approached and the study will be verbally explained to them by the researcher. In addition they will be provided with a participant information sheet and will be encouraged to ask any questions. They will be informed that they are under no obligation to take part and that they are free to withdraw from the study at any time. Potential participants will be asked to take the information sheet, read it, and consider whether or not they wish to participate for 24 hours before making a decision, and will thereafter be asked to sign a consent form if they wish to participate (see attached Participant Information Sheet and Consent Form).

13. Methodology
Investigations governed by the Code of Practice that involve any of the types of projects listed in B1(a) must be submitted to the University Ethics Committee for prior approval. Where an independent reviewer is not used, then the UEC/ DEC reserves the right to scrutinise the methodology.

Are any of the categories mentioned in the Code of Practice Section B1(a) (project considerations) applicable in this investigation?
☐ Yes
☒ No
If 'yes' please detail: N/A

Design: what kind of design/research method(s) is/are to be used in the investigation?

This study will use healthy adult participants wearing a device that simulates claw toe deformity. The proposed device will consist of a modified Thera-Band (elasticated resistance band) which will be wrapped around the most distal part of the lower limb such that it does not impede the ankle joint. Protruding from this loop will be three smaller loops, which will hook over and underneath the the hallux and digits 3 and 5. The tightness of the device will then be adjusted such that they gently pull back the toes so the metatarsophalangeal joints are hyperextended and the proximal (and distal) interphalangeal joints are flexed, thus simulating claw toe deformity in the wearer. See Appendix 1 for further detail of the device.

The device will be tested by the researchers named on this application to ensure that it both replicates claw toe deformity, and that it does not cause any pain or discomfort for the participant. The ability of the device to replicate claw toe deformity will be carried out using an F-Scan pressure mapping system to measure plantar pressure while wearing the device. The recorded plantar pressure profiles will then be compared with claw toe plantar pressures described in the literature, and the device adjusted until a comparable pressure profile is achieved. The level of pain or discomfort caused by wearing the device will be assessed by the researchers during this testing.

Data will be obtained regarding participant's kinetic, kinematic and spatiotemporal characteristics and ground reaction forces using the Vicon 3D gait analysis system with a Kistler force platform. Participants will act as their own control, and experimental and

control data will be statistically compared.

Techniques: what specific techniques will be employed and what exactly is required of participants?

Participants will have a set of reflective markers adhered to specific anatomical sites on both lower limbs and will be asked to wear a device over their foot to simulate claw toe deformity. They will walk at a self selected pace in a straight line along a walkway, which incorporates a force platform, whilst data is captured using the systems described above. Trials will be carried out, first without and then with, the claw toe deformity simulating device worn, and in each case will be repeated until two clean force plate strikes are obtained.

Has this methodology been participant to independent scrutiny?
☐ Yes
☒ No
Please provide the name and contact details of the independent reviewer: N/A

14. Data collection, storage and security

Explain how data are handled, specifying whether it will be fully anonymised, pseudo-anonymised, or just confidential, and whether it will be securely destroyed after use:
Each participant will be assigned a unique identification number. The consent form will contain both the patients name and the unique ID number. All other documentation will contain the unique participant ID number only. Consent forms, participant documentation and unique ID numbers will be stored securely in a locked filing cabinet on university premises. Gait parameter data obtained will be used anonymously (i.e. will contain ID number only) and will be accessible only to the named investigators. Results from gait parameter data will be presented as part of the researcher's thesis and may be used as part of future research or within published literature. Participants will be advised of this via the participant information sheet and will be asked if they agree to these terms on the consent form. Hardcopies of the consent forms will be securely destroyed at the end of the study.

Explain how and where it will be stored, who has access to it, and how long it will be stored:
Participant personal data will include a consent form, health questionnaire and physical assessment. These will be given an ID number and stored (as described above) in a locked filing cabinet on university premises that may only be accessed by the named investigators. Gait parameter data gathered as part of the study will be anonymous (ID only) and will be stored electronically on a secure server. As previously described, the anonymous gait parameter data and results obtained from the study will be presented as part of the researcher's thesis and participants will be requested to give consent for this on the consent form. Gait data will be stored for no longer than 10 years.

Will anyone other than the named investigators have access to the data? Yes ☐ **No** ☒
If 'yes' please explain: As detailed above personal data will be accessible only to named investigators during the study and, except from the consent form, all documentation will be anonymous and ID coded only. Anonymous ID coded gait data will be used as part of the researcher's thesis with prior permission of the participant being obtained on the consent form. No personal details will be published.

15. Potential risks or hazards

- Trip or fall during completion of trials. This will be minimised by ensuring a clear walkway and by allowing the participant time to get used to the device prior to trial completion.
- Reaction to reflective marker adhesive. This will be minimised by using hypoallergenic tape. Marker removal should cause no more discomfort than removing a first-aid plaster.
- Slight discomfort whilst wearing the device that simulates claw toe deformity. This will be minimised by assessing discomfort using a visual analogue scale following device application, and by restricting the amount of time that the device is worn. Participants will be advised to inform a researcher immediately if they are in any discomfort so that the device can be removed.

16. Ethical issues

Possible hazards will be explained in the participant information form. There will be no pressure on individuals to take part in the study and it will be explained that if they choose to take part they may withdraw from the study at any time. Potential participants will be given the opportunity to ask questions and take the participant information sheet away for further consideration before making a decision and prior to signing the consent form. The physical assessment will involve minimal physical contact, will be non-invasive and painless and will be carried out by a qualified Health Professions Council registered podiatrist (Beverley Caie) who is experienced in assessing the foot and lower limb. It is possible that the device will cause the wearer pain or discomfort, however every effort will be made by the researchers to ensure that this does not happen.

17. Any payment to be made

No

18. What debriefing, if any, will be given to participants

After the initial screening session the researcher will explain the data gathering procedure to the participant. After the gait data gathering session participants will be briefly advised about how the data will be used. On completion of the study all participants will be sent an email summarising the general findings of the study.

19. How will the outcomes of the study be disseminated (will you seek to publish the results)

The results will be used as part of the researcher's thesis, and may be used in presentations, lectures, conferences and journal articles in the future. No personal details will be used and data presented will remain anonymous.

20. Nominated person to whom participants' concerns/ questions should be directed before, during or after the investigation (please also provide contact details)

Beverley Caie, Bioengineering Department, University of Strathclyde

Email: beverely.caie@strath.ac.uk

21. Previous experience of the investigator(s) with the procedures involved

Beverley Caie (MSc Bioengineering)

- Full training in use of the Vicon gait analysis and Kistler force platform systems will be completed prior to data collection.
- Qualified Health Professions Council registered podiatrist who is experienced in assessing foot and lower limb pathology.

Dr Bruce Carse

- PhD in Biomechanics, with 5+ years' experience of using various 3D motion analysis systems in gait and upper limb studies.
- Experience in designing and conducting clinical research trials (ISRCTN52126764).
- Up-to-date training in Good Clinical Practice.

Professor Philip Rowe

- Professor of Rehabilitation Science in the Bioengineering Unit with a 25-year track record in rehabilitation-related clinical biomechanics
- Experience of managing multiple research grants.
- Up-to-date training in Good Clinical Practice.

Information for participants

Project Title: Investigating the effect of simulated diabetic claw toe deformity on the gait patterns of healthy adults

Introduction
We would like to invite you to take part in a research study. This information sheet will explain the research being done and what it would involve for you. Please take time to read the following information about the study carefully. You can also show this information to carers, friends or relatives if you wish. Please ask us if there is anything that is unclear or if you would like more information.

This research is being carried out by Beverley Caie (MSc Bioengineering student), Dr Bruce Carse and Professor Philip Rowe (Bioengineering Department , University of Strathclyde).

What is the purpose of this investigation?
This purpose of this study is to find out if foot deformity (and specifically claw toe deformity) changes the way that people with diabetes walk, and how their muscles and joints react during walking. It will also measure the amount of pressure on the sole of the foot during walking to see if this is higher in people who have a foot deformity.

Do you have to take part?
No, it is up to you to decide, and participation is totally voluntary. We will describe the study and go through this information sheet, which we will then give to you. If you are willing to participate you will be asked to sign a consent form to show you have agreed to take part. You are free to withdraw at any time, without giving a reason. This would not affect any future participation in studies.

What will you do in the project?
If you decide to take part in this study you will be asked to attend two sessions. These will take place in the Biomechanics laboratory which is located in the Wolfstone building (part of the University of Strathclyde, Bioengineering Department. Please see the top left of this page for the full address).

The sessions are described below and will take place in June/July 2012:

Session 1: This will take no longer than 45min. The researcher will complete a health questionnaire with you and will carry out a short assessment to see how flexible your joints are. They will also test the circulation and feeling in your feet. None of the assessments will be painful. It is recommended to bring shorts and a t-shirt.

Session 2: This will last no longer than 90 min and will study the way you walk. Again, we request that you bring shorts and a t-shirt. Because people with diabetes might have other health problems it is often difficult to measure how deformity changes the way they walk. For this reason the study will use subjects who **do not** have diabetes. If you decide to take part in the study, a foot deformity will be simulated by placing a device around your foot that pulls your toes back into a clawed position.
In this session a number of reflective markers will be placed on the skin on both of your legs. The device will be placed over your foot (one foot only) and you will have a chance to walk around the room to get used to the device, as it may feel a little strange at first. You will then be asked to walk barefoot in a straight line for about 10 paces while cameras record how the markers move as you walk. The device will be removed and you will walk down the walkway again. Each part will be repeated 2-8 times. After this session the information will be used to make a 3D image of the way you walk and this can be used to measure your walking characteristics.

You will not be paid for taking part in this study, but travelling expenses can be reimbursed.

Why have you been invited to take part?
You have been asked to take part in this study because you are a healthy adult with no foot conditions. Listed below are some of the conditions that the researcher will ask you about to decide if you can take part in the trial.

You will be able to take part in the study if:

- You are a healthy adult
- You have good mobility and balance (i.e. you are able to walk normally without assistance up and down a walkway).

You will not be able to take part in the study if you have any of the following:

- Diabetes, rheumatoid arthritis (or other forms of arthritis), neurological abnormalities or other conditions which significantly affect the circulation or nerve function in your foot and/or leg
- Previous foot/leg surgery or any medical condition which significantly alters the way you walk
- Significant foot deformity or callus
- Poor movement in the joints of your leg and/or foot

The researcher will complete a health questionnaire and a short physical examination (of your feet and lower legs only) with you to make sure you do not have any health issues or foot problems that might affect the way you walk.

How long will I be involved in the study?
If you choose to participate, you will be involved in the study for a maximum period of 4 weeks, with attendance at two sessions which in total should last no longer than 2 hours 15 min. You will be free to withdraw at any time, without giving a reason.

What are the potential risks to you in taking part?

The risks involved in taking part in this study are minimal; however, the following list describes possible risks and how these will be minimised:

1. Trips or falls whilst moving down the walkway will be minimised by ensuring a clear walkway and by allowing time for you to get used to the device.

2. Wearing the device should not hurt but it may feel strange and may cause mild discomfort as it is designed to pull the toes back to simulate having a foot deformity. Discomfort will be minimised by assessing your level of comfort once the device has been placed on your foot, and by minimising the amount of time that you wear it. You will be advised to inform a researcher if you are in any discomfort so that the device can be removed immediately.

3. Skin irritation caused by the reflective marker adhesive will be minimised by using hypoallergenic tape. Marker removal should cause no more discomfort than removing a first-aid plaster.

What are the possible benefits of taking part?
It is hoped that information gathered during this study (i.e. how deformity changes the way that people with diabetes walk) may help clinicians to better understand the importance of deformity and how they can best care for patients who have foot deformity.

What happens to the information in the project?

Your identity and personal information will be completely confidential and known only to the named investigators. Personal information will be securely stored on university premises. The consent form will contain your name but all other documents will be anonymously coded. The data are held in accordance with the Data Protection Act, which means that we keep it safely and cannot reveal it to other people, without your permission.

Information collected about the way you walk will be anonymous. It will be stored electronically on the university computer system which requires password access, and may only be accessed by the named investigators/researcher. The results of this study will be published as part of the researcher's MSc thesis and may be used in future presentations, lectures, conferences or journal articles. No personal details will be given and results will be anonymous. Once the study is complete a short summary of the study results will be e-mailed to you and you may request an electronic copy of the whole thesis by replying to this e-mail.

The University of Strathclyde is registered with the Information Commissioner's Office who implements the Data Protection Act 1998. All personal data on participants will be processed in accordance with the provisions of the Data Protection Act 1998.

Thank you for reading this information – please ask any questions if you are unsure about what is written here.

What happens next?
If you are happy to be involved in this study you will be asked to sign a consent form to confirm this. You may take the information away (for 24 hours) to read before deciding whether to take part.
If you do not want to be involved in this project then we thank you for your interest and for taking the time to read this project information.

If you would like to take part then at the end of the study a short summary of the results will be e-mailed to you. You may request an electronic copy of the full thesis and/or give feedback on what you thought of the study by replying to the e-mail.

Researcher Contact Details:
Beverley Caie (MSc Bioengineering student)
Bioengineering Department
University of Strathclyde
106 Rottenrow
Glasgow G4 0NW
Tel: 07812131893
Email: beverley.caie@strath.ac.uk

Chief Investigator Details:
Professor Philip Rowe
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106 Rottenrow
Glasgow G4 0NW
Tel: 0141 548 3032
E-mail: philip.rowe@strath.ac.uk

Other Starthclyde Investigator Details:
Dr Bruce Carse
Bioengineering Department
University of Strathclyde
106 Rottenrow
Glasgow G4 0NW

Tel: 0141 548 3028
Email: bruce.carse@strath.ac.uk

This investigation was granted ethical approval by the Bioengineering Department Ethics Committee, University of Strathclyde.
If you have any questions/concerns, during or after the investigation, or wish to contact an independent person to whom any questions may be directed or further information may be sought from, please contact:

Secretary to the University Ethics Committee
Research & Knowledge Exchange Services
University of Strathclyde
Graham Hills Building
50 George Street
Glasgow
G1 1QE
Telephone: 0141 548 3707
Email: ethics@strath.ac.uk

Participant Name: Participant Identification Code:

Consent Form

Project Title: Investigating the Effect of Simulated Diabetic Claw Toe Deformity on the Gait Patterns of Healthy Adults

I have read and understood the form 'Information for Participants', and I have had the opportunity to discuss this study.	☐
I have spoken to a researcher regarding any questions I have about this study and I have received satisfactory answers.	☐
I have read and understood the inclusion and exclusion criteria and confirm that I do not suffer from any of the medical conditions listed and do not have any significant foot/mobility problems.	☐
I understand that I will be wearing a device over one foot to simulate deformity while completing a series of walking trials.	☐
I am aware that I am free to withdraw from this study at any time, and I agree to participate in this study.	☐
I give my consent to the use of my data anonymously in various theses, presentations, lectures, conferences and journal articles.	☐

To be completed at first screening (please sign and date box):

I agree to the completion of a health questionnaire and physical assessment to determine my suitability for this study.

Sign:

Date:

Participant Print name: Sign: Date:

Witness Print name: Sign: Date:

Address (optional): .....
Contact number: ..... **E-mail:** .....

Participant Identification Code:

Health Questionairre

Project Title: Investigating the effect of simulated diabetic claw toe deformity on the gait patterns of healthy adults

Introduction

You have been asked to take part in this study because you are a healthy adult with no foot conditions.

This questionnaire is designed to ensure you are suitable to take part in the study and do not have any conditions which would affect the way that you walk. We appreciate your help in completing this questionnaire and recommend that you have a look over the questions before coming to the screening session so that you can check any you are unsure of. The researcher will fill in the health questionnaire with you and then carry out a short physical assessment (of your feet and lower legs only) to see if your joints are flexible and to check the circulation and sensation in your feet.

In total these should take no more than 45min.

Listed below are some of the conditions that the researcher will ask you about to decide if you can take part in the trial.

You will be able to take part in the study if:

- You are a healthy adult
- You have good mobility and balance (i.e. you are able to walk normally without assistance up and down a walkway).

You will not be able to take part in the study if you have any of the following:

- Diabetes, rheumatoid arthritis (or other forms of arthritis), neurological abnormalities or other conditions which significantly affect the circulation or nerve function in your foot and/or leg
- Previous foot/leg surgery or any medical condition which significantly alters the way you walk
- Significant foot deformity or callus
- Poor movement in the joints of your leg and/or foot

For any more information please contact:

Researcher
Beverley Caie (MSc Bioengineering student)
Bioengineering Department
University of Strathclyde
106 Rottenrow
Glasgow G4 0NW
Tel: 07812131893
Email: beverley.caie@strath.ac.uk

Health Questions

1. Please list any medical condition(s) which you have been diagnosed with.

2. Please list any medications you are currently taking.

3. Do you have poor circulation and/or sensation in your feet or legs? Please detail.

4. Do you have any problems with walking? Please detail.

5. Do you have severe stiffness in any joints, and if yes which joints?

6. Have you had any surgery on your legs or feet and, if yes, has the surgery altered you leg/foot function or ability to walk?

7. Do you have any foot problems which would prevent you from wearing a device over your foot?

8. Having read the information sheet, do you have any other health or foot problems which you think might prevent you from taking part in this study?

If you agree that the above statements are true and confirm that you are happy to participate in the physical assessment then please sign and date the appropriate section on the consent form.

Participant Identification Code:

Physical Assessment

Project Title: Investigating the Effect of Simulated Diabetic Claw Toe Deformity on the Gait Patterns of Healthy Adults

Range of Motion (ROM) Assessment

Significantly restricted dorsiflexion/plantarflexion at:

- first metatarsophalangeal joint Yes/No
- first ray (approx. 1 cm range) Yes/No
- lesser metatarsophalangeal joints Yes/No

Significantly restricted ankle ROM:

- dorsiflexion/plantarflexion Yes/No
- inversion/eversion Yes/No
- abduction/adduction Yes/No

Significantly restricted knee ROM:

- flexion/extension Yes/No
- tibial rotation Yes/No

Significantly restricted hip ROM:

- Flexion/extension Yes/No
- abduction/adduction Yes/No

Significant gastrocnemius/soleus equinus Yes/No

Circulation and Sensation

Pedal pulses absent Right foot-DP/PT Left foot- DP/PT
Abnormal skin colour/hair growth/condition Yes/No
Sensation absent with 10g monofilament Right foot - /5 Left foot- /5

Other

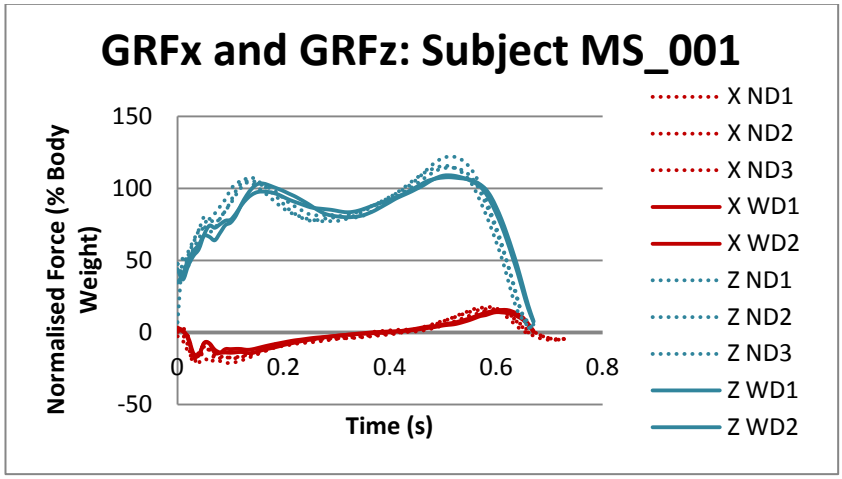
Significant callus Yes/No
Significant foot deformity Yes/No
Poor balance Yes/No

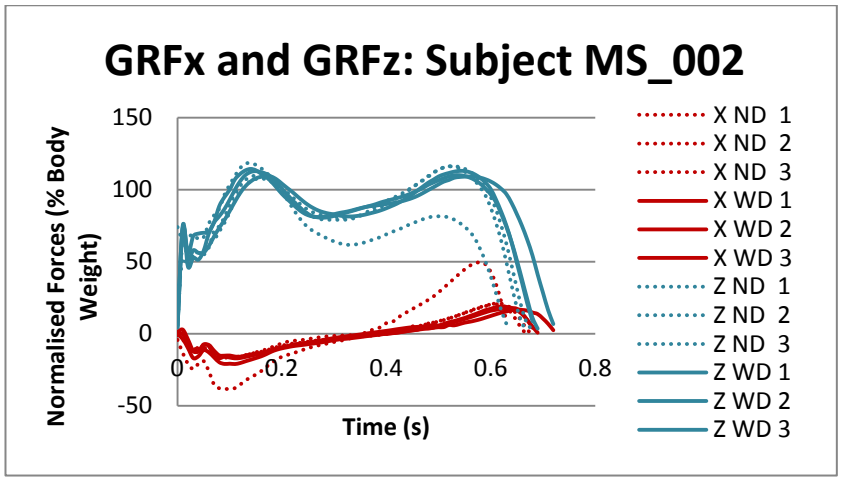
Key:

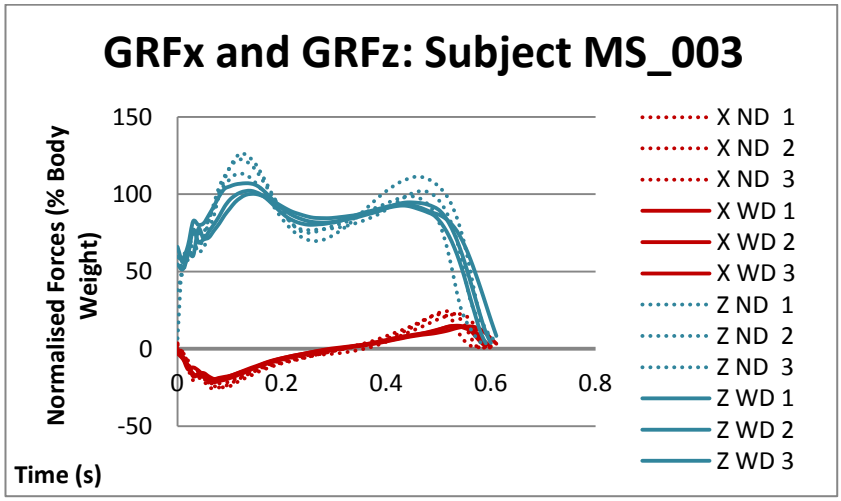
- DP/PT: dorsalis pedis/posterior tibial pulse – pulse present in either indicates normal circulation
- Monofilament sites: hallux, 1^{st} /3^{rd}/5^{th} MTPJ, heel – if absent in 2 or more sites in either foot this indicates neuropathy

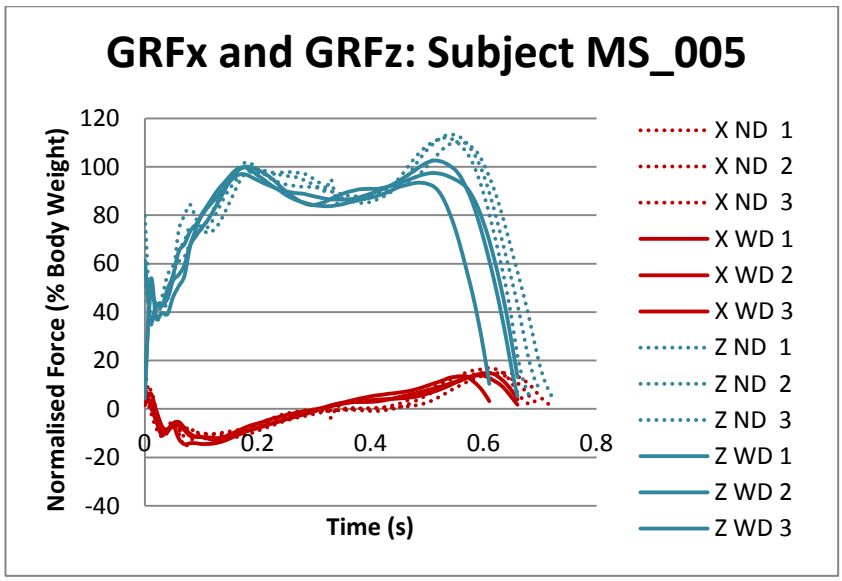
APPENDIX D

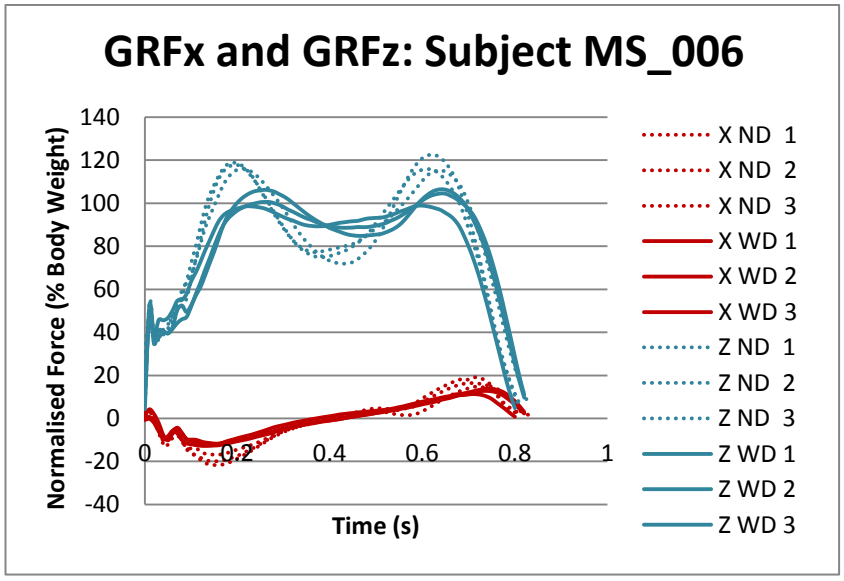
GRFx (bottom) and GRFz (top) ground reaction force components for all subjects without (dotted line) and with (solid line) CTDSD.

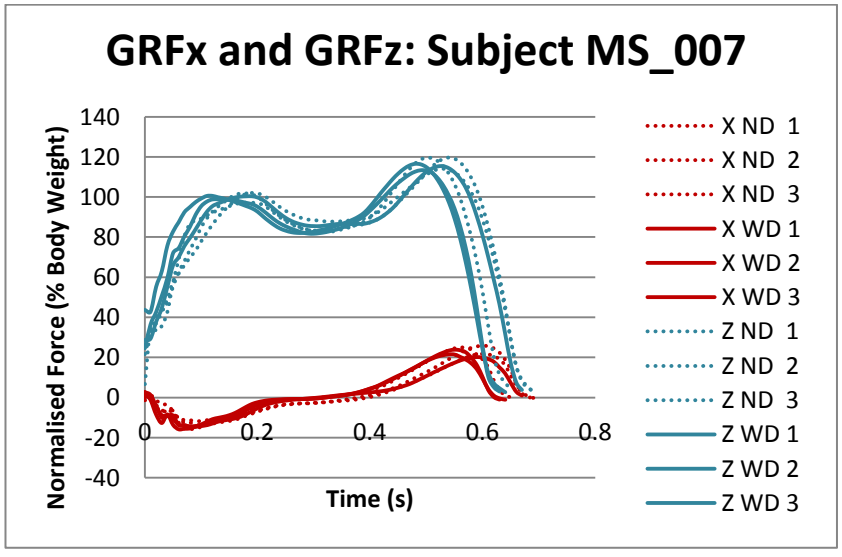







GRFx and GRFz: Subject MS_005
Normalised Force (% Body Weight)
120
100
80
60
40
20
0
-20
-40
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3


GRFx and GRFz: Subject MS_006
Normalised Force (% Body Weight)
140
120
100
80
60
40
20
0
-20
-40
0
0.2
0.4
0.6
0.8
1
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3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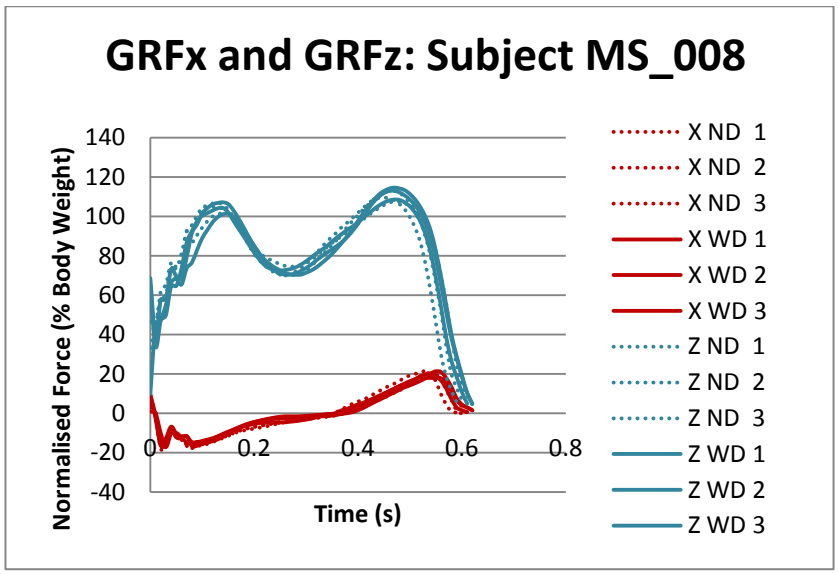
Normalised Force (% Body Weight)
140
120
100
80
60
40
20
0
-20
-40
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3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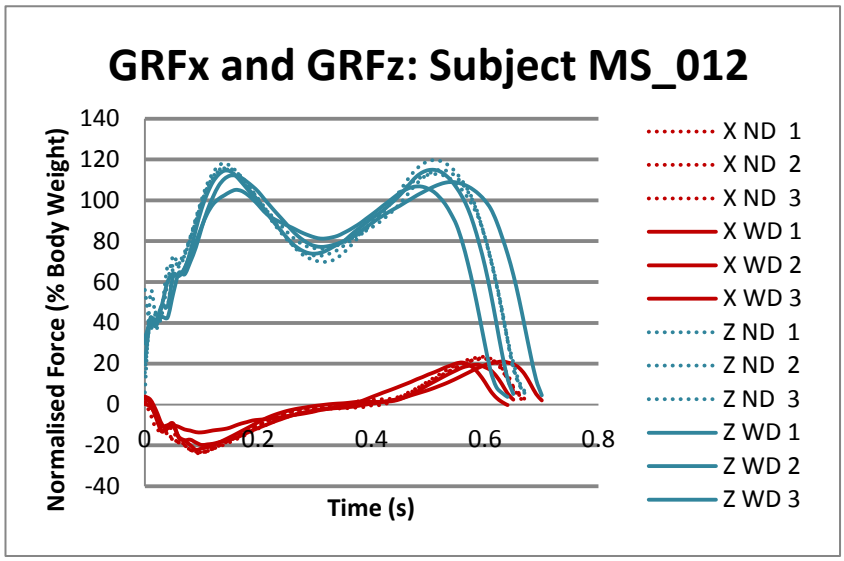
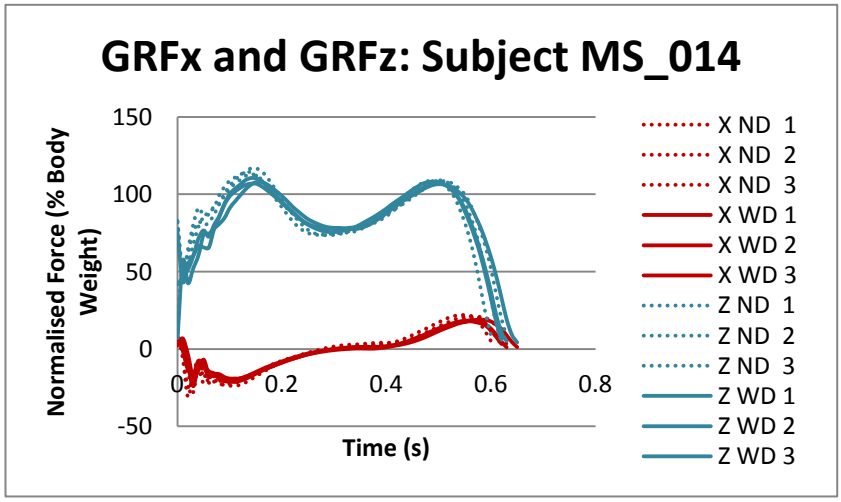
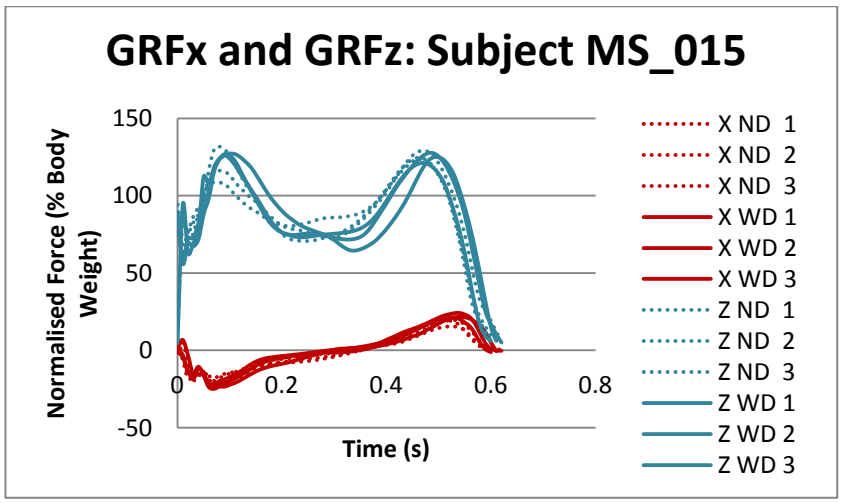
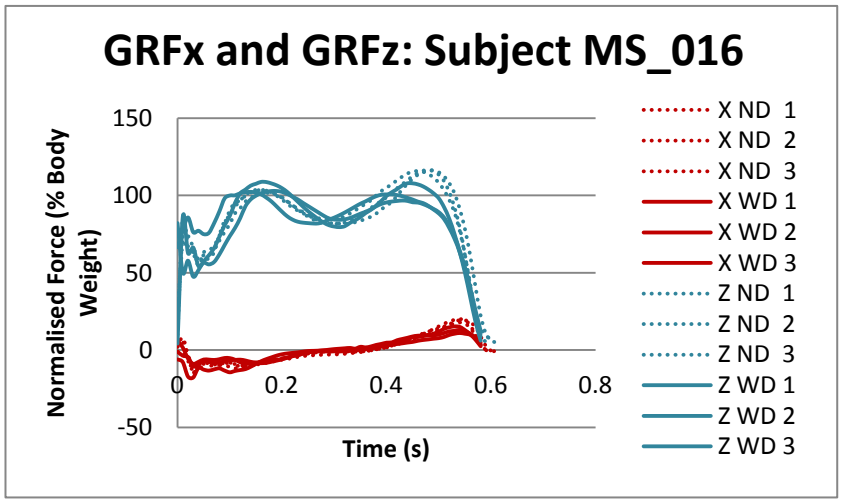
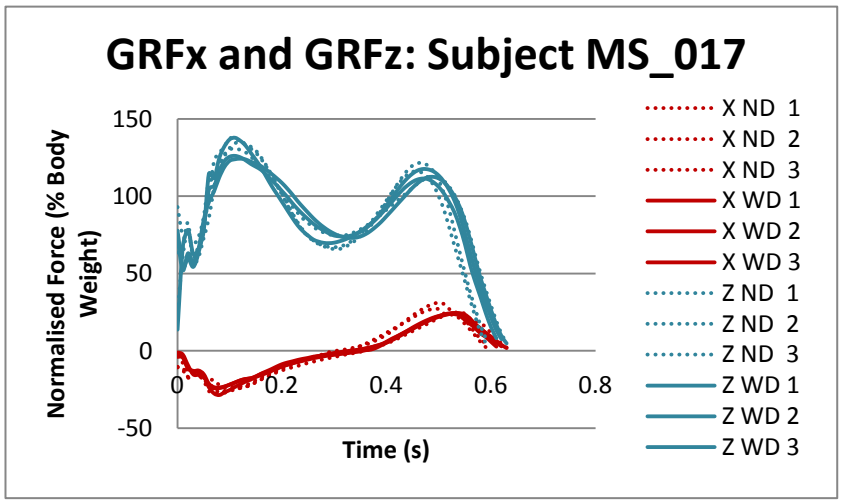
Normalised Force (% Body Weight)
140
120
100
80
60
40
20
0
-20
-40
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3


GRFx and GRFz: Subject MS_012
Normalised Force (% Body Weight)
140
120
100
80
60
40
20
0
-20
-40
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3


GRFx and GRFz: Subject MS_014
Normalised Force (% Body Weight)
150
100
50
0
-50
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3


GRFx and GRFz: Subject MS_015
Normalised Force (% Body Weight)
150
100
50
0
-50
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3


GRFx and GRFz: Subject MS_016
Normalised Force (% Body Weight)
150
100
50
0
-50
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3


GRFx and GRFz: Subject MS_017
Normalised Force (% Body Weight)
150
100
50
0
-50
0
0.2
0.4
0.6
0.8
Time (s)
X ND 1
X ND 2
X ND 3
X WD 1
X WD 2
X WD 3
Z ND 1
Z ND 2
Z ND 3
Z WD 1
Z WD 2
Z WD 3

APPENDIX E

Minitab datasheets showing descriptive statistics (count, mean, SE, SD, min., max) and results of paired t-tests comparing no device and with device groups for each gait parameter (NB variables denoted '_1' describe with device trial values).

Results for: Spatiotemporal Parameters

Descriptive Statistics: Walking Speed, Walking Speed, Stride Lengt, ...

Variable	Total Count	Mean	SE Mean	StDev	Minimum	Maximum
Walking Speed	12	1.2788	0.0419	0.1452	1.0508	1.5717
Walking Speed_1	12	1.2074	0.0520	0.1802	0.8601	1.5717
Stride Length	12	1.3969	0.0362	0.1255	1.1638	1.6628
Stride Length_1	12	1.3340	0.0450	0.1559	1.1529	1.6628
Step Length	12	0.7032	0.0186	0.0643	0.5800	0.8422
Step Length_1	12	0.6709	0.0245	0.0849	0.5568	0.8422
Stride Time	12	1.0986	0.0238	0.0823	0.9967	1.3100
Stride Time_1	12	1.1142	0.0301	0.1044	1.0100	1.3750
Step Time	12	0.5528	0.0114	0.0396	0.4933	0.6467
Step Time_1	12	0.5613	0.0138	0.0476	0.5000	0.6550
Left Single Support Dura	12	0.44306	0.00769	0.02665	0.39667	0.48333
Left Single Support Dura	12	0.4564	0.0130	0.0450	0.4050	0.5550
Right Single Support Dur	12	0.4442	0.0103	0.0342	0.4033	0.5300
Right Single Support Dur	12	0.4300	0.0172	0.0571	0.3000	0.5150
Left Double Support Dura	12	0.2126	0.0114	0.0379	0.1567	0.2850
Left Double Support Dura	12	0.2247	0.0128	0.0424	0.1567	0.3100
Right Double Support Dur	12	0.2120	0.0129	0.0427	0.1533	0.3050
Right Double Support Dur	12	0.2267	0.0130	0.0432	0.1533	0.3050
SSD Symmetry (LSSD/RSSD)	12	0.95594	0.00827	0.02742	0.90750	0.98582
SSD Symmetry (LSSD/RSSD)	12	0.9214	0.0211	0.0699	0.7230	0.9707
DSD Symmetry (LDSD/RDSD)	12	0.94803	0.00997	0.03308	0.88165	1.00000
DSD Symmetry (LDSD/RDSD)	12	0.94479	0.00960	0.03185	0.88312	0.98077

Paired T-Test and CI: Walking Speed, Walking Speed_1

Paired T for Walking Speed - Walking Speed_1

	N	Mean	StDev	SE Mean
Walking Speed	12	1.2788	0.1452	0.0419
Walking Speed_1	12	1.2074	0.1802	0.0520
Difference	12	0.0714	0.1009	0.0291

95% CI for mean difference: (0.0073, 0.1355)
T-Test of mean difference = 0 (vs not = 0): T-Value = 2.45 P-Value = 0.032

Paired T-Test and CI: Stride Length, Stride Length_1

Paired T for Stride Length - Stride Length_1

	N	Mean	StDev	SE Mean
Stride Length	12	1.3969	0.1255	0.0362
Stride Length_1	12	1.3340	0.1559	0.0450
Difference	12	0.0630	0.1014	0.0293

95% CI for mean difference: (-0.0015, 0.1274)
T-Test of mean difference = 0 (vs not = 0): T-Value = 2.15 P-Value = 0.055

Paired T-Test and CI: Step Length, Step Length_1

```
Paired T for Step Length - Step Length_1

               N    Mean   StDev  SE Mean
Step Length    12  0.7032  0.0643   0.0186
Step Length_1  12  0.6709  0.0849   0.0245
Difference     12  0.0323  0.0541   0.0156


95% CI for mean difference: (-0.0021, 0.0666)
T-Test of mean difference = 0 (vs not = 0): T-Value = 2.06  P-Value = 0.063
```

Paired T-Test and CI: Stride Time, Stride Time_1

```
Paired T for Stride Time - Stride Time_1

               N     Mean   StDev  SE Mean
Stride Time    12   1.0986  0.0823   0.0238
Stride Time_1  12   1.1142  0.1044   0.0301
Difference     12  -0.0156  0.0362   0.0105


95% CI for mean difference: (-0.0386, 0.0075)
T-Test of mean difference = 0 (vs not = 0): T-Value = -1.49  P-Value = 0.165
```

Paired T-Test and CI: Step Time, Step Time_1

```
Paired T for Step Time - Step Time_1

              N       Mean    StDev  SE Mean
Step Time    12    0.5528   0.0396   0.0114
Step Time_1  12    0.5613   0.0476   0.0138
Difference   12  -0.00847  0.02499  0.00721


95% CI for mean difference: (-0.02435, 0.00741)
T-Test of mean difference = 0 (vs not = 0): T-Value = -1.17  P-Value = 0.265
```

Paired T-Test and CI: Left Single Support Dura, Left Single Support Dura

```
Paired T for Left Single Support Duration (S - Left Single Support Duration _1

                          N      Mean    StDev  SE Mean
Left Single Support Dura  12    0.4431   0.0266   0.0077
Left Single Support Dura  12    0.4564   0.0450   0.0130
Difference                12  -0.01333  0.02803  0.00809


95% CI for mean difference: (-0.03114, 0.00448)
T-Test of mean difference = 0 (vs not = 0): T-Value = -1.65  P-Value = 0.128
```

Paired T-Test and CI: Right Single Support Dur, Right Single Support Dur

```
Paired T for Right Single Support Duration ( - Right Single Support Duration_1

                          N    Mean   StDev  SE Mean
Right Single Support Dur  11  0.4442  0.0342   0.0103
Right Single Support Dur  11  0.4300  0.0571   0.0172
Difference                11  0.0142  0.0347   0.0105


95% CI for mean difference: (-0.0091, 0.0376)
T-Test of mean difference = 0 (vs not = 0): T-Value = 1.36  P-Value = 0.203
```

Paired T-Test and CI: Left Double Support Dura, Left Double Support Dura

```
Paired T for Left Double Support Duration (D - Left Double Support Duration _1
```

```
                           N      Mean    StDev  SE Mean
Left Double Support Dura  11    0.2126   0.0379   0.0114
Left Double Support Dura  11    0.2247   0.0424   0.0128
Difference                11  -0.01212  0.03151  0.00950


95% CI for mean difference: (-0.03329, 0.00905)
T-Test of mean difference = 0 (vs not = 0): T-Value = -1.28  P-Value = 0.231
```

Paired T-Test and CI: Right Double Support Dur, Right Double Support Dur

```
Paired T for Right Double Support Duration ( - Right Double Support Duration_1

                           N      Mean    StDev  SE Mean
Right Double Support Dur  11    0.2120   0.0427   0.0129
Right Double Support Dur  11    0.2267   0.0432   0.0130
Difference                11  -0.01470  0.02619  0.00790


95% CI for mean difference: (-0.03229, 0.00290)
T-Test of mean difference = 0 (vs not = 0): T-Value = -1.86  P-Value = 0.092
```

Paired T-Test and CI: SSD Symmetry (LSSD/RSSD), SSD Symmetry (LSSD/RSSD)_1

```
Paired T for SSD Symmetry (LSSD/RSSD) - SSD Symmetry (LSSD/RSSD)_1

                           N    Mean   StDev  SE Mean
SSD Symmetry (LSSD/RSSD)  11  0.9559  0.0274   0.0083
SSD Symmetry (LSSD/RSSD)  11  0.9214  0.0699   0.0211
Difference                11  0.0345  0.0751   0.0226


95% CI for mean difference: (-0.0159, 0.0850)
T-Test of mean difference = 0 (vs not = 0): T-Value = 1.53  P-Value = 0.158
```

Paired T-Test and CI: DSD Symmetry (LDSD/RDSD), DSD Symmetry (LDSD/RDSD)_1

```
Paired T for DSD Symmetry (LDSD/RDSD) - DSD Symmetry (LDSD/RDSD)_1

                           N     Mean    StDev  SE Mean
DSD Symmetry (LDSD/RDSD)  11  0.94803  0.03308  0.00997
DSD Symmetry (LDSD/RDSD)  11  0.94479  0.03185  0.00960
Difference                11   0.0032   0.0415   0.0125


95% CI for mean difference: (-0.0246, 0.0311)
T-Test of mean difference = 0 (vs not = 0): T-Value = 0.26  P-Value = 0.801
```

Results for: Kinetic Parameters – Ground Reaction Forces
Descriptive Statistics: Z GRF Peak 1, Z GRF Peak 1, Z GRF Peak 2, ...

```
                    Total
Variable            Count    Mean  SE Mean  StDev  Minimum  Maximum
Z GRF Peak 1 (FZ1)     23  112.56     2.11  10.11    99.47   134.23
Z GRF Peak 1 (FZ1)_1   23  110.84     1.82   8.75    98.88   129.34
Z GRF Peak 2 (FZ2)     23    49.6     14.3   68.8    -27.8    125.8
Z GRF Peak 2 (FZ2)_1   23   66.85     9.31  44.64    15.64   124.53
X GRF MIN (FX1)        23    41.5     13.9   66.6    -27.8    129.3
X GRF MIN (FX1)_1      23    42.5     13.5   64.6    -25.5    124.5
X GRF MAX (FX2)        23    2.58     4.27  20.47   -25.45    30.43
X GRF MAX (FX2)_1      23  17.949    0.803  3.851   12.648   24.132
```

Paired T-Test and CI: Z GRF Peak 1 (FZ1), Z GRF Peak 1 (FZ1)_1

```
Paired T for Z GRF Peak 1 (FZ1) - Z GRF Peak 1 (FZ1)_1
```

	N	Mean	StDev	SE Mean
Z GRF Peak 1 (FZ1)	23	112.56	10.11	2.11
Z GRF Peak 1 (FZ1)_1	23	110.84	8.75	1.82
Difference	23	1.72	9.69	2.02

95% CI for mean difference: (-2.47, 5.91)
T-Test of mean difference = 0 (vs not = 0): T-Value = 0.85 P-Value = 0.404

Paired T-Test and CI: Z GRF Peak 2 (FZ2), Z GRF Peak 2 (FZ2)_1

Paired T for Z GRF Peak 2 (FZ2) - Z GRF Peak 2 (FZ2)_1

	N	Mean	StDev	SE Mean
Z GRF Peak 2 (FZ2)	23	49.6	68.8	14.3
Z GRF Peak 2 (FZ2)_1	23	66.9	44.6	9.3
Difference	23	-17.20	25.55	5.33

95% CI for mean difference: (-28.25, -6.15)
T-Test of mean difference = 0 (vs not = 0): T-Value = -3.23 P-Value = 0.004

Paired T-Test and CI: X GRF MIN (FX1), X GRF MIN (FX1)_1

Paired T for X GRF MIN (FX1) - X GRF MIN (FX1)_1

	N	Mean	StDev	SE Mean
X GRF MIN (FX1)	23	41.5	66.6	13.9
X GRF MIN (FX1)_1	23	42.5	64.6	13.5
Difference	23	-1.02	6.12	1.28

95% CI for mean difference: (-3.67, 1.62)
T-Test of mean difference = 0 (vs not = 0): T-Value = -0.80 P-Value = 0.431

Paired T-Test and CI: X GRF MAX (FX2), X GRF MAX (FX2)_1

Paired T for X GRF MAX (FX2) - X GRF MAX (FX2)_1

	N	Mean	StDev	SE Mean
X GRF MAX (FX2)	23	2.58	20.47	4.27
X GRF MAX (FX2)_1	23	17.95	3.85	0.80
Difference	23	-15.37	21.02	4.38

95% CI for mean difference: (-24.46, -6.28)
T-Test of mean difference = 0 (vs not = 0): T-Value = -3.51 P-Value = 0.002

Results for: Kinematic Parameters – Joint Angles

Descriptive Statistics: Ankle Angles, Ankle Angles, Ankle Angles, ...

Variable	Total Count	Mean	SE Mean	StDev	Minimum	Maximum
Ankle Angles Peak(P1)	12	15.62	1.09	3.79	9.36	20.85
Ankle Angles Peak(P1)_1	12	21.05	2.93	10.14	9.59	49.08
Ankle Angles Min (M1)	12	-22.33	2.90	10.03	-47.42	-11.80
Ankle Angles Min (M1)_1	12	-7.99	2.23	7.73	-20.77	8.08
Hip Angles Peak (P1)	12	27.92	2.92	10.12	13.92	42.79
Hip Angles Peak(P1)	12	33.93	2.28	7.89	19.08	43.94
Hip Angles Min (M1)	12	-9.11	2.63	9.10	-31.00	0.29
Hip Angle Min (M1)	12	-10.40	2.94	10.18	-31.00	7.79
Knee Angle Peak (P1)	12	62.19	1.72	5.97	55.28	72.25
Knee Angle Peak (P1)_1	12	59.50	1.82	6.32	49.14	73.21
Knee Angle Min (M1)	12	-0.61	3.29	11.38	-31.00	14.16
Knee Angle Min (M1)_1	12	1.09	3.55	12.28	-31.00	16.06

Paired T-Test and CI: Ankle Angles Peak(P1), Ankle Angles Peak(P1)_1

```
Paired T for Ankle Angles Peak(P1) - Ankle Angles Peak(P1)_1

                          N   Mean  StDev  SE Mean
Ankle Angles Peak(P1)    12  15.62   3.79     1.09
Ankle Angles Peak(P1)_1  12  21.05  10.14     2.93
Difference               12  -5.43   8.16     2.36


95% CI for mean difference: (-10.61, -0.24)
T-Test of mean difference = 0 (vs not = 0): T-Value = -2.30  P-Value = 0.042
```

Paired T-Test and CI: Ankle Angles Min (M1), Ankle Angles Min (M1)_1

```
Paired T for Ankle Angles Min (M1) - Ankle Angles Min (M1)_1

                          N    Mean  StDev  SE Mean
Ankle Angles Min (M1)    12  -22.33  10.03     2.90
Ankle Angles Min (M1)_1  12   -7.99   7.73     2.23
Difference               12  -14.35  10.96     3.16


95% CI for mean difference: (-21.31, -7.38)
T-Test of mean difference = 0 (vs not = 0): T-Value = -4.53  P-Value = 0.001
```

Paired T-Test and CI: Hip Angles Peak (P1), Hip Angles Peak(P1)

```
Paired T for Hip Angles Peak (P1) - Hip Angles Peak(P1)

                       N   Mean  StDev  SE Mean
Hip Angles Peak (P1)  12  27.92  10.12     2.92
Hip Angles Peak(P1)   12  33.93   7.89     2.28
Difference            12  -6.01  13.80     3.98


95% CI for mean difference: (-14.78, 2.76)
T-Test of mean difference = 0 (vs not = 0): T-Value = -1.51  P-Value = 0.160
```

Paired T-Test and CI: Hip Angles Min (M1), Hip Angle Min (M1)

```
Paired T for Hip Angles Min (M1) - Hip Angle Min (M1)

                      N    Mean  StDev  SE Mean
Hip Angles Min (M1)  12   -9.11   9.10     2.63
Hip Angle Min (M1)   12  -10.40  10.18     2.94
Difference           12    1.29   5.17     1.49


95% CI for mean difference: (-2.00, 4.58)
T-Test of mean difference = 0 (vs not = 0): T-Value = 0.86  P-Value = 0.406
```

Paired T-Test and CI: Knee Angle Peak (P1), Knee Angle Peak (P1)_1

```
Paired T for Knee Angle Peak (P1) - Knee Angle Peak (P1)_1

                          N   Mean  StDev  SE Mean
Knee Angle Peak (P1)     12  62.19   5.97     1.72
Knee Angle Peak (P1)_1   12  59.50   6.32     1.82
Difference               12   2.69   7.37     2.13


95% CI for mean difference: (-1.99, 7.38)
T-Test of mean difference = 0 (vs not = 0): T-Value = 1.27  P-Value = 0.232
```

Paired T-Test and CI: Knee Angle Min (M1), Knee Angle Min (M1)_1

Paired T for Knee Angle Min (M1) - Knee Angle Min (M1)_1

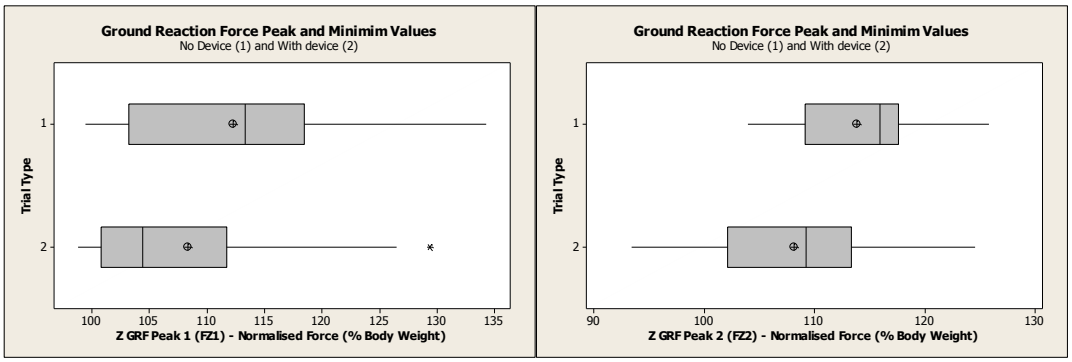
	N	Mean	StDev	SE Mean
Knee Angle Min (M1)	12	-0.61	11.38	3.29
Knee Angle Min (M1)_1	12	1.09	12.28	3.55
Difference	12	-1.703	2.782	0.803

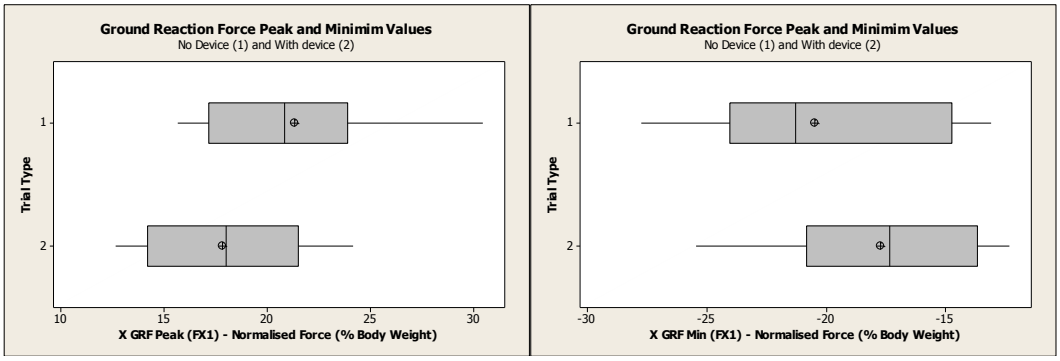
95% CI for mean difference: (-3.470, 0.064)
T-Test of mean difference = 0 (vs not = 0): T-Value = -2.12 P-Value = 0.058

APPENDIX F

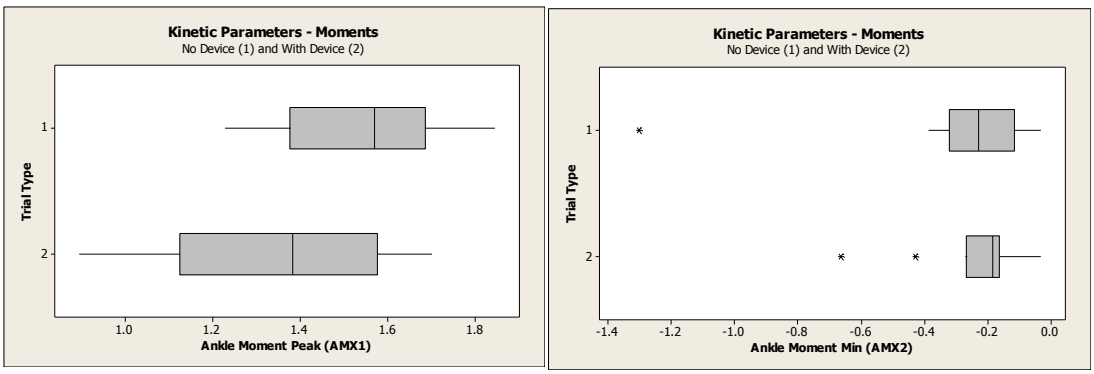
Box and whisker plots for each parameter showing max (furthermost point of line to right), min (furthermost point of line to left), interquartile (left and right margins of box), medial (vertical line within box), mean (⊕) values, and outliers (*) for no device and with device groups.

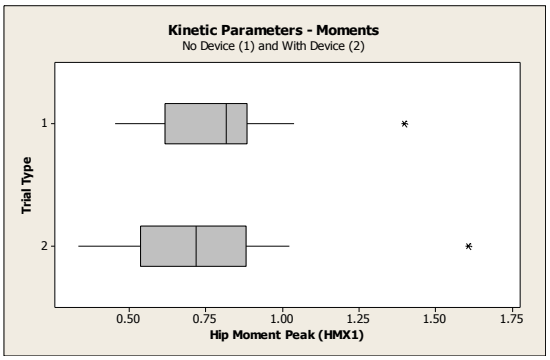
Kinetic Parameters: Ground Reaction Forces

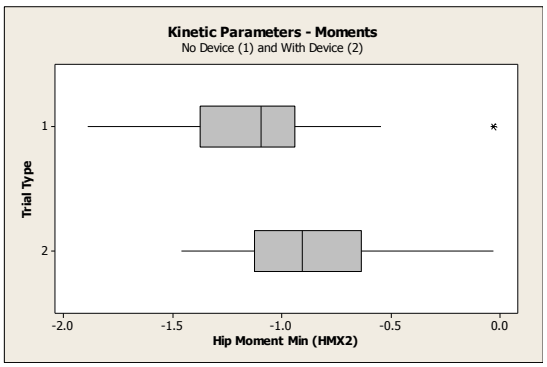


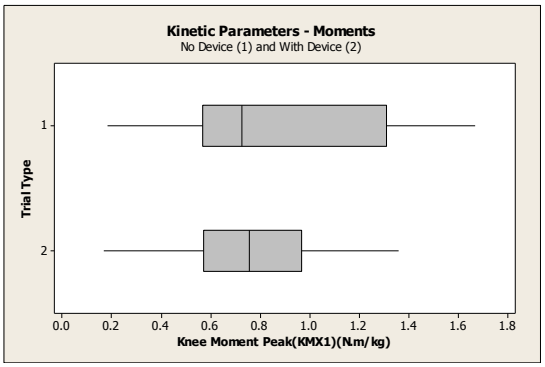


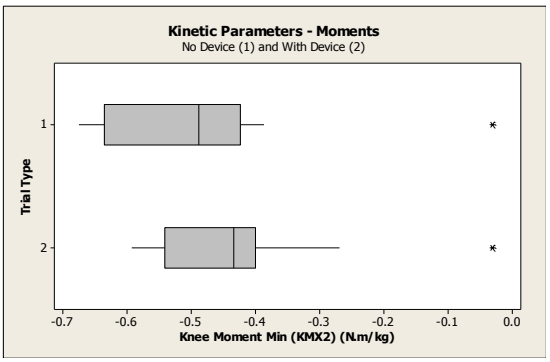
Kinetic Parameters: Joint Moments



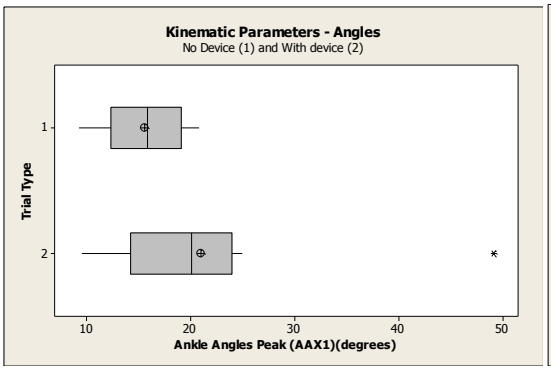


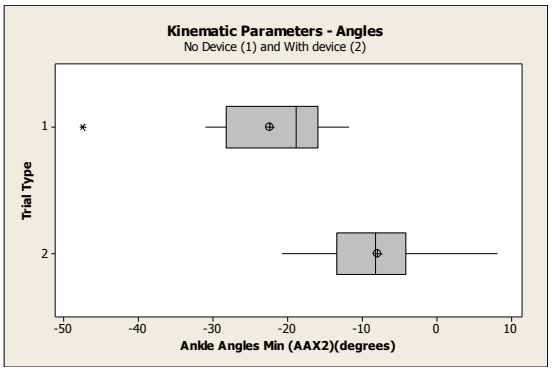


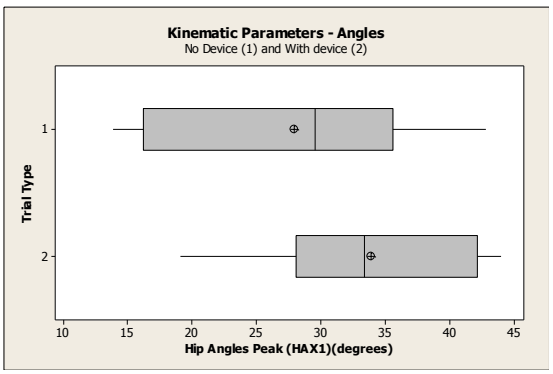


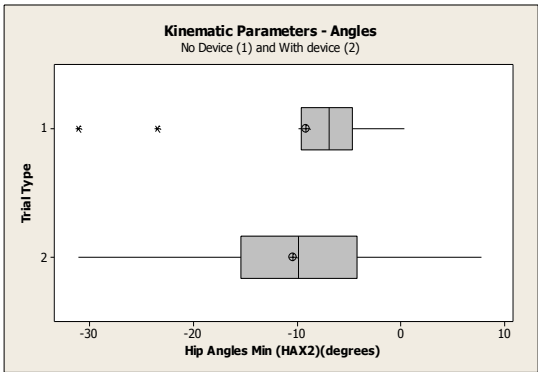


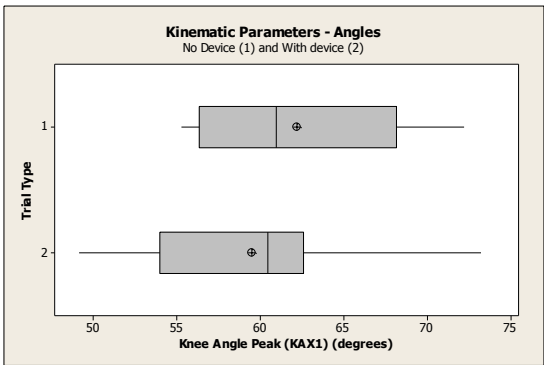
Kinematic Parameters: Joint Angles

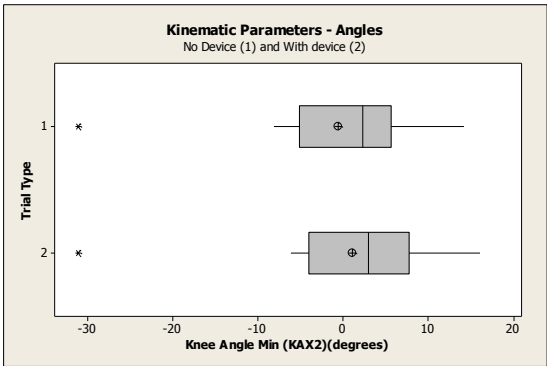




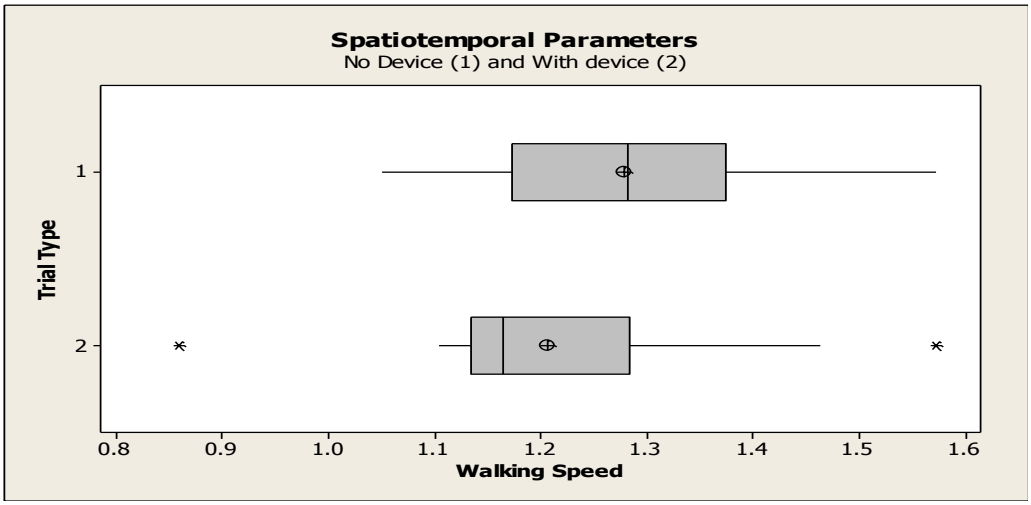


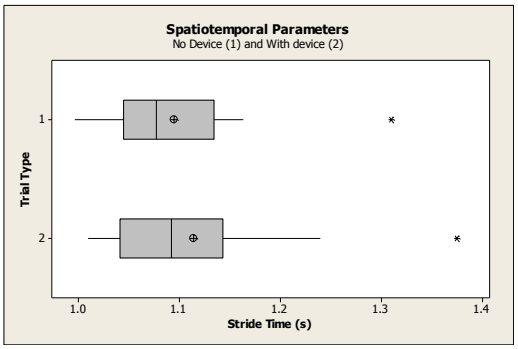


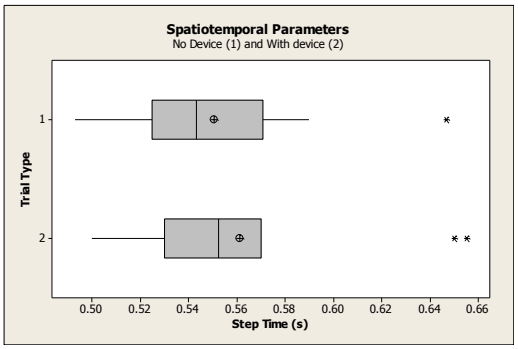


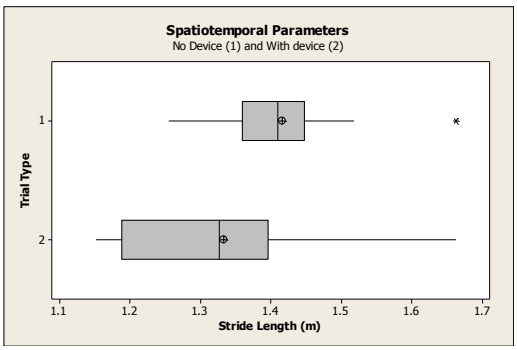


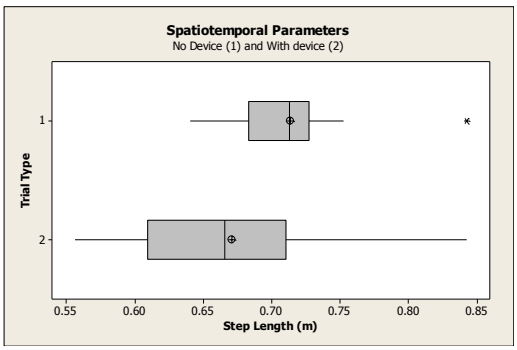
Spatiotemporal Parameters

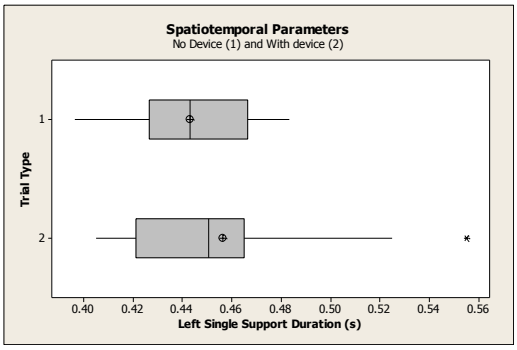


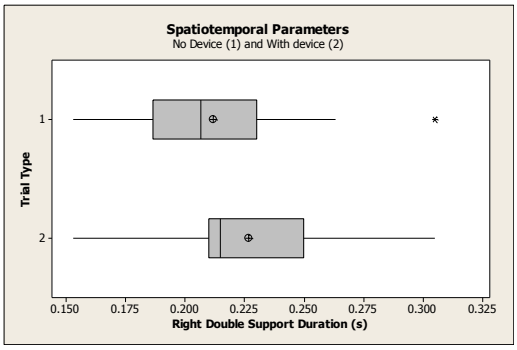


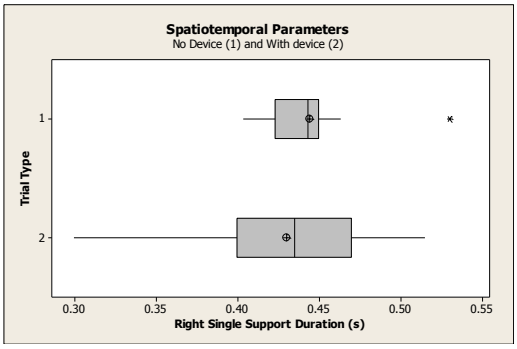


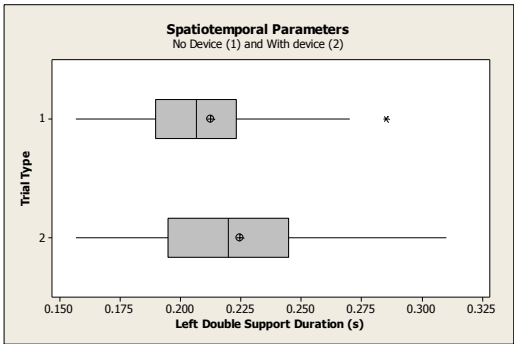

Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
1.1
1.2
1.3
1.4
1.5
1.6
1.7
Stride Length (m)

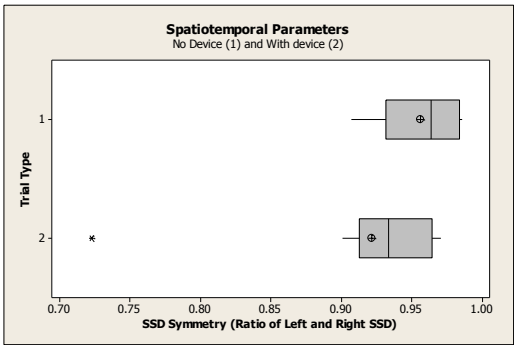

Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.55
0.60
0.65
0.70
0.75
0.80
0.85
Step Length (m)

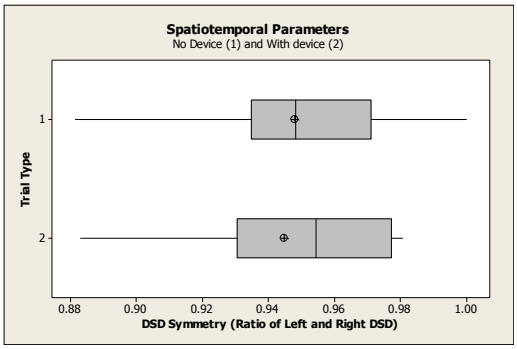

Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.40
0.42
0.44
0.46
0.48
0.50
0.52
0.54
0.56
Left Single Support Duration (s)


Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.150
0.175
0.200
0.225
0.250
0.275
0.300
0.325
Right Double Support Duration (s)


Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.30
0.35
0.40
0.45
0.50
0.55
Right Single Support Duration (s)


Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.150
0.175
0.200
0.225
0.250
0.275
0.300
0.325
Left Double Support Duration (s)


Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.70
0.75
0.80
0.85
0.90
0.95
1.00
SSD Symmetry (Ratio of Left and Right SSD)


Spatiotemporal Parameters
No Device (1) and With device (2)
Trial Type
1
2
0.88
0.90
0.92
0.94
0.96
0.98
1.00
DSD Symmetry (Ratio of Left and Right DSD)

APPENDIX G

Spatiotemporal, kinetic and kinematic parameter intra-participant mean values for all participant trials, with intra-group mean and standard deviation values indicated by bold typeface.

Spatiotemporal Parameters –No Device

GAIT PARAMETER	Walking Speed	Left Single Support Duration (SSD)	Left Double Support Duration (DSD)	Right Single Support Duration (SSD)	Right Double Support Duration (DSD)	Stride Length	Step Length	SSD Symmetry	DSD Symmetry	Stride Time	Step Time
PARTICIPANT	m/s	s	s	s	s	m	m	Ratio	Ratio	s	s
MS_001	1.234	0.440	0.207	0.450	0.220	1.366	0.694	0.964	0.944	1.107	0.563
MS_002	1.271	0.470	0.223	0.463	0.210	1.453	0.721	0.986	0.941	1.143	0.573
MS_003	1.393	0.397	0.197	0.403	0.197	1.388	0.704	0.984	0.935	0.997	0.493
MS_005	1.051	0.427	0.270	0.427	0.263	1.164	0.580	0.985	0.882	1.120	0.567
MS_006	1.088	0.480	0.285	0.530	0.305	1.425	0.729	0.908	0.907	1.310	0.647
MS_007	1.155	0.433				1.256	0.641			1.083	0.543
MS_008	1.308	0.427	0.193	0.423	0.190	1.358	0.680	0.922	1.000	1.040	0.523
MS_012	1.231	0.483	0.223	0.450	0.230	1.432	0.720	0.932	0.971	1.163	0.590
MS_014	1.322	0.447	0.190	0.440	0.187	1.418	0.720	0.970	0.948	1.073	0.540
MS_015	1.428	0.447	0.177	0.443	0.170	1.518	0.752	0.935	0.966	1.063	0.543
MS_016	1.292	0.410	0.217	0.407	0.207	1.323	0.653	0.960	0.954	1.023	0.520
MS_017	1.572	0.457	0.157	0.450	0.153	1.663	0.842	0.971	0.980	1.060	0.530
Mean	**1.279**	**0.443**	**0.213**	**0.444**	**0.212**	**1.397**	**0.703**	**0.956**	**0.948**	**1.099**	**0.553**
SD	**0.145**	**0.027**	**0.038**	**0.034**	**0.043**	**0.126**	**0.064**	**0.027**	**0.033**	**0.082**	**0.040**

Spatiotemporal Parameters-With device

GAIT PARAMETER	Walking Speed	Left Single Support Duration (SSD)	Left Double Support Duration (DSD)	Right Single Support Duration (SSD)	Right Double Support Duration (DSD)	Stride Length	Step Length	SSD Symmetry	DSD Symmetry	Stride Time	Step Time
PARTICIPANT	m/s	s	s	s	s	m	m	Ratio	Ratio	s	s
MS_001	1.150	0.465	0.195	0.470	0.210	1.317	0.667	0.948	0.931	1.145	0.550
MS_002	1.133	0.525	0.235	0.490	0.225	1.404	0.722	0.933	0.957	1.240	0.650
MS_003	1.141	0.405	0.190	0.390	0.215	1.153	0.605	0.963	0.883	1.010	0.500
MS_005	1.105	0.445	0.245	0.400	0.250	1.210	0.617	0.901	0.981	1.095	0.560
MS_006	0.860	0.555	0.275	0.515	0.305	1.183	0.612	0.928	0.901	1.375	0.655
MS_007	1.181	0.440				1.303	0.609			1.105	0.555
MS_008	1.292	0.405	0.220	0.420	0.210	1.337	0.668	0.964	0.955	1.035	0.515
MS_012	1.183	0.465	0.235	0.440	0.235	1.347	0.675	0.924	0.958	1.140	0.570
MS_014	1.258	0.440	0.210	0.435	0.215	1.371	0.665	0.966	0.977	1.090	0.550
MS_015	1.464	0.460	0.200	0.420	0.185	1.559	0.812	0.913	0.935	1.065	0.570
MS_016	1.150	0.415	0.310	0.300	0.290	1.161	0.557	0.723	0.935	1.010	0.530
MS_017	1.572	0.457	0.157	0.450	0.153	1.663	0.842	0.971	0.980	1.060	0.530
Mean	**1.207**	**0.456**	**0.225**	**0.430**	**0.227**	**1.334**	**0.671**	**0.921**	**0.945**	**1.114**	**0.561**
SD	0.180	0.045	0.042	0.057	0.043	0.156	0.085	0.070	0.032	0.104	0.048

Kinetic Parameters-No device

GAIT PARAMETER	GRFz Peak 1 (FZ1)	GRFz Peak 2 (FZ2)	GRFx Peak (FX1)	GRFx Min (FX2)	Ankle Moment Peak(P1)	Ankle Moment Min (M1)	Hip Moment Peak(P1)	Hip Moment Min (M1)	Knee Moment Peak(P1)	Knee Moment Min (M1)
PARTICIPANT	% bw	% bw	% bw	% bw	Nm/kg	Nm/kg	Nm/kg	Nm/kg	Nm/kg	Nm/kg
MS_001	105.787	117.291	16.285	-21.243	1.571	-1.302	0.856	-1.399	1.333	-0.491
MS_002	111.945	104.632	30.427	-23.919	1.682	-0.174	0.747	-1.083	0.682	-0.467
MS_003	120.705	104.009	23.021	-24.049	1.230	-0.214	0.591	-1.275	1.250	-0.409
MS_005	99.466	111.775	15.640	-13.092	1.282	-0.242	0.798	-1.010	0.574	-0.475
MS_006	117.521	117.827	16.849	-19.467	1.844	-0.031	0.511	-0.031	1.668	-0.031
MS_007	100.319	117.748	24.181	-13.782	1.688	-0.093	0.706	-0.915	0.186	-0.590
MS_008	104.445	110.129	20.375	-17.664	1.372	-0.286	0.888	-1.302	0.773	-0.485
MS_012	116.511	115.839	22.794	-23.508	1.569	-0.211	0.456	-1.019	0.565	-0.387
MS_014	114.699	108.788	21.278	-27.751	1.528	-0.095	1.038	-0.546	0.200	-0.675
MS_015	118.823	125.809	18.034	-21.333	1.602	-0.334	1.399	-1.551	0.658	-0.552
MS_016	102.888	116.072	19.126	-13.672	1.398	-0.273	0.878	-1.103	0.789	-0.662
MS_017	134.229	116.553	27.599	-26.291	1.708	-0.385	0.837	-1.888	1.561	-0.651
MEAN	**112.278**	**113.873**	**21.301**	**-20.481**	**1.539**	**-0.303**	**0.809**	**-1.093**	**0.853**	**-0.490**
SD	**10.206**	**6.235**	**4.570**	**5.013**	**0.186**	**0.331**	**0.250**	**0.475**	**0.492**	**0.174**

Kinetic Parameters-With device

GAIT PARAMETER	GRFz Peak 1 (FZ1)	GRFz Peak 2 (FZ2)	GRFx Peak (FX1)	GRFx Min (FX2)	Ankle Moment Peak(P1)	Ankle Moment Min (M1)	Hip Moment Peak(P1)	Hip Moment Min (M1)	Knee Moment Peak(P1)	Knee Moment Min (M1)
PARTICIPANT	% bw	% bw	% bw	% bw	Nm/kg	Nm/kg	Nm/kg	Nm/kg	Nm/kg	Nm/kg
MS_001	100.536	108.346	14.860	-16.299	1.315	-0.271	1.608	-1.457	0.580	-0.420
MS_002	112.129	110.628	17.561	-18.045	1.364	-0.163	0.707	-1.133	0.702	-0.425
MS_003	103.119	93.461	14.716	-20.346	0.898	-0.169	0.816	-1.198	0.997	-0.270
MS_005	98.879	97.760	14.054	-13.243	0.900	-0.241	1.022	-1.093	0.585	-0.428
MS_006	101.808	103.302	12.648	-12.334	1.105	-0.031	0.487	-0.031	0.886	-0.031
MS_007	99.928	115.095	21.868	-14.964	1.616	-0.186	0.664	-0.973	0.206	-0.594
MS_008	104.275	112.105	19.620	-16.658	1.597	-0.428	0.576	-0.839	0.815	-0.481
MS_012	110.568	110.224	20.309	-18.560	1.512	-0.162	0.866	-0.840	0.568	-0.393
MS_014	108.453	107.281	18.477	-20.954	1.470	-0.183	0.524	-0.602	0.170	-0.588
MS_015	126.497	124.526	22.394	-23.328	1.404	-0.664	0.888	-0.591	1.187	-0.542
MS_016	104.631	101.737	13.203	-12.339	1.192	-0.251	0.730	-0.975	0.854	-0.440
MS_017	129.342	113.804	24.132	-25.453	1.702	-0.137	0.335	-0.742	1.358	-0.542
MEAN	**108.347**	**108.189**	**17.820**	**-17.710**	**1.340**	**-0.240**	**0.769**	**-0.873**	**0.742**	**-0.429**
SD	**10.052**	**8.332**	**3.905**	**4.244**	**0.268**	**0.163**	**0.327**	**0.365**	**0.354**	**0.155**

Kinematic Parameters-No device

GAIT PARAMETER	Ankle Angles Peak (AAX1)	Ankle Angles Min (AAX2)	Hip Angles Peak (HAX1)	Hip Angles Min (HAX2)	Knee Angle Peak (KAX1)	Knee Angle Min (KAX2)
PARTICIPANT	degrees	degrees	degrees	degrees	degrees	degrees
MS_001	19.580	-12.911	31.618	-7.626	62.119	0.082
MS_002	14.988	-11.800	36.204	-7.060	59.206	5.684
MS_003	19.105	-47.423	33.782	-6.762	67.510	4.653
MS_005	19.034	-20.311	42.788	0.288	68.589	5.448
MS_006	12.229	-30.997	27.524	-30.997	59.793	-30.997
MS_007	11.301	-28.710	33.566	-8.805	56.345	-5.655
MS_008	20.847	-23.649	39.984	-0.670	68.362	-1.269
MS_012	13.342	-26.426	21.496	-23.386	55.369	-8.112
MS_014	12.756	-16.279	25.426	-9.889	55.283	-3.408
MS_015	9.355	-16.286	13.917	-4.638	65.015	5.052
MS_016	16.804	-17.331	14.212	-4.817	72.245	14.156
MS_017	18.155	-15.868	14.508	-4.999	56.447	6.995
MEAN	**15.625**	**-22.333**	**27.919**	**-9.113**	**62.190**	**-0.614**
SD	**3.627**	**9.607**	**9.690**	**8.710**	**5.719**	**10.900**

Kinematic Parameters-With device

GAIT PARAMETER	Ankle Angles Peak (AAX1)	Ankle Angles Min (AAX2)	Hip Angles Peak (HAX1)	Hip Angles Min (HAX2)	Knee Angle Peak (KAX1)	Knee Angle Min (KAX2)
PARTICIPANT	degrees	degrees	degrees	degrees	degrees	degrees
MS_001	24.079	-6.079	29.392	-8.940	60.533	2.948
MS_002	19.412	0.852	35.418	-6.397	60.396	7.235
MS_003	49.083	-4.024	31.361	-5.966	62.706	5.645
MS_005	23.722	-7.661	42.595	-3.593	58.292	2.943
MS_006	13.841	-20.769	25.994	-30.997	49.144	-30.997
MS_007	14.149	-12.413	27.663	-16.367	52.405	-4.747
MS_008	25.014	-13.705	37.041	-2.151	62.334	-1.870
MS_012	9.589	-10.982	19.079	-24.295	53.357	-6.117
MS_014	14.633	-8.817	30.510	-12.358	56.031	-0.991
MS_015	15.199	-15.936	43.942	-10.781	63.211	7.889
MS_016	23.048	8.083	40.629	7.785	62.360	15.067
MS_017	20.840	-4.391	43.489	-10.782	73.208	16.058
MEAN	**21.051**	**-7.987**	**33.926**	**-10.403**	**59.498**	**1.089**
SD	**9.708**	**7.401**	**7.556**	**9.746**	**6.047**	**11.760**