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A Comparison of the Aircast™ and Airstep™ Walkers Off-Loading Capabilities; Pilot Study

Master Thesis

by

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Declaration

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Abstract

It is estimated that there are currently 3.2 million individuals in the UK who are diagnosed with Diabetes Myelitis (DM), whereof 2.5 percent are affected by foot complications. Ischemia, neuropathy and infection are the three pathological components that lead to diabetic foot complications. The most common trauma, plantar ulceration, arise from extrinsic or intrinsic mechanical stress, with high peak pressures identified as a major contributor. Consequently, total contact casts, or removable walkers have been clinically used to relieve these pressures.

This project has investigated the commercially available Aircast™ and Airstep™ Walker Systems in relation to their respective off-loading capabilities. The validated Tekscan™ in-shoe pressure measurement system was utilised to monitor loading patterns during static and dynamic walking tests. In addition the off-loading effect from the embedded adjustable air bladder system and product specific rocker sole profile was investigated.

Results indicated that the Airstep™ from Promedics Orthopaedics ltd. demonstrated a superior offloading and greater reduced peak pressures for the tested (n=2) subjects. It is acknowledged that a more extensive study with a substantial sample size is required to draw any clinical relevant and significant conclusions.

Contents

1 Introduction

Diabetes mellitus (DM), commonly referred to as diabetes, is a group of metabolic diseases in which there are high blood sugar levels over a prolonged period of time. Diabetes is a debilitating medical condition affecting millions each year (Diabetes UK, 2013). This is caused by the pancreas not producing enough insulin or the cells of the body not responding properly to the insulin produced. If left untreated, diabetes can cause many complications (National Health Service (NHS), 2012). Acute complications include diabetic ketoacidosis and non-ketotic hyperosmolar coma (Diabetes UK, 2013). Serious long-term complications include cardiovascular disease, stroke, chronic kidney failure, damage to the eyes and last but not least foot ulcers (NHS, 2012). Ischemia, neuropathy and infection are the three pathological components that can lead to diabetic foot complications, which can then result in chronic ulcers.

In the UK 2.5% of diabetics suffer from ulcers on the planter surfaces of their feet at any given time, commonly under the metatarsal heads; the probability of these ulcers resulting in amputation is very high. There are over 100 amputations a week in England alone, the majority of these preceded by a foot ulcer (Diabetes UK, 2014). There are many ways in which to treat these plantar surface ulcers, but in order for them to heal the foot must be off-loading long enough for the treatment to be effective. When off-loading the foot, the primary goals are to preserve foot function and rectify abnormal gait patterns; all while the forces produces during gait are evenly distributed over the entire surface of the plantar foot. This reduces peak pressures and therefore will protect the foot from further damage and allow the ulcer time to heal (Cavanagh & Bus 2010).

Off-loading can be achieved in different forms but there is a consensus that a rigid total contact Plaster of Paris (POP) cast is considered to be the gold standard of off-loading devices (Ho et al. 2013; Mrdjenovich 2010). While it is

true that the pressure reductions on the plantar surfaces are great, the cast is contraindicated in cases of soft tissue infections or osteomyelitis. In addition, the POP cast has to be applied by a specialist clinician and has the potential to affect patients' quality of life.

The removable, commercially available cast walker is proven as effective as the total contact POP cast, as long as the patient is committed wearing it. The removable walkers claim to provide similar off-loading and enable monitoring of ulcers and daily hygienic care (Armstrong et al. 2005).

The following project aims are identified:

(i) To explore the mechanisms contributing to off-loading of the selected Aircast™ and Airstep™ Walker Systems.

(ii) To explore the differences in rocker sole design utilising kinetic and kinematic motion analysis.

(iii) To provide Promedics Orthopeadics ltd with an awareness of off-loading behaviour in the Airstep™ Walker system and possible optimisation.

2 Background

2.1 Diabetes myelitis (DM)

Diabetes mellitus (DM) is a disease that has reached worldwide epidemic proportions, caused by genetic factors or lifestyle actions. It is a disease with a wide range of disastrous complications, placing a substantial burden on each countries healthcare system, and can devastate people's lives (Boulton et al. 2005). The World Health Organisation estimated that 347 million people worldwide suffer from diabetes, expecting it to be the 7th leading cause of death by 2030 (World Health Organisation (WHO), 2013).

DM is caused by the bodies' inability to transport glucose from within the blood into the cells that require it. Glucose is the human bodies' primary source of energy, which can be stored as glycogen, but it needs to be actively transported into a cell (McInnes 2012). To be actively transported, the glucose needs insulin to bind to its corresponding receptor on the cell surface. With the insulin bound, the glucose transport facilitators are allowed to move to the cell surface by the phosphorylation of intracellular proteins, thus creating the ability for the glucose to diffuse into a cell. This illustrates how important the insulin structure is, without it the human body cannot store energy to be used after food has been consumed; therefore the blood contains a high glucose level (Van Belle et al. 2011). Insulin is a structure, made up of two peptide chains containing a total of 51 amino acids, and produced within the endocrine pancreas, specifically from the beta cells within the Islets of Langerhans (Martini, F.H & Nath 2009). Diabetics can suffer from either an absolute deficiency of insulin secretion or a biological resistance to the effectiveness of insulin, called type 1 or type 2 respectively. Other types of DM include gestational and maturity onset diabetes of the young (MODY). This review refers only to T1DM and T2DM.

Type 1 diabetes (T1DM) used to be commonly known as juvenile diabetes, as it was more likely to occur in younger people (depending on who reports, either

under 15 or under 30), though it can occur at any age. More recently is has begun to occur more widely throughout different age categories. Type 1 is a chronic autoimmune disorder thought to manifest in genetically susceptible individuals through environmental factors. This autoimmune disorder selectively attacks and destroys the beta cells within the Islets of Langerhans, resulting in a reduction of insulin production and eventually eliminates production of the insulin entirely (Van Belle et al. 2011).

Type 2 diabetes (T2DM) used to be commonly known as adult onset diabetes from the same premise as above; it was more likely to occur later in life. Now that it is known to onset increasingly because of obesity, which can unfortunately occur at any age. Defining diabetes by the age of the patient has become redundant. Type 2 diabetes is commonly associated with an unhealthy lifestyle, while also resulting from a genetic predisposition. The resistance to insulin action can present in the patient years prior to the actual development of diabetes. First, as a response to the lack of action, the patients' beta cells will overproduce insulin cells to maintain the correct level of glucose. Overtime, these beta cells will produce less insulin, resulting in higher glucose levels (Lindsay & Bennett 2001; Schaper et al. 2000; Vinik 1999).

One of the major complications associated with both T1DM and T2DM is diabetic neuropathy (DN). DN is a major contributor to the morbidities and mortality of those suffering diabetes. Morbidities closely related to DN are ulceration and amputation. DM is responsible for more than half of all lower limb amputations (Diabetes UK, 2012, Perkins & Bril 2003) making it the single most common cause of lower limb amputation (Diabetes UK, 2012). Of those that undergo amputations, approximately 70% will die within the 5 following years (Diabetes UK, 2012). It is estimated that approximately half of DM sufferers will develop neuropathy, most commonly diabetic sensorimotor polyneuropathy (DSPN) a combination of motor, sensory and autonomic neuropathy (Tracy & Dyck 2008). Between 60-70% of diabetic foot ulcers are neuropathy related and, when considering that 80-85% of amputations are

preceded by ulceration, shows the risk DN poses to the diabetic (Pritchard et al. 2011; Alavi et al. 2014; Edwards et al. 2008).

In the UK it is estimated that more than one in seventeen people have diabetes, about 3.2 million people were diagnosed in Britain with diabetes in 2013 (UK gov, 2015). In the U.S., there was an estimated 21 million people diagnosed with diabetes in 2012, and an estimated 8.1 million undiagnosed; that is 9.3% of the population (Prevention, 2014).

Diabetes has risen steeply in the last 10 years, and is forecast to keep increasing to 366 million suffers worldwide by 2030. Of the people suffering from Diabetes about 3 out of 4 will suffer from some stage of kidney disease, while diabetic sufferers are twice as likely as other people to develop cardiovascular disease and have a 12-25% lifetime risk of developing a foot ulcer(Leung 2007). Because of these complications, diabetic sufferers are 15 times more likely to undergo a lower extremity amputation than people without diabetes(Van Schie et al. 2004).

It cost the American healthcare system $176 billion in direct medical costs, to care for people with diabetes (Prevention, 2014), 33% of this figure is attributed to the treatment of foot ulcers. Apelqvis, et al., reported in 1994 on a study that monitored diabetic patients with ulcers in the US and estimated that treatment for an ulcers came to an average of $6,664 per treatment, but that amount increase to $44,790 when the treatment for healing included amputation (Apelqvist, et al., 1994). It is estimated that the UK National Health Service spends £10 billion a year on diabetes (Kerr, 2012). The estimated cost of ulceration and amputation alone for diabetics in England is £661,767,195, which rises to £985,600,282 when including the rest of the UK (McInnes, 2012). At Southampton university hospital, when new procedures for better diabetic foot care were put in place, they calculated their total annual financial savings. This figure was estimated to be 889,000 pounds (Kerr, 2012).

It can be said, that if proper care is taken with diabetes from the beginning, with regular checks to the feet, both financial expenditure and pain could be reduced. If clinicians can treat an ulcer effectively and early, then the likelihood of amputation is reduced, saving the patient harm but also saving the healthcare system millions of pounds.

2.2 Complications from Diabetes

Without constant monitoring, this life-long disease results in high blood sugar levels, which can be damaging, resulting in failure of organs and tissues. Other complications can result in cardiovascular diseases, damage to the retina, peripheral nerve damage and foot complications.

The high blood sugar levels can result in a number of different cardiovascular problems, including an increased risk of angina, myocardial infarctions and stroke. Diseases of the heart and circulation are common and can lead to hardening and narrowing of the arteries supplying the legs, known as peripheral vascular disease. In the UK, cardiovascular disease accounts for about 50% of the fatalities in people with diabetes (Diabetes UK, 2015).

The high blood sugar levels can also damage the blood vessels surrounding the eye, which can cause cataracts to develop and glaucoma, resulting in impaired vision.

Ischemia, neuropathy and infection are the three pathological components that lead to diabetic foot complications.

The first component, peripheral vascular disease is a major cause of amputation worldwide; risk factors include smoking and obesity, which develop atherosclerosis. Atherosclerosis is a gradual process of fatty material building within arteries. This fatty material mixes with calcium and scar tissue to form hard plaques, narrowing and blocking the arterial vessels. Diabetic patients

frequently exhibit high blood pressure and high fats within the blood, in addition, they are thought to have metabolic abnormalities, which together accelerate the problem (Schaper et al. 2000).

Diabetic induced peripheral arterial disease is due to hypoglycaemia, an elevation in blood glucose levels, which will damage the blood vessels and is a restriction in blood supply to the tissue. As hypoglycaemia results in the decrease of endothelium derived nitric oxide (NO). Endothelial cells line the blood vessels and therefore regulate vascular function and structure, NO is normally synthesized by these cells in order to initiate vasodilation. In addition, NO protects these blood vessels from atherosclerosis, therefore since diabetics possess decreased NO, blood flow cannot be successfully regulated, or vessels protected from endogenous injury (Creager et al. 2003). The basement membrane of arteriolar and capillary vessels can thicken, hampering normal hyperaemic or vasodilatory response (Kalish & Hamdan 2010). The dermal vascular permeability also increases with diabetes, resulting in pericapillary albumin deposition, furthering the diminished diffusion of oxygen and nutrients to the tissue. With the restriction in blood and nutrients to tissue, extremities are vulnerable to injury. Additionally, once injured, diabetics show a decrease in wound healing (Broughton et al. 2006).

It was once thought that ischemia in diabetes was only a microcirculatory dysfunction (Lavery & Armstrong 2012). This meant that amputation was frequently recommended, as surgical or other procedures would not be able to re-vascularise. However, it is now known that patients typically possess both peroneal and tibial arterial occlusive disease (Kalish & Hamdan 2010).

Diabetes increases the incidence of limb ischaemia by about 400% (Lüscher et al. 2003). Ischemia in diabetes can frequently develop early in life with dysfunction-non-occlusive impairment of arterioles and capillaries (Ahmad 2015). Capillary capacity can be limited because of increased micro-arterial pressure and flow that leads to endothelial injury. Once the large arteries, which deliver blood to the foot (the posterior/anterior tibial artery and the

peroneal artery) are compromised, this environment places the foot at the risk of ulceration. After ulceration if there is not an adequate blood supply the ulcer will take longer to heal, placing it at risk of infection or increasing depth (Ahmad 2015).

The second component, peripheral neuropathy is a result of interacting metabolic abnormalities, demonstrated by Diabetes Control and Complications Trial, (1993). This verifies that hyperglycaemia and insulin deficiency are significant contributors. Persistent hyperglycaemia is thought of as the metabolic theory in which it increases activity of the polyol pathway. This pathway accumulates excess sorbitol in the nerves because of the presence of hyperglycaemia, while sorbitol and the accompanying fructose damage the nerves. Accumulation of sorbitol consequently produces a decrease in myo-inositol and taurine, which results in a reduced nerve conduction velocity. This metabolic theory of peripheral neuropathy is accompanied by a vascular theory, which is thought to exacerbate the problem. Ischemic injury to the nerves is caused by occlusion of the supplying nutrient vessels, from thickening of the vessel walls (Kalish & Hamdan 2010; Vinik 1999; Greene et al. 1999).

Neuropathy is the most common complication among diabetic patients. It is thought to affect up to 60% of patients, with the possibility increasing with the age of the patient and the duration of the disease. It can and will usually affect all three types of nerves; motor, sensory and autonomic, thus initiating a multitude of problems.

Sensory neuropathy leads to minor injuries, which will then lead into greater complications. The earliest appearances are thought to be predominantly the small fibres, which affect the sense of pain and temperature, followed by fibre degeneration which, affects vibration and proprioception (Greene et al. 1999). Since the sensitivity of pain and temperature fibres are affected first, this leaves the patients more vulnerable to unnoticed mechanical or thermal injury. As they will not only be unable to feel pain in order to start healing the foot,

they will continue to walk on the insensitive foot, impairing subsequent healing (Apelqvist et al. 2008).

Motor neuropathy will damage the innervations of the foot, affecting the intrinsic foot muscles and the leg muscles to some extent, as it is restricted to distal extremities. This change in mechanical balance between extrinsic and intrinsic musculature leads to a knock on affect, altering the biomechanics and the anatomy of the foot. These deformities result in abnormal loading of the foot which will lead to high stresses, calluses and ulcers (Greene et al. 1999; Malhotra et al. 2012).

Autonomic neuropathy is the most overlooked of the neuropathies, but can be equally as damaging. It can create an opening for bacteria or add to the injury of the foot, because demyelination of the autonomic nerves instigates breakdown and fissures of the skin. This is due to the loss of sweat and oil gland function of the skin, causing it to become dry and cracked (Greene et al. 1999; Malhotra et al. 2012). With autonomic demyelination comes the loss of normal sympathetic innervation to the vascular supply in the foot, which is known to contribute to the development of foot ulcers (Greene et al. 1999; Jeffcoate & Harding 2003).

The diagnosis of peripheral vascular disease is conducted using a handheld Doppler device, and obtaining the arterial brachial index of the patient. Doppler device measures flow of signals in the dorsalis pedis and posterior tibial arteries, with an absent or monophasic signal indicating severe peripheral vascular disease. The ankle brachial index (ABI) is a measurement of the systolic blood pressure of these arteries and additionally the brachial arteries using the Doppler device and pneumatic cuffs. If the ABI is below 0.6 then the wound has a low probability of healing with significant ischaemia (Schaper et al. 2012).

Detected neuropathy should ideally been done as early as possible, with assessments every 6 months, using the 10-g Semmes-Weinstein

monofilament. This device attaches a nylon monofilament to a plastic handle and when applied under pressure on a patients' foot, tests 10 different dermatome points for a level of sensation. A different assessment can be conducted with a tuning fork, testing for vibratory sensation but this is less predictive of ulceration (Kalish & Hamdan 2010; Apelqvist et al. 2008).

It is the combination of neuropathy, ischemia, neuro-ischemia and trauma, which will lead to foot ulcers and abnormal biomechanical loading of the foot. Infection can be an initiating factor but is nearly always a consequence of diabetic foot complications (Lepantalo et al. 2011; Ahmad 2015). Abnormal biomechanical loading of the foot can lead to deformities within the foot, which can compound the risks for an ulcer.

2.3 Ulcers

Trauma to a diabetic's foot can be dangerous. In a person not suffering from diabetes, a wound on the plantar surface of the foot can be slower to heal that a wound anywhere else, but it will still heal because of a combination of inflammatory, proliferating and remodelling phases. However the cellular response to the same tissue injury in a diabetic's foot will fail to heal in the same timeline or even in the same manner (Rosenberg 1990).

In 2007, Brem and Tomic-Canic state how there are 'over 100 known physiologic factors that contribute to wound healing deficiencies' in patients with diabetics. These include the obvious hypoglycaemia caused by the lack of insulin, as well as a decrease in growth factor production and abnormal immune functions.

For any wound on a diabetic, the disease will negatively affect the majority of cellular processes. Wound tensile strength is decreased as a result of a prolonged inflammatory phase and thereby extrinsic factors such as excessive

or repetitive pressure can prolong the healing stage or exacerbate the wound. Macrophage function is decreased, increasing the likelihood of callus formation (Acosta et al. 2008).

Once a callus has formed over a diabetic wound, there is a high chance of a neuropathic ulcer forming underneath. As the soft tissue underneath the callus will have a higher pressure exerted upon it, without removal of the callus, inflammatory autolysis and haematomas may develop. This is a destruction of the tissue and a swelling of clotted blood, which results in tissue necrosis and the formation of a cavity. If this callus is then removed, an ulcer will be revealed. Not all ulcers are predated by a callus, but they are highly predictive of neuropathic ulcers (Edmonds & Foster 2006).

There are many types of ulcers, such that clinicians have attempted to classify them in many different ways, in order to have a universal treatment of diabetic ulcers. A system by the International Working Group on the Diabetic Foot was developed, as illustrated in table 2-1, in order to classify the risk of developing an ulcer if diabetic.

The International Working Group's Diabetic Foot Risk Classification System	
Risk Group 0	No neuropathy No peripheral arterial No foot deformity or limited joint mobility
Risk Group 1	Peripheral neuropathy No peripheral arterial No foot deformity or limited joint mobility
Risk Group 2	Peripheral neuropathy and foot deformity or limited joint mobility and/or Peripheral arterial disease
Risk Group 3	History of ulcer or amputation or Charcot

Table 2-1 The Classification of risks for a diabetics foot by the International Working Group (Lavery & Armstrong 2012)

This is used along with diagnosis systems in order to develop a standardised method of description, based on physical finding. A system by Wagner is currently, the most widely used classification system, which gives an ulcer a classification based on the depth of the ulcer (Oyibo et al. 2001). See table 2-2. Unfortunately it is only based on the depth and other factors such as infection are more likely to affect the severity of the wound and the length of time it will take to heal. Another system devised by University of Texas therefore includes the presence of infection or ischemia in the increasing grades. See table 2-3.

The Wagner Ulcer Classification System	
Grade	**Description**
Grade 0	Pre or post-ulcerative lesion
Grade 1	Partial/full skin thickness. Superficial diabetic ulcer
Grade 2	Ulcer involving underlying tissues (fascia, ligaments, tendons) involved
Grade 3	Deep ulcer with abscess or osteomyelitis
Grade 4	3 + Gangrene to portion of forefoot
Grade 5	4 + Whole foot gangrene

Table 2-2; Classification by ulcer depth and gangrene tissues. The Wagner ulcer classification system (Clayton & Elcasy 2009; Oyibo et al. 2001)

The University of Texas Wound Classification System	
Grade	**Description**
Grade 0	Pre- or postulcerative site that has healed
Grade 1	Superficial wound not involving tendon, capsule, or bone
Grade 2	Wound penetrating to tendon or capsule
Grade 3	Wound penetrating bone or joint
Within each grade there are four stages	
Stage	**Description**
Stage A	Clean wounds, no infection or ischemia
Stage B	Non-ischemic infected wounds
Stage C	Ischemic non-infected wounds
Stage D	Ischemic infected wounds

Table 2-3; Classification of wounds based on depth with additional infection and vascular status. The University of Texas Classification System (Oyibo et al. 2001; Clayton & Elcasy 2009)

For the manifestation of an ulcer to occur, trauma is necessary, diabetic trauma can come in the form of an extrinsic mechanical stress, such as a sharp or rough object breaking the skin of the foot. Alternatively, trauma can be an intrinsic mechanical stress, either of repetitive pressure or a high peak pressure. The extrinsic trauma can originate from a sharp object, such as a nail or glass, or it can originate from inappropriately fitted shoes that rub the skin. With any of these extrinsic traumas, they would instantly be identified if they occurred in a person with adequate sensation. Having experienced the pain of the trauma, such a person would likely treat the wound and remove the offending object (be it shoe or glass). However, when the same trauma occurs in a diabetic with neuropathy or any loss of sensation, there is no warning pain. Consequently, the trauma or persistent pressure goes unnoticed, leading to a complete breakdown of the tissue and ulceration.

Whereas it was once thought that trauma would only occur after a very high peak pressure resulting from a foreign object piercing the skin, it was Brand, et al., in 1983 that proposed differently. Brand put forward the concept that

repetitive stresses from lower pressures were just as dangerous as high peak pressures. This stress applied to a discrete area of the plantar surface over an extended period of time would cause ‘a local inflammatory response, focal tissue ischemia, tissue destruction and ulceration’.

Foot deformity or abnormality can instigate these high peak pressures without any extrinsic trauma. A normal foot morphology is devised in such a way to best spread the pressure evenly over the plantar surface. Even so, there are still peak pressures on the balls of the feet at push off and on the heel as the foot makes contact with the ground. These areas of the plantar surface have evolved in such a way to act as shock absorbers. In the normal biomechanics of a non-pathological foot, gait has evolved to neatly distribute and disperse shearing, tensile and compressive forces.

When standing still, the ground reaction force is about equal to the person’s body weight. With a normal morphology each foot experiences roughly 50% of the bodies’ weight distributed over the entire planter surface of the foot. This results in lower peak pressures, though with a slight peak on the heel of the foot.

The normal morphology of the foot includes 26 bones, divided into the hind-foot, mid-foot and forefoot. The talus and calcaneus (subtalar) make up the hind-foot and part of the ankle, which is joined by a modified hinge joint. See Figure 2.1. This ankle joint serves to transfer the forces from the foot to the leg. The subtalar joint plays a role in allowing the foot to adapt to uneven ground by allowing three planes of motion. The navicular, cuboid and the three cuneiforms consist of the mid-foot, while the five metatarsals and nine phalangeal bones make up the forefoot. The foot can act as a shock absorber, because of the many fat pads and bursa within it. The main fat pad underneath the calcaneus bone, allows for shock absorption during heel strike while the foot is unstable. The foot can also act as a strong lever, able to propel the body forwards. This is made possible by the plantar fascia, which is a strong connective tissue running along the plantar surface. See Figure 2.2. The

plantar fascia is strong enough to hold the bones of the foot in place and stabilize the arches of the foot, allowing for the foot to act like a lever when load is applied (Martini, F.H & Nath 2009).

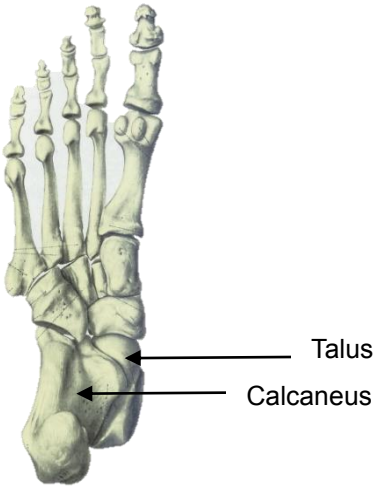


Figure 2-1; Anatomical Bones of the Foot. Hindfoot Indicated (Martini, F.H & Nath 2009)

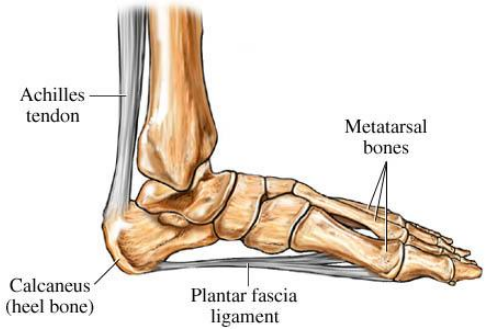


Figure 2-2; Anatomy of the Foot. Plantar Fascia Indicated (Martini, F.H & Nath 2009)

During gait, the ground force reaction varies predictably, at certain points to above body weight. Consequently for only 22% of the time are both feet in contact with the floor, which increases the weight borne on only one foot for a substantial interval. The gait cycle consist of the stance phase and the swing phase. Of these, the latter swing phase incurs no plantar pressure, as it is the movement of the lower limb to swing forward to take another step. The stance phase can be split into a debated number of parts, in this thesis three parts are discussed: the first contact, the mid-stance and the toe-off (or propulsion phase). With first contact or heel-strike, the heel is acting as a shock absorber under considerable pressure and begins to pronate allowing for adaption of the terrain. The heel stays in contact with the ground for approximately the first 64% of the stance phase. During mid-stance, the opposite foot is in toe-off and the entire weight of the body is transferred over to the new foot. This involves the heel and the forefoot together in contact with the ground for only 23% of the stance phase. This phase ends with propulsion, which begins with the heel lifting off the ground and placing the greatest amount of force directly over the

relatively small area of the forefoot and ends with the heel-strike of the opposite foot. The forefoot is therefore in contact with the ground for approximately 59% of the stance phase (van Deursen 2004; Randolph et al. 2000; Leardini et al. 2014).

The stance phase is characterised by the rocker movement of the foot, which can be once again divided into three parts. See Figure 2-3. First is the heel rocker as the foot rotates over the heel. Here, the heel acts as an axis to allow plantar flexion to occur smoothly and for the foot to make full contact with the ground. Subsequently, the ankle rocker occurs during mid-stance, which allows for dorsiflexion of the ankle as the tibia is able to propel over the foot. As the centre of pressure advances along the foot the ground force reaction progresses from the heel to the forefoot. Last of the rocker momentum is the forefoot rocker, which occurs as the foot rolls over the metatarsal heads and hallux. This forefoot rocker is centred over the first metatarsal-phalangeal joint (MTPJ) and allows the limb to progress over the forefoot and for the heel to lift off the ground. With this dorsiflexion of the first MTPJ the joint takes the full force of the body weight, which is not distributed evenly over the other MTPJs, The first MTPJ consists of the first metatarsal head and the base of the proximal phalanx (named the hallux). The rocker motion highlights the importance of the load-bearing function of both the hallux and the first metatarsal head (van Deursen 2004; Dawe & Davis 2011).

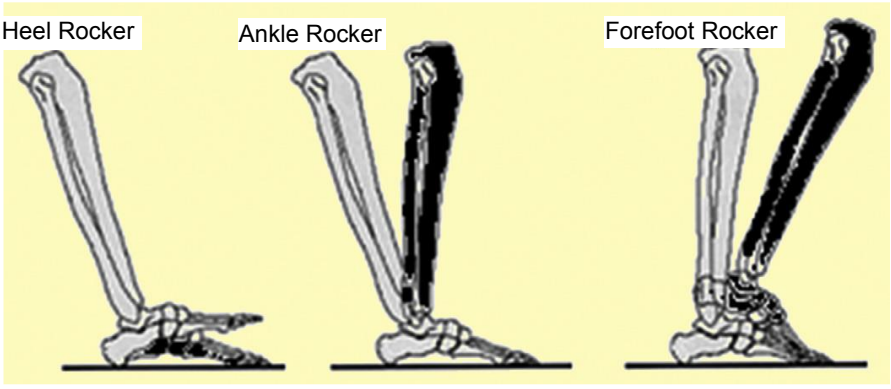


Figure 2-3; Depicting the three 'Rocker' motions during gait (Dawe & Davis 2011).

Therefore the two peaks of pressure during gait (normally graphed as the vertical force component) occur first on the heel and later on the first metatarsal head and hallux. See Figure 2-4. As well as this vertical force, there is of course shear forces occurring during gait, acting directly anteriorly at the forefoot and posteriorly on the heel. With a non-pathological foot these peak pressures are dealt with successfully, but in a diabetic patient with neuropathy, ischemia or a deformity these pressures will exceed the foot capabilities.

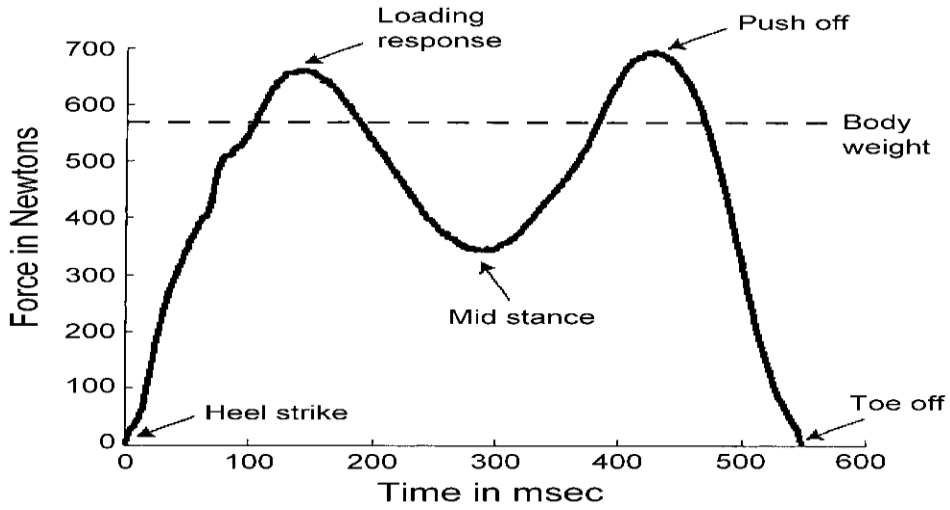


Figure 2-4; Vertical ground force reactions during gait (van Deursen 2004)

The occurrence of diabetic complications can lead to patients developing foot deformities which can lead to further foot complications such as ulcers and amputations. These deformities can come in many different forms and severities.

A diabetic patient with neuropathy or with any damage to their nerves can as a result have weak or uncoordinated muscle of the feet. This can lead to the foot deforming, resulting in claw toes, hammer toes and bunions among others. Deformities of the foot will result in badly fitted shoes and create unnatural pressures on the foot. These unnatural pressures will preclude the higher peak pressures or a repetitive stress injury that produces an ulcers.

It has been suggested by many authors that muscle weakness in the toe flexors precludes toe deformities. Though van Schie, et al., suggests that only once the plantar aponeurosis and plantar plate, as the most significant stabilizing force on the plantar aspect of the metatarsophalangeal joint, becomes ineffective that the toe flexors and extensors will have a significant effect on the position of the toes (Van Schie et al. 2004). Claw toes and hammer toes are the most common toe deformities in patients with diabetes.

Claw toes as defined by Magee (2008) is a 'hyperextension of the metatarsophalangeal joints and flexion of the proximal and distal interphalangeal joints' and resulting in the foot becoming curved and 'claw-like'. See Figure 2-5. The absence or reduction of midfoot support that develops results in the forefoot undertaking more support, leading to an increase in pressure under the metatarsal heads. Schuster (1939) demonstrated how the plantar flexion of the first ray was a factor in claw toes and Root et al (1977) confirmed how the first ray can be plantar flexed by the spasticity of the peroneus longus muscle. Badly fitted shoes, defective lumbrical and interosseous muscles and abnormally short peroneus longus muscle are also common causes for developing claw toes. Claw toe can be common in patients with diabetes, as well as a result of Charcot-Marie-Tooth disease which is an inherited muscular dystrophy disease.

Hammer toes were defined as "an extension contracture at the metatarsophalangeal joint with a flexion deformity of the proximal interphalangeal joint and hyperextension of the distal interphalangeal joint," by Magee (2008). It is very similar to claw toes, but results in only the one toe developing a deformity. It too develops following muscle neuropathy and atrophy of the small muscles responsible for metatarsophalangeal plantar flexion(Van Schie et al. 2004).

Forefoot varus or valgus deformities are another type of foot abnormality which develops from diabetic neuropathy and leads to increased stresses on the plantar foot, resulting in callus formation.

A Forefoot valgus deformity involves a mid-tarsal joint deviation which gives rise to the forefoot everting onto the hind-foot when the subtalar joint is in the neutral position. When there is no STJ compensation the medial aspect of the forefoot will bear all of the body weight before the lateral foot loads rendering the foot unstable. With STJ supinatory compensation the lateral side of the foot is brought to the ground, increased weight bearing on the fifth metatarsal head(Mueller et al. 1990; Magee 2008).

Forefoot varus deformity is similar but the mid-tarsal joint deviation brings about an inversion of the forefoot onto the hind-foot when the subtalar joint is in the neutral position. During gait the mid-tarsal joint is completely pronated in order to bring the first metatarsal head into contact with the ground, with peak pressure generated under the second and third metatarsal heads, as the first metatarsals is thought to be hyper mobile, according to Root, et al.,(Mueller et al. 1990; Magee 2008).

Charcot arthropathy is a deformity in the diabetic foot, which is one of the most misdiagnosed diseases, commonly misdiagnosed as osteomyelitis or tendinitis (Ahmad 2015). See Figure 2-5. It is a progressive degenerating disease, which if left untreated can cause a complete collapse or destruction of the foot and ankle joints. There are two theories, neurotraumatic and neurotrophic, which try to clarify the pathogenesis, though it is now thought that it is the two combined which will result in a Charcot foot (Kaynak et al. 2013). But it is the neuropathic insensitive foot, which is the root cause, enhancing a local inflammatory response triggered by trauma. The local inflammatory response which in the general population would diminish once the foot was immobilize and allowed to heal, would be possible because the patient felt the pain of a trauma. In a diabetic with peripheral neuropathy they would not feel the pain of trauma and the foot would not be immobilized in time allowing the inflammatory response to flourish. This is compounded by a compromised Charcot patients' immune system, allowing for an abnormally intense and prolonged response. This response will induce osteoclast

production which are responsible for reabsorbing bone. The neurotrophic theory suggests that it is the failure of the autonomous nerve function, because of diabetic neuropathy which is responsible for Charcot foot. It leads to the breakdown of smooth muscle tone on the arterial wall which then results in a failure to vasoregulate and ends up increasing blood flow to the bone. With increased blood flow, there are more osteoclasts which will accelerate bone reabsorption. As the bone grows weaker, minor injuries will turn into larger fractures and joint deterioration because of the instability of the foot architecture (Kaynak et al. 2013; Clayton & Elcasy 2009). The neurotraumatic theory suggest that joint deterioration results from repetitive trauma which once again because of the diabetic sensory neuropathy goes unnoticed. This together with abnormal loading of the joints because of an inability to judge the foot pressure capacity leads to Charcot foot abnormality. If Charcot foot is left untreated a complete collapse of the joints affected can be anticipated. If the midfoot is affected, with fractures to the tarsal joints, a complete collapse of the arches causes the plantar region to become convex, with bony prominences at the midfoot. The result is a flatfoot called a rocker-bottom foot and high pressure can be expected followed by ulceration in the midfoot region (Mueller et al. 1990).

The result of all these abnormalities is a significant disruption in the architecture of the foot, which can lead to excessive peak plantar pressure which will then produce a chronic ulcer on the plantar surface of the foot.

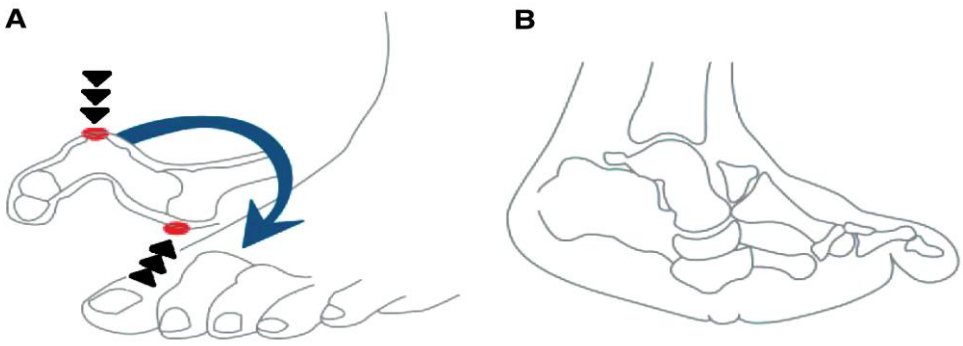


Figure 2-5; Depiction of (A) Claw toe and (B) Charcot Foot (Clayton & Elcasy 2009)

2.4 Correcting Complications

Prevention

It is important to keep a diabetic in good health, regular check-up are necessary, as this will save the patient much distress and harm in terms of complications, as well as money in the long run. Prevention of foot complications can be achieved by educating patients, with good foot hygiene, proper protection of pressure points, with well-fitted shoes (Leung 2007). But above all patients need to have routine surveillance and improved blood-glucose control as this reduced microvascular complications and therefore reducing the probability of ischaemia in the foot and other problems that quickly lead to ulcers (Jeffcoate & Harding 2003). Comprehensive foot examinations should be conducted annually, with assessments into peripheral neuropathy status and any problems should be reported to a doctor especially any changes in the architecture of the foot, pain or loss of sensation (Ahmad 2015). Even with good care and regular check-ups complications are possible, in that case quick assessment is required in order to prevent an exponential decrease in quality of life or eventual amputations. If potential ulceration risk factors are detected early, wound development can be reduced and the ease of treatment increased (Clayton & Elcasy 2009).

When treating a diabetic foot ulcer, first be aware of infection, which is very likely and can come in many different forms. Usually divided into one of three categories, either superficial and local, soft tissue and cellulitis, or osteomyelitis (Jeffcoate & Harding 2003). Once any infection is under control, the vasculature of the patient should be assessed, if ischaemia is present, revascularisation should be considered in order to restore skin perfusion. A proper supply of blood is very important in order to allow the ulcer to heal. Force on the ulcer should then be keep to a minimum, relieving pressure on an ulcer will allowing healing to begin without exacerbating the wound. The condition the ulcer is kept in should be carefully monitored and prepared, with debridement, removal of any callus and topical medicine applied.

If infection is cleared up on a large ulcer and the wound has a growth of granulation, a skin graft could be considered. Likewise surgery can be considered, though usually only for the younger patients as they are less likely to have neuropathy and more likely to have better blood circulation, suited for recovering after surgery (Leung 2007).

If the patient has osteomyelitis it needs to be caught and assessed quickly, as it can very likely lead to amputation, four times more likely than a normal soft tissue infection. Once assessed, antibiotics and surgery can now successfully treat osteomyelitis, as long as the foot is properly stabilised and cared for after (Malhotra et al. 2014).

More attention should be focused on controlling oedema, which can be detrimental in the healing of ulcers, as the presence oedema was more common in patients who required amputation than not (Ho et al. 2013). A foot-compression device, like an intermittent pneumatic compression device has been shown to facilitate oedema reduction. Currently there is no reliable evidence that supports the use of hyperbaric oxygen in reducing oedema (Ho et al. 2013; Jeffcoate & Harding 2003).

Debridement is an important first step in the healing of any ulcer, specifically if necrotic or unhealthy tissue is present. It is the removal of foreign material and particulate matter from the wound, including the removal of surrounding callus. This provides an ideal healing environment, with removal of bacteria from the wound and additionally decreases the pressure points on the ulcer (Wu et al. 2007).

As oxygen-rich environments has been demonstrated to provide higher replication rates of fibroblasts, endothelial cells and keratinocytes, all which will increase the rate of healing in a diabetic ulcer, there have been several attempts to provide oxygen as a treatment. A polyethylene bag can create a chamber over an ulcer, with ten litres per minute of oxygen passed through, this kind of diffuse of oxygen is called a topical oxygen treatment, and has not

of yet been verified (Leung 2007). Hyperbaric oxygen is more conventional but as doubts on the true value, there has been recent multiple small studies into its use with ulcers. Over 30-40 sessions, lastly 1-2h, a patient breaths 100% oxygen intermittently within a chamber while atmospheric pressure is increased to 2-3 atmospheres. As of yet there is insufficient data to validate whether it is cost-effective but there have been significant improvement in amputation rates when hyperbaric oxygen treatment was used (Alexiadou & Doupis 2012; Kalish & Hamdan 2010).

Negative pressure wound therapy is another treatment to improve the wound bed of an ulcer, and therefore improve active healing, by the removal of wound fluid. Negative pressure wound therapy works by distributing local negative pressure evenly across an ulcer, usually by an electoral pump and an airtight film wound dressing. Not only is this meant to improve the wound bed but there have been studies suggesting that the flow of blood can be optimized along with swelling in local tissue reduced and the removal of harmful bacteria. While there have been promising studies completed, there is still only a cautious agreement on the success of negative pressure wound therapy and as such it will not replace methods such as debridement or off-loading the foot (Lavery & Armstrong 2012; Vikatmaa et al. 2008; Wu & Armstrong 2005).

There is also a possible future in growth factors and stem cell therapy healing diabetic ulcers. There have been studies into the used of basic fibroblast growth factor, epidermal growth factor and granulocyte colony stimulation factor, with each of these studies producing varying results and limited evidence of success so far (Alexiadou & Doupis 2012).

If there has been prolonged disuse of the ankle, or a deformity in the foot, the Achilles tendon can becomes very taught causing plantar flexion. This instigates increased pressure under the metatarsal head as it reduces the ability of the heel to maintain contact with the ground. Surgical intervention is then required in order to lengthen the Achilles tendon to allow the heel to

resume its normal position and release plantar pressure on the metatarsal heads (Lewis & Lipp 2013; Leung 2007).

Offloading

When treating a diabetic ulcer, one of the most important steps in rehabilitation is to reduce the pressure put on the ulcer, allowing it time to heal without mechanical stress. Pressure reduction can be achieved by distributing the weight over a wider area, allowing the pressure to be shared, best achieved by maintaining contact with the entire plantar surface of the foot and the lower leg.

A surgeon named Dr Paul W. Brand first made the link between ulcers, insensitive feet and offloading. While he was working with leprosy patients in southern India, he noticed that patients with plantar ulcers walked without a limp, unable to feel the injury. With this information he connected the healing of a plantar neuropathic ulcer with the offloading of the plantar surface, using a total contact cast (Boulton 2004; Boulton 2012; Hunt n.d.).

Where treating a patient with foot ulcers the best prescription for offloading could be assumed to be bed-rest or a wheelchair as they would totally remove the pressure from the plantar surface of the foot. However, this is not the most practical treatment for patients wanting to keep a similar quality of life or for overweight patients, which is a high percentage of diabetics with foot complications or for patients with other debilitation factors.

There are many ways in which to offload a diabetic foot, some more effective than others, though others are more widely used. Offloading a foot can be achieved by: a total contact cast, a removable walking cast, cast shoe, and different types of footwear such as forefoot offloading shoes, therapeutic footwear, felted-foam dressings and specialized insoles.

Even though Brand started using these total contact cast (TCC) in the 1960s it wasn’t until the 1980s when Boulton et al., and Sinacore et al., conducted

studies into the effectiveness that his observations was corroborated. Once Mueller, et al., (1989) published their findings, total contact cast was seen as the gold standard for off-loading the plantar surface of the foot, by the American Diabetic Association. Mueller, et al., observed in a randomized controlled trial (RCT) how plantar ulcers treated with TCC healed at a significant greater rate than those treated with traditional therapeutic shoes, 'with an absolute risk reduction of 59%'.TCC are below-knee casts which are applied over minimum padding. These casts are anatomically conforming, ensuring a careful fit following the natural contours of the foot and lower leg, usually made of Plaster of Paris or fiberglass (Mrdjenovich 2010; Lewis & Lipp 2013). They require a well-trained experienced clinician to cast, with regular monitoring and weekly visits as usually require replacing every 5 to 7 days. They are very effective in distributing plantar pressure along the cast evenly with one study able to directly measure the load on the cast wall, which receives 23-34% of the lower limb load (Begg et al. 2012). There are many studies in the last 30 years into the effectiveness of the TCC, demonstrating how TCC heal a higher percentage of plantar ulcers at a faster rate and with higher healing proportion than therapeutic footwear. Between Sinacore et al. 1987, Walker et al. 1987 and Birke et al.2002 they have established that 73% and 100% of plantar wounds are reported to heal with the use of TCC (Bus et al. 2008). TCC are thought to be able to eliminate or reduce shear stress as there should be no movement within the cast, but currently there is no way to measure the shear to confirm this (Lavery et al. 1997; Begg et al. 2012).

They are also thought to be effective because they are able to hold the ankle at 90°, limiting motion and therefore correction any gait deformities as well as shortening stride length and velocity which limit peak pressure on the plantar foot (Cavanagh & Bus 2010; Begg et al. 2012). However TCC are shown to be less effective in offloading the plantar heel and are contraindicated for patients with deep infections, abscess, active osteomyelitis and gangrene. As well as should be avoided for use in patients with insufficient venous or arterial supply (Mrdjenovich 2010). As these TCC are non-removable, patients may have

trouble sleeping or with daily hygiene, they have also been shown to reduce activity levels in patients, which may be why they have proven to be as effective as they are compelling the patients to manually offload.

The Charcot restraint orthotics walker is an option meant to provide a bridge between treatment of diabetic Charcot foot and the use of a total contact cast. Though not widely used they can provide for a 50% reduction in plantar forefoot and midfoot pressure and have had success in patients with fluctuating edema (Mrdjenovich 2010).

Another option for offloading a diabetic foot is the removable cast walker, this cast is not custom made and therefore does not need a qualified clinician for application. The advantage of this device is the ability to remove it, allowing for monitoring of infected or non-infected wounds, this also allows a better standard of living for the patient, rendering sleep and good hygiene easier. It still keeps the ankle at a 90° angle and can contain accommodative padding with extra custom made padding at the insole, therefor works to the same pressure reliving concepts that the TCC does. Studies report the removable cast walker is as effective as the TCC but doesn't produce the same results, as patient compliance is difficult to enforce. Armstrong, et al., 2003 report that patients wear the removable boot for only 29% of total steps taken in any 24 hour period. The RCW could become as efficient with healing as the TCC by a small adjustment of a fiberglass wrap or bandage around the outside of the cast, which now renders the cast irremovable. A study by Armstrong, et al., 2005 demonstrated how once a RCW became irremovable, ulcers healed at a significant increased proportions compared with a standard RCW. With a study by Katz, et al., 2005 establishing how irremovable RCW have comparable healing rates of a TCC and another by Martin, et al., 2005 resulting in no significant difference in peak pressures between a RWC and a TCC. The multitude of studies comparing the two cast all come to similar conclusions, the RWC compares with the TCC but has the additional advantage of easy removal for regular monitoring, avoiding the need for a qualified casting

technician and is far more cost efficient. Katz, et al., 2005 asses the cost of an irremovable RCW at 24.8% less than a TCC.

2.5 Static and Dynamic Measurement Systems

In order to offload the foot in patients with arthritis or injury, pain and discomfort are taken as measurements, in order to design or correct the pressure distribution. In patients with diabetics this distinction is unusable because of the patient's neuropathy, which is partly responsible for the ulcer in the first place. Therefore in order to be able to correctly fit an offloading device, a pressure-mapping system is needed. There are many different ways in which to pressure map a person's gait, one of the more useful ways is to use an in-shoe system. Other potential systems are a pressure distribution platform or expensive imaging technologies. Pressure distribution platforms are embedded within the floor of a laboratory and consist of a matrix configuration of an array of pressure sensing elements. They can be used for both static and dynamic experiments and are easy to use. Though this pressure system does require a certain familiarity of use before a natural gait can be recorded, as it is important for the foot to land on the appropriate area. While pressure platforms reflect the interface between the subject and the platform, an in-shoe pressure system reflects the pressure distribution between the foot and the shoe, far better for off-loading experiments. There is the possibility of the sensor slipping as well as the lower spatial resolution compared to the pressure platforms due to fewer sensors, but the in-shoe pressure system is flexible and highly portable allowing for a wider variety of experiments.

The key specifications for sensor performance include: linearity, hysteresis, size of sensors, pressure range, temperature sensitivity as well as the operating frequency, creep and repeatability. It is important for pressure sensors used in gait experiments to have low hysteresis, a linearity of output and a recommended pressure range of approximately 1 GPa for walking with

a higher pressure range for other activities (Abdul Razak et al. 2012). The resolution is important for plantar foot measurements because of the variations in the size of anatomical structures such as the metatarsal heads between patients. There should be a high number of small sensors in order to achieve a more accurate pressure reading (Orlin & McPoil 2000).

There are different types of sensor technologies. The most common are capacitive sensors, resistive sensors and piezoelectric sensors. In this study the sensor technology uses for an in-shoe measurements for a force sensing resistor (FSR) from Tekscan™.

A force sensing resistor works in one of two ways, either it is made from two Mylar sheets, one with metal tracks on the surface, the other with a conductive polymer permeating the surface. When force is applied and this thin sandwich is pressed together, the conductive particles form a resistive path between the metal; the higher the force the more pressure and the lower the resistance. The other approach is to have the two metal pattern on both Mylar sheets and the conductive polymer infused between the two. This time when pressure is applied the flow of electrons has less resistance thanks to the conductive layer, allowing pressure to decrease resistance (Abdul Razak et al. 2012; Cavanagh et al. 1992).

Capacitance transducers can be used as another type of sensor system. This electrical device consists of two conductive plates that store charge and are separated by a dielectric layer, which is non-conductive. When force is applied the distance between the two electrical plate’s decreases and the dielectric layer is compressed, allowing for the capacitance to increase and the resulting change in voltage is measured (Cavanagh et al. 1992).

Piezoelectric sensors can be found from natural materials or manufactured, such as quartz or PZT. When a piezoelectric material is deformed from pressure, there is a generation of charge, which can be collected on electrodes. The pressure can be measured as proportional to the voltage

which is converted from these electrodes. While no external voltage is necessary for these sensors, for each individual transducer a charge amplifier is required (Abdul Razak et al. 2012).

Historically capacitance transducers are more widely used with in-shoe sensors, as are thought to be more reliable. Since early versions of force sensing resistors worked more like a switch and therefore offered poor resolution. The chance is sensitivity during use and the difference in sensitivity between transducers incurs more problems for clinical investigations remaining accurate. While some investigators may prefer the FSR over the capacitance sensors as they then to produce a thicker insole which may compromise some studies (Orlin & McPoil 2000).

3 Methods and Materials

The aim of this pilot study is to evaluate the differences and similarities between the Aircast™ and the Airstep™ Walker Systems. For this study, a convenient sample size of two (n=2) participants was used. This pilot work will be used in order to calculate which sample size would be required in future clinical studies. The two volunteers were healthy, without any deviations from normal gait. This study explored the mechanisms contributing to off-loading abilities of the selected Aircast™ and Airstep™ walker systems, additionally examining the differences in rocker sole design utilising kinetic and kinematic motion analysis. Therefore, it was appropriate to use an in-shoe pressure sensor for one test and a force platform for the other.

Figure 3-1 Airstep™

Figure 3-2 Aircast™

The Airstep™ Walker™ Promedics Orthopaedics ltd is a newly designed cast brought to the department for investigation. See Figure3-1 The Pneumatic Walker™ from Aircast™, an older cast already on the market, was chosen as a comparative walker. See Figure3-2. This was because this walker has been studied before and compared to other devices, the department also had ready access to the walker. Studies show that this removable cast can match a total contact cast in offloading capabilities, healing ulcers in approximately 50 days

(Armstrong et al. 2005). Both casts are removable and readily usable with different patients, they both use the concept of total contact in addition to air pressurized bladders in order to distribute pressure and offload the plantar surface of the foot.

3.1 F-Scan In-Shoe Pressure Sensor

Static and dynamic tests were performed to provide insight into the offloading effectiveness of each cast. For the first trial, the sensor system F-scan was used. This is a 0.7mm thick sensor composed of pressure-sensitive, resistive, and conductive silver-based inks, arranged in 60 columns and 21 rows, embedded in Mylar coating that can be cut to fit into the subjects shoes. See Figure 3-3.

As the subject walks with the pressure sensor within their shoe/cast, the changes in resistance on the sensor reflects the pressure differences on the cells. This information is then transmitted via the embedded wires attached to the top of the sensor, which in turn is connected to the preamplifier. The preamplifier is attached to the subjects' ankle by a blue Velcro strap, where the data undergo analogue to digital conversion, which is then transmitted to the computer for analysis.

The F-scan system is advantageous to use because of the mobility of the device, it eliminates the problem of targeting footprints, allowing for a normal gait response. Furthermore, when analysing it allows for easy targeting of specific anatomical areas (Mueller & Strube 1996). While it may have a reduced resolution compared to a pressure platform, because of the number of sensor that can be incorporated, it is still validated to provide an accurate picture of the pressure within a cast device (Orlin & McPoil 2000).

Reported disadvantages with the F-scan in-shoe system are the possibility of movement within the shoe and the sensor becoming creased (Randolph et al. 2000). The poor durability of the sensor may affect data, but that was not a problem with the number of test undertaken in this thesis. There is the possibility of large differences between sensors used, as discovered by Woodburn & Helliwell (1996), therefore each subject used their one sensor for all the tests undertake. With the new Tekscan™ system, temperature does not influence the data collection, so no wait time is needed between tests. But the time taken to switch between shoes and different cast should be sufficient for the sensor to rest between tests, if such a period is really necessary.

Figure 3-3; Tekscan™, F-scan in-shoe pressure sensor

3.2 Method for In-Shoe Sensor Test

The first trials were conducted with the inserted F-scan sensor into each cast, and the subject walking on a treadmill. Calibration was performed by having the subject stand for the required period of time (10 seconds), with their barefoot fully on the sensor, which was laid flat on the floor. The subject then walked at their optimal speed on a standard treadmill. The optimal speed was 0.8 m/s for subject B and 0.49 m/s for subject A.

Data was recorded from the subjects during a 10 second period, which consisted of approximately 5 full steps. The data from the first and the last

stance was not included. Three attempts were recorded for each experimental condition, and the data averaged to produce a final value. Control data was collected by having the subjects walk for 10 seconds on the treadmill at their self-chosen speed, in their normal shoes. These shoes had a very slight heel and raised sole, but were generally flat, comfortable shoes. The subject performed this three times for accuracy. The subjects then repeated this trial, first wearing the old style orthosis without the air bladders inflated, then with the air bladders inflated at 30 mm/Hg, at 40 mm/Hg and finally at an uncomfortably high air pressure. This uncomfortably high air pressure measurement was taken as off the pressure gauge measurement chart. The trail was repeated for the Airstep™ without, then with, the air bladders inflated. The Airstep™, unlike the Aircast™, does not provide a pump with a barometer in order to calculate the pressure of air within the bladders. The pressure was also recorded with the subject standing still, with the sensor in each shoe/cast for 10 seconds.

3.3 Force Plate and Rocker Bottom

It is useful to add a contoured ‘rocker’ to the underside of an offloading cast, in order to mimic the normal anatomical gait of rocker phases. The weight of the body causes the momentum of the foot to rock over the fulcrum of the cast. The forefoot rocker is capable of reliving pressure under the metatarsal heads by distributing the force over a larger area, and reducing anterior displacement of the metatarsal head soft tissue. The placement of the apex of the rocker, or the angle of the rocker, may contribute to the offloading effectiveness of the cast.

In order to understand the differences between the two cast rocker bottoms, test were conducted walking over a force plate. A force plate is generally considered the gold standard device in gait analysis and force measurements. The Kistler™ force plate was used, which utilizes piezoelectric technology and is sunk into the floor in the centre of the gait lab used. This allowed as least 10m of space to walk. The floor platform itself is composed of four plates

constructed into a larger square. Each plate is made up of a top plate, a middle plate and a bottom plate, with quartz transducers in the four corners. See Figure 3-4.

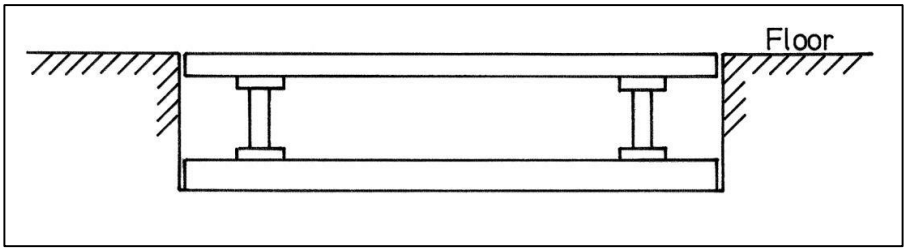


Figure 3-4; Cross-sectional diagram depicting the force plate sunk into the gait lab floor

The extent of the lab, allows subjects time to feel comfortable in their own gait, ensuring the recorded measurements were during free movement. The space allows the subject time to accelerate and decelerate at the beginning and end of each walk without these inconsistencies occurring over the force plate. The accuracy of the force plate is assessed each day by way of a static measurement; the subject is asked to stand on one leg over the force plate. This equates to the body weight of the subject, previously established, therefore confirming the accuracy of the system. This ensures that the subsequent gait data will also be accurate. The data collected from the force plate can be calculated into the vectors of the force, demonstrating how the force will start with a backwards motion and continually tilt as the foot moves forward through a gait cycle. The centre of pressure can be calculated from the force plate data. This is the mean of all the pressure applied to the foot which, in a normal gait pattern, will move from the lateral side of the heel to the toe on the medial side of the foot.

3.4 Method for Force Plate Test

The subject walks the length of the lab three times with their normal shoes on, three times with the Aircast™, and three times with the Airstep™. If there is not a clean footprint recorded on any single force plate, the subject is asked to walk again. Once the necessary data is recorded, it can be extracted from the system for analyses.

3.5 Analysis

For the trials using the F-scan insoles, Tekscan™ software was used to interpret the results. This software recorded roughly 6 steps, or 'footprints' in each 10s test. In each test these footprints are all approximately the same. Therefore without access to more sophisticated software, one footprint was chosen to study at random, based on their visual similarity. Within each footprint, two different regions were selected, using the Tekscan™ software to identify an area under the metatarsal heads and an area under the heel. These regions were selected as the areas of the planter foot most at risk of ulceration, therefore the areas requiring the greatest reduction in plantar pressure. Contact pressure and the peak contact pressure were both chosen to analyse. Contact pressure is an average pressure value, providing an understanding of the typical pressures acting on each chosen anatomical region during the gait cycle. The peak pressure results represent the highest pressure value recorded by each sensor over the footprint. Peak pressures are therefore selected to determine the effectiveness of each walker at offloading the metatarsal heads and heel.

For the trials using the force plate, data was extracted from the computer and then imported into a spreadsheet before Pedotti diagrams could be constructed and analysed.

4 Results

The following graph in Figure 4-1 presents an example of a subjects 10 second walk on the treadmill, repeated three times. The figure is generated by the Tekscan™ software, in which it produces 50 frames per second. Within this 10 second period (or 500 frames), the subject takes, on average, 6 steps with the right foot (the foot with the cast on). Each colour represent a repeated 10 second walking test.

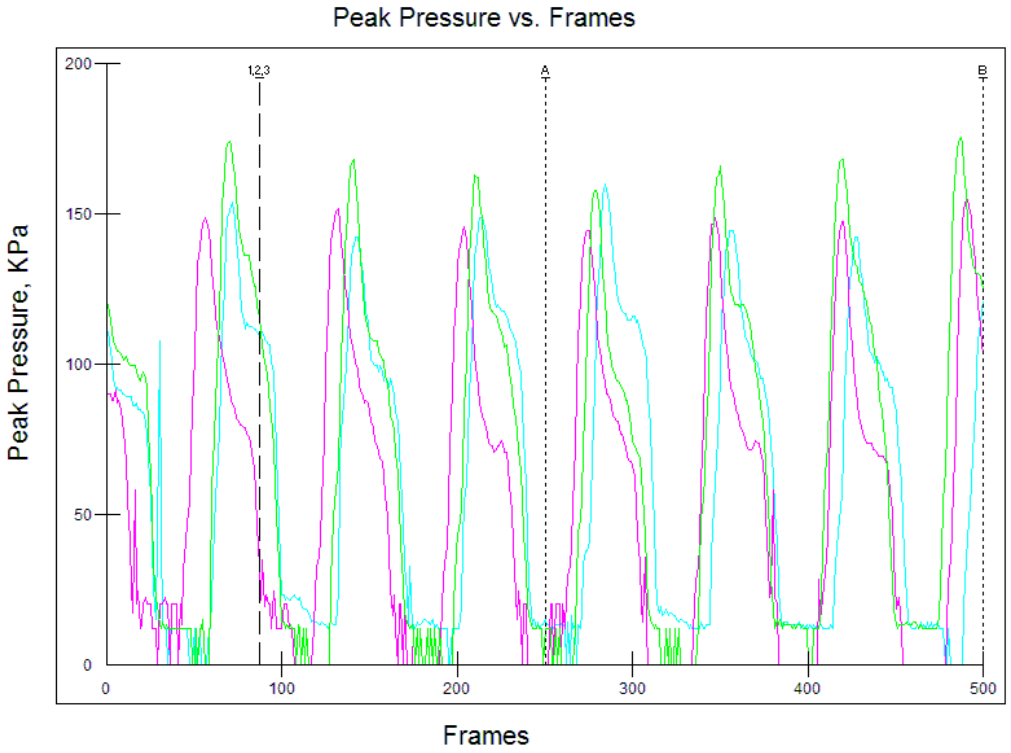


Figure 4-1; An example first output from 10 second treadmill test

Visual inspection and selection of a single step, which was representative of the complete set of 6 steps, was then analysed. The 3 selected steps, one from each test, are displayed in an example graph in Figure 4-2. This figure represents the peak pressures on the whole plantar surface of the foot the number of frames it takes for one step.

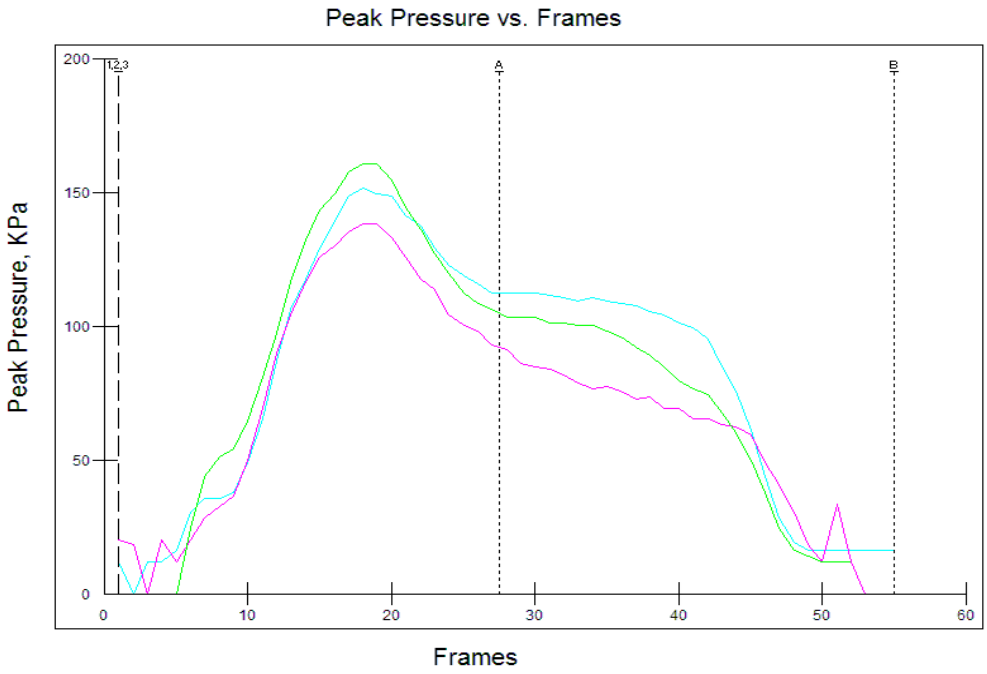


Figure 4-2; The subsequent manual selection of single step from 10second treadmill test

Following selection of a single step, the Tekscan™ software is used to select the region under the metatarsal heads and the region under the heel. These two regions have been selected as the most vulnerable to developing an ulcer.

For the following 28 pages, each graph in the upper left corner represents the pressures under the metatarsal head across one step. The three repeated tests are displayed in red, blue and green. Each graph in the lower left corner represents the pressures under the heel across one step, the three repeated tests are displayed again in red, blue and green. Each of these graphs correspond to an example footprint on the right of the page, which is taken at random from one of the three test. This sensor footprint is displayed within the generic white footprint of the software and displays two boxed areas, representing the metatarsal heads and the heel. The highest pressures from across the step are presented with a colour range to illuminate the most extreme pressures.

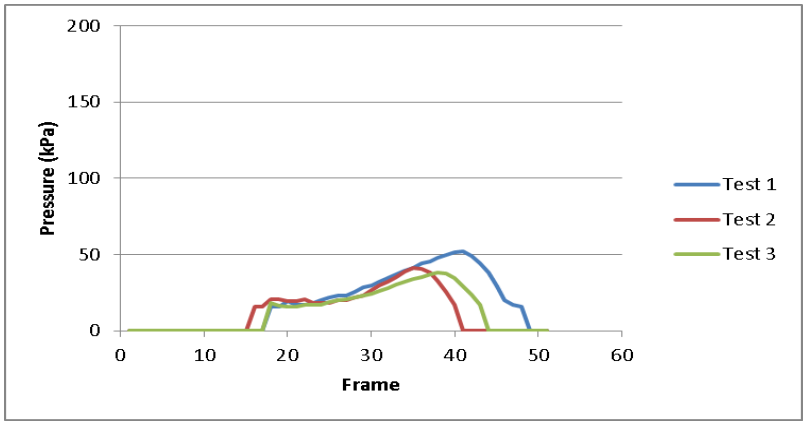


Figure 4-3; Subject A wearing their Normal Shoe. Contact Pressure under the Metatarsal Heads

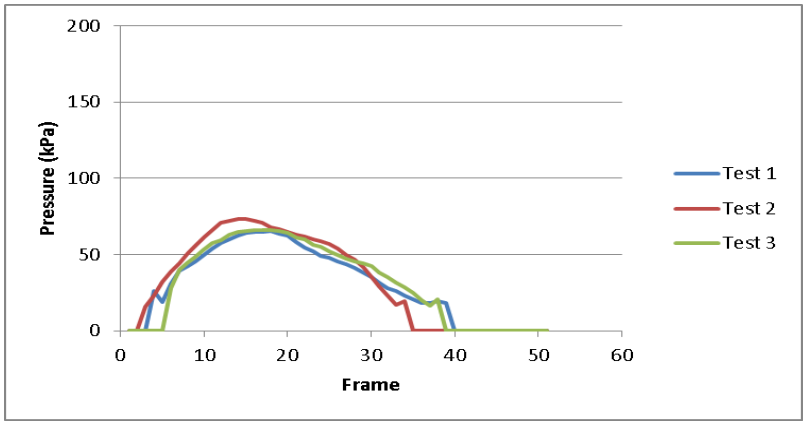


Figure 4-4; Subject A wearing their Normal Shoe. Contact Pressure under the Heel

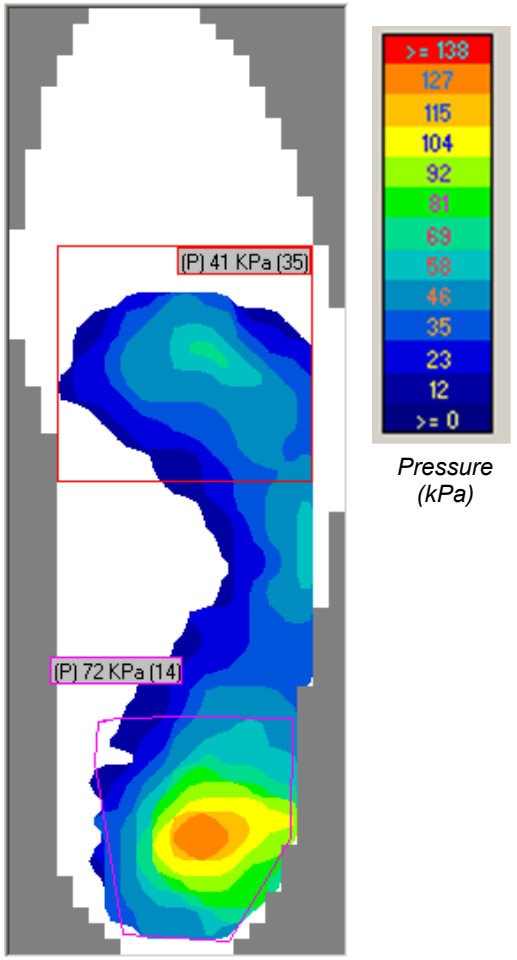


Figure 4-5; Subject A Footprint of Contact Pressure Distribution across the Plantar Surface, wearing their Normal Shoe

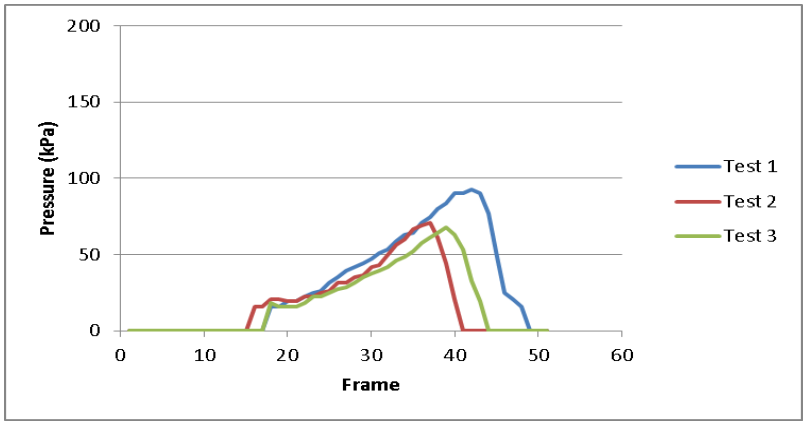


Figure 4-6; Subject A wearing their Normal Shoe. Peak Contact Pressure under the Metatarsal Heads

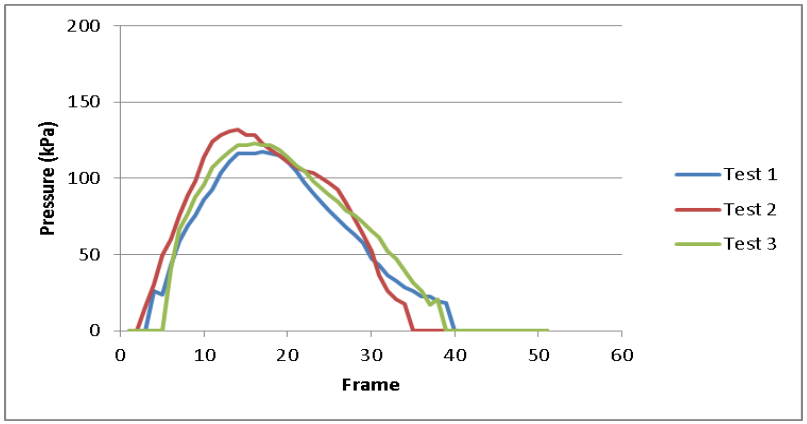
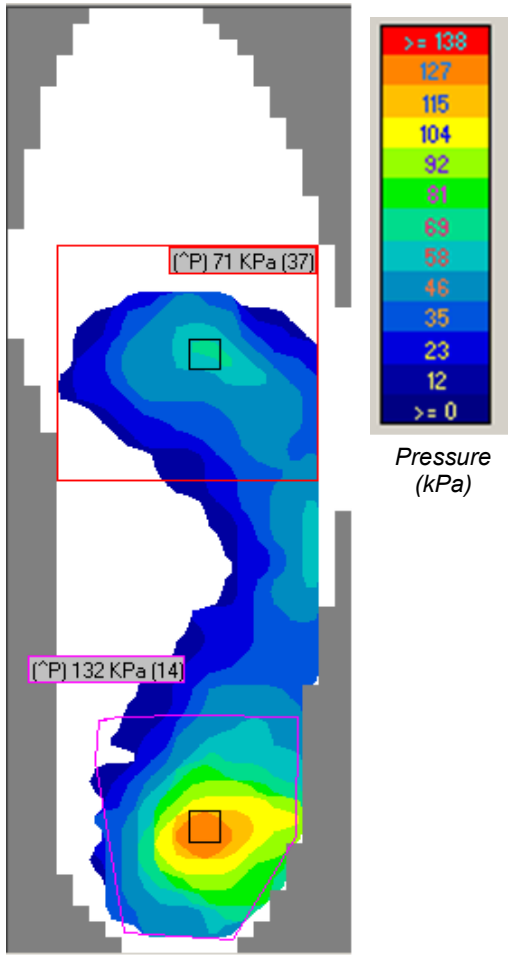


Figure 4-7; Subject A wearing their Normal Shoe. Peak Contact Pressure under the Heel



Pressure (kPa)

Figure 4-8; Subject A Footprint of Peak Contact Pressure Distribution across the Plantar Surface, wearing their Normal Shoe

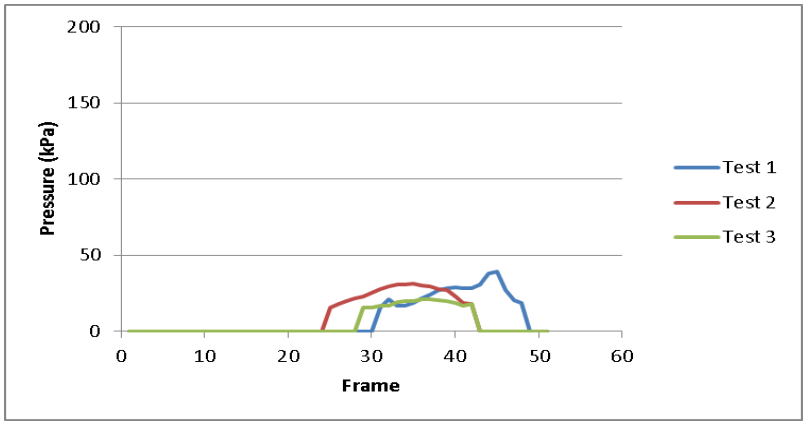


Figure 4-9; Subject A wearing the Aircast™ with No Added Air, Contact Pressure under the Metatarsal Heads

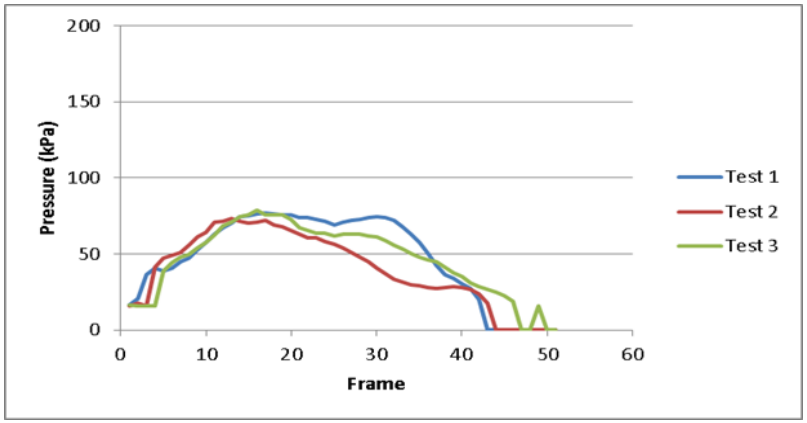


Figure 4-10; Subject A wearing the Aircast™ with No Added Air, Contact Pressure under the Heel

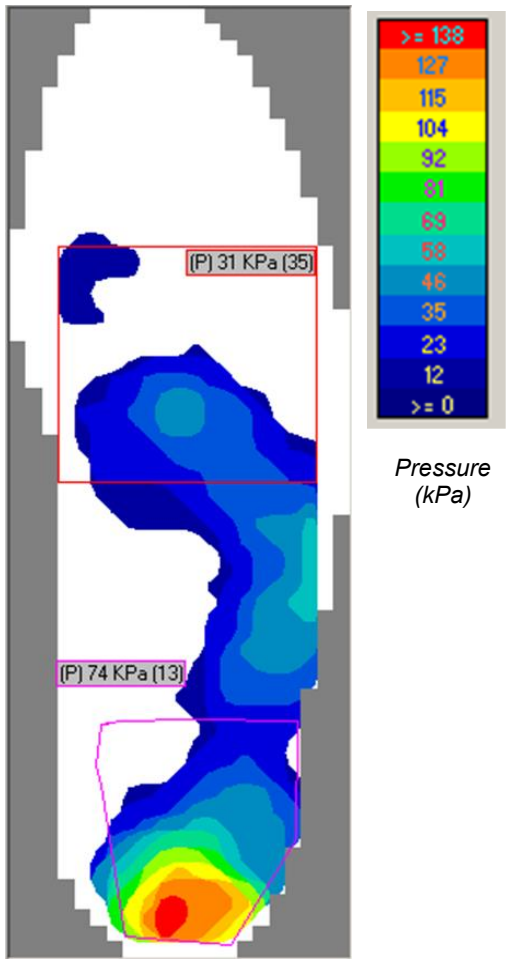


Figure 4-11; Subject A Footprint of Contact Pressure Distribution across Plantar Surface, wearing the Aircast™ with no added air

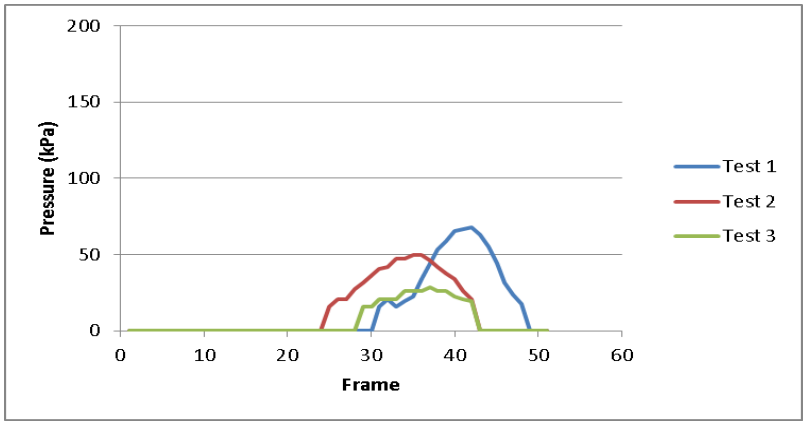


Figure 4-12; Subject A wearing the Aircast™ with No Added Air, Peak Contact Pressure under the Metatarsal Heads

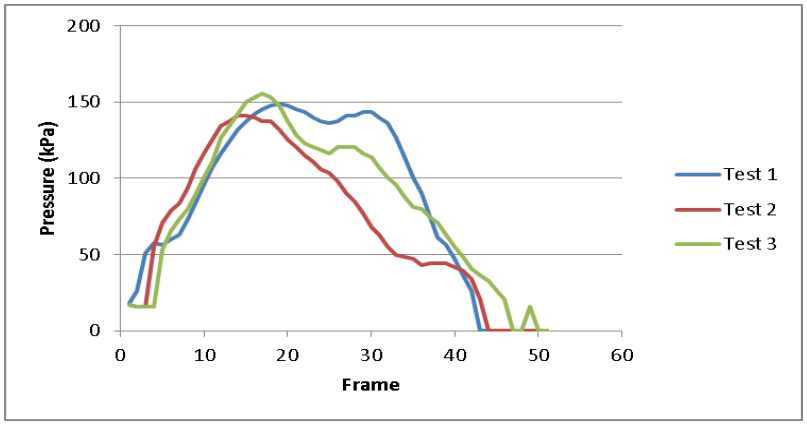


Figure 4-13; Subject A wearing the Aircast™ with No Added Air. Peak Contact Pressure under the Heel

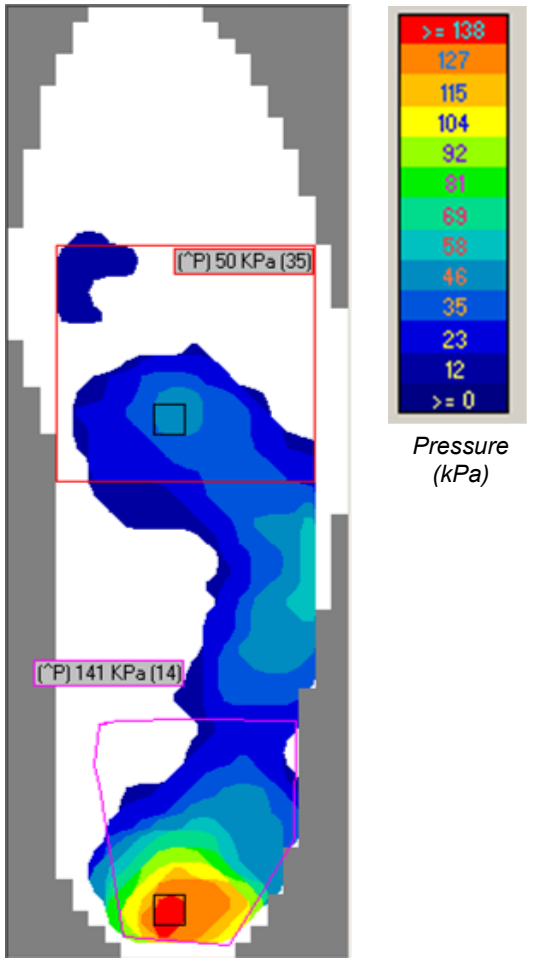


Figure 4-14; Subject A Footprint of Peak Contact Pressure Distribution across the Plantar Surface, wearing the Aircast™ with no added Air

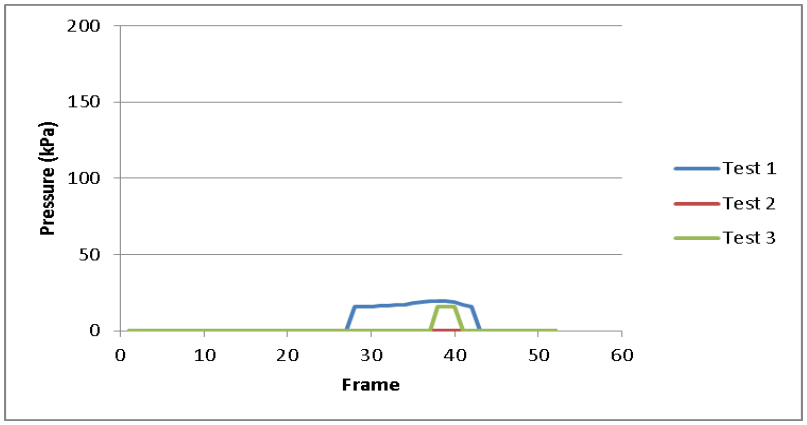


Figure 4-15; Subject A wearing the Aircast™ with 30mmHg Air. Contact Pressure under the Metatarsal Heads

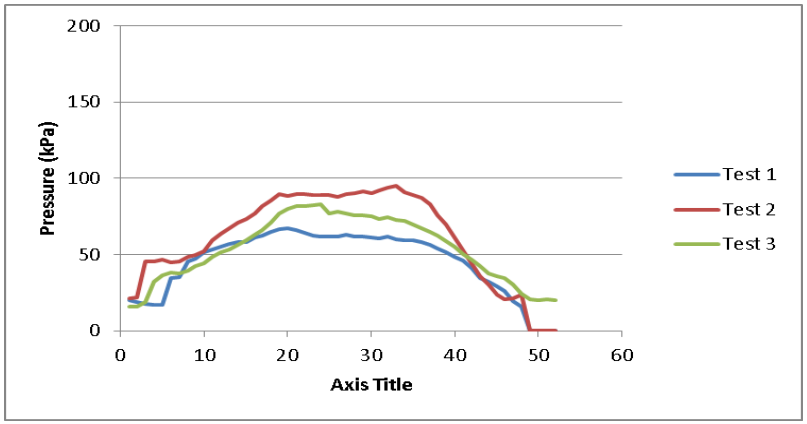


Figure 4-16; Subject A wearing the Aircast™ with 30mmHg Air. Contact Pressure under the Heel

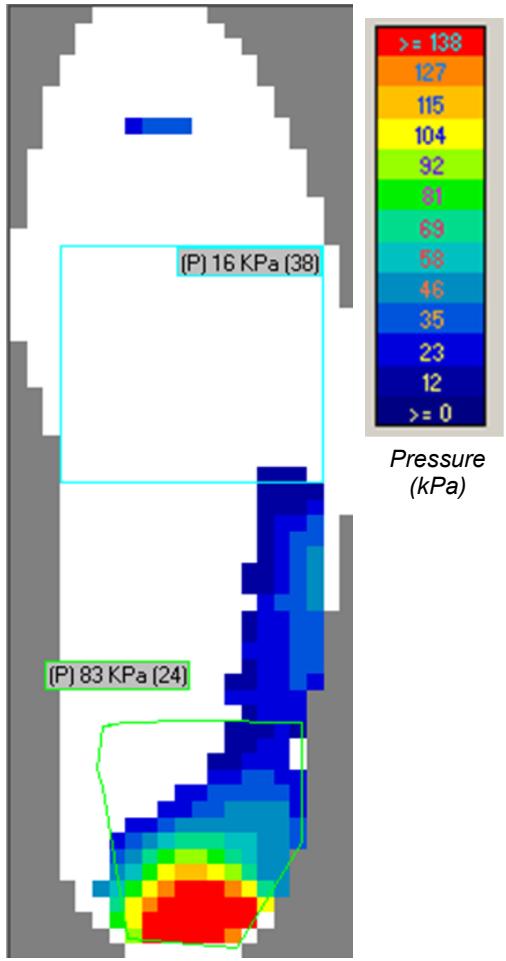


Figure 4-17; Subject A Footprint of Contact Pressure Distribution across Plantar Surface, wearing the Aircast™ with 30mmHg

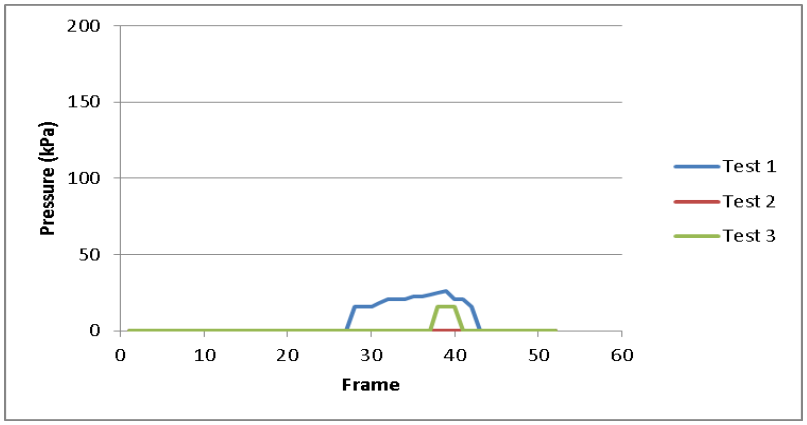


Figure 4-18; Subject A wearing the Aircast™ with 30mmHg Air. Peak Contact Pressure under the Metatarsal Heads

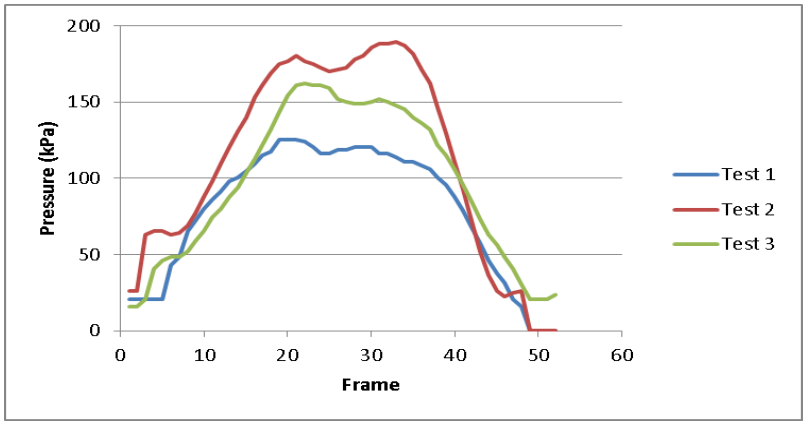


Figure 4-19; Subject A wearing the Aircast™ with 30mmHg Air. Peak Contact Pressure under the Heel

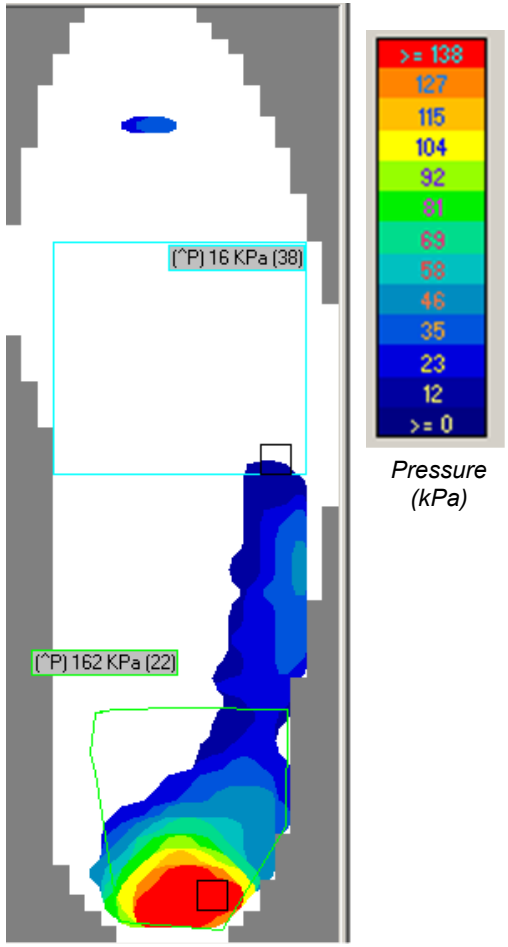


Figure 4-20; Subject A Footprint of Peak Contact Pressure Distribution across the Plantar Surface, wearing the Aircast™ with 30mmHg Air

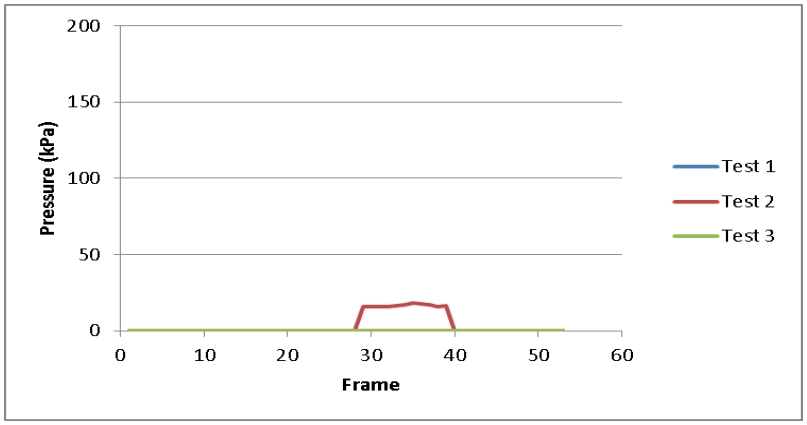


Figure 4-21; Subject A wearing the Aircast™ with 40mmHg Air. Contact Pressure under the Metatarsal Heads

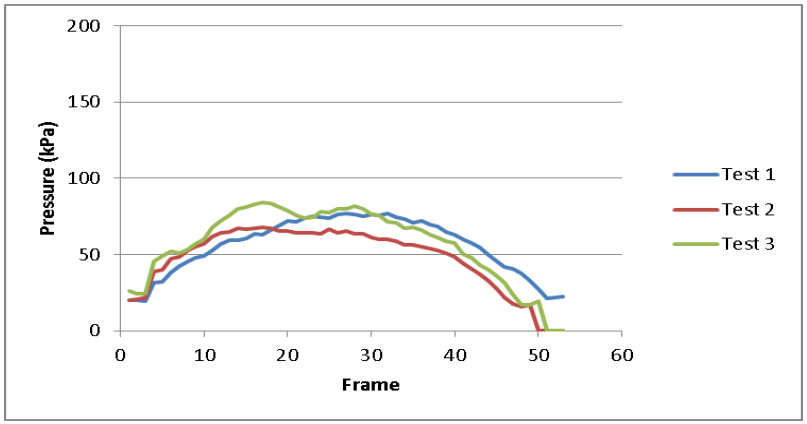


Figure 4-22; Subject A wearing the Aircast™ with 40mmHg. Contact Pressure under the Heel

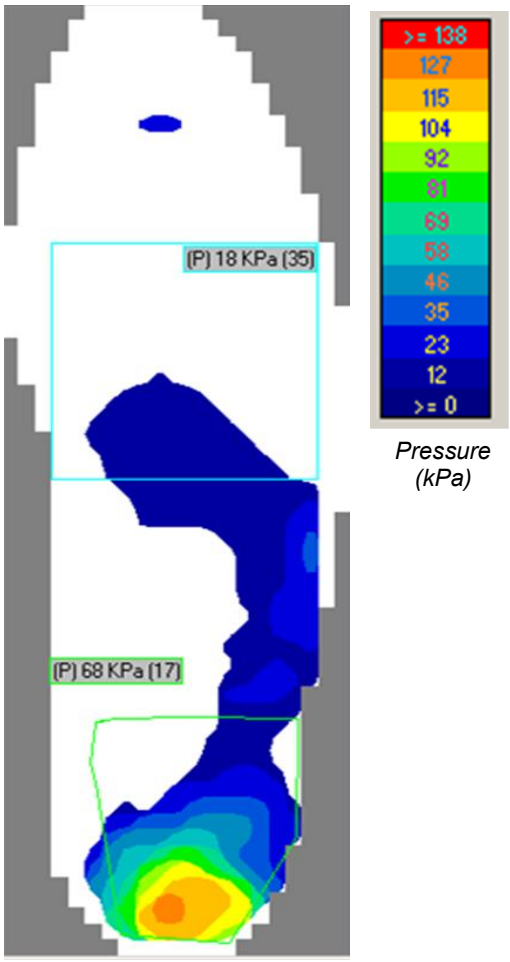


Figure 4-23; Subject A Footprint of Contact Pressure Distribution across Plantar Surface, wearing the Aircast™ with 40mmHg

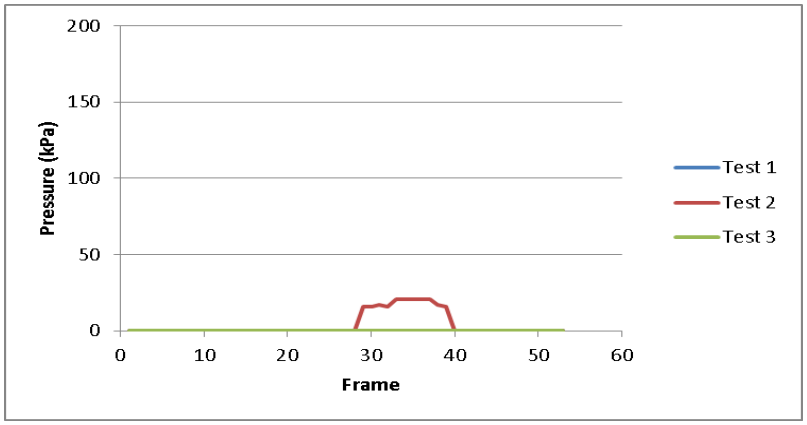


Figure 4-24; Subject A wearing the Aircast™ with 40mmHg Air. Peak Contact Pressure under the Metatarsal Heads

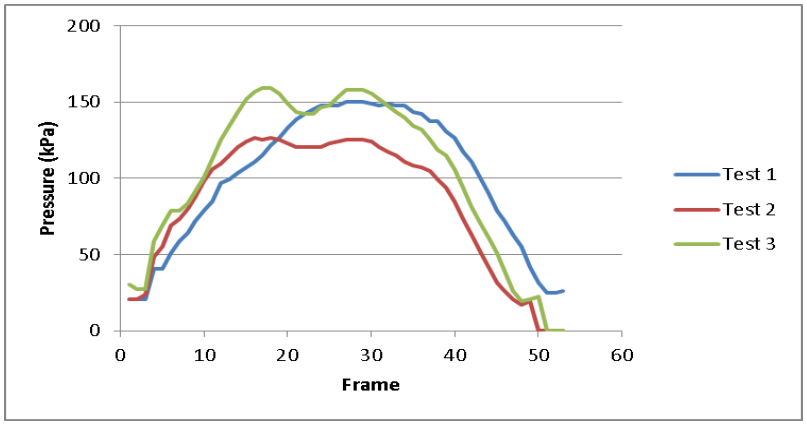


Figure 4-25; Subject A wearing the Aircast™ with 40mmHg Air. Peak Contact Pressure under the Heel

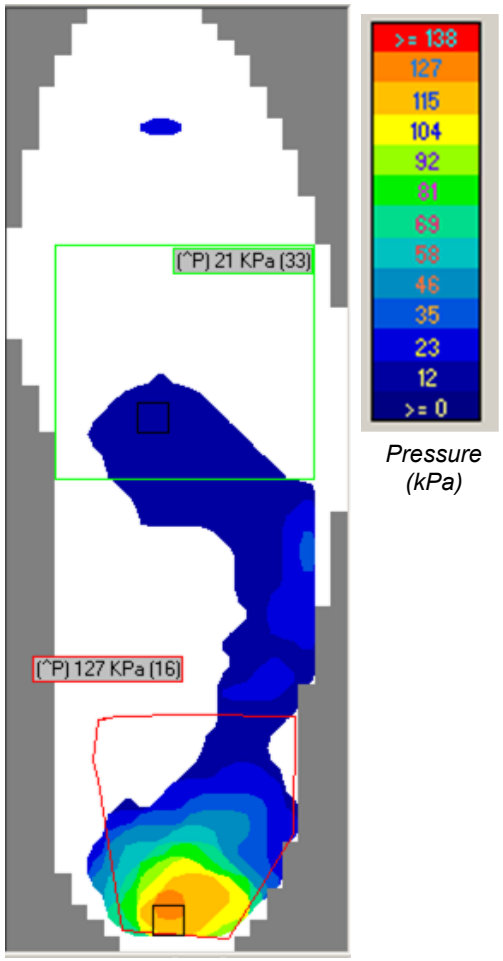


Figure 4-26; Subject A Footprint of Peak Contact Pressure Distribution across the Plantar Surface, wearing the Aircast™ with 40mmHg Air

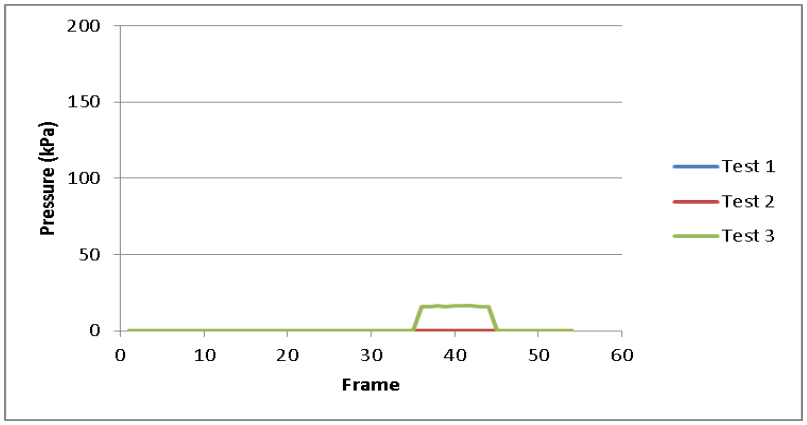


Figure 4-27; Subject A wearing the Aircast™ with High Air Pressure. Contact Pressure under the Metatarsal Heads

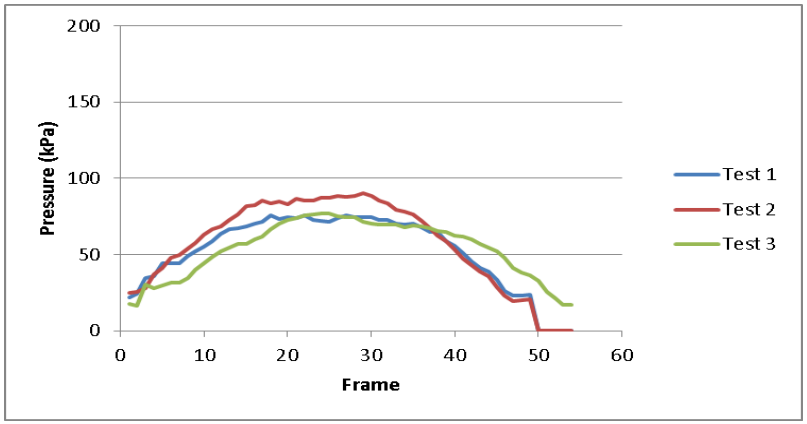


Figure 4-28; Subject A wearing the Aircast™ with High Air Pressure. Contact Pressure under the Heel

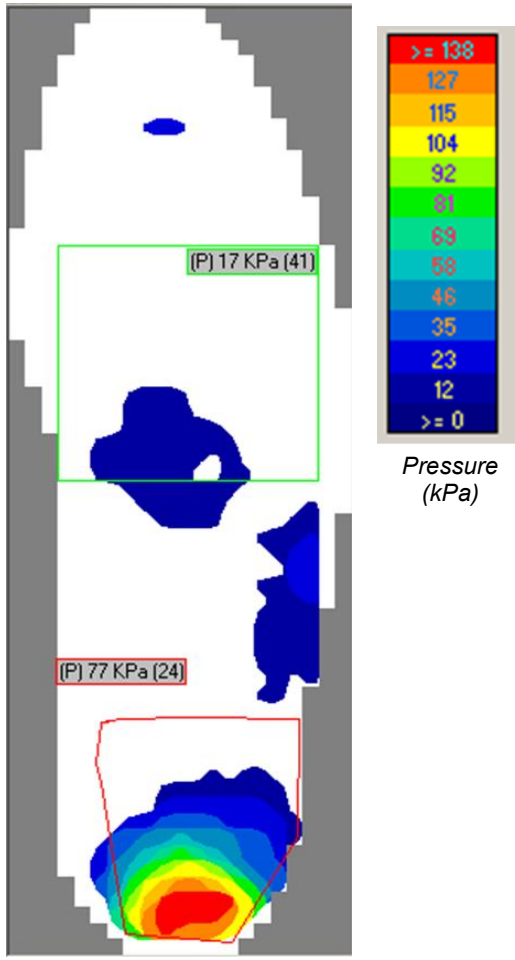


Figure 4-29; Subject A Footprint of Contact Pressure Distribution across Plantar Surface, wearing the Aircast™ with High Air Pressure

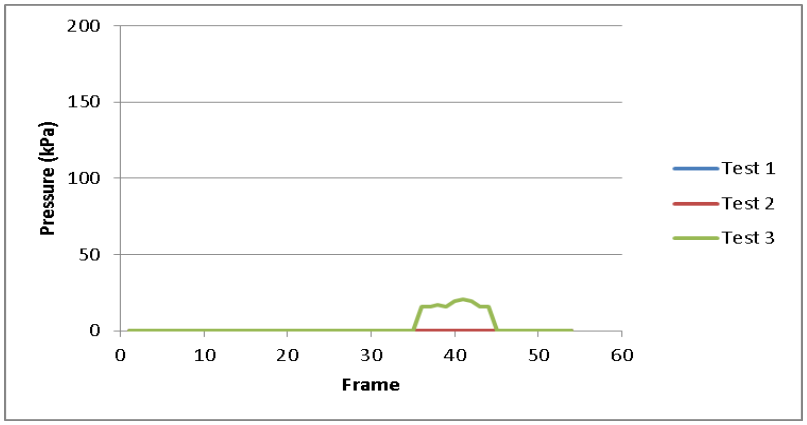


Figure 4-30; Subject A wearing the Aircast™ with High Air Pressure. Peak Contact Pressure under the Metatarsal Heads

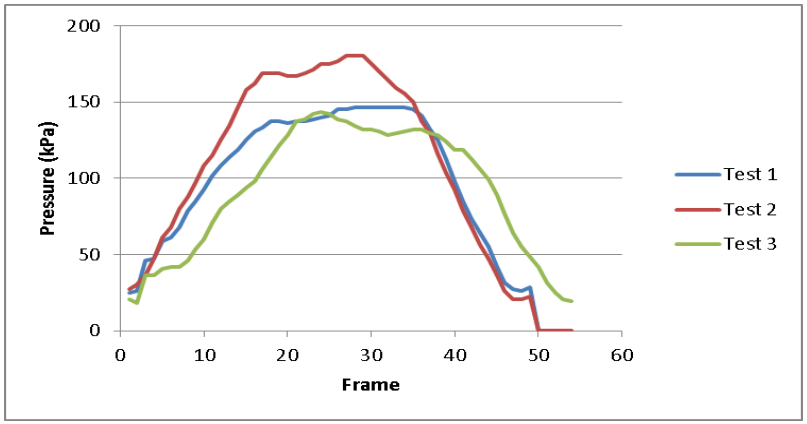
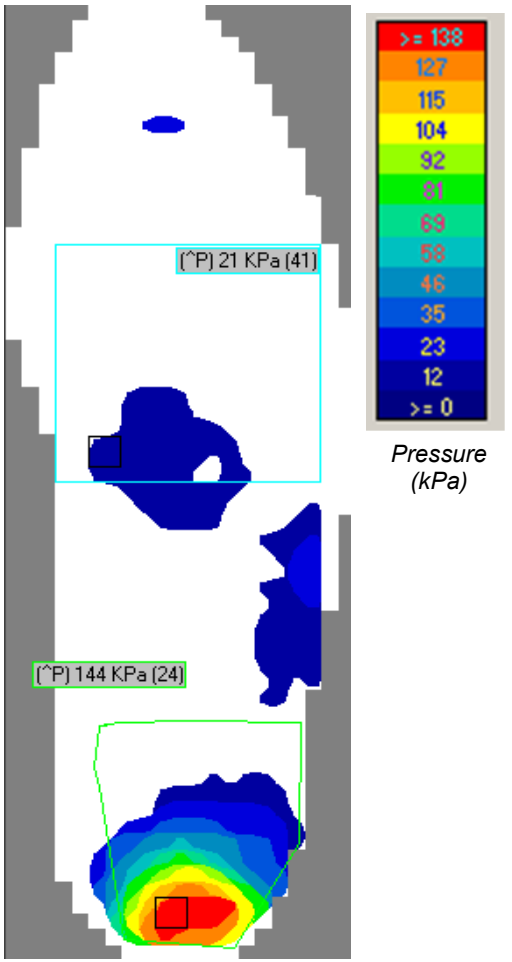


Figure 4-31; Subject A wearing the Aircast™ with High Air Pressure. Peak Contact Pressure under the Heel



Pressure (kPa)

Figure 4-32; Subject A Footprint of Peak Contact Pressure Distribution across Plantar Surface, wearing the Aircast™ with High Air Pressure

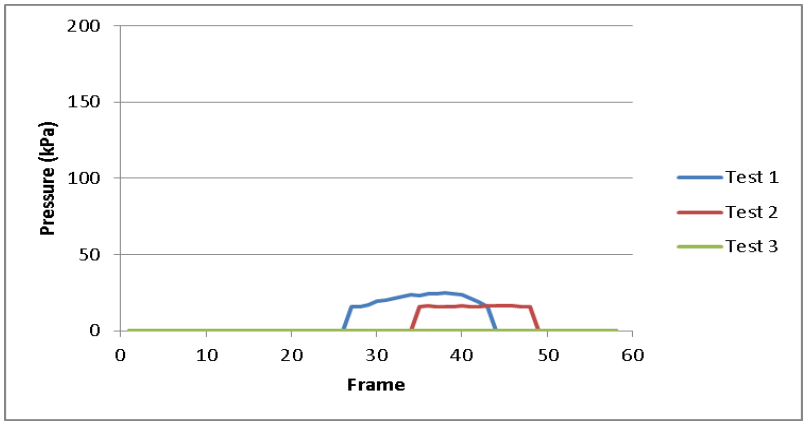


Figure 4-33; Subject A wearing the Airstep™ with no added air. Contact Pressure under the Metatarsal Heads

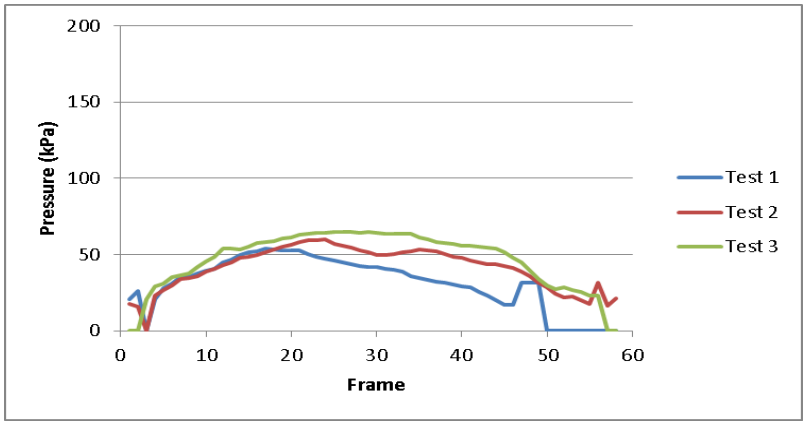


Figure 4-34; Subject A wearing the Airstep™ with no added air. Contact Pressure under the Heel

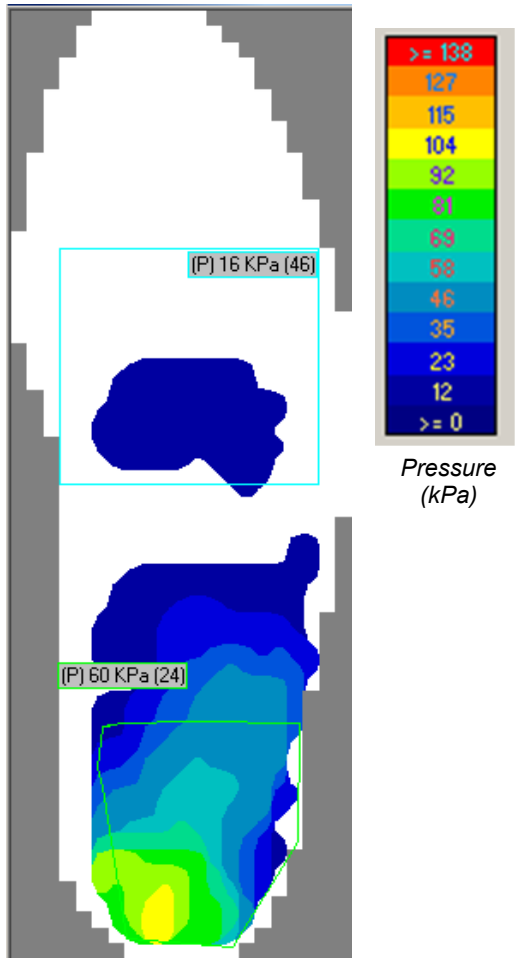


Figure 4-35; Subject A Footprint of Contact Pressure Distribution across the Plantar Surface, wearing the Airstep™ with no added air.

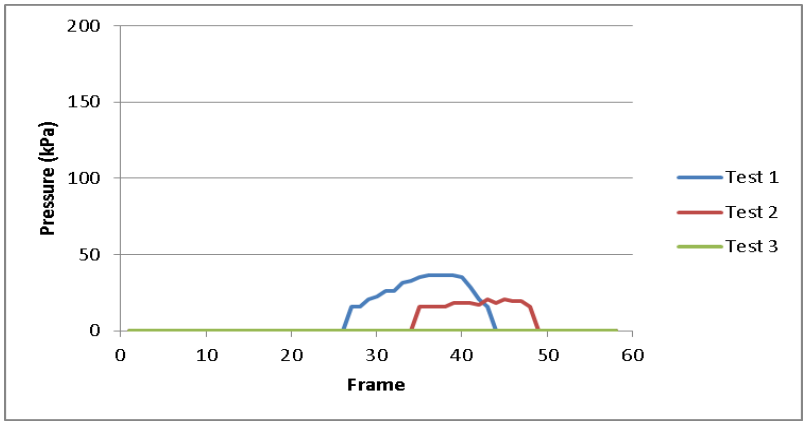


Figure 4-36; Subject A wearing the Airstep™ with no added air. Peak Contact Pressure under the Metatarsal Heads

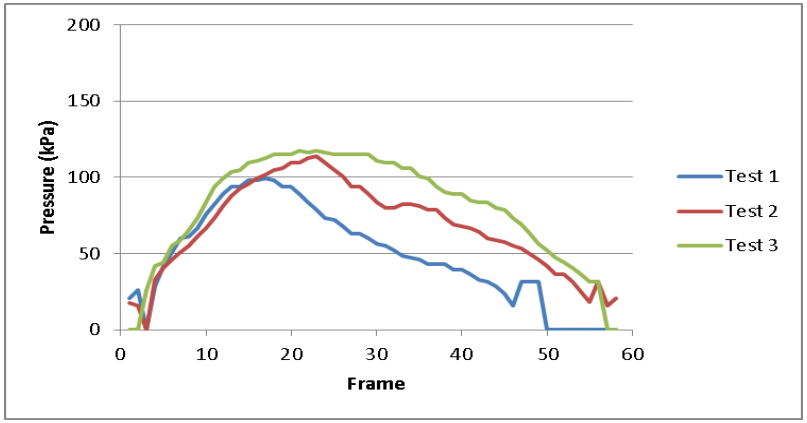


Figure 4-37; Subject A wearing the Airstep™ with no added air. Peak Contact Pressure under the Heel

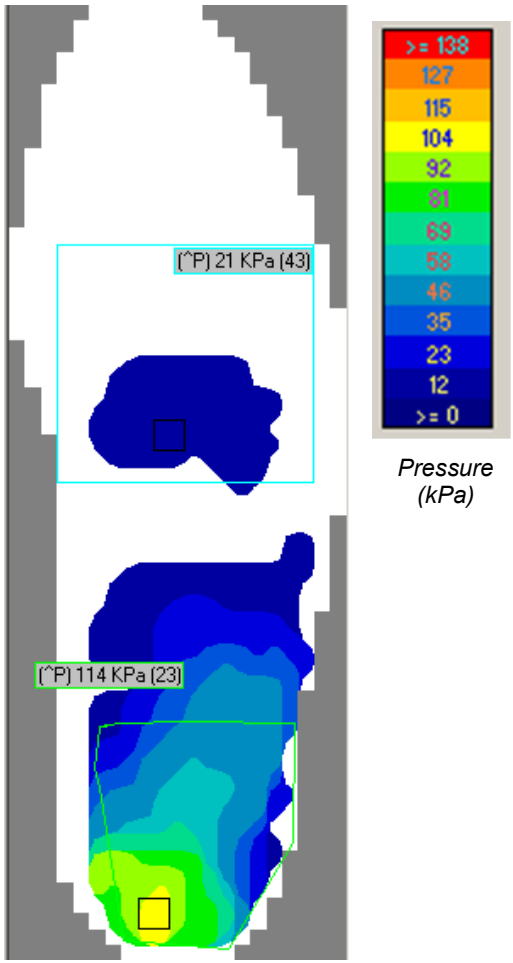


Figure 4-38; Subject A Footprint of Peak Contact Pressure Distribution across the Plantar Surface, wearing the Airstep™ with no added air.

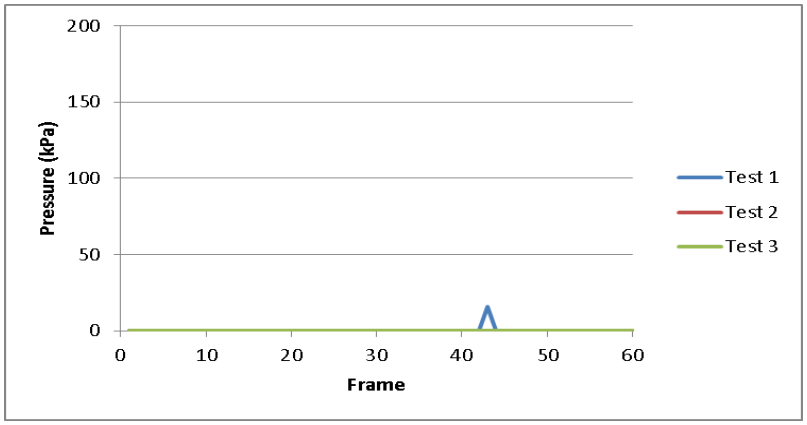


Figure 4-39; Subject A wearing the Airstep™ with added air. Contact Pressure under the Metatarsal Heads

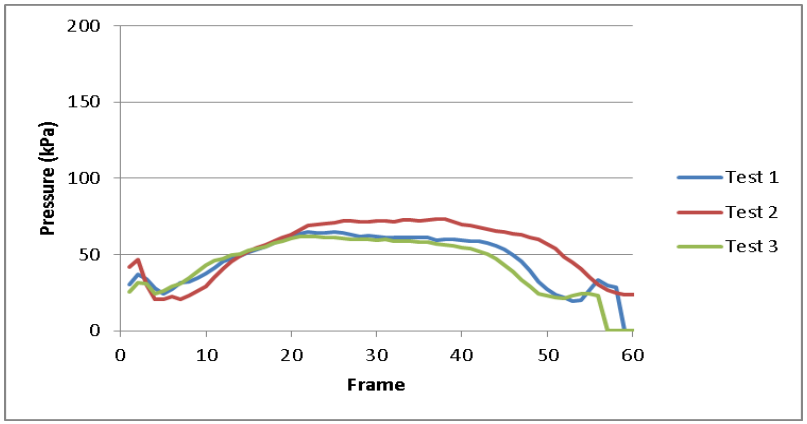


Figure 4-40; Subject A wearing the Airstep™ with added air. Contact Pressure under the Heel

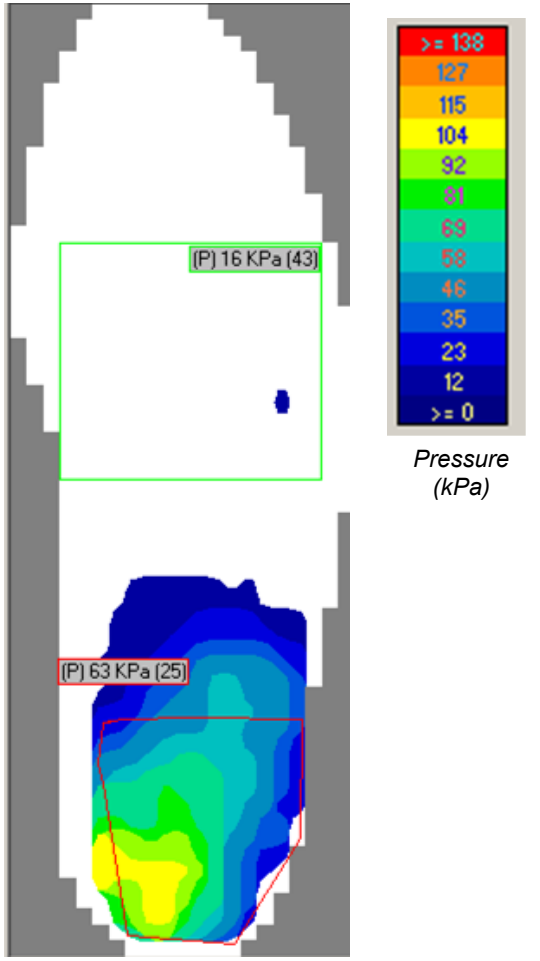


Figure 4-41; Subject A Footprint of Contact Pressure Distribution across the Plantar Surface, wearing the Airstep™ with added air.

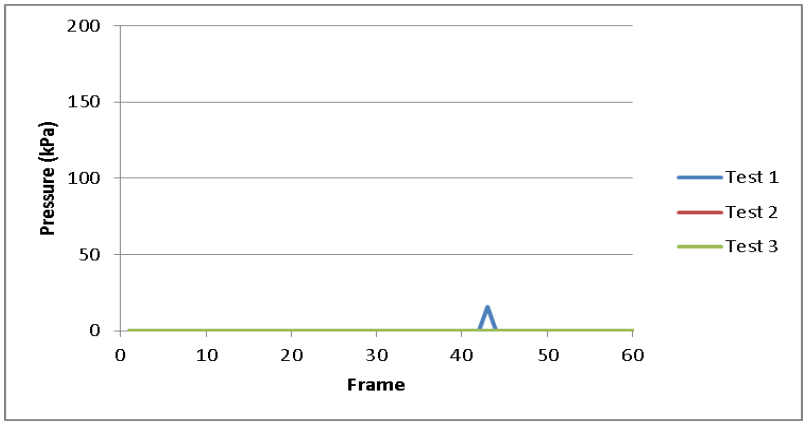


Figure 4-42; Subject A wearing the Airstep™ with added air. Peak Contact Pressure under the Metatarsal Heads

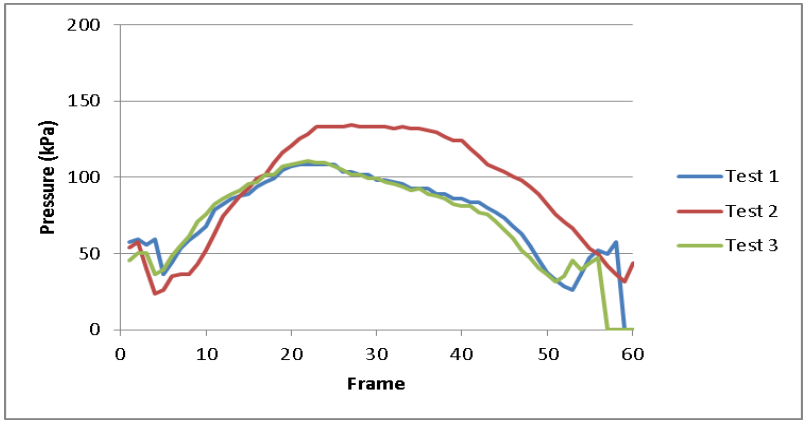


Figure 4-43; Subject A wearing the Airstep™ with added air. Peak Contact Pressure under the Heel

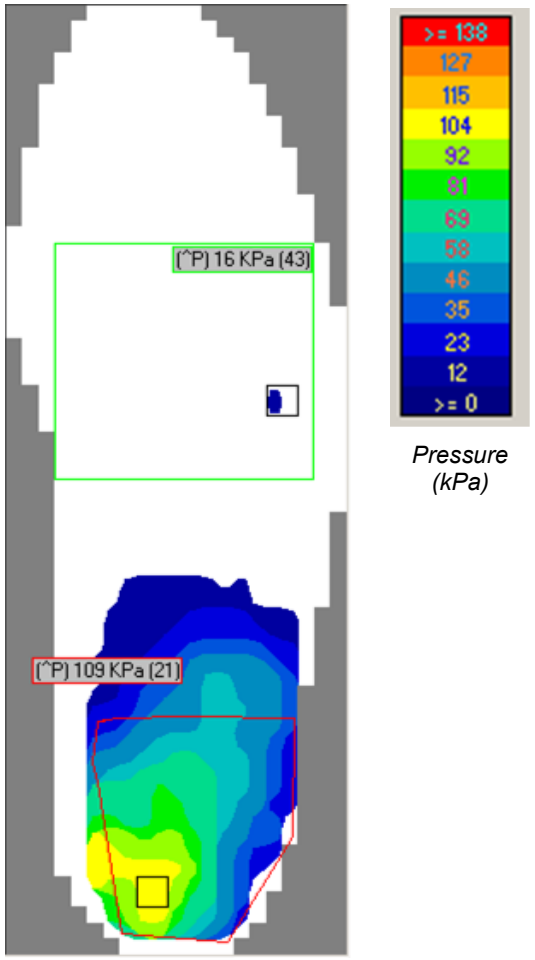


Figure 4-44; Subject A Footprint of Peak Contact Pressure Distribution across the Plantar Surface, wearing the Airstep™ with Added air

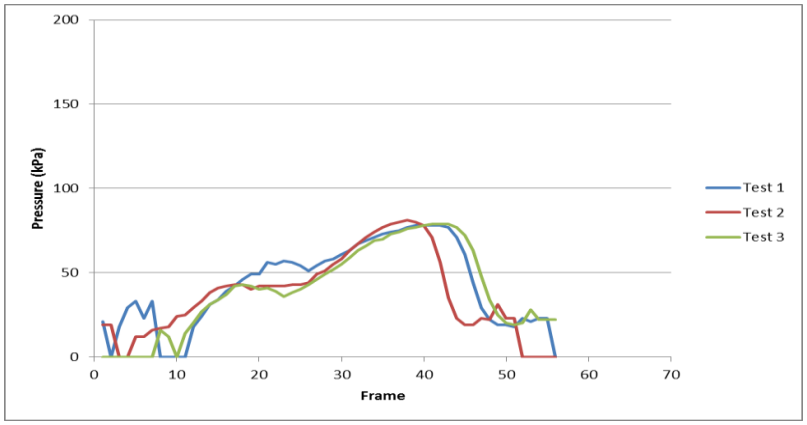


Figure 4-45; Subject B wearing their Normal Shoe, Contact Pressure under the Metatarsal Heads

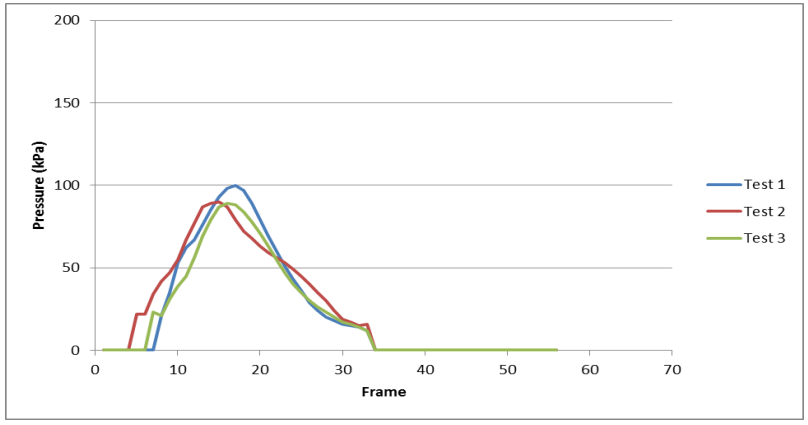
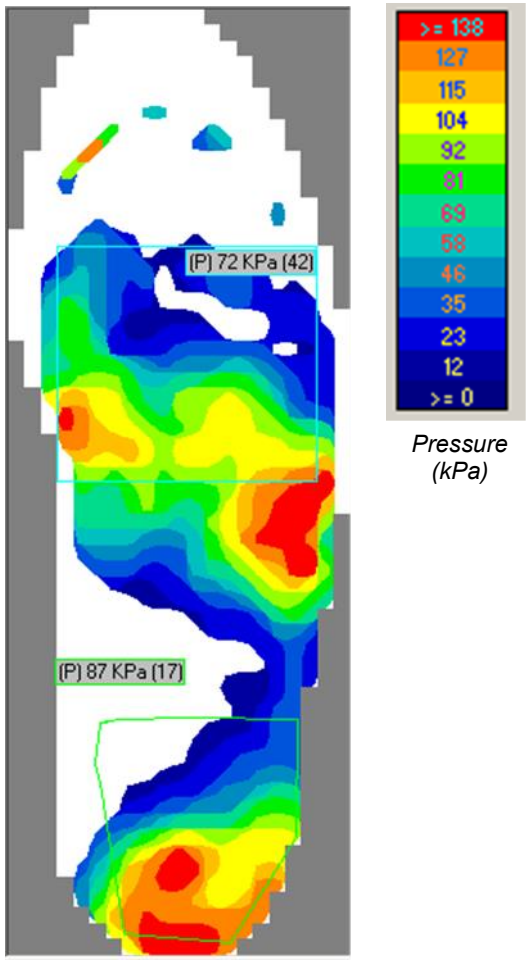


Figure 4-46; Subject B wearing their Normal Shoe, Contact Pressure under the Heel



Pressure (kPa)

Figure 4-47; Subject B Footprint of Contact Pressure Distribution across Plantar Surface, wearing the Normal Shoe

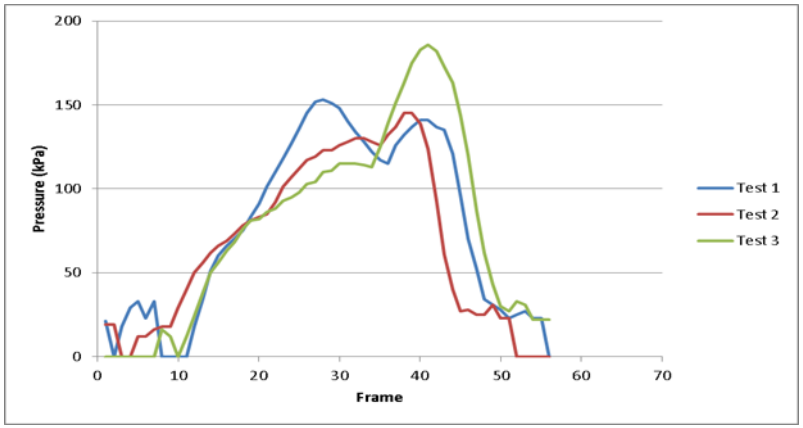


Figure 4-48; Subject B wearing their Normal Shoe, Peak Contact Pressure under the Metatarsal Heads

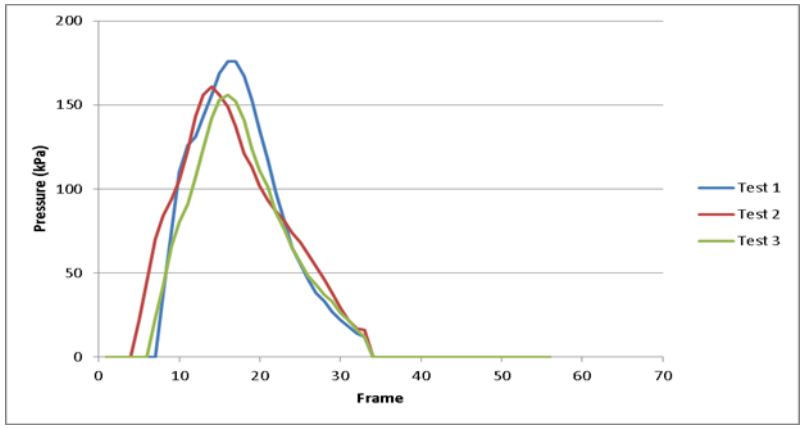


Figure 4-49; Subject B wearing their Normal Shoe, Contact Pressure under the Heel

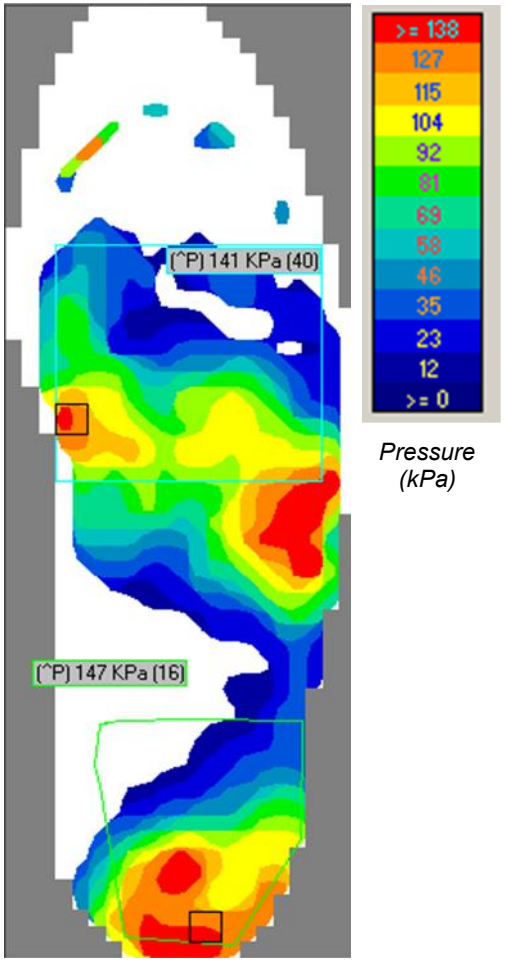


Figure 4-50; Subject B Footprint of Peak Contact Pressure Distribution across Plantar Surface, wearing the Normal Shoe

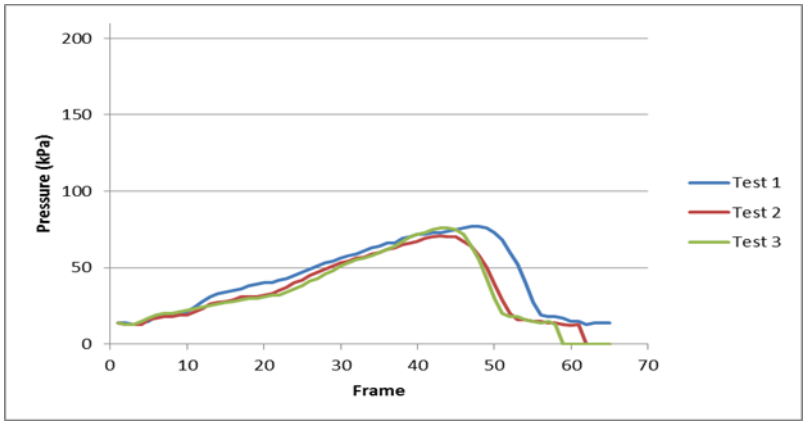


Figure 4-51; Subject B wearing the Aircast™ with no added air. Contact Pressure under the Metatarsal Heads

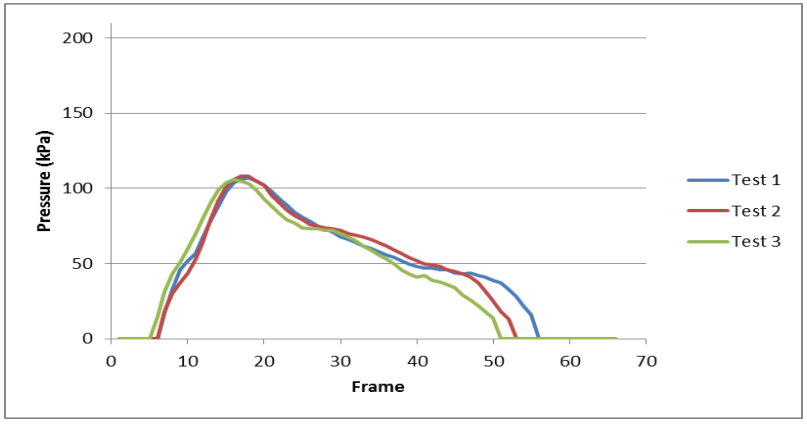


Figure 4-52; Subject B wearing the Aircast™ with no added air. Contact Pressure under the Heel

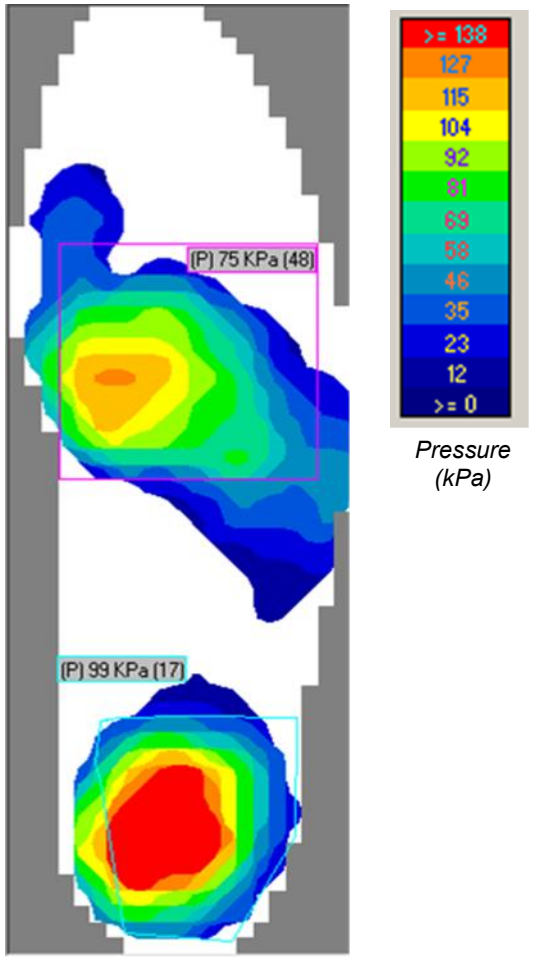


Figure 4-53; Subject B wearing the Aircast™ with no added air. Footprint of Contact Pressure Distribution across Plantar Surface

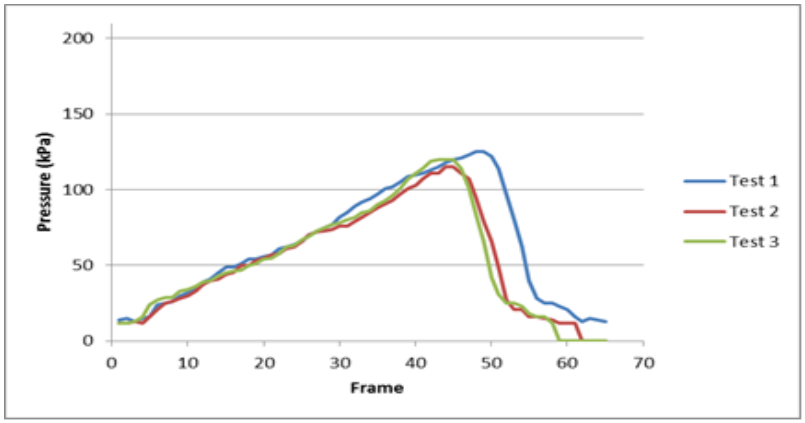


Figure 4-54; Subject B wearing the Aircast™ with no air. Peak Contact Pressure under the Metatarsal Heads

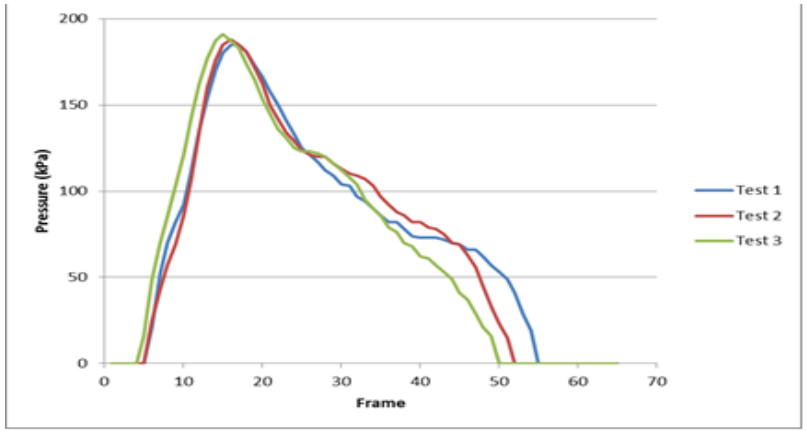


Figure 4-55; Subject B wearing the Aircast™ with no air. Peak Contact Pressure under the Heel

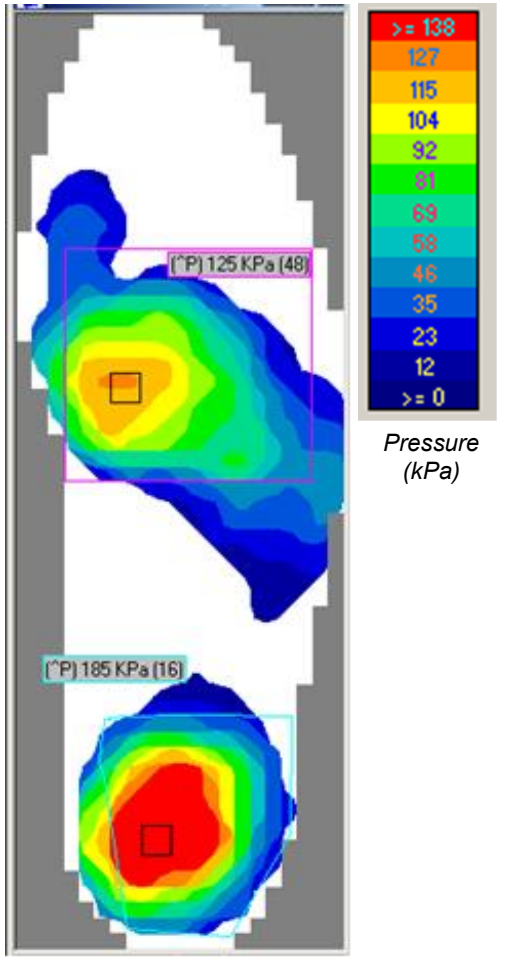


Figure 4-56; Subject B wearing the Aircast™ with no air. Footprint of Peak Contact Pressure Distribution across Plantar Surface

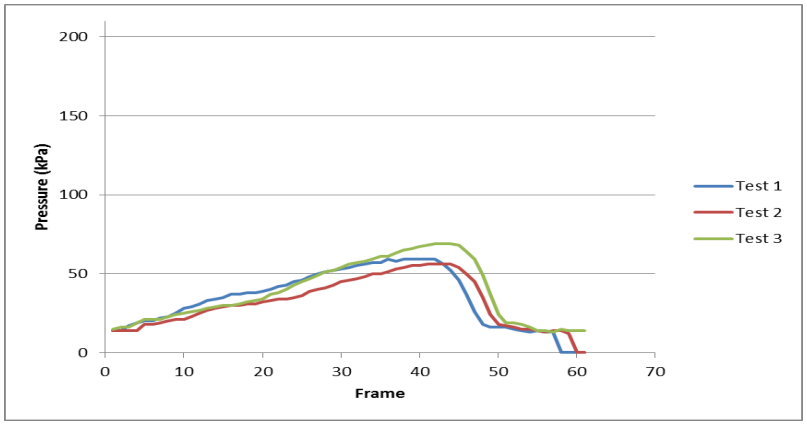


Figure 4-57; Subject B wearing the Aircast™ with 30mmHg. Contact Pressure under the Metatarsal Heads

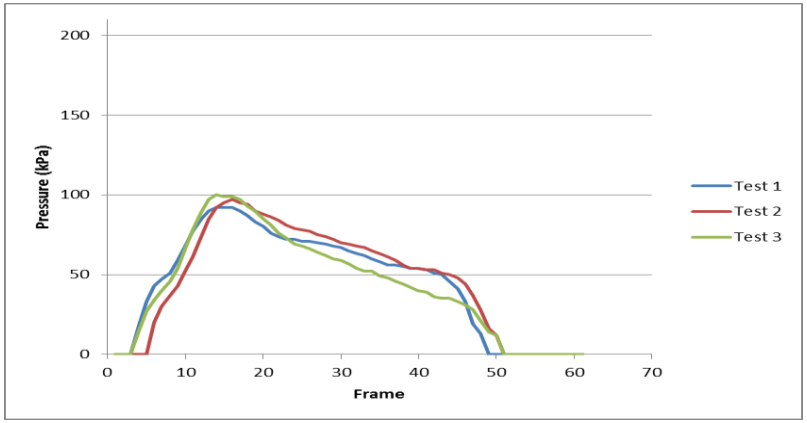


Figure 4-58; Subject B wearing the Aircast™ with 30mmHg. Contact Pressure under the Heel

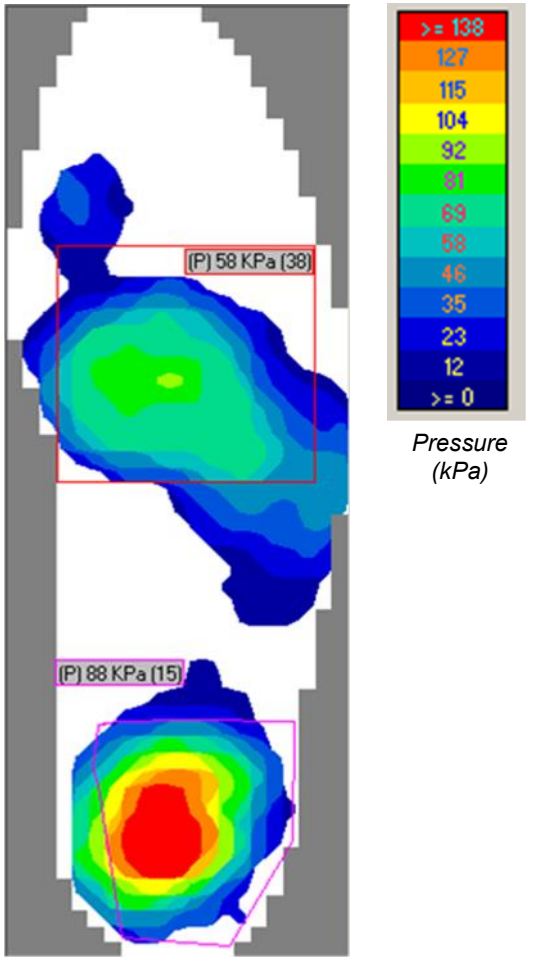


Figure 4-59; Subject B wearing the Aircast™ with 30mmHg. Footprint of Contact Pressure Distribution across Plantar Surface

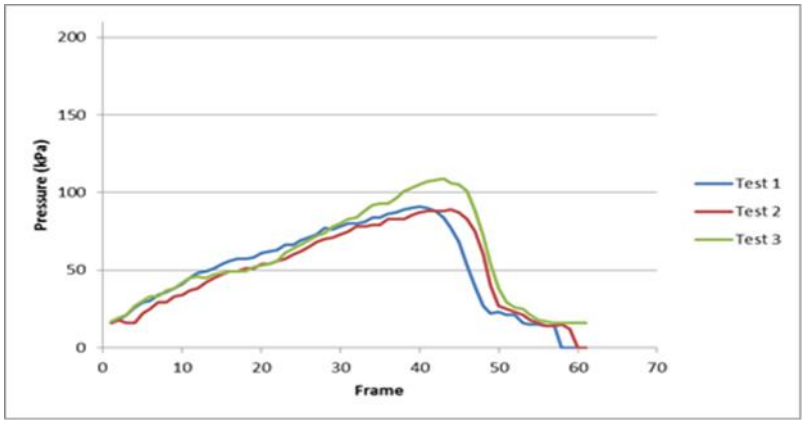


Figure 4-60; Subject B wearing the Aircast™ with 30mmHg of Air. Peak Contact Pressure under the Metatarsal Heads

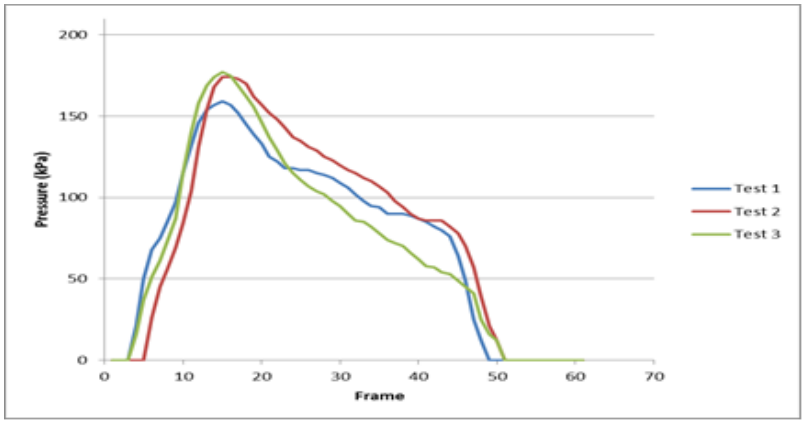


Figure 4-61; Subject B wearing the Aircast™ with 30mmHg of Air. Peak Contact Pressure under the Heels

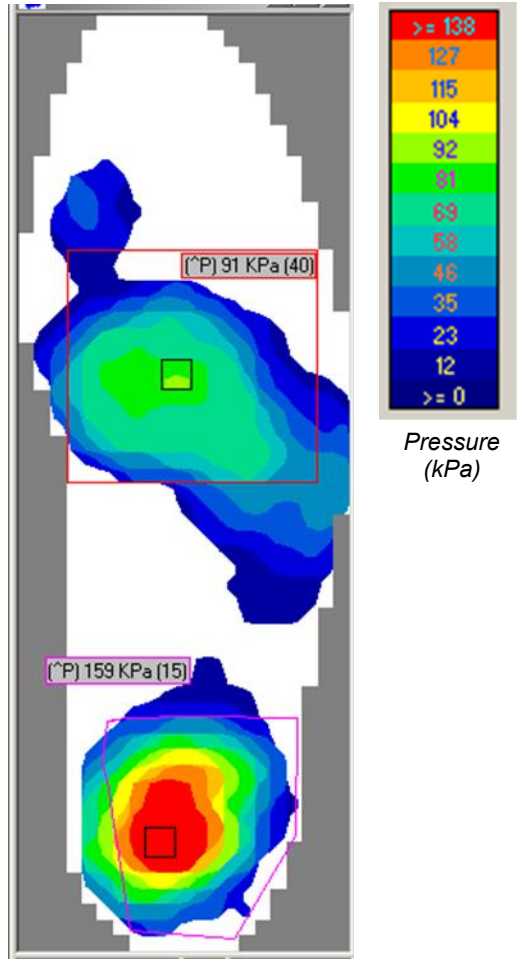


Figure 4-62; Subject B wearing the Aircast™ with 30mmHg. Footprint of Peak Contact Pressure Distribution across Plantar Surface

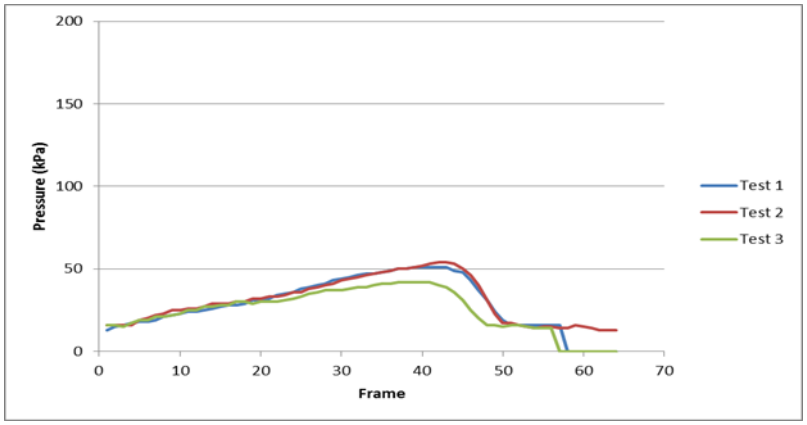


Figure 4-63; Subject B wearing the Aircast™ with 40mmHg. Contact Pressure under the Metatarsal Heads

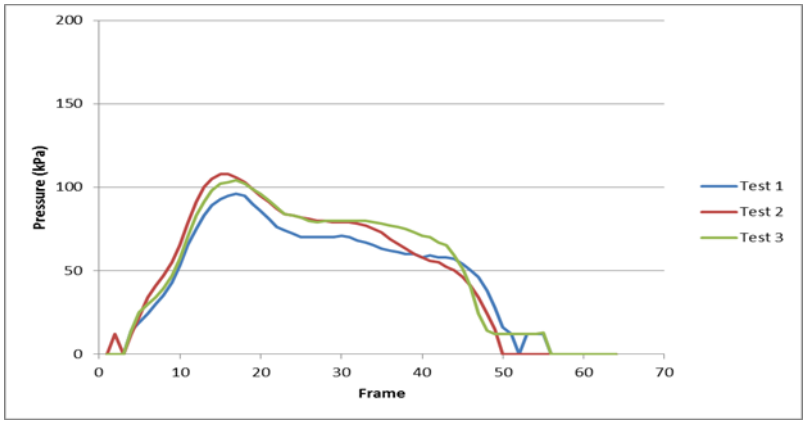


Figure 4-64; Subject B wearing the Aircast™ with 40mmHg of air. Contact Pressure under the Heel

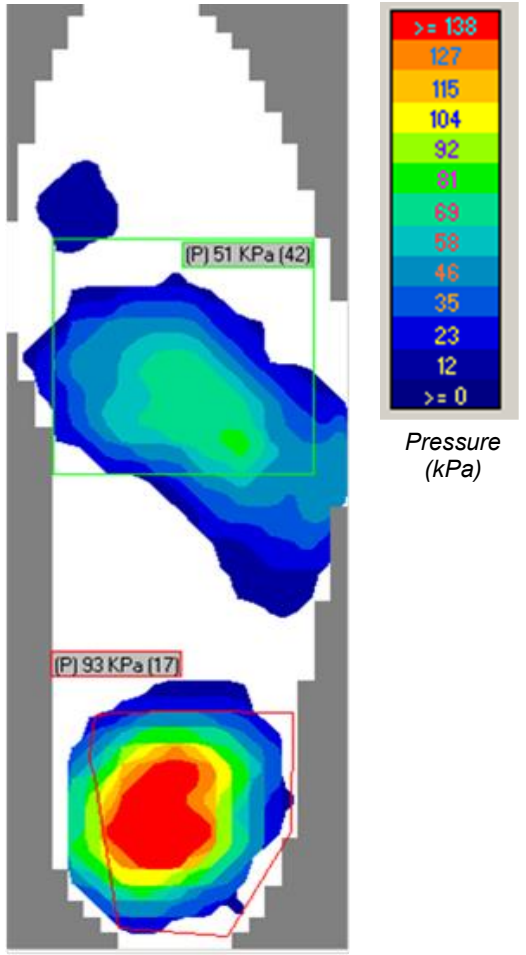


Figure 4-65; Subject B wearing the Aircast™ with 40mmHg of air. Footprint of Contact Pressure Distribution across Plantar Surface

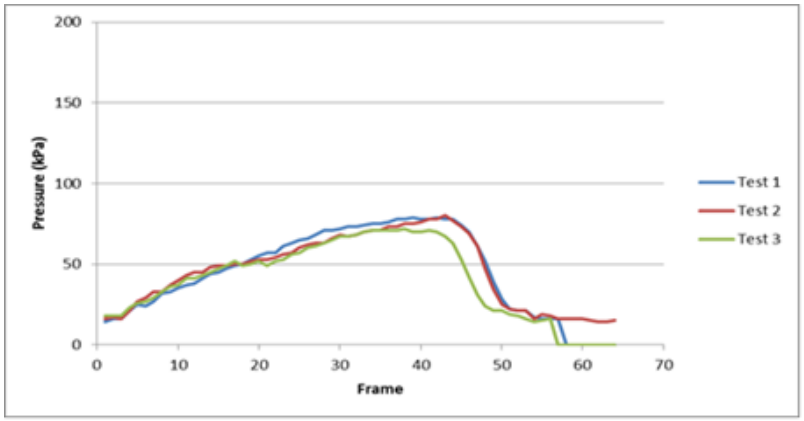


Figure 4-66; Subject B wearing the Aircast™ with 40mmHg. Peak Contact Pressure under the Metatarsal Heads

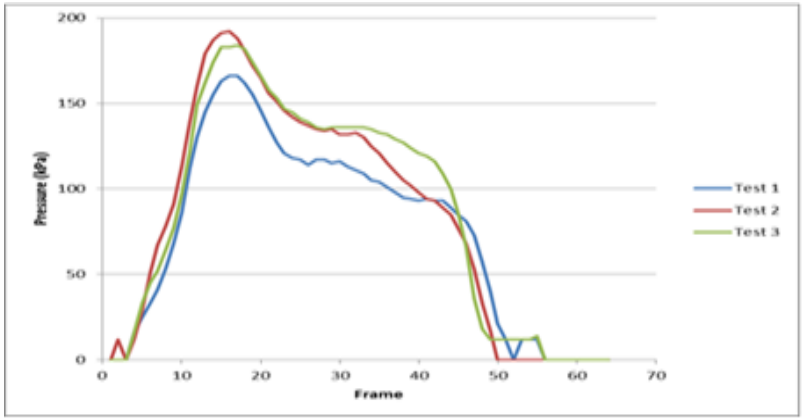


Figure 4-67; Subject B wearing the Aircast™ with 40mmHg. Peak Contact Pressure under the Heel

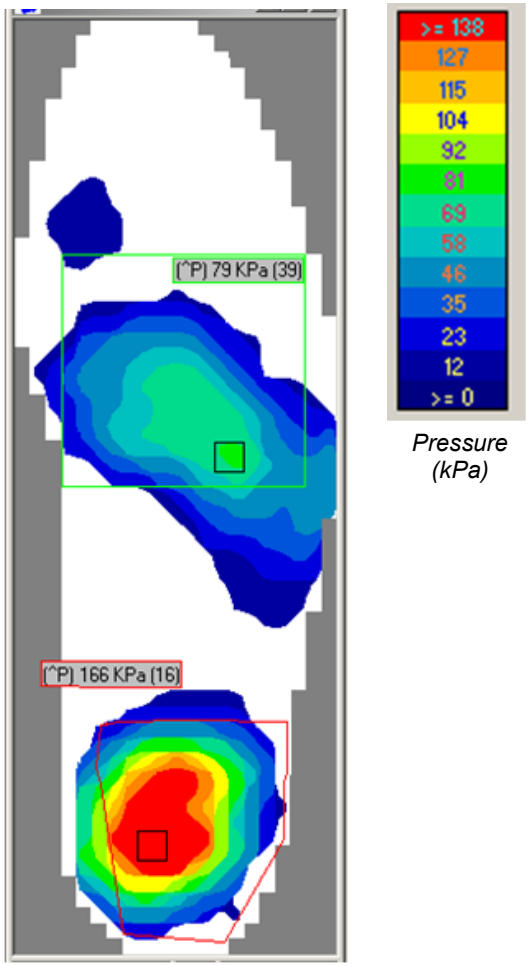


Figure 4-68; Subject B wearing the Aircast™ with 40mmHg. . Footprint of Peak Contact Pressure Distribution across Plantar Surface

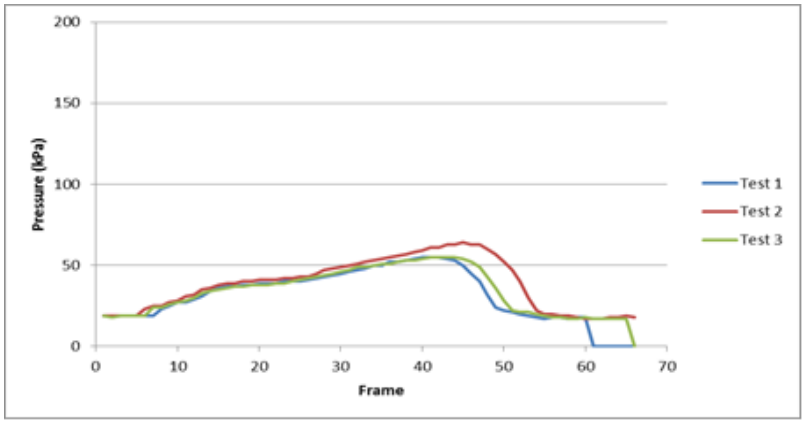


Figure 4-69; Subject B wearing the Aircast™ with High Air. Contact Pressure under the Metatarsal Heads

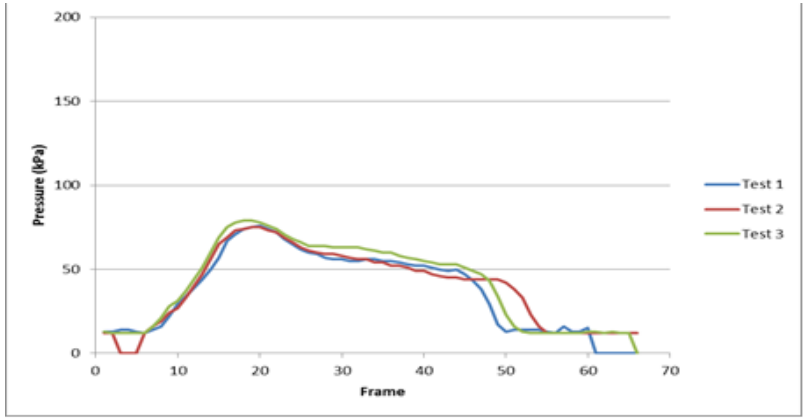


Figure 4-70; Subject B wearing the Aircast™ with High Air. Contact Pressure under the Heel

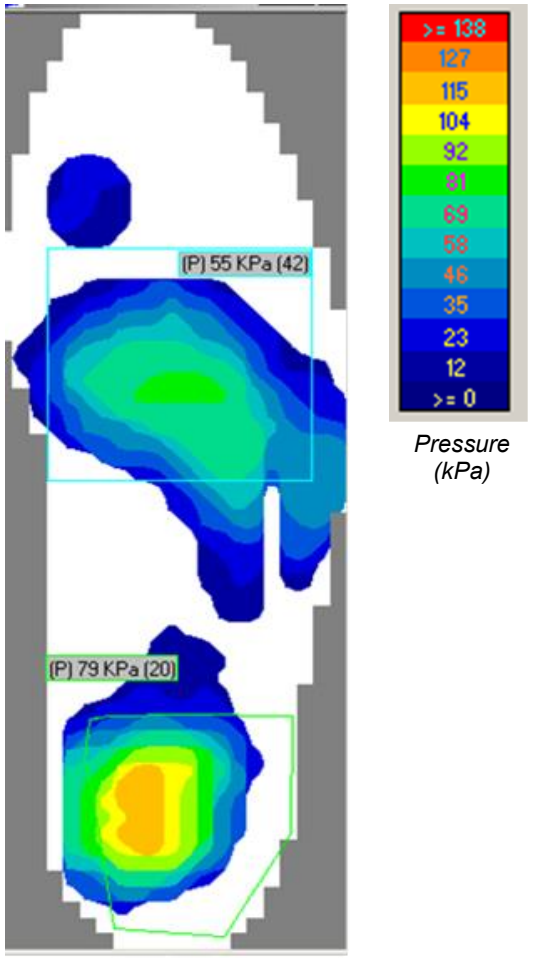


Figure 4-71; Subject B wearing the Aircast™ with High Air. Footprint of Contact Pressure Distribution across Plantar Surface

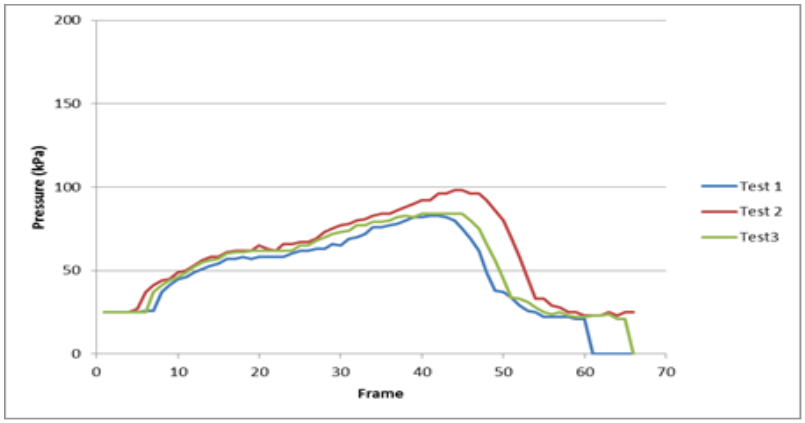


Figure 4-72; Subject B wearing the Aircast™ with High Air. Peak Contact Pressures under the Metatarsal Heads

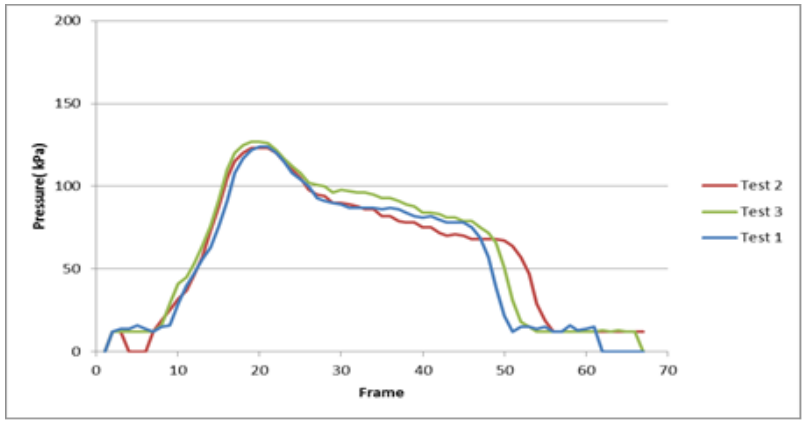


Figure 4-73; Subject B wearing the Aircast™ with High Air. Peak Contact Pressures under the Heel

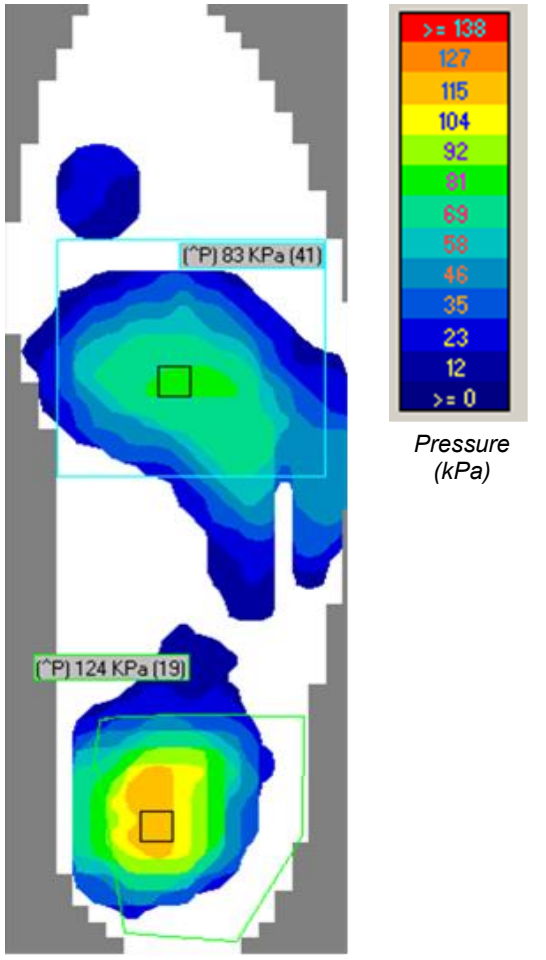


Figure 4-74; Subject B wearing the Aircast™ with High Air. Footprint of Peak Contact Pressure Distribution across Plantar Surface

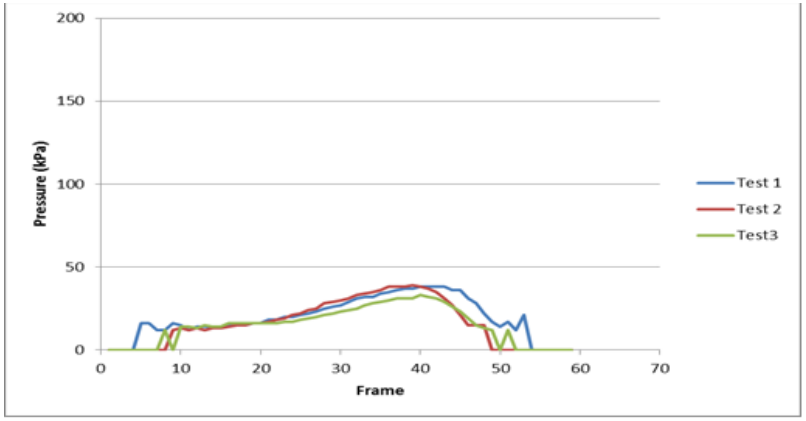


Figure 4-75; Subject B wearing the Airstep™ with No added Air. Contact Pressure under the Metatarsal Heads

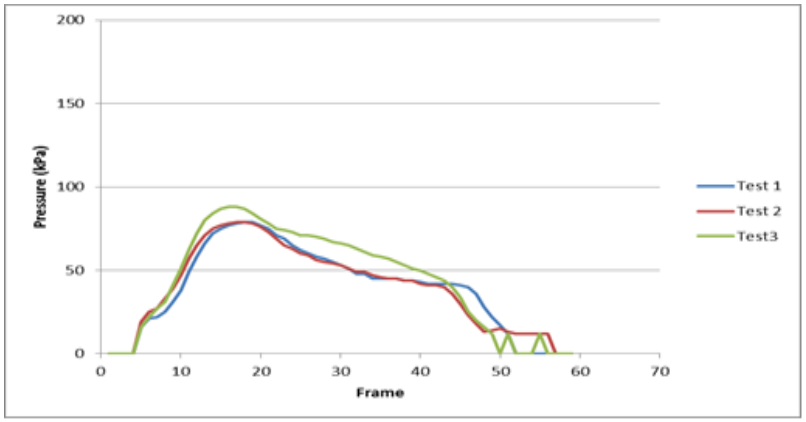


Figure 4-76; Subject B wearing the Airstep™ with No added Air. Contact Pressure under the Heel

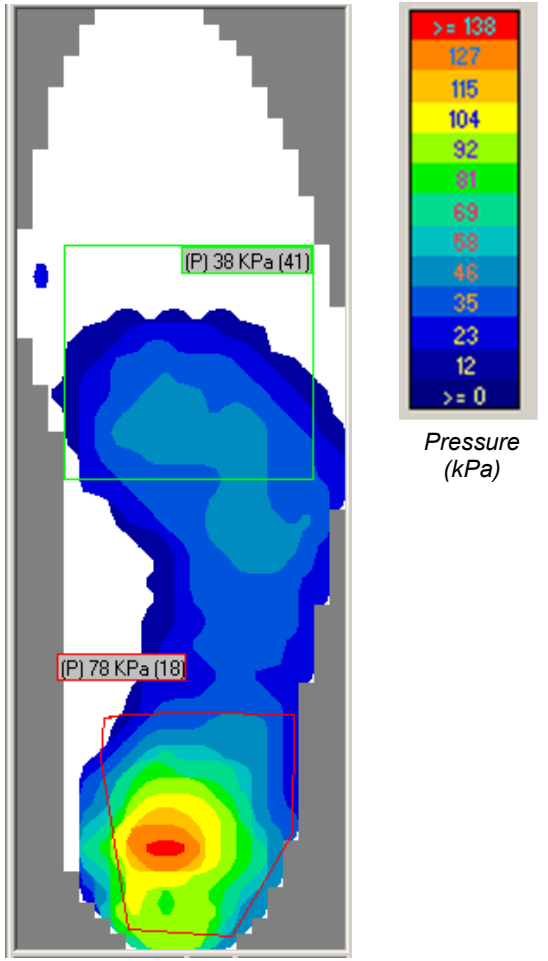


Figure 4-77; Subject B wearing the Airstep™ with No added Air. Footprint of Contact Pressure Distribution across Plantar Surface

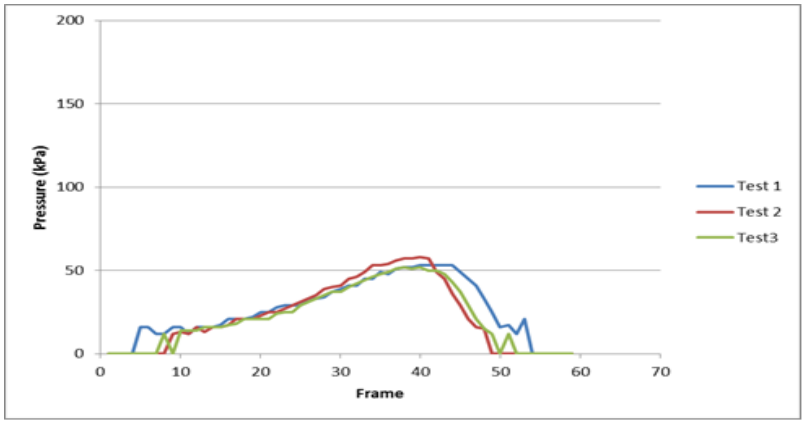


Figure 4-78; Subject B wearing the Airstep™ with No added Air. Peak Contact Pressures under the Metatarsal Heads

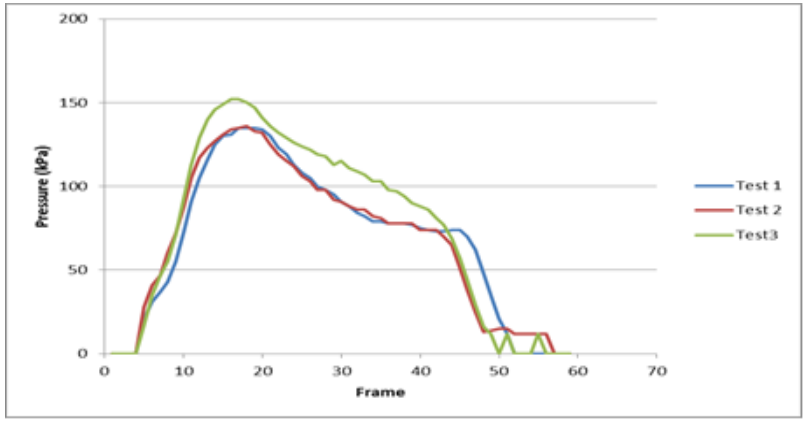


Figure 4-79; Subject B wearing the Airstep™ with No added Air. Peak Contact Pressures under the Heel

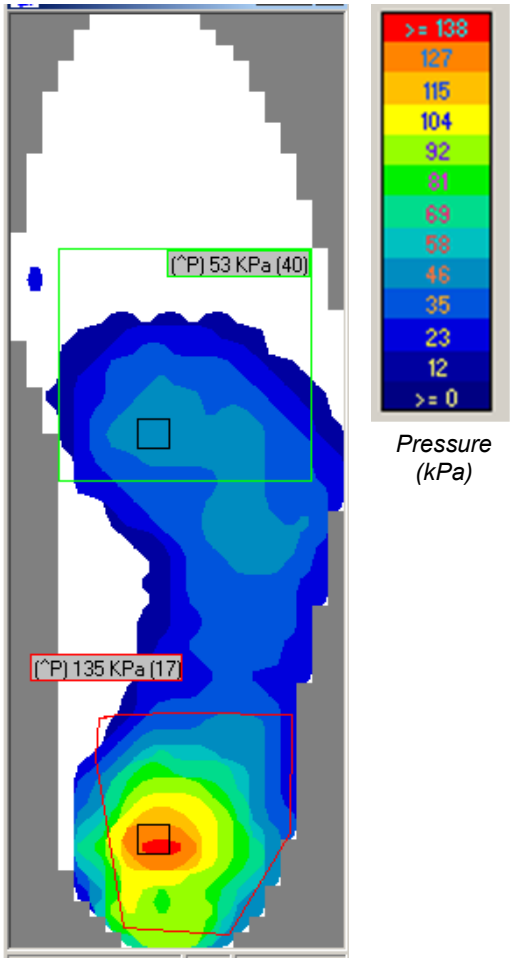


Figure 4-80; Subject B wearing the Airstep™ with No added Air. Footprint of Peak Contact Pressure Distribution across Plantar Surface

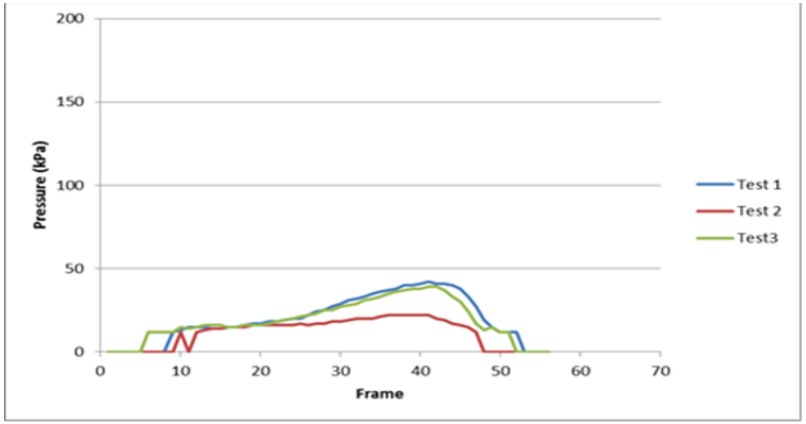


Figure 4-81; Subject B wearing the Airstep™ with added Air. Contact pressures under the Metatarsal Heads

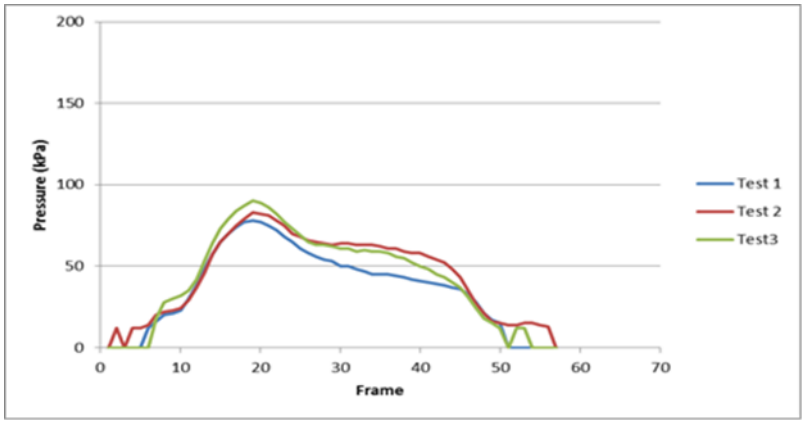


Figure 4-82; Subject B wearing the Airstep™ with added Air. Contact pressures under the Heel

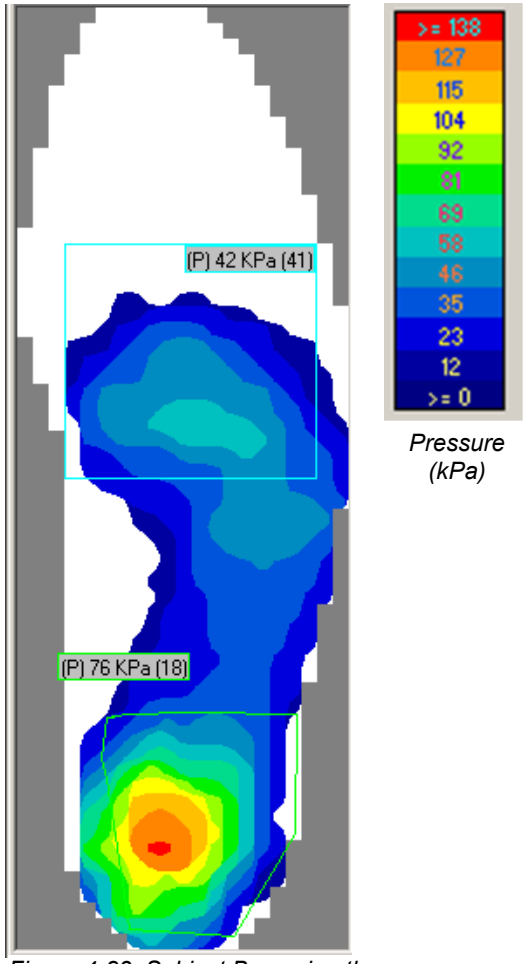


Figure 4-83; Subject B wearing the Airstep™ with added Air. Footprint of Contact Pressure Distribution across Plantar Surface

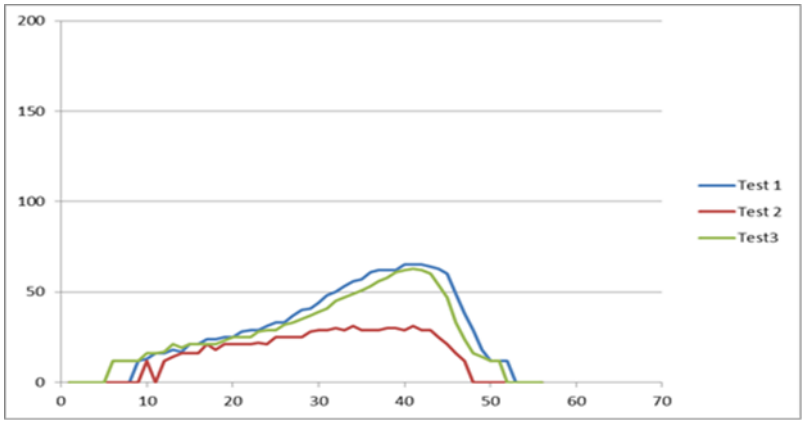


Figure 4-84; Subject B wearing the Airstep™ with added Air. Peak Contact Pressures under the Metatarsal Heads

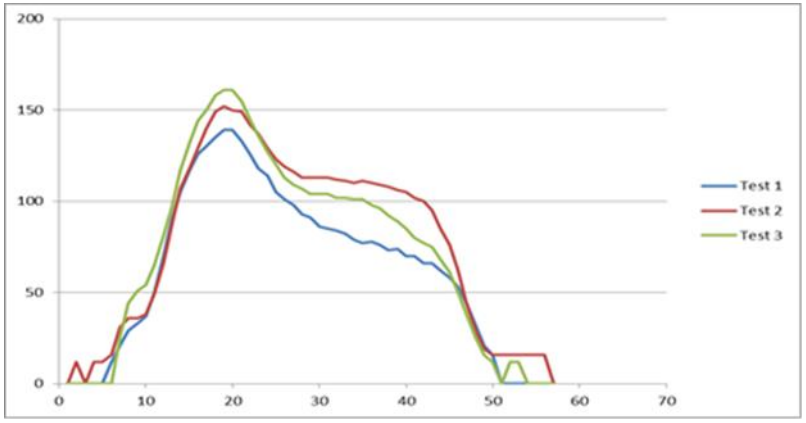
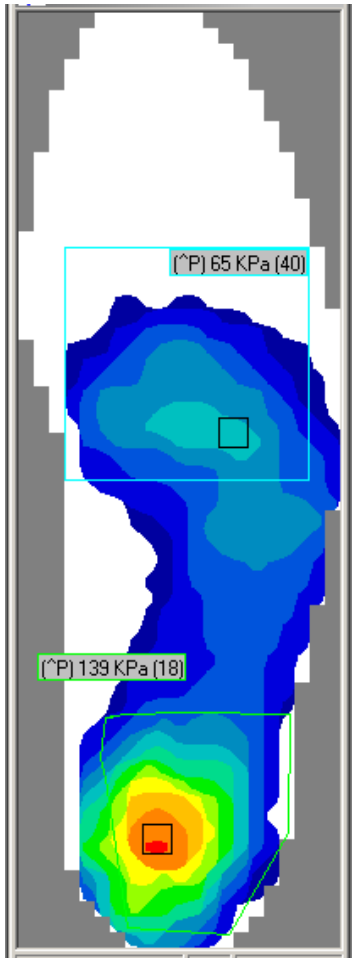


Figure 4-85; Subject B wearing the Airstep™ with added Air. Peak Contact Pressures under the Heel



Pressure (kPa)

Figure 4-86; Subject B wearing the Airstep™ with added Air. Footprint of Peak Contact Pressure Distribution across Plantar Surface

The highest pressures from each test are recorded numerically with the frame taken as reference to the position in the gait cycle. A low frame (around 12-30) is at heel strike and a high frame (around 35-45) is at toe off.

Aircast™		Contact Pressure (kPa)		Peak Contact Pressure (kPa)	
		Metatarsal Head	Heel	Metatarsal Head	Heel
No Air	1	37.8 at Frame 44	76.8 at Frame 17	68 at Frame 42	149.1 at Frame 19
	2	31.1 at Frame 35	73.6 at Frame 13	49.7 at Frame 35	141.2 at Frame 14
	3	21.0 at Frame 36	78.9 at Frame 16	28.8 at Frame 37	155.6 at Frame 17
Air 30mmHg	1	0	95.1 at Frame 33	0	189.6 at Frame 33
	2	19.5 at Frame 37	67.1 at Frame 20	26.2 at Frame 39	125.5 at Frame 19
	3	15.7 at Frame 38	83.0 at Frame 24	15.7 at Frame 38	162.1 at Frame 22
Air 40mmHg	1	0	76.9 at Frame 27	0	150.4 at Frame 27
	2	18 at Frame 35	67.7 at Frame 17	20.9 at Frame 33	126.8 at Frame 16
	3	0	84.1 at Frame 17	0	159.5 at Frame 17
Air High	1	0	75.7 at Frame 27	0	146.4 at Frame 28
	2	0	90.0 at Frame 29	0	180.4 at Frame 27
	3	16.7 at Frame 41	77.1 at Frame 24	20.9 at Frame 41	143.8 at Frame 24

Airstep™		Contact Pressure (kPa)		Peak Contact Pressure (kPa)	
		Metatarsal Head	Heel	Metatarsal Head	Heel
No Air	1	25.5 at Frame 38	53.5 at Frame 17	36.6 at Frame 36	99.4 at Frame 17
	2	16.4 at Frame 46	59.7 at Frame 24	20.9 at Frame 43	113.8 at Frame 23
	3	0	64.7 at Frame 26	0	117.7 at Frame 21
Air	1	0	63.5 at Frame 25	0	108.5 at Frame 21
	2	0	72 at Frame 38	0	134.7 at Frame 27
	3	0	60.4 at Frame 23	0	111.1 at Frame 22

Normal Shoe		Contact Pressure (kPa)		Peak Contact Pressure (kPa)	
		Metatarsal Head	Heel	Metatarsal Head	Heel
	1	52.2 at Frame 41	65.4 at Frame18	92.8 at Frame 42	117.7 at Frame 17
	2	38.4 at Frame 38	68.1 at Frame 17	68 at Frame 39	122.9 at Frame 16
	3	41.2 at Frame35	73.3 at Frame 14	70.6 at Frame 37	132.1 at Frame 14

Table 4-1; Numerical Results from Subject A

T-tests were performed for each trail to determine if there was a significant difference in the reduction of plantar pressure from the normal gait plantar pressure, when the air pressure of the cast is changed.

For all analyses the significance (alpha) level was 0.05.

For the Aircast™: The mean of contact pressure under the metatarsal heads for no air, 30mmHg and 40mmHg were not significantly different contact pressure in normal gait. Whereas the mean of contact pressure under the metatarsal heads for high air pressure within the bladders, is significantly different from normal gait, p=0.042. None of the results for under the heel were significantly different from the mean contact pressure of normal gait.

The mean of the peak contact pressure under the metatarsal heads for no air and 30mmHg were not significantly different from normal gait. Whereas the mean of the peak contact pressure under the metatarsal heads for 40mmHG, and high air pressure in the bladders is significantly different from normal gait, p=0.034 and p=0.03 respectively. The mean of the peak contact pressures under the heel with no added air in the bladders, is significantly different, p=0.023. None of the peak contact pressures under the heel with 30mmHG, 40mmHg, and high added to the bladders, are significantly different from normal gait.

For the Airstep™: The mean of the contact pressure under the metatarsal heads, with no added air and with added air are significantly different from the mean of normal gait, with p=0.035 and p=0.009 respectively. The mean of the contact pressure under the heel, with no added air was significantly different from the mean of normal gait, p=0.02, whereas under the heel with added air was not significantly different from normal gait.

The mean of the peak contact pressures under the metatarsal heads with no added air and added air are significantly different from the mean of normal gait, with p=0.014 and p=0.010 respectively. The mean of the peak contact pressure

under the heel with no added air was significantly different from the mean of normal gait, p=0.04, whereas the mean of the peak contact pressures under the heel with added air was not significantly different from normal gait.

Aircast™		Contact Pressure (kPa)		Peak Contact Pressure (kPa)	
		Metatarsal Head	Heel	Metatarsal Head	Heel
No Air	1	77 at Frame 48	107 at Frame 17	125 at Frame 48	185 at Frame 16
	2	70 at Frame 43	108 at Frame 16	115 at Frame 44	188 at Frame 16
	3	76 at Frame 43	106 at Frame 15	120 at Frame 43	191 at Frame 15
Air 30mmHg	1	59 at Frame 39	92 at Frame 15	109 at Frame 43	177 at Frame 15
	2	56 at Frame 43	97 at Frame 16	89 at Frame 44	174 at Frame 15
	3	69 at Frame 43	100 at Frame 14	91 at Frame 40	159 at Frame 15
Air 40mmHg	1	51 at Frame 39	96 at Frame 17	72 at Frame 38	184 at Frame 17
	2	54 at Frame 42	108 at Frame 16	80 at Frame 43	192 at Frame 16
	3	42 at Frame 38	104 at Frame 17	79 at Frame 39	166 at Frame 16
Air High	1	55 at Frame 42	76 at Frame 20	83 at Frame 41	124 at Frame 19
	2	64 at Frame 45	75 at Frame 20	98 at Frame 44	123 at Frame 18
	3	55 at Frame 43	79 at Frame 18	84 at Frame 40	127 at Frame 18

Airstep™		Contact Pressure (kPa)		Contact Pressure (kPa)	
		Metatarsal Head	Heel	Metatarsal Head	Heel
No Air	1	38 at Frame 41	79 at Frame 18	53 at Frame 40	135 at Frame 17
	2	39 at Frame 39	79 at Frame 18	58 at Frame 40	136 at Frame 18
	3	33 at Frame 40	88 at Frame 17	52 at Frame 38	152 at Frame 16
Air	1	42 at Frame 41	78 at Frame 18	63 at Frame 41	161 at Frame 18
	2	22 at Frame 39	83 at Frame 18	31 at Frame 34	152 at Frame 18
	3	39 at Frame 41	90 at Frame 18	65 at Frame 40	139 at Frame 18

Normal Shoe		Contact Pressure (kPa)		Peak Contact Pressure (kPa)	
		Metatarsal Head	Heel	Metatarsal Head	Heel
	1	78 at Frame 41	100 at Frame 17	153 at Frame 28	176 at Frame 16
	2	81 at Frame 38	90 at Frame 15	145 at Frame 38	161 at Frame 14
	3	79 at Frame 42	89 at Frame 16	186 at Frame 41	156 at Frame 16

Table 4-2; Numerical Results from Subject B

More T-test were conducted on the results for subject B.

For the Aircast™: The mean of the contact pressure under the metatarsal heads when no air and 30mmHg are added to the bladders, were not significantly different from the mean of normal gait. Whereas the mean of the contact pressures under the metatarsal heads for added 40mmHg and high air are significantly different from the mean of normal gait, p=0.012 and p=0.010. None of results for contact pressure under the heel were significantly different from the mean of normal gait.

The mean of the peak contact pressures under the metatarsal heads when no air and 30mmHg are added to the bladders are not significantly different from the mean of normal gait. Whereas the mean of the peak contact pressures under the metatarsal heads when 40mmHG, and high air are added, are significantly different from the mean of normal gait, p=0.020 and p=0.045 respectively. For the mean of the peak contact pressures under the heel with no added air, 30mmHg and 40mmHg in the bladders, there was no significant difference. But for the peak contact pressures under the heel with high added air in the bladders, there was a significant difference, p=0.027, from normal gait.

For the Airstep™: The mean of the contact pressures under the metatarsal heads with no air pressure and air pressure are significantly different from the mean of normal gait, with p=0.002 and p=0.024 respectively. None of results for contact pressure under the heel were significantly different from the mean of normal gait.

The mean of the peak contact pressures under the metatarsal heads with no air pressure and air pressure, are significantly different from the mean of normal gait, with p=0.017 and p=0.007 respectively. The mean of the peak contact pressures under the heel, with no air in the bladders, was not significantly different from normal gait. Whereas the mean of the peak contact pressures under the heel with added air in the bladders was significantly different from the mean of normal gait, p=0.03.

The graphs below compare the mean of the peak contact pressures for each subject, in each trial. They visually show the vast difference under the metatarsal heads, while under the heel the peak contact pressures are more similar.

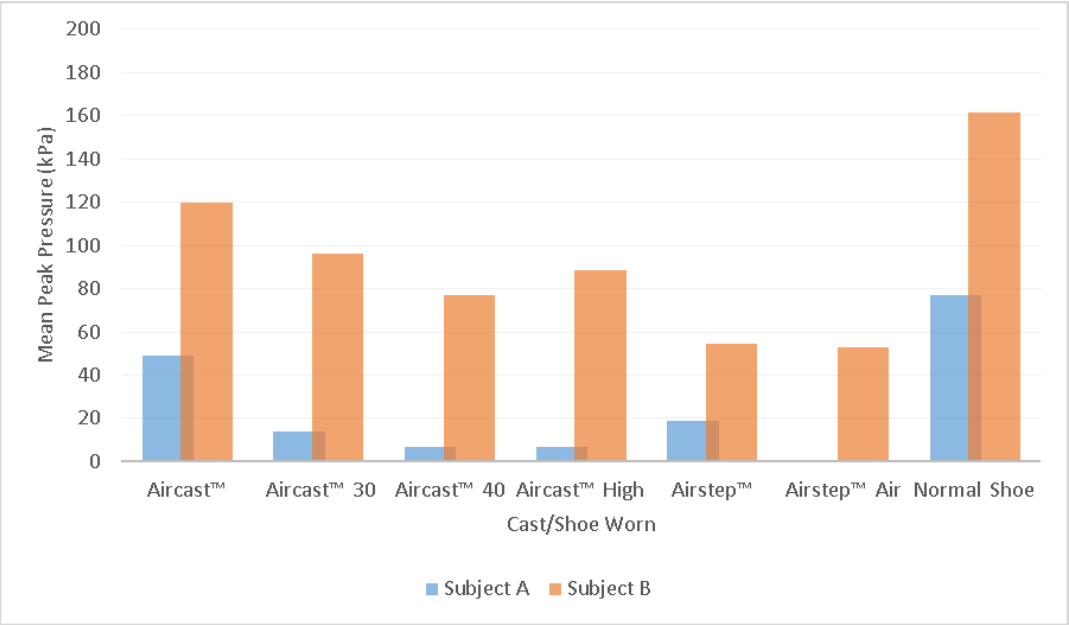


Figure 4-87; Mean of the Peak Pressures under the Metatarsal Heads

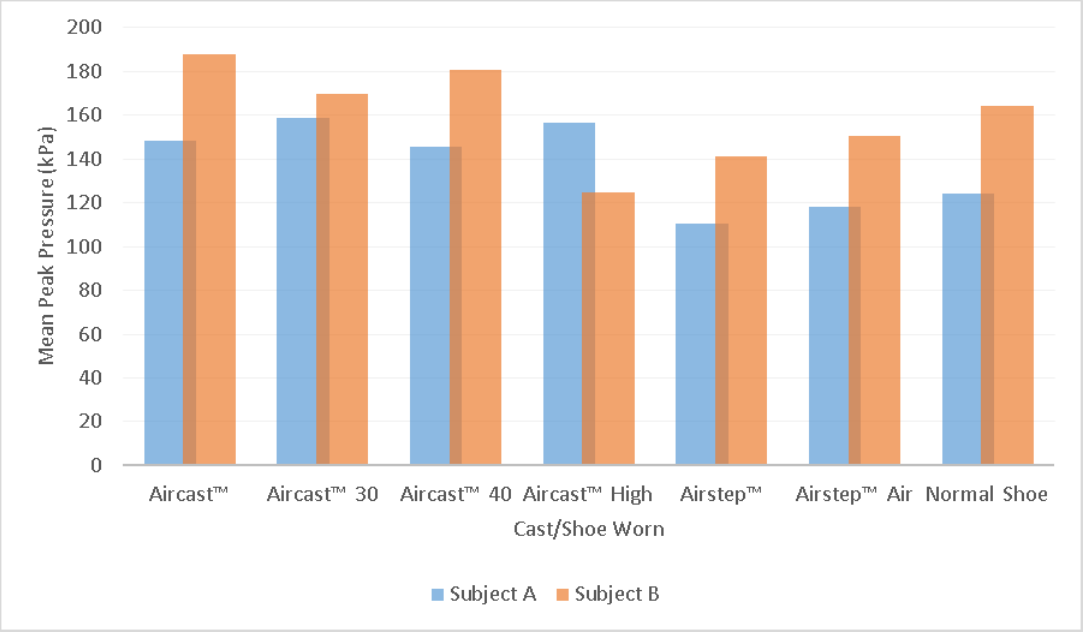


Figure 4-88; Mean of the Peak Pressures under the Heel

The table below normalises the data, in order to visually display the difference in plantar pressure between each casts trial and the plantar pressure during the subjects' normal gait. The data in red, is when the cast increases pressure on the plantar foot rather than offloading the pressure.

		Subject A		Subject B	
Cast	**Pressure (kPa)**	**Metatarsal Head**	**Heel**	**Metatarsal Head**	**Heel**
Aircast™– No Air	Contact Pressure	-31.7%	+10.8%	-6.3%	+15%
	Peak Contact Pressure	-36.7%	+19.6%	-25.6%	+14.4%
Aircast™– 30mmHG	Contact Pressure	-73.3%	+18.5%	-22.6%	+3.5%
	Peak Contact Pressure	-81.9%	+27.9%	-40.3%	+3.4%
Aircast™– 40mmHG	Contact Pressure	-86.3%	+10.6%	-38.2%	+9.7%
	Peak Contact Pressure	-90.9%	+17%	-52.3%	+9.9%
Aircast™– HighAir	Contact Pressure	-87.1%	+17.4%	-26.9%	-17.5%
	Peak Contact Pressure	-90.9%	+26.2%	-45.2%	-24.1%
Airstep™–No Air	Contact Pressure	-68.2%	-14%	-53.8%	-11.8%
	Peak Contact Pressure	-75.1%	-11.2%	-66.3%	-14.2%
Airstep™- Air	Contact Pressure	-100%	-5.3%	-56.7%	-10%
	Peak Contact Pressure	-100%	-4.9%	-67.1%	-8.3%

Table 4-3; The percentage plantar pressure difference in each walker from the plantar pressure within the normal shoe

5 Discussion

This thesis investigated the plantar pressure distribution during gait, influenced by two different offloading casts, compared to each other as well as to the normal plantar pressure distribution. The results show clearly that the new Airstep™ offloads more plantar pressure.

While the pressure offloaded for subject A was much greater than subject B, the age and weight of the subjects should be considered; subject A was older and lighter than subject B. While the difference in weight defiantly accounts for the difference in peak plantar pressures, it does not explain the dissimilarity in the subjects' percentage offloaded from normal gait. Subject A, who is older, walked at a much lower speed than subject B. This fact, combined with subject A's far lower normal plantar pressure, might explain why an increase of pressure in a casts bladder resulted in exponential increases in offloading capabilities for this subject. This is unlike the results observed for subject B, when pressure offloaded in a more gradual fashion. Because at an assumed normal walking speed of just over 1m/s, a normal gait pattern has the two distinctive peaks of a gait diagram or Perdotti diagram, see Figure 2-4. However when walking speed is slowed, these peaks become flatter. Rosenbaum, et al., in (1994) demonstrated that reduced walking speed reduced peak plantar pressures over the entire plantar surface of the foot, with 11% reduced under the metatarsal heads (van Deursen 2004).

5.1 Aircast™

When the Aircast™ pressure distribution for subject B is observed, it is in accordance with predictions to see that the metatarsal heads are increasingly offloaded with the increased amount of air pumped into the cast. Yet it appears that the pressurised air does not successfully re-distributing the pressure up the calf, but merely onto the heel of the foot, increasing rather than decreasing peak pressures on the heel. This cast could therefore be especially dangerous to any diabetic with a history of ulcers on the whole plantar surface of the foot.

Additionally any diabetic without a history of heel ulcers should still be made aware of this and they should be encouraged to examine the heel of their foot regularly. For subject B, it was not until an uncomfortably high air pressure was created in the Aircast™ that the plantar pressure reduced over all areas of the plantar surface, including the heel. However, this reduction in heel plantar pressure corresponded with a reduction in the pressure offloaded at the forefoot; which is thought to transpire because of a pressure distribution threshold of the cast, preventing greater simultaneous offloading.

Subject A produced very similar results in the Aircast™; as overall there continues to be increased forefoot offloading as the bladders air pressure is increased, all the while, the heel is placed under increasing plantar pressure. Unlike subject B however, subject A never achieved in offloading the heel, despite being closer to complete offload of the forefoot.

5.2 Airstep™

When the subjects walked with the Airstep™, their metatarsal heads were significantly more offloaded than with the Aircast™. For subject A, this resulted in the metatarsal heads completely offloading with added air pressure. While the plantar heel was also offloaded with the Airstep™, the pressure offloaded actually decreased when air pressure was added to the bladders. This may be due to the increased offloading of the metatarsal heads, with some of that pressure redistributed to the heel.

When subject B walked with the Airstep™, the forefoot offloading capabilities were higher than with any of the Aircast™ results. While the heel did offload regardless of whether or not air was added, the results in this plantar region could not reach the peak offloading that the Aircast™ achieved with an uncomfortably high air pressure. Additionally, it should be noted that the results for the Airstep™ with and without added air pressure were not remotely different.

5.3 Variations in Design

As there does not seem to be significant difference between the Airstep™ without air and the Airstep™ with air, it draws the question, why is the Airstep™ offloading more that the Aircast™?

This is when the ground force reaction of the two casts was investigated. In order to help clear up the question into whether it was the outer design or the inner padding of the cast which allowed for the Airstep™ to offload more plantar pressure. Unfortunately analysis of pedotti diagrams did not yield any plausible explanations.

When considering what might make one cast offload better than the other, the differences of the cast should be noted.

The Aircast™ is a slightly taller cast, with thinner padding around the top of the cast, with air bladders run the full height of the cast, either side of the leg, and along the back of the leg;. There is also an air bladder on the underside of the shin guard and tongue, which is not refillable. Additionally there is no additional padding inside the cast. The sole measures 280mm in length and 70mm in thickness. The sole has thin padding that wraps around the forefoot and encloses the toes within the cast. The sole of the cast is a constant curve, making for a circular rocker.

The Airstep™ on the other hand is a slightly shorter cast, it has thick padding all around the leg and foot, on the inside of the cast. The air bladders within the cast are only 150mm in height, on each side of the ankle, with an additional non-refillable air bag, on the underside of the skin guard and tongue. The length of the sole is 225mm, shorter than the Aircast™, with a 70mm thick sole, identical to the Aircast™, but with an additional 12mm of memory foam padding on top of the sole. The forefoot is wrapped in the same thick padding, with no air bladders and with an open toe. There is a toe guard to protect from

stubbed toes. The actual sole of the Airstep™ is far flatter than the Aircast™, producing an entirely different rocking motion.

Superficial elements for the Airstep™ to improve on are the straps used to tighten the cast. These have only one rung to hold them and therefore possess little security in the instance of the Velcro accidently unstrapping. An alternative fastening, with an additional rung could make use of friction crated there to serve as extra security. The air pump does not include a pressure gauge, therefore it does not give any indication on how high the air pressure is within the bladders. This should be rectified immediately, or patients will not be able to confidently pump up the casts to the correct pressure. The patients may be concerned that the cast is too tight, when in fact the pressure is insufficient to achieve the desired results. Alternatively, patients may pump to an overly high pressure such that they infringe on the blood flow to their feet. Reduced blood flow is an existing problem for many diabetics, as discussed in Section 2.2.

Both subjects found the Airstep™ easier to walk in than the Aircast™. This is of particular importance if prescribing an offloading cast to an elderly, or otherwise partially disabled, patient. As they require a device with greater stability, allowing them to feel more confident in walking, permitting for better quality of daily life.

From the substantial quantity of papers that report on the effectiveness of difference offloading devices, and on the differences between casts, very few have quantified their pressure reductions. A review conducted by Bus et, al., in 2008 identify 1608 papers on offloading devices, they narrowed their review down to 160 papers, of which they reported only 5 papers which gave quantified measurements of plantar pressure reductions.

In a study by Lavery, et al., in 1997, they looked into the off-loaded pressure using total contact casts with diabetic patient’s suffering from ulcers on the balls of their feet. They conducted this study using the Novel Pedar in-shoe pressure measuring system, which they reported does not interfere with

normal gait. When comparing the Novel Pedar system to the Tekscan™ system used in this study, there are some obvious differences. One notable difference is their size, since the Novel system is far thicker. When looking at the results from Lavery, et al., it appears possible that the novel pedar system while not interfering with normal gait, might interfere with the off-loading pressure. It is a fundamental principle of off-loading casts that, the more contact the foot has with the cast the more the pressure is distributed. This relationship might be strengthened by using a cushioned shoe and the thicker Novel Pedar system may contribute to this cushioning effect. However, the Novel Pedar system also uses capacitance sensors, which are seen to be more reliable/exact than the force resistor sensors used in the Tekscan™ system. Lavery, et al., report mean peak pressures under the 1st metatarsal, 2nd-5th metatarsal, and under the great toe when using a total contact cast and a therapeutic shoe. When the patients wear a total contact cast, the mean peak pressure under the 1st metatarsal hover around 60kPa, under the 2nd – 5th metatarsal the mean peak pressure ranges from 50-85kPa and under the great toe at 35kPa (unless it is the site of an ulcer, then it increases to 50kPa). These results are similar to the results obtained in this study with the Aircast™ and Airstep™, validating the use of the Tekscan™ system.

Armstrong, et al., (1998) investigated using peak pressure as a screening mechanism for the potential development of plantar users, using an Ermed SF pressure platform. Therefore, very different results were found compared to those obtained using an in-shoe pressure sensor system. They reported results around 627kPa when their patient had an ulcer, and 831kPa without. These are extremely different from the results in this study but also very difficult to compare, with the use of a pressure platform, instead of an in-shoe system.

Fleischil, et al., (1997) explored different strategies into reducing pressure at the site of ulcers on diabetic patients. They examined a removable cast walker, a total contact cast, a half-shoe and accommodative dressing. Their resounding conclusion reached was that the removable walker and the total

contact cast offload the most pressure, as also concluded by subsequent studies. However, they also state that the removable walker seems the logical solution, as the total contact cast has too many contraindications and disadvantages. While they draw similar conclusions to those drawn here, their results are only taken for forefoot or great-toe ulcers and do not take into consideration the simultaneous peak pressures at the heel. Despite this, their forefoot pressure offloading results are fairly comparable to the results obtained here. They report mean peak pressures of 77kPa with the removable walker and 124kPa with the total contact cast (Fleischli et al. 1997).

Conversely to the study above, Armstrong & Stacpoole-Shea (1999) only explored the results on the plantar heel, when offloading the foot. Here they summarise that a well confined heel will offload more of the planter heel than a cast without lateral or medial support. Without such support, the heels loading time decreases with surface of the heel increasing, this alters the pressure-time integral, leading to lesser offloading abilities. Their results indicate a peak pressure of 180kPa with a total contact cast and 200kPa with an Aircast™ Pneumatic Walker, both of which confined the heel. These values are slightly higher than those obtained in this thesis.

As useful as this thesis is regarding which cast may offload the most pressure, without a threshold value to compare to, the potential use for real ulcerated patients is not known. While a few studies have touched upon this topic, non have given a satisfactory solution. While a few report on a threshold to ulceration, this implies the pressure the plantar foot will exceed in order to develop an ulcer. We now know this threshold would be useful in only a few cases, since a neuropathic diabetic can develop an ulcer even under very normal plantar pressures, by a case of repetitive stress. What is useful for the treatment of an ulcer is the threshold of pressure required in order to sufficiently offload an ulcer for it to heal. A study by Owings (2009) stated a provisional pressure threshold of 200kPa. This is a result of their findings into peak pressures on patients with healed plantar ulcerations. They state that

while plantar ulcerations remain healed as patients continually walk with peak pressures of 200kPa, this must be a safe stress level. While our study found the peak pressure in a normal gait do not exceed 200kPa, but instead average around 150kPa. This provisional threshold is very conservative, but until further evidence is available, it may serve as a useful initial value.

6 Conclusions

In conclusion, this pilot study presents evidence pointing to the Airstep™ from Promedics Orthopaedics ltd as a superior offloading walker compared to the Aircast™ for diabetic ulcers. The Airstep™ had greater reduced peak pressures for both subjects compared to the Aircast™, with additional reduced contact pressures. The Airstep™ succeeded in a complete offload of the forefoot on occasion, indicating superior reduction in pressure. With this greater reduction in pressure, it can be assumed the healing rate of ulcers could be increased within the Airstep™ walker. Clinical studies are therefore advised. After a preliminary look, some minor adjustments need to be considered in order to optimise the cast. New straps and a pressure gauge first, with subsequent adjustments into the type of bladders used. Further study needs to take place to confirm this finding and investigation into the reasons behind the success. This would allow for future casts to increase in offloading ability, together with increased care and comfort for the patient.

7 Further Works

Time constraints prevented investigation into the optimum size of the air bladders within the Airstep™ and the medium of which they were filled. Further works should investigate these design variables.

As air is a compressible substance, there is reason to believe that water is a more suitable medium. As diabetics are likely to suffer from PVD, a compressible substance might be dangerous. This is why, prior to time constraints being met, another test of the pressure at the calf was planned. In such an investigation, Tekscan™ pressure sensors should be attached around the calf of the subject, and the tests used here should be performed.

Shear stresses result from forces acting parallel to the foot, and are very difficult to investigate in a removable cast walker. A few studies have designed different methods, if the Airstep™ walker was to be properly optimised, it is necessary to investigate the amount of shear stress produced.

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