THE EVALUATION OF ULTRASONOGRAPHIC FINDINGS IN THE MANAGEMENT OF PLANTAR FASCIITIS IN RUNNERS AND THE ASSOCIATION WITH CLINICAL FINDINGS

By

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Dissertation submitted in partial compliance with the requirements for the Master's Degree in Technology: Chiropractic in the Faculty of Health, at the Durban Institute of Technology.

I, Shoshanna Dunn, do hereby declare that this dissertation represents my own work in both conception and execution.

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Dedication

To Mom and Dad, thank you.
Acknowledgments

I would like to thank the following people for their contribution:

Dr Charmaine Korporaal for her supervision of this study, and her dedication to chiropractic and her students, your hard work is greatly appreciated.

Dr Petrie de Villiers for his time, generosity and knowledge.

Simiso Skosana, for his time and hard work, and the Radiography Department of DIT.

Ms M. Terry Carey-Loghmani for reviewing this dissertation.

Sally Stewart for her help in editing this dissertation.

Kirsten Maartens, my research partner, for her support and encouragement.

To all the patients who took part in the study, without whom this would not be possible.

To my class, for your friendship and the good times, thank you.
Abstract

Plantar Fasciitis (PF), also called ‘the painful heel syndrome’, is a common cause of heel pain (Barrett and O’Malley, 1999:2200), accounting for between 7% and 10% of all running injuries (Batt and Tanji, 1995:77; Chandler and Kibler, 1993:345). Primarily an overuse injury, resulting from tensile overload, it involves inflammation and micro-tears of the plantar fascia at its insertion on the calcaneus (Lillegard and Rucker, 1993:168; Barrett and O’Malley, 1999:2200). The body’s attempt to heal these micro-tears leads to chronic inflammation and the formation of adhesions (Ambrosius and Kondracki, 1992:30).

Transverse friction massage has been found to be beneficial in the treatment of PF (Hyde and Gegenbach, 1997:478,481; Hertling and Kessler, 1996:137). Cyriax (1984) and Prentice (1994) state the effect of frictions to include the breakdown of adhesions (scar tissue), as well as preventing the formation of further adhesions.

Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM), based on the principles of frictions, aims to break down adhesions, realign collagen fibres and aid in the completion of the inflammatory process (Carey-Loghmani, 2003:31, 51-62; Hammer, 2001). Enabling us to see changes on ultrasonography, which has been found to be an objective, non-invasive way of evaluating PF (Wall and Harkness, 1993:468; Tsai et al, 2000:259; Cardinal et al, 1996:258). These changes include decreased thickness of the fascia.

The aim of the study was to see the effect of GISTM on PF in runners, in terms of ultrasonography, and identify any correlation between these findings and other objective and subjective findings.

Ultrasonography was used to evaluate changes in the plantar fascia. The algometer and weight-bearing ankle dorsiflexion made up the remainder of the objective measurements. Subjective measurements were the Foot Function Index (FFI) and the Numerical Rating Scale – 101 (NRS-101).
The study consisted of a total of 30 subjects diagnosed with PF. Participants were runners between the ages of 25 to 50, and were required to run a minimum of five kilometres a week.

There were a total of ten treatments over a period of eight weeks. Subjects underwent a clinical assessment (objective and subjective findings) and an ultrasound evaluation in week one. The second and third clinical assessment and ultrasound evaluation took place in week five and week eight.

Data was captured in MS Excel and exported to SPSS version 11.5 (SPSS Inc., Chicago, Ill, USA) for analysis. Demographic variables were compared between the two treatment groups using independent chi square tests, Fisher’s exact tests or independent t-tests where appropriate. Repeated measures ANOVA was used to assess factors associated with the change over time in outcome measurements. Spearman’s correlation was used to examine the relationships between the changes in the various measurements intra-group.

This study showed evidence for a beneficial effect of the GISTM on the plantar fascia, but only demonstrated on ultrasonography. GISTM was effective in decreasing the thickness of the plantar fascia, indicating scar tissue reduction. The other outcomes measured were not significantly affected by the technique, which showed no benefit over and above that of the placebo.

There was no correlation between ultrasonography and the other objective or subjective outcomes. However, correlations between NRS-101, FFI-1 (worst pain), FFI-2 (morning pain) and FFI-11 (pain getting up from a chair) were found.

In conclusion, Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM) was found to be beneficial in certain aspects of the treatment of PF in runners.
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List of Abbreviations

PF: Plantar fasciitis

GISTM: Graston Technique Instrument-assisted Soft Tissue Mobilisation

NRS-101: Numerical Rating Scale – 101

FFI: Foot Function Index

Definition of Terms

Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM)

An advanced form of soft tissue mobilisation that is used to detect and release scar tissue, adhesions and fascial restrictions (Carey-Loghmani, 2003:7).

Ultrasonography

The imaging of deep structures of the body by recording the echoes of pulses of ultrasonic waves directed into the tissues and reflected by tissue planes where there is a change in density (Dorland’s Medical Dictionary, 1995).
Chapter One

Introduction

1.1) Introduction

Plantar Fasciitis (PF) is a relatively common condition that occurs in both athletes and non-athletes (Batt and Tanji, 1995:77). It has been classified as a syndrome resulting from overload of the plantar fascia at its insertion into the calcaneus (Kibler et al, 1991:66). The patient complains of a sharp pain of insidious onset with maximal tenderness at the anterior medial border of the calcaneus (Batt and Tanji, 1995:77). The pain is typically worst on the first few steps in the morning (Brantingham et al, 1992:76).

One of the findings seen on ultrasonography of the plantar fascia is thickening of the fascia, this is due to the reactive fibrosis and angiofibroblastic proliferation that results when the body attempts to heal itself following the repetitive micro-trauma (Tsai et al, 2000:259; Pollard and So, 1999:95; Ambrosius and Kondracki, 1992:30).

However, to date no correlation exists between the changes seen on ultrasonography and the clinical findings. This research was aimed at investigating the changes in the plantar fascia seen on ultrasonography, following treatment with Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM). Once these changes were identified, the correlation between these changes and the objective and subjective clinical findings was determined.

1.2) Aims and Objectives

This study was a pre-post clinical investigation assessing the effect of GISTM on PF in runners and determining the correlation between ultrasonography and clinical outcomes.
1.2.1) The first objective
The first objective was to evaluate the ultrasonography findings, pre and post GISTM treatment, in the management of plantar fasciitis in runners.

1.2.2) The second objective
The second objective was to determine the effect of GISTM in the treatment of PF in terms of subjective and objective clinical findings.

1.2.3) The third objective
The third objective was to correlate the ultrasonography findings with the subjective and objective clinical findings.

1.3) Hypothesis

1.3.1) The first hypothesis
The first hypothesis was that GISTM would result in a decrease in the thickness of the plantar fascia, seen on the ultrasonography findings of the plantar fascia post treatment.

1.3.2) The second hypothesis
The second hypothesis was that GISTM would be effective in the treatment of PF in terms of subjective and objective clinical findings.

1.3.3) The third hypothesis
The third hypothesis was that the ultrasonography findings would correlate with the subjective and objective clinical findings.

1.4) Rationale

Ultrasonography has been found to be an objective, non-invasive way of evaluating PF (Wall and Harkness, 1993:468; Tsai et al, 2000:259; Cardinal et al, 1996:258). It can therefore be used to determine the effectiveness of treatment and could assist in the assessment of the effect of GISTM on PF.
These findings could then be compared to the objective and subjective data to determine whether the subjective and objective measurements could be accurately used by practitioners as a reliable indicator of the progress/effectiveness of the treatment.

An association between a decrease in scar tissue and the clinical presentation would provide a guideline for use in practice, to indicate what the degree of progress should be in terms of healing and scar tissue breakdown.
Chapter Two

Literature Review

2.1) Introduction

This chapter follows a detailed discussion with regards to the condition of PF, including relevant anatomy and biomechanics and the most frequent forms of treatment.

2.2) Definition

Plantar Fasciitis (PF), also called ‘the painful heel syndrome’, is a common cause of heel pain (Barrett and O’Malley, 1999:2200). Primarily an overuse injury, resulting from tensile overload, it involves inflammation of the plantar fascia at its insertion on the calcaneus (Lillegard and Rucker, 1993:168; Barrett and O’Malley, 1999:2200).

As a repetitive micro-trauma overload injury it is commonly associated with running sports (Krickvikas, 1997:141; Nicholas and Hershman, 1995:451; Chandler and Kibler, 1993:345). However it can be a cause of heel pain in both athletes and non-athletes (Gill and Kiebzack, 1996:527; Batt and Tanji, 1995:77).

PF most characteristically presents as localised plantar heel pain, typically on the first few steps in the morning (Batt and Tanji, 1995:77; Brantingham et al, 1992:76).

2.3) Incidence, Prevalence and Epidemiology

PF accounts for between 7% and 10% of all running injuries (Batt and Tanji, 1995:77; Chandler and Kibler, 1993:345). It also associated with sports requiring a ‘push-off’ motion: for example tennis, hockey and soccer (Batt and Tanji, 1995:77).
PF occurs in both males and females, with Barrett and O’Malley (1999) and Brown (1996) suggesting a slight predominance in females. An association with obesity has however also been noted (Brown, 1996:877; Wolgin et al, 1994:100), which is supported by Barrett and O’Malley (1999) who found a correlation between obesity and the incidence and severity of PF.

Brown (1996) describes two distinct groups of people who are most likely to present with this type of heel pain, each group with a different mechanism of injury. The first group consists of the athlete with an overuse type injury, especially common in runners.

The second group, as stated by Brown (1996), is an older age group. Here the problem is more biomechanical in nature, with the majority of PF patients falling into this group. He states most commonly between the ages of 40 and 60 years. Young et al (2001) and Reid (1992) concur with this older age group of over 40 years, while Ambrosius and Kondracki, (1992) state that persons over 45 years of age fall into this category. In this group Brown (1996) also notes a higher incidence in women and an association with obesity and prolonged periods of standing or walking.

2.4) Anatomy

The plantar fascia, also known as the plantar aponeurosis, is a multilayered fibrous aponeurosis (Kwong et al, 1998:118; Young et al, 2001:467, Batt and Tanji, 1995:79). It is part of the deep fascia of the foot, consisting of strong sheets of dense connective tissue (Ambrosius and Kondracki, 1992:30; Polkinghorn, 1999:45).

The plantar fascia is composed of three segments, namely central, medial and lateral, of which the central is thickest. Clinically, when referring to the plantar fascia it is generally the central portion that is considered. Proximally, at its insertion, the fibres are longitudinally arranged. Here it is thickest but at it’s most narrow, the fascia then thins and fans out distally (Brown, 1996:877; Batt and Tanji, 1995:79).
Originating from the medial calcaneal tubercle, the central portion then divides distally to form five tracts, inserting onto the plantar plates of the metatarsophalangeal joints and the proximal phalanges (Delee and Drez, 1994:1817; Kwong et al, 1988:118; Brown, 1996:877). These distal tracts have a superficial and a deep component. The superficial portion anchors the skin providing additional support. The deep component forms the attachment to the phalanges and helps maintain the flexor tendons under the metatarsal heads (Kwong et al, 1988:118; Batt and Tanji, 1995:79).

The medial and lateral portions of the plantar fascia are smaller and less significant. The lateral segment arises from the lateral margin of the medial calcaneal tubercle and inserts into the base of the fifth metatarsal. The medial segment is thin and forms the fascial covering of abductor hallucis (Kwong et al, 1988:118; Delee and Drez, 1994:1818).

2.5) **Biomechanics**

The plantar fascia is involved in the normal biomechanics of the foot and ankle. It functions in the static stabilisation, support and maintenance of the medial longitudinal arch (Brown, 1996:876; Young et al, 2001:467), where it provides the highest contribution of arch stability, aided by the plantar and spring ligaments (Polkinghorn, 1999:45; Cheung et al, 2004). The fascia is also involved in dynamic shock absorption during weight-bearing, its design allowing it to absorb and direct forces during the gait cycle (Brown, 1996:876; Young et al, 2001:467; Reiner et al, 2002).

The anatomical attachments of the plantar fascia, extending between the calcaneus and the phalanges results in the fascia acting like a ‘bowstring’. By tensing as a bowstring it helps maintain the longitudinal arch (Batt and Tanji, 1995:79). Biomechanical alterations in the region of the foot and ankle can lead to increased tension in the plantar fascia resulting in stretching and tearing at its site of bony attachment and of the fascia itself (Polkinghorn, 1999:45; Arnheim and Prentice, 1993:480; Garrick and Webb, 1999:392;
Brown, 1996:876). When the plantar fascia becomes torn, inflamed or irritated, the condition is known as PF (Warren, 1990:339).

Hyperextension of the toes and metatarsophalangeal joints causes tension in the plantar fascia and raises the longitudinal arch, this process is involved in forward propulsion and is known as the ‘windlass mechanism’ (Delee and Drez, 1994:1818).

The fascia also gives firm attachment to the overlying skin, as well as protecting the underlying vessels, nerves and tendons (Ambrosius and Kondracki, 1992:30; Brown, 1996:876).

2.6) **Pathology**

PF is characterised by inflammation of the plantar fascia, progressing to collagen degeneration, at the origin of the fascia at the medial tubercle of the calcaneus (Wilk et al, 2000:27; Young et al, 2001:467). The cause of this inflammation is repetitive micro-trauma resulting from tensile overload at the fascial insertion, this leads to micro-tears in the fascia itself (Batt and Tanji, 1995:78; Kibler et al, 1991:66). The pain is secondary to these micro-tears and the inflammatory response (Lillegard and Rucker, 1993:168).

The lesion of PF is an enthesitis i.e. inflammation at the bone-ligament or in this case bone-fascial interface (Batt and Tanji, 1995:78). The body’s attempt to heal these micro-tears leads to chronic inflammation and the formation of adhesions (Ambrosius and Kondracki, 1992:30). Scar tissue contracts over time, this can lead to stiffness and decreased functional ability (Lachmann and Jenner, 1994:28; Batt and Tanji, 1995:78). The tightness of the plantar fascia may overstress the insertion sites at the calcaneus and lead to the development of focal heel pain (Cheung et al, 2004).

PF can present as an acute problem, where repetitive micro-trauma manifests as an acute inflammatory lesion with the presence of inflammatory cells. With persistent overuse it often develops into a protracted chronic lesion.
characterised by degenerative collagen changes, including loss of cell continuity, increased ground substance and fibroblasts (Batt and Tanji, 1995:78; Khan et al, 2000:39).

Chronic stress at the fascial insertion can lead to calcium deposition and the formation of a spur. These spurs are not the primary cause of the pain, they are as a result of the chronic inflammation and calcification rather than the cause of it (Batt and Tanji, 1995:79; Lillegard and Rucker, 1993:168; Nicholas and Hershman, 1995:453).

Histologically the plantar fascia consists of collagen fibres and fibroblasts arranged in orderly multiple layers. In response to the tensile overload and the insertion and the resultant micro-tears, new connective tissue is laid down (Warren, 1990:342).

2.7) **Aetiology**

Many different factors can be involved in the aetiology of PF. These can be broken down into three broad categories; environmental, biomechanical and anatomical as per Kibler et al (1991).

2.7.1) **Environmental**

Training errors in athletes associated with PF include:
- a sudden increase in volume
- increased hill running
- increased intensity
- inadequate time to recover between training
- increased frequency of workouts.

All these factors result in overuse and tensile overload at the fascial insertion, leading to inflammation and the development of PF (Warren, 1990:340; Batt and Tanji, 1995:78).
Poor footwear is also considered an important cause of PF in runners as explained by Warren (1990) and Ambrosius and Kondracki (1992), where worn out shoes result in poor shock absorption while inadequate arch support causes the arch to flatten. Excessively flexible shoes result in greater toe flexion and increased tension in the fascia. Rajput and Abboud (2004) believe that this factor is often overlooked as a possible contributory factor in the development and aggravation of the condition and has the potential to cause injury or worsen an already existing condition.

2.7.2) Biomechanical

Hyperpronation is widely stated to predispose to PF. Kwong et al (1998) showed that excessive or prolonged pronation leads to increased strain on the plantar fascia resulting in PF. A tight Achilles tendon and the associated limited ankle dorsiflexion can cause compensatory pronation therefore also predisposing to the condition (Krivickas, 1997:141; Batt and Tanji, 1995:78; Warren, 1990:341).

2.7.3) Anatomical

The pes cavus foot has a high rigid arch, being less flexible it is less able to absorb forces and adapt to the ground resulting in greater stress placed on the plantar fascia. Pes planus, also known as the flat foot, can be as a result of weakened ligaments in the arch, therefore also resulting on greater tensile stress on the plantar fascia. Leg length difference is another contributing anatomical factor as it results in pronation on the side of the short leg predisposing to PF (Krivickas, 1997:141; Batt and Tanji, 1995:78; Warren, 1990:341).

2.7.4) Other

PF has also been associated with systemic disorders, included are Reiter’s syndrome, ankylosing spondylitis, psoriatic arthritis, rheumatoid arthritis, gout and nutritional osteomalacia (Ambrosius and Kondracki, 1992:31-32; Hyde
and Gengenbach, 1997:478). There is also an association with obesity and prolonged periods of standing or walking (Brown, 1996:877).

2.8) **Clinical Presentation and Diagnosis**

Clinically PF presents as pain on the plantar surface of the heel, with maximal tenderness at the fascial insertion on the anterior medial border of the calcaneus. The pain is usually localised to the origin of the fascia, but may extend along the medial portion distally towards the metatarsals (Arnheim and Prentice, 1993:481; Krivickas, 1997:141).

Characteristically the patient complains of a sharp localised pain, which is described as knife-like in nature. It is commonly of insidious onset, and may initially be described as burning in nature (Chandler and Kibler, 1993:344; Batt and Tanji, 1995:78).

The pain is worst following prolonged rest, with maximal pain experienced on the first few steps in the morning. It is generally worse at the start of an activity and lessens as you warm up. This pattern of pain is related to the stiffness and contracture of the plantar fascia, which subsequently eases during activity (Batt and Tanji, 1995:78). Pain may also be experienced on prolonged standing (Young et al, 2001:467).

In general, PF is known to be a self-limiting condition (Young et al, 2001:468; Wolgin et al, 1994:100). Natural history dictating that, with time, injuries heal and strengthen (Lachmann and Jenner, 1994:28).

Young et al (2001) state that dorsiflexion of the toes and standing on tips of the toes may exacerbate the pain. PF has also been shown to limit range of motion in big toe and ankle dorsiflexion (Lillegard and Rucker, 1993:169).

The diagnosis of PF is based mainly on medical history and clinical presentation, combined with severe tenderness over the medial calcaneal tubercle on palpation (Barrett and O'Malley, 1999:2201). In recent years
ultrasound diagnosis of PF has been found to be a reliable method of confirming the diagnosis (Wall and Harkness, 1993:468; Tsai et al, 2000:259; Cardinal et al, 1996:258). Using ultrasonography, physicians can see if the fascia is torn or thickened (Cohen, 2004).

This thickening of the fascia is due to the reactive fibrosis and angio-fibroblastic proliferation that results when the body attempts to heal itself following the repetitive micro-trauma. Other findings include hypoechoic changes of the fascia, this refers to the increased blackness of the fascia seen on ultrasound and is as a result of the hypercellular inflammatory response (Tsai et al, 2000:259; Pollard and So, 1999:95; Ambrosius and Kondracki, 1992:30).

2.9) Differential Diagnosis

PF must be differentiated from other conditions causing heel pain. Young et al (2001) described the basic categories: soft tissue, skeletal and neurological causes of heel pain. PF has also been associated with certain systemic disorders (Ambrosius and Kondracki, 1992:31).

2.9.1) Soft Tissue

Rupture of the Plantar Fascia
Although uncommon, when it does occur, patients report a sudden severe pain usually following physical trauma and often accompanied by a popping sound. Ecchymosis over the area may also be noted. A diagnosis can be made on MRI (Barrett and O'Malley, 1999:2204; Young et al, 2001:468; Nicholas and Hershman, 1995:451).

Fat Pad Syndrome
Atrophy of the heel fat pad leading to decreased shock absorption can also result in pain on the plantar surface of the heel with maximum tenderness directly over the fat pad (Young et al, 2001:468; Nicholas and Hershman, 1995:451).
Bursitis
Usually retro-calcaneal, this presents with pain, swelling and erythema over the posterior aspect of the heel and is an inflammation of the bursa between the Achilles tendon and the calcaneus (Young et al, 2001:468; Pollard and So, 1999:96).

2.9.2) Skeletal

Calcaneal Stress Fracture
Usually associated with a sudden increase in daily exercise. Patients complain of pain on weight-bearing. Pain is often increased with medial to lateral compression of the calcaneus (Barrett and O'Malley, 1999:2204; Young et al, 2001:468). This pain increases with exercise unlike that of PF, which decreases with exercise (Batt and Tanji, 1995:80). A positive bone scan can confirm the diagnosis (Nicholas and Hershman, 1995:451).

Calcaneal Apophysitis
Also known as Sever’s disease, this condition usually affects boys between the ages of six and ten years, usually affecting either those who are overweight or those who are very active. These patients experience posterior heel pain that is worse following athletic activity (Barrett and O'Malley, 1999:2204; Young et al, 2001:468).

2.9.3) Neurological

Sciatica
Pressure on the L5-S1 nerve root may result in a sharp pain radiating from the buttocks, down the posterior thigh and leg towards the heel. Sciatica may be differentiated from PF by the presence of weakness of certain muscles, decreased ankle reflex (S1) and decreased sensation over the areas supplied by the L5-S1 nerve root (Barrett and O'Malley, 1999:2203).
Tarsal Tunnel Syndrome
This is as a result of compression of the posterior tibial nerve within the tarsal tunnel. This may result from a number of causes including inflammation, callus formation and a soft tissue mass. It usually presents as a more diffuse pain and unlike in PF, patients may have difficulty pin-pointing their pain. Patients may experience a burning or tingling sensation on the plantar surface, and often experience nocturnal pain (Barrett and O'Malley, 1999:2204; Young et al, 2001:468). A positive Tinel’s sign may help with the diagnosis (Nicholas and Hershman, 1995:451; Batt and Tanji, 1995:80).

Entrapment of the Lateral Plantar Nerve
The first branch of the lateral plantar nerve can become entrapped between abductor hallucis and quadratus plantae. This results in a burning sensation on the plantar surface of the heel (Barrett and O’Malley, 1999:2204; Young et al, 2001:468).

2.9.4) Systemic Disorders

Systemic arthritides
These may also present with heel pain, such as rheumatoid arthritis, gout and the sero-negative arthritides ankylosing spondylitis and Reiter’s syndrome. PF, due to systemic disorders, is often found to be bilateral and these patients often present with inflammation and joint pain in others areas as well (Ambrosius and Kondracki, 1992:34; Batt and Tanji, 1995:80; Barrett and O'Malley, 1999:2206). Blood tests including ESR, rheumatoid factor and uric acid can rule out these conditions (Hyde and Gengenbach, 1997:478). These conditions, while co-existing with PF can also be the cause of it and therefore may also be considered aetiological factors (Ambrosius and Kondracki, 1992:35).

Nutritional Osteomalacia
This results from a deficiency in Vitamin D, this condition may predispose to PF by either causing softening of the calcaneus or causing weakening of the intrinsic muscles of the feet leading to increased strain on the fascia. It can
therefore be considered as both an aetiological factor and a differential diagnosis (Ambrosius and Kondracki, 1992:34).

2.10) Treatment

Treatment should address the inflammatory component causing the discomfort as well as the biomechanical factors producing the disorder (Barrett and O’Malley, 1999:2201).

2.10.1) Conservative

Conservative treatment is generally recommended as the initial treatment of choice for PF (Ambrosius and Kondracki, 1992:35; Hyde and Gengenbach, 1997:479; Wolgin, 1994:102). In a study conducted by Davis et al (1994), 89.5% of patients were found to have significantly improved following a nonoperative treatment protocol.

Rest
Relative rest is an important aspect in the initial conservative approach to the treatment of PF, especially where the cause is thought to be overuse. This involves avoidance of aggravating activities such as running, substituting this with non weight-bearing activities such as swimming and cycling, which do not increase the tensile stress on the fascia (Batt and Tanji, 1995:83; Young et al, 2001:468; Nicholas and Hershman, 1995:453).

Correction of Environmental Factors
If training errors are the cause of this overuse syndrome, then runners should decrease intensity and mileage of their training. Similarly, use of appropriate footwear can be an important factor in the prevention and treatment of PF (Batt and Tanji, 1995:83; Wilk et al, 2000:24; Young et al, 2001:471).

Ice and Ultrasound
Applying ice to the plantar aspect of the heel helps to decrease the inflammation associated with the condition. Some authors recommend daily
ice massage of the area for 10 to 15 minutes (Nicholas and Hershman, 1995:453; Barrett and O'Malley, 1999:2202). Ultrasound may also aid in decreasing pain and inflammation (Hyde and Gengenbach, 1997:479; Pollard and So, 1999:96).

**Stretching and Strengthening**

Stretching was found to be the most effective form of treatment in studies conducted by both Wolgin *et al* (1994) and Davis *et al* (1994). It is effective as it also addresses functional risk factors such as tight Achilles tendon and triceps surae muscles. Stretching of these areas helps decrease tension on the plantar fascia. It is recommended to follow the stretching program for six to eight weeks. Strengthening of the intrinsic muscles may also decrease stress on the plantar fascia, and should include exercises such as towel curls (Young *et al*, 2001:469; Barrett and O'Malley, 1999:2201; Krivickas, 1997:141; Hyde and Gengenbach, 1997:479).

**Orthotics, Strapping and Splints**

Orthotics help correct the biomechanical factors contributing to the condition. They can aid in shock absorption and provide relief by decreasing tension on the plantar fascia (Young *et al*, 2001:471; Barrett and O'Malley, 1999:2202). Strapping can provide significant relief, with the aim to decrease the tensile stress on the plantar fascia, allowing the healing process to occur (Ambrosius and Kondracki, 1992:36; Brown, 1996:885). Night splints prevent contraction of the plantar fascia during sleep, allowing the fascia to heal in an elongated position therefore minimising tension on the first few steps in the morning (Young *et al*, 2001:472; Barrett and O'Malley, 1999:2202; Batt and Tanji, 1995:83). Morris (2000), found that the combination of adjustment and splint gave a greater improvement in terms of pain, disability and function than splinting alone.

**Manipulation**

Manipulation and mobilisation of the joints of the foot and ankle can restore the normal mechanics and alignment, therefore supporting the fascia and decreasing tension across it (Hyde and Gengenbach, 1997:479).

2.10.2) Anti-inflammatories

Non-steroidal anti-inflammatory drugs are often part of the initial treatment plan for PF, aiding in decreasing pain and inflammation. Their primary function is to control pain and not to treat the underlying problem. Disadvantages include gastrointestinal pain and bleeding and renal damage (Young et al, 2001:473; Barrett and O’Malley, 1999:2202).

2.10.3) Corticosteroid Injections

The use of steroid injections in the treatment of PF is controversial and they should be used with caution. These injections may only provide temporary relief and potential risks include rupture of the plantar fascia and fat pad atrophy (Young et al, 2001:473; Barrett and O’Malley, 1999:2202; Batt and Tanji, 1995:84).

2.10.4) Surgery

Surgery should be a last resort in the treatment of PF and should only be considered after at least six to nine months of conservative treatment has failed. Only 5% of patients require surgical treatment, and although the outcome is good, recovery is slow (Nicholas and Hershman, 1995:453; Ambrosius and Kondracki, 1992:38; Brown, 1996:885).

2.10.5) Transverse Friction Massage

Both Cyriax (1984) and Prentice (1994) state the effect of frictions to include the breakdown of adhesions (scar tissue), as well as preventing the formation of further adhesions. Frictions also aid in increased tissue perfusion, facilitating absorption of local edema and effusions (Cyriax, 1984:8; Prentice, 1994:351).

Lillegard and Rucker (1993) and Brantingham et al (1992) agree, saying that friction massage can be used to break down and reduce fibrotic scar tissue in the plantar fascia. In this regard Wilk et al (2000) utilise soft tissue mobilisation and deep friction massage of the plantar fascia to decrease inflammation. This is congruent with Pollard and So (1999), who note that deep friction massage can aid in reducing the inflammatory response and enhance tissue healing. Transverse friction massage can further enhance recovery, as stated by Hyde and Gengenbach (1997).

Prentice (1994) notes the importance of inflammation in the healing process. He states: ‘The purpose of transverse friction massage is to increase the inflammation to a point where the inflammatory process is complete and the injury can progress to the later stages of the healing process’.

2.10.6) Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM)

GISTM relates most closely to transverse friction massage and therefore it is hypothesised that it too could be beneficial in the treatment of PF. This is achieved by reducing scar tissue, aiding inflammation and allowing healing and the tissue remodelling process to begin, and ultimately decreasing the patient’s pain and improving function (Carey-Loghmani, 2003:31, 51-62; Hammer, 2001).

Transverse friction massage, and so GISTM, can also improve pain levels by increasing large-fibre input and, therefore, decrease nociceptive transmission through the ‘Gate Control Theory’ put forward by Melzack and Wall (Lynch and Kessler, 1990:48; Melzack and Wall,1965:971).
The instruments assist in locating areas in need of treatment and directing the clinician in the precise treatment of the lesion (Carey-Loghmani, 2003:7; Hammer, 2004). One of the most important effects of the instruments is the soft tissue loading which stimulates fibroblasts and the synthesis and maintenance of collagen (Hammer, 2004; Hammer, 2003).

Recent studies using various types of microscopy have shown that after friction massage there is fibroblastic proliferation and realignment of collagen fibres (Davidson et al, 1997:319).

When considering the phases of tissue healing, the role of GISTM can be more clearly explained. Phase two, the repair phase, lasts between 48 hours and six weeks and involves the synthesis and deposition of collagen. The role of GISTM during this phase includes the prevention of adhesion formation and aiding in the correct orientation of repair tissue. The remodelling phase (phase three), lasts from three weeks to twelve months or more. This stage involves fibroblastic activity and fibrosis, here GISTM is used to break down adhesions and aid in proper alignment of repair collagen. (Carey-Loghmani, 2003:31; Vizniak, 2003:165).

In terms of PF, tightness of the fascia may overstress the insertion sites at the calcaneus and lead to the development of focal heel pain. A reduction of fascia stiffness, through break down of adhesions and restrictions, is able to relieve the focal stress at the calcaneal insertion and so provide relief from pain (Cheung et al, 2004).

By breaking down adhesions in the plantar fascia, GISTM may assist in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62). This reduces the tension in the plantar fascia and at the fascial insertion, thereby reducing pain (Cheung et al, 2004).
GISTM, based on the principles of frictions, aims to break down adhesions, realign collagen fibres and aid the completion of the inflammatory process.

2.11) Conclusion

The efficacy of GISTM in the treatment of PF has not yet been clinically tested. This research aimed to determine the effect of GISTM on PF with respect to the changes seen on ultrasonography of the plantar fascia. Secondly, it aimed to identify any correlation between these ultrasonographic changes and the other objective and subjective findings.
Chapter Three: Materials and Methods

3.1) Introduction

This chapter outlines the general procedure utilised in carrying out this study. This includes the patient eligibility criteria, a description of the intervention received as well as the data types and statistical methods.

3.2) Research Design

A prospective pre-post quasi-experiment of a quantitative nature.

3.3) Sampling

Advertisements informing the public of the study being conducted were placed in newspapers, and posters were placed around the Durban Institute of Technology campus and at sports and running clubs (Appendix A). Talks were done at running clubs in the area informing the public of the study. Pamphlets were handed out at local race meetings (Appendix A).

3.3.1) Randomisation

The method was that of self-selection as subjects responded to the advertisements and the sample group was filled consecutively until there were a total of 30 subjects who fitted the study’s criteria.

The sample was stratified according to treatment intervention. Subjects were either placed into a treatment group, receiving an active treatment in the form of GISTM (Group A) or into a placebo group, receiving a placebo treatment in the form of de-tuned ultrasound (Group B).
3.3.2) Patient Screening

The evaluation and selection process began with all possible subjects undergoing a cursory telephonic discussion with the examiner to exclude subjects who did not fit the criteria of the study.

Questions asked:

- How old are you?
- With reference to the pain: location? Is it aggravated by dorsiflexion or walking on toes? Is it worse on the first few steps in the morning?
- Do you have a history of foot or ankle fracture, dislocation, surgery, peripheral neuropathy, nerve root entrapment or any other condition causing foot pain?
- Do you suffer from any systemic disease e.g. ankylosing spondylitis, Reiter’s disease, psoriatic arthritis, rheumatoid arthritis and/or gout?
- Do you run at least five kilometres per week?
- Have you been diagnosed with Diabetes?

Subjects suitable for the study were then evaluated at an initial consultation, at which they received a letter of information (Appendix B) and an informed consent form (Appendix C) to sign. These explained the study and the right of the patient to withdraw from the study at any time.

At this consultation a diagnosis was made based on case history (Appendix D), relevant physical examination (Appendix E) and foot and ankle regional examination (Appendix F).

3.4) Inclusion and Exclusion Criteria

3.4.1) Inclusion Criteria

1) Participants had to be between the ages of 25-50 years (Young et al, 2001:467 and Reid, 1992:196).
2) Participants had to have a diagnosis of PF based on:
- maximal pain located at the antero-medial aspect of the plantar surface of the calcaneus
- aggravation of pain by passive dorsiflexion of the great toe
- aggravation of pain when patient stands or walks on their toes
- pain that is worst on the first few steps in the morning.
(Young et al, 2001:467; Ried, 1992:196; Barrett and O'Malley, 1999:2201)

3) According to Warren (1990), it was suggested that participants run 35km per week, but for the purposes of this study, a minimum of five kilometres per week was required.

4) In tissue healing, Phases two and three, the repair and remodelling phases, may last from 48 hours to six weeks and three weeks to twelve months respectively. Clinical objectives in these phases include the proper alignment of repair tissue and collagen and the prevention and breakdown of fibrotic adhesions (Vizniak, 2003:165). Therefore, for purposes of this study patients were required to present with discomfort for a minimum of four weeks and a maximum of twelve months so as to fall within these boundaries.

5) All suitable subjects were required to complete and sign an informed consent form in order to be accepted into the study.

3.4.2) Exclusion Criteria

1) Participants were excluded from the study if they received any form of therapy, manual or medicinal, for their PF during the course of the research period (e.g. strapping, orthotics).

2) Participants with a history of foot or ankle fracture, dislocation, surgery, peripheral neuropathy, nerve root entrapment or any other condition
other than PF causing foot pain were excluded from the study (Davis et al, 1994:531; Wolgin et al, 1994:98).

3) Participants suffering from systemic disease causing foot pain were excluded from the study. This included ankylosing spondylitis, Reiter’s disease, psoriatic arthritis, rheumatoid arthritis and gout (Reid, 1992:196; Ambrosius and Kondracki, 1992:31; Davis et al, 1994:531).

4) Any systemic condition with the potential for causing peripheral neuropathy e.g. Diabetes, resulted in exclusion from the study.

5) The presence of any contraindications to GISTM, as listed in the Graston Technique Instruction Manual resulted in exclusion from the study. This included haematoma, open wounds, unhealed fractures, thrombophlebitis, uncontrolled hypertension, myositis ossificans and osteomyelitis (Carey-Loghmani, 2003:11).

6) Subjects accepted into the study were asked not to change their lifestyle, daily activities; regular medication and exercise programs to avoid being excluded from the study.

7) Individuals with bilateral PF were not excluded from the study. However, this factor was taken into account statistically.

3.5) Intervention Type and Manner

3.5.1) Intervention Type/Method

All subjects in the study underwent a number of clinical testing measurements, including subjective and objective data and an ultrasound evaluation of the plantar fascia. Thirty patients between the ages of 25 and 50 were randomly allocated into two groups, fifteen in each. Group A received the active treatment in the form of GISTM, while group B received a placebo treatment in the form of de-tuned ultrasound.
A) GISTM involves the use of stainless steel instruments, which aid in the location and breakdown of soft tissue adhesions (Carey-Loghmani, 2003:31, 7). It is thought to be beneficial in the treatment of PF by reducing scar tissue, aiding inflammation and allowing healing and the tissue remodelling process to begin, and ultimately decreasing the patient’s pain and improving function (Carey-Loghmani, 2003:31, 51-62).

The treatment was conducted using the GT-4 instrument (scanner) and involved the use of sweeping strokes over the sole of the foot, the focus being on the arch and heel. An emollient was used to decrease friction between the surface of the skin and the instrument. Treatment time was five minutes.

B) The placebo treatment was given in the form of de-tuned ultrasound, to act as a control in the study and to which to compare the results from the Group A. De-tuned ultrasound was chosen to be the placebo, firstly because it would have no physical effect on the plantar fascia and therefore would provide an effective control. And secondly because of its similarities in procedure to the active treatment, both involve the use of metal surface and a form of lubrication over the area being treated. The de-tuned ultrasound was applied superficially and without any pressure.

3.5.2) Intervention frequency

There were a total of ten treatments over a period of eight weeks. Subjects underwent a clinical assessment (objective and subjective findings) and an ultrasound evaluation in week one. The second and third clinical assessment and ultrasound evaluation took place in week five and week eight.
Week 1:
- clinical assessment no.1
- ultrasound evaluation no.1

Week 2:
- treatments 1, 2 & 3

Week 3:
- treatments 4, 5 & 6

Week 4:
- treatment 7

Week 5:
- ultrasound evaluation no.2
- clinical assessment no.2
- treatment 8

Week 6:
- treatment 9

Week 7:
- treatment 10

Week 8:
- ultrasound evaluation no.3
- clinical assessment no.3

3.6) **Data Type**

3.6.1) **Descriptive Data**

This includes: age, gender, race, weight and height.

3.6.2) **Inferential Data**

3.6.2.1) **Objective Data**

1) Ultrasonography was used to determine whether there were any changes in the plantar fascia pre and post treatment (Wall and
Harkness, 1998; Tsai et al, 2000; Cardinal et al, 1996). Measurements were taken at the point of maximum thickness and measured in millimetres (mm).

A linear probe was used, scanned at a frequency of 14mHz (Toshiba, Aplio).

In this study, ultrasonography was one of the objective measurements used to determine the effectiveness of the treatment. It is considered useful in the diagnosis of PF as it can provide an objective, non-invasive assessment of the inflammation. Increased plantar fascia thickness and hypoechoic fascia are consistent sonographic findings associated with PF, as well as poorly defined edges of the plantar fascia having also been noted in PF patients (Wall and Harkness, 1993:468; Tsai et al, 2000:259; Cardinal et al, 1996:258).

(Appendix G)

2) An algometer reading was taken at the point of maximum tenderness, usually over the insertion of the plantar fascia, to determine the degree of pain/tenderness in this region and to note any improvements with regard to treatment (Fischer, 1986:207). This was done by slowly increasing the pressure until the patient indicated they were starting to feel pain or discomfort (pressure threshold measurement). (Appendix H)


Participants stood on the involved leg and dorsiflexed the ankle while flexing the knee up to a point where no further dorsiflexion took place, without lifting the heel from the ground. A set square was used to measure the horizontal distance (x) from the back of the heel to the front of the knee. The vertical distance (y) from the ground to the front
of the knee was measured similarly. The degree of ankle dorsiflexion was calculated using: $\tan \theta = y/x$.
(Appendix H & I)

3.6.2.2) Subjective Data

1) The Functional Foot Index (FFI) was used to obtain information on the impact of the patients’ foot pain on their daily activities and to note any improvement in their functional ability with regards to treatment (Budiman-Mak, 1991:570; Saag et al, 1996:506). Patients were asked to answer a series of questions, rating the level of their pain between 0 and 10 for each activity. A zero (0) would mean ‘no pain at all’, and ten (10) indicating, ‘pain as bad as it could be’.
(Appendix J)

2) The Numerical Rating Scale – 101 (NRS-101) was used to record the patients’ perception of their foot pain (Jensen et al, 1986:118). Patients were asked to give their pain a rating of between 0 and 100. Firstly for when their pain was at it’s worst and secondly, when their pain was at it’s least. A zero (0) would mean ‘no pain at all’, and one hundred (100) would mean, ‘pain as bad as it could be’.
For statistical analysis the average of the first and the second reading where taken.
(Appendix K)

3.7) Data Collection Frequency

Data collection took place at three points during the research period, forming three data sets:

Data set 1: clinical assessment, ultrasound evaluation (week 1)
Data set 2: ultrasound evaluation, clinical assessment (week 5)
Data set 3: ultrasound evaluation, clinical assessment (week 8)
3.8) **Statistical Methods**

Data was captured in MS Excel and exported to SPSS version 11.5 (SPSS Inc., Chicago, Ill, USA) for analysis.

Demographic variables were compared between the two treatment groups using independent chi square tests, Fisher’s exact tests or independent t-tests where appropriate.

Repeated measures ANOVA was used to assess factors associated with the change over time in outcome measurements, particularly whether the change over time was related to the group (active or placebo) of the individual. Whether each subject was used once in the study (one affected ankle) or twice in the study (two affected ankles) was also controlled for as a factor in the analysis.

FFI items were summed at each time point to create a total FFI score for each time point.

Spearman’s correlation was used to examine the relationships between the changes in the various measurements intra-group.
Chapter Four

Results

4.1) Introduction

The statistical findings and results obtained from the data will be discussed in this chapter.

Demographic data consisting of age, race, gender, height and weight was analysed. Objective and subjective findings were also analysed, and the correlation between findings evaluated.

4.2) Demographics

Thirty participants who met eligibility criteria were randomised into two groups: GISTM and placebo. Demographic characteristics were compared between the two groups to ensure that the randomization process was complete in eliminating confounding variables between the groups.

4.2.1) Gender

There were 22 males (73.3%) and 8 females (26.7%). There was no significant difference between the proportions of each gender in each group (p = 0.682)(Table 1).

<table>
<thead>
<tr>
<th>GENDER</th>
<th>Total</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Count</td>
<td>10</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Row %</td>
<td>66.7%</td>
<td>33.3%</td>
</tr>
<tr>
<td>GISTM</td>
<td>Count</td>
<td>12</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Row %</td>
<td>80.0%</td>
<td>20.0%</td>
</tr>
<tr>
<td>Placebo</td>
<td>Count</td>
<td>22</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>Row %</td>
<td>73.3%</td>
<td>26.7%</td>
</tr>
</tbody>
</table>

Fisher’s exact p = 0.682
Males made up the greater numbers in both treatment groups of this study. This is consistent with the gender distribution as stated by Ambrosius and Kondracki (1992), although other studies show a majority of females (Davis et al., 1992:531; Wolgin et al., 1992:97). As this study was conducted on runners, this predominance of males could be a result of a possible greater number of male runners. A study conducted by Kibler et al. (1991) on PF in athletes (mainly runners), also reflected a higher proportion of males. Therefore, although not totally congruent with the literature, the data implies a similar trend.

4.2.2) Race

The majority of the participants in the study were White (53.3%). Indian participants made up the next 43.3%. There was only one Coloured participant and no African participants. There was no significant difference in the racial distribution between the groups (p = 0.515)(Table 2).

Table 2: Racial distribution by group

<table>
<thead>
<tr>
<th></th>
<th>RACE</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>White</td>
<td>Indian</td>
</tr>
<tr>
<td>GROUP</td>
<td>Count</td>
<td></td>
</tr>
<tr>
<td>GISTM</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>Row %</td>
<td>60.0%</td>
<td>40.0%</td>
</tr>
<tr>
<td>Placebo</td>
<td>Count</td>
<td>7</td>
</tr>
<tr>
<td>Row %</td>
<td>46.7%</td>
<td>46.7%</td>
</tr>
<tr>
<td>Total</td>
<td>Count</td>
<td>16</td>
</tr>
<tr>
<td>Row %</td>
<td>53.3%</td>
<td>43.3%</td>
</tr>
</tbody>
</table>

Lehohla (2004) states that the largest racial group in the province of Kwa-Zulu Natal is made up by the African population, followed by the Indian race and thirdly the White population. However, this racial distribution was not reflected in this study. The inversed ethnic ratio demonstrated may be due to a number of different reasons.
Firstly, the African population of KZN may not be familiar with Chiropractic, which is derived from a western culture. Secondly, participants in this study were required to make a number of trips to Pietermaritzburg, where one of the objective measurements was conducted. This may have resulted in a sample that is representative of only the highly mobile portion of the population, rather than the population in general. Lastly, as this study was conducted on runners, the racial profile may not reflect that of the general population of KZN.

4.2.3) Age, weight, height

There was also no significant difference in mean age, weight and height between the two groups (p = 0.835, 0.968 and 0.508 respectively). This is shown in Table 3.

<table>
<thead>
<tr>
<th>Group</th>
<th>N</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>AGE</td>
<td>GISTM 15</td>
<td>35.73</td>
<td>6.112</td>
<td>1.578</td>
<td>0.835</td>
</tr>
<tr>
<td></td>
<td>Placebo 15</td>
<td>36.27</td>
<td>7.658</td>
<td>1.977</td>
<td></td>
</tr>
<tr>
<td>WEIGHT</td>
<td>GISTM 15</td>
<td>75.53</td>
<td>14.803</td>
<td>3.822</td>
<td>0.968</td>
</tr>
<tr>
<td></td>
<td>Placebo 15</td>
<td>75.73</td>
<td>12.320</td>
<td>3.181</td>
<td></td>
</tr>
<tr>
<td>HEIGHT</td>
<td>GISTM 15</td>
<td>1.693</td>
<td>0.0704</td>
<td>0.0182</td>
<td>0.508</td>
</tr>
<tr>
<td></td>
<td>Placebo 15</td>
<td>1.713</td>
<td>0.0915</td>
<td>0.0236</td>
<td></td>
</tr>
</tbody>
</table>

The mean age of participants in this study was 36 years, with a youngest of 25 and the oldest being 49 years of age. This is congruent with another study conducted on PF in athletes (Kibler et al, 1991:67), who found an average age of 31. Brantingham et al (1992) also reported an average age of 36 in their study. While other studies show a higher mean age of 48 years (Davis et al, 1992:531; Wolgin et al, 1992:97), the younger mean age reported in this study reflects the athletic nature of the participants, who were required to be runners (Hertling and Kessler, 1996:426).
The weight of participants ranged from 55kg to 98kg with a mean of 75.63kg. While a correlation between PF and obesity has been noted (Barrett and O’Malley, 1999:2200; Brown, 1996:877), the fact that this was not reflected in this study could also be due to the athletic nature of the participants.

The mean height was found to be 1.703m. No literature reviewed provided data on this variable, thus no comparison is possible.
4.3) **Objective Data**

4.3.1) **Ultrasonography**

There was a statistically significant time*group interaction for ultrasonography ($p = 0.002$)(Table 4). Figure 1 shows that the placebo group experienced an increase in mean thickness over time, while the GISTM group showed a sharp decrease.

**Table 4: Between-subject and within-subject effects for ultrasonography**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.947</td>
<td>0.504</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.615</td>
<td>0.002</td>
</tr>
<tr>
<td>Group</td>
<td>$F=0.146$</td>
<td>0.706</td>
</tr>
</tbody>
</table>

![Figure 1: Profile plot of time by group for ultrasonography](image)

GISTM, based on the principles of transverse friction massage, was hypothesised to reduce the thickness of the plantar fascia. As Figure 1 clearly
demonstrates a sharp decrease in this thickness in the GISTM group, it would appear that this hypothesis holds true.

Lillegard and Rucker (1993) and Brantingham et al (1992) advocate the use of friction massage to break down and reduce fibrotic scar tissue in the plantar fascia. GISTM, through the breakdown of scar tissue, prevention of adhesion formation and aid in the correct orientation of the repair collagen (Carey-Loghmani, 2003:31; Vizniak, 2003:165) can result in a decrease in the thickness of the plantar fascia. The improved quality of the fascia was commented on regularly by the radiographer (de Villiers, 2005).

As participants in this study were asked not to change their daily activities or exercise programs, the increase in thickness noted in the placebo group may be as a result of the continued repetitive strain of the plantar fascia. While intervention with GISTM, prevented the formation of these further adhesions as well as breaking down existing fibrotic tissue.
4.3.2) Algometer

Algometer measurements showed a significant time effect but lack of a treatment effect (p=0.548), as shown in Table 5. Figure 2 shows that both groups showed a general increase in values over time, however the increase shown by the GISTM group levelled off after time 2. However, the two profiles were statistically parallel, thus there was no effect of GISTM treatment relative to placebo on algometer scores.

Table 5: Between-subject and within-subject effects for algometer measurement

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.775</td>
<td>0.041</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.953</td>
<td>0.548</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.766</td>
<td>0.389</td>
</tr>
</tbody>
</table>

The algometer was used to quantitatively evaluate local tenderness i.e. sensitivity to pressure (Fischer, 1986:207). The higher the pressure tolerated by the participant, the lower the level of local tenderness.
There was an increase in the pressure readings of the GISTM group, indicating an improvement in the level of pain and sensitivity of the area. Wilk et al (2000:24) utilise soft tissue mobilisation and deep friction massage of the plantar fascia to decrease inflammation. GISTM aids the inflammatory process allowing healing and the tissue remodelling to occur, ultimately decreasing the patient’s pain and improving function (Carey-Loghmani, 2003:31, 51-62; Hammer; 2001).

The improvement in pain levels can also be attributed to Melzack and Wall’s ‘Gate Control Theory’ (1965). Input from the small nerve fibres (from nociceptors) is blocked at the substantia gelatinosa (SG) by sensory input from the large nerve fibres (Melzack and Wall, 1965:971). Therefore, by increasing large fibre inputs through interventions such as frictions (Lynch and Kessler, 1990:48) and GISTM, can result in a decrease in pain.

However, this increased tolerance leveled off after T2. This may be accounted for by referring to the intervention frequency. From T1 and T2, participants received seven treatments in three weeks. While from T2 to T3 there were only three treatments in three weeks, possibly resulting in the plateau in improvement demonstrated in Figure 2.

The placebo group also showed a continual increase in the amount of pressure tolerated. This could be attributed to natural history, with the healing and strengthening of an injury over time (Lachmann and Jenner, 1994:28). The use of the placebo, in the form of de-tuned ultrasound, may also have had an effect. Although the placebo would result in no therapeutic effect, the tactile stimulation of the ultrasound head on the sole of the foot, could increase large nerve fibre input. Resulting in decreased pain according to Melzack and Wall’s theory (Melzack and Wall 1965:971).
4.3.3) Weight-bearing Ankle dorsiflexion

Ankle dorsiflexion showed no significant treatment effect ($p = 0.674$), as shown in Table 6. Figure 3 shows that both groups showed an increase in mean values between time 1 and 2, but thereafter the GISTM group decreased values while the placebo group levelled off. However, statistically the profiles of the two groups over time were not different.

**Table 6: Between-subject and within-subject effects for ankle dorsiflexion**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.964</td>
<td>0.633</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.969</td>
<td>0.674</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.622</td>
<td>0.437</td>
</tr>
</tbody>
</table>

**Figure 3: Profile plot of time by group for ankle dorsiflexion**

Repetitive micro-trauma to the plantar fascia results in the formation of scar tissue and adhesions (Ambrosius and Kondracki, 1992:30), the ensuing stiffness and contracture can limit ankle dorsiflexion (Batt and Tanji, 1995:78; Lillegard and Rucker, 1993:169).
By reducing adhesions, frictions and similarly GISTM, may assist in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62). This may be seen in an increase in ankle dorsiflexion, as demonstrated between T1 and T2 of the GISTM group. The improved dorsiflexion may reflect the intensive treatment during this time period.

T2 to T3 of this group, however, showed a decrease in ankle dorsiflexion. While one may expect the level of improvement to plateau during this second, less intensive phase of treatment, this sharp decrease was not anticipated.

Breaking down adhesions, and therefore reducing fascia stiffness, may have an impact on arch stability and cause other biomechanical effects. It is possible that this may induce significant strains or stresses in the muscular, ligamentous and bony structures (Cheung et al, 2004). This could then possibly affect the ankle dorsiflexion measurement by causing overload and stiffness in the triceps surae muscles.

Another explanation for this may be post exercise stiffness. Tightness of the triceps surae muscles and the associated Achilles tendon can also contribute to limited ankle dorsiflexion (Pope et al, 1998:166). As participants were asked to continue with their exercise programs, it is reasonable to expect a certain amount muscle stiffness to be experienced.

The placebo group showed an improvement in ankle dorsiflexion. This may be accounted for by Melzack and Wall’s theory of pain inhibition already discussed. The tactile stimulation of the de-tuned ultrasound, although not having any actual therapeutic effect, may stimulate the large nerve fibres resulting in decreased pain (Melzack and Wall, 1965:971). With decreased pain levels patients may have a greater mobility and improved dorsiflexion.

This method of measuring ankle dorsiflexion has been found to be reliable (Bennell et al, 1998:178). Making use of the functional, weight-bearing...
position, it is more relevant to PF than other non weight-bearing methods (Pope et al, 1998:170). However, it is an indirect measure of plantar fascia flexibility, and affected by flexibility or stiffness of the triceps surae muscles (Blake, 2003:81).

PF has also been shown to limit dorsiflexion of the big toe (Lillegard and Rucker, 1993:169; Hyde and Gengenbach, 1997:479). Measuring great toe extension may be a more accurate measurement of plantar fascia flexibility (Carey-Loghmani, 2005).
4.4) **Subjective Data**

4.4.1) **NRS-101**

There was a significant change over time for both groups for NRS-101 (p<0.001), but no treatment effect (p=0.983)(Table 7). Thus the profiles of the two groups were parallel. This is reflected in Figure 4.

**Table 7: Between-subject and within-subject effects for NRS-101**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.288</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.999</td>
<td>0.983</td>
</tr>
<tr>
<td>Group</td>
<td>F=1.008</td>
<td>0.325</td>
</tr>
</tbody>
</table>

**Figure 4: Profile plot of time by group for NRS-101**

There is an overall decrease in pain levels experienced by the GISTM group. The mechanism by which GISTM works is by reducing scar tissue, aiding inflammation and allowing healing and the tissue remodelling process to begin (Carey-Loghmani, 2003:31, 51-62). By aiding the inflammatory process, GISTM allows the injury to progress to the later stages of healing and finally to
resolution. In doing so there is an improvement in the level of the patients’ pain (Carey-Loghmani, 2003:31; Hammer, 2001).

As for algometer, the improvement in pain levels may also be attributed to the ‘Gate Control Theory’, by increasing large-fibre input GISTM can result in decrease nociceptive transmission and lower pain levels (Melzack and Wall, 1965:971).

There is an initial sharper decrease in pain from T1 to T2, followed by a more gradual decrease to T3. This may be attributed to the more frequent treatments during the first phase of treatment, tapering off during the second phase.

The placebo group follows the same pattern as the GISTM group: steeper decline from T1 to T2, levelling off to T3. This may be accounted for by natural history, which involves the natural resolution of an injury over time (Lachmann and Jenner, 1994:28).

When considering placebo, doctor-patient interaction must also be taken into account. There is an element of touch that occurs during treatment. Ventegodt et al (2004) believes that holistic treatment is facilitated when there is a combination of touch and therapeutic work on mind and feelings. Improvements in both psychological and physical functioning were found in a study on healing by gentle touch conducted by Weze et al (2005).

The Hawthorne effect may also come into play here. Human beings, aware that they are being studied, may demonstrate atypical behavior (Mouton, 2002:152). The tactile stimulation of the placebo may also decrease pain through the ‘Gate Control Theory’ already discussed (Melzack and Wall, 1965:971).
4.4.2) Foot Function Index (FFI)

The Functional Foot Index (FFI) evaluates foot pathology in terms of three main categories: foot pain, disability and activity restriction (Budiman-Mak, 1991:570; Saag et al, 1996:506). Patients were asked to answer a series of questions, rating the level of their pain between 0 and 10 for each activity.

4.4.2.1) FFI-1: Worst pain

Worst pain showed a significant decrease over time, which was independent of group (p=0.621)(Table 8). Thus the GISTM treatment had no effect over and above the placebo for this outcome. Figure 5 shows almost parallel profiles of the two groups.

**Table 8: Between-subject and within-subject effects for FFI-1: Worst pain**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.345</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.963</td>
<td>0.621</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.949</td>
<td>0.339</td>
</tr>
</tbody>
</table>

![Figure 5: Profile plot of time by group for FFI-1: Worst pain](image-url)
The GISTM group showed an overall decrease in worst pain. Prentice (1994) states that transverse friction massage increases inflammation to a point where the inflammatory process is complete, allowing the injury to progress to the later stages of healing. GISTM works on the same theory, initiating a new inflammatory cascade to promote healing (Hammer, 2001; Carey-Loghmani, 2003:31). The result is an improvement in pain levels experienced by the patient, as demonstrated in Figure 5.

There is an initial greater decrease from T1 to T2, which levels off towards T3. This corresponds with the change in intervention frequency from phase one to phase two already discussed. This pattern was demonstrated by the majority of the FFI questions.

The improvement demonstrated by the GISTM group is not as rapid as that of the placebo group. This may be attributed to the initial inflammation that GISTM stimulates (Hammer, 2001; Carey-Loghmani, 2003:31), in order to allow progression to the later stages of healing and finally, resolution.

The placebo group also improved in terms of worst pain levels. As for NRS, this could be attributed to natural history (Lachmann and Jenner, 1994:28), the Hawthorne effect (Mouton, 2002:152) and Melzack and Wall’s theory (Melzack and Wall 1965:971). These theories can be applied to each of the FFI questions, in a possible explanation for the improvement demonstrated by the placebo group.
4.4.2.2) FFI-2: Morning pain

Morning pain was significantly decreased in both groups, irrespective of the group of the participant (p = 0.942), as shown in Table 9. The profiles are parallel for the two groups (Figure 6).

**Table 9: Between-subject and within-subject effects for FFI-2: Morning pain**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.672</td>
<td>0.007</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.995</td>
<td>0.942</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.639</td>
<td>0.431</td>
</tr>
</tbody>
</table>

**Figure 6: Profile plot of time by group for FFI-2: Morning pain**

Morning pain is the most characteristic complaint of PF, typically patients complain that the pain is worst on the first few steps in the morning (Young *et al*, 2001:467). Kibler and Chandler (1993) attribute this morning pain to the fact that the plantar fascia is contracted and stiff, the pain is due to the stretching of damaged tissues.
GISTM facilitates the healing of these damaged tissues by promoting resolution of the inflammatory process, thereby decreasing the level of pain experienced. Furthermore, by reducing fibrotic scar tissue, GISTM will help to limit contracture and stiffness of the fascia resulting in decreased fascial tension and therefore decreased pain (Carey-Loghmani, 2003:31; Cheung et al, 2004).

A reduction of fascia stiffness, through break down of adhesions and restrictions, is able to relieve the focal stress at the calcaneal insertion, which is believed to be associated with heel pain (Cheung et al, 2004).

For the majority of the patients in this study, morning pain equaled their worst pain. Resulting in similar profiles in Figure 5 (4.4.2.1) and Figure 6.
4.4.2.3) FFI-3: Pain walking barefoot

There was a highly significant change over time in both groups (p<0.001). There was no evidence of a treatment effect (p=0.692), as shown in Table10. Figure 7 shows that both groups decreased to the same extent over time.

**Table 10: Between-subject and within-subject effects for FFI-3: Pain walking barefoot**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk's lambda=0.465</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk's lambda=0.971</td>
<td>0.692</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.017</td>
<td>0.897</td>
</tr>
</tbody>
</table>

**Figure 7: Profile plot of time by group for FFI-3: Pain walking barefoot**

By breaking down adhesions in the plantar fascia, GISTM may assist in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62).

Greater strain is placed on the plantar fascia when walking barefoot as the fascia does not have the support of the shoe or orthotic. PF results from
tensile overload at the insertion of the plantar fascia, resulting in inflammation and pain (Batt and Tanji, 1995:78). Increased tension at the inflamed insertion of the fascia exacerbates this pain.

By breaking down the adhesions in the fascia, greater extensibility is achieved (Hertling and Kessler, 1996:134). This reduces the tension in the plantar fascia and at the fascial insertion, thereby reducing pain (Cheung et al, 2004).

Therefore the improvement demonstrated when walking barefoot, would seem to confirm the positive effect that GISTM has on mobility, extensibility and function. Furthermore, the decrease in pain indicates an improvement of the level of inflammation at the fascial insertion.
4.4.2.4) **FFI-4: Pain walking with shoes**

Whilst pain walking with shoes decreased significantly, this decrease rate was the same in both groups (p=0.487)(Table 11). Figure 8 shows that the rate of decrease was similar for both groups.

**Table 11: Between-subject and within-subject effects for FFI-4: Pain walking with shoes**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.378</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.944</td>
<td>0.487</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.066</td>
<td>0.799</td>
</tr>
</tbody>
</table>

**Figure 8: Profile plot of time by group for FFI-4: Pain walking with shoes**

Pain when walking with shoes demonstrated a similar pattern to that of pain when walking barefoot. This can also be attributed to the decreased tension in the plantar fascia following the break down in adhesions (Cheung *et al*, 2004). Resulting in improved extensibility and function and improved pain levels (Hertling and Kessler, 1996:134).
The starting and finishing points demonstrated by the GISTM group in Figure 8, are lower than those of the same group in Figure 7 (4.4.2.3). Indicating less pain is experienced when walking with shoes. This may be due to the support that is provided to the plantar fascia by the shoe and/or orthotic.

When answering this section of the FFI, patients often commented that their pain levels when walking with shoes was dependant on the type of shoe and the presence or absence of orthotics. More flexible shoes and the absence of orthotics were often said to result in greater pain levels than shoes of good support.

This concurs with the literature that finds that shoes, especially in runners, can be a causative factor as well as perpetuating an already existing condition (Wilk, 2000:27; Rajput and Abboud, 2004). Worn out shoes result in poor shock absorption while excessively flexible shoes result in greater toe flexion and increased tension at the fascial insertion (Warren, 1990:340; Ambrosius and Kondracki, 1992:34).
4.4.2.5) **FFI-5: Pain standing with shoes**

There was no treatment effect (p=0.744) for pain standing with shoes (Table 12). Figure 9 shows that the rate of decrease was similar for both groups.

**Table 12: Between-subject and within-subject effects for FFI-5: Pain standing with shoes**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.521</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.977</td>
<td>0.744</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.000</td>
<td>0.985</td>
</tr>
</tbody>
</table>

**Figure 9: Profile plot of time by group for FFI-5: Pain standing with shoes**

As the pathology of PF is due to tensile overload of the fascial insertion (Batt and Tanji, 1995:78), it would follow that any factor increasing the tension in the fascia would aggravate the condition. Young *et al* (2001) note that pain can be experienced with prolonged standing and may be accompanied by stiffness. Weight-bearing increases tension in the fascia, exacerbating the symptoms (Hyde and Gegenbach, 1997:478).

Secondly, with prolonged standing the plantar fascia may become cold and start to contract, increasing the tension. By reducing the adhesions, this contraction of the fascia may be limited (Cheung et al, 2004; Carey-Loghmani, 2003:31, 51-62; Hertling and Kessler, 1996:134).
4.4.2.6) FFI-6: Pain walking inside

Figure 10 shows that the profiles of the two groups were very similar over time (p=0.667), as shown in Table 13. Thus there was no treatment effect for this outcome.

Table 13: Between-subject and within-subject effects for FFI-6: Pain walking inside

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.548</td>
<td>0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.967</td>
<td>0.667</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.003</td>
<td>0.960</td>
</tr>
</tbody>
</table>

Discussed under the following section, 4.4.2.7.
4.4.2.7) FFI-7: Pain walking outside

There was no treatment effect for walking outside (p = 0.750) (Table 14). This is shown in Figure 11.

Table 14: Between-subject and within-subject effects for FFI-7: Pain walking outside

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.608</td>
<td>0.002</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.977</td>
<td>0.750</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.500</td>
<td>0.486</td>
</tr>
</tbody>
</table>

GISTM demonstrated a greater improvement for walking outside than walking inside. ‘Walking inside’ would be associated with more level, even surfaces. On the other hand ‘walking outside’ would include more uneven surfaces such as beach sand and grass. Walking on these types of bumpy, irregular surfaces would require greater flexibility from the plantar fascia as the foot has to conform and adapt to the changing surface as opposed to a smooth, regular surface.
By breaking down adhesions in the plantar fascia, GISTM may assist in restoring mobility, increasing extensibility and pliability of structures and thereby improving function (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62).

This reduces the tension in the fascia, thereby reducing pain (Cheung et al, 2004). The greater improvement demonstrated when walking on uneven surfaces (outside) as opposed to even surfaces (inside), would seem to corroborate the positive effect that GISTM has on mobility, extensibility and function of the plantar fascia.
4.4.2.8) **FFI-8: Pain climbing stairs**

There was both a significant time effect ($p=0.001$) and a significant group effect ($p=0.015$) for climbing stairs (Table 15). This does not imply a treatment effect, though. It means that the group’s means were significantly different at each time point. This could be due to large baseline differences between the groups for this outcome. Figure 12 shows that whilst the profiles were far apart, they were essentially parallel over time.

**Table 15: Between-subject and within-subject effects for FFI-8: Pain climbing stairs**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.561</td>
<td>0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.957</td>
<td>0.586</td>
</tr>
<tr>
<td>Group</td>
<td>F=6.818</td>
<td>0.015</td>
</tr>
</tbody>
</table>

![Figure 12: Profile plot of time by group for FFI-8: Pain climbing stairs](image)

Discussed under the following section, 4.4.2.9.
4.4.2.9) **FFI-9: Pain descending stairs**

There was no treatment effect for this outcome (p=0.923), as shown in Table 16. Figure 13 shows that the profiles were parallel over time.

**Table 16: Between-subject and within-subject effects for FFI-9: Pain descending stairs**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
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<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.786</td>
<td>0.050</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.994</td>
<td>0.923</td>
</tr>
<tr>
<td>Group</td>
<td>F=3.437</td>
<td>0.075</td>
</tr>
</tbody>
</table>

![Figure 13: Profile plot of time by group for FFI-9: Pain descending stairs](image)

**Figure 13: Profile plot of time by group for FFI-9: Pain descending stairs**

A study conducted by Riener *et al* (2002) on ground reaction force patterns during ascent and descent of stairs, found that forces were significantly higher during descent than during ascent.

In terms of the GISTM group, there was a greater improvement with regards to pain with climbing stairs as opposed to pain with descending stairs. The ascending task consists primarily of a transfer of muscle energy, whereas
during descent, the energy has to be absorbed (Reiner et al, 2002). This results in a large amount of stress on the plantar fascia. Since descending stairs puts the plantar fascia under greater tension, it can be assumed that symptoms would be worse on this activity than on ascending (Cheung et al, 2004). With this in mind, the greater improvement demonstrated on climbing stairs would seem reasonable.
4.4.2.10) FFI-10: Pain standing on toes

There was no significant treatment effect for this outcome (p=0.489), although both groups improved significantly over time (p=0.020)(Table 17). Figure 14 shows that both groups changed at the same rate over time.

Table 17: Between-subject and within-subject effects for FFI-10: Pain standing on toes

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.731</td>
<td>0.020</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.944</td>
<td>0.489</td>
</tr>
<tr>
<td>Group</td>
<td>F=3.481</td>
<td>0.073</td>
</tr>
</tbody>
</table>

Figure 14: Profile plot of time by group for FFI-10: Pain standing on toes

The plantar fascia, spanning the distance between the calcaneus and the forefoot, has been likened to a ‘bowstring’ (Hyde and Gegenbach, 1997:477; Garrick and Webb, 1999:392). Dorsiflexion of the toes and metatarsophalangeal joints tenses the fascia (Delee and Drez, 1994:1818). It is this position that is achieved by toe standing, and is known to aggravate the pain of PF (Young et al, 2001:468; Garrick and Webb, 1999:394).
4.4.2.11) **FFI-11: Pain getting up from a chair**

There was no treatment benefit in the GISTM group over the placebo \( (p = 0.947) \) for this outcome (Table 18). Both groups showed significant changes over time \( (p = 0.011) \). This is shown in Figure 15 where the group means are very similar at all time points.

**Table 18: Between-subject and within-subject effects for FFI-11: Pain getting up from a chair**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.698</td>
<td>0.011</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.996</td>
<td>0.947</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.028</td>
<td>0.868</td>
</tr>
</tbody>
</table>

![Figure 15: Profile plot of time by group for FFI-11: Pain getting up from a chair](image)

This can be likened to ‘morning pain’ (4.4.2.2). After prolonged sitting or non-weight-bearing, the plantar fascia can become cold or contracted resulting in pain on the first few steps (Hyde and Gegenbach, 1997:478; Kibler *et al*, 1991:66). As activity continues and the fascia warms up the symptoms ease, reflecting the change in degree of stiffness and contracture in the fascia (Batt

GISTM enhances the recovery process, resulting in decreased inflammation and therefore decreased pain (Carey-Loghmani, 2003:31, 51-62; Hammer, 2001). Secondly, through the break down of scar tissue, GISTM helps to limit the amount of contraction that occurs during rest periods. Resulting in decreased fascial tension and a resultant decrease in pain (Carey-Loghmani, 2003:31; Cheung et al, 2004).
4.4.2.12) FFI-12: Pain climbing curbs

There was no significant change over time (p=0.172), or treatment effect for climbing curbs (p=0.926), as shown in Table 19. Figure 16 shows that the values did not change much over time, or between the groups.

**Table 19: Between-subject and within-subject effects for FFI-12: Pain climbing curbs**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.869</td>
<td>0.172</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.994</td>
<td>0.926</td>
</tr>
<tr>
<td>Group</td>
<td>F=1.601</td>
<td>0.217</td>
</tr>
</tbody>
</table>

**Figure 16: Profile plot of time by group for FFI-12: Pain climbing curbs**

This can be likened to ‘pain climbing stairs’ (4.4.2.8). Similarly, decreased adhesions and decreased inflammation would result in lower pain levels on climbing curbs (Carey-Loghmani, 2003:31; Cheung et al, 2004; Hammer, 2001). The more gradual graph represented here, may be due to the lower initial pain level.
4.4.2.13) FFI total score

FFI items were summed at each time point to create a total FFI score for each time point.

Both groups showed a significant decrease in score over time, but this decrease was not related to the group that the participant was in ($p = 0.861$), as shown in Table 20. In Figure 17 the profiles over time of the two groups showed similar rates of decrease.

Table 20: Between-subject and within-subject effects for FFI total score

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.422</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.988</td>
<td>0.861</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.318</td>
<td>0.578</td>
</tr>
</tbody>
</table>

![Figure 17: Profile plot of time by group for FFI total score](image)

Although both groups closely parallel each other, the GISTM group appears to have a more rapid overall improvement indicated by the steepness of the graph.
It would seem that the effect that GISTM has on pain and function can be attributed to a number of factors. Firstly, by breaking down adhesions in the plantar fascia, GISTM may assist in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62).

Secondly, the decrease in pain indicates an improvement of the level of inflammation at the fascial insertion. By initiating a new inflammatory cascade to promote healing (Hammer, 2001; Prentice, 1994:351), GISTM can result in improved in pain levels experienced by the patient.

By increasing large-fibre input, GISTM may decrease nociceptive transmission through the ‘Gate Control Theory’ put forward by Melzack and Wall, resulting in decreased pain levels (Lynch and Kessler, 1990:48; Melzack and Wall, 1965:971).

Lastly, by reducing fascia stiffness and tension, through break down of adhesions and restrictions, GISTM may be able to relieve the focal stress at the calcaneal insertion, improving pain and function levels (Cheung et al, 2004; Carey-Loghmani, 2003:31, 51-62).

There is an initial greater improvement from T1 to T2, which levels off towards T3. This corresponds with the change in intervention frequency from phase one (seven treatments in three weeks) to the less intensive phase two (three treatments in three weeks).

The placebo group also improved in terms of FFI total score. This could be attributed to natural history (Lachmann and Jenner, 1994:28), the Hawthorne effect (Mouton, 2002:152) and Melzack and Wall’s gate control theory (Melzack and Wall (1965:971).
4.5) **Intra-group correlations between changes in objective and subjective findings**

**Table 21: Spearman correlation between changes in subjective and objective outcomes in the GISTM group**

<table>
<thead>
<tr>
<th>Change in algometer</th>
<th>Correlation Coefficient</th>
<th>Change in ultrasonography</th>
<th>Change in ankle dorsiflexion</th>
<th>Change in NRS-101</th>
<th>Change in FFI total</th>
<th>Change in FFI-1</th>
<th>Change in FFI-2</th>
<th>Change in FFI-11</th>
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</thead>
<tbody>
<tr>
<td>1.000</td>
<td>.016</td>
<td>-.418</td>
<td>-.007</td>
<td>-.255</td>
<td>.031</td>
<td>-.296</td>
<td>-.253</td>
<td></td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.955</td>
<td>.121</td>
<td>.980</td>
<td>.360</td>
<td>.913</td>
<td>.284</td>
<td>.363</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in ultrasonography</th>
<th>Correlation Coefficient</th>
<th>Change in ankle dorsiflexion</th>
<th>Change in NRS-101</th>
<th>Change in FFI total</th>
<th>Change in FFI-1</th>
<th>Change in FFI-2</th>
<th>Change in FFI-11</th>
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</thead>
<tbody>
<tr>
<td>.016</td>
<td>1.000</td>
<td>.097</td>
<td>-.085</td>
<td>-.373</td>
<td>.043</td>
<td>-.269</td>
<td>-.340</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.955</td>
<td>.732</td>
<td>.762</td>
<td>.171</td>
<td>.879</td>
<td>.332</td>
<td>.215</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in ankle dorsiflexion</th>
<th>Correlation Coefficient</th>
<th>Change in NRS-101</th>
<th>Change in FFI total</th>
<th>Change in FFI-1</th>
<th>Change in FFI-2</th>
<th>Change in FFI-11</th>
</tr>
</thead>
<tbody>
<tr>
<td>-.418</td>
<td>.097</td>
<td>1.000</td>
<td>.192</td>
<td>.267</td>
<td>.367</td>
<td>.305</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.121</td>
<td>.732</td>
<td>.493</td>
<td>.336</td>
<td>.179</td>
<td>.269</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in NRS-101</th>
<th>Correlation Coefficient</th>
<th>Change in FFI total</th>
<th>Change in FFI-1</th>
<th>Change in FFI-2</th>
<th>Change in FFI-11</th>
</tr>
</thead>
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<tr>
<td>-.007</td>
<td>-.085</td>
<td>.192</td>
<td>.100</td>
<td>.116</td>
<td>.698(**)</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.980</td>
<td>.762</td>
<td>.493</td>
<td>.682</td>
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</table>

<table>
<thead>
<tr>
<th>Change in FFI total</th>
<th>Correlation Coefficient</th>
<th>Change in FFI-1</th>
<th>Change in FFI-2</th>
<th>Change in FFI-11</th>
</tr>
</thead>
<tbody>
<tr>
<td>-.255</td>
<td>-.373</td>
<td>.267</td>
<td>.116</td>
<td>.000</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.360</td>
<td>.171</td>
<td>.336</td>
<td>.682</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in FFI-1</th>
<th>Correlation Coefficient</th>
<th>Change in FFI-2</th>
<th>Change in FFI-11</th>
</tr>
</thead>
<tbody>
<tr>
<td>.031</td>
<td>.043</td>
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<td>Sig. (2-tailed)</td>
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<td>.179</td>
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<th>Correlation Coefficient</th>
<th>Change in FFI-11</th>
</tr>
</thead>
<tbody>
<tr>
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<td>.305</td>
</tr>
<tr>
<td>Sig. (2-tailed)</td>
<td>.284</td>
<td>.332</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in FFI-11</th>
<th>Correlation Coefficient</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>-.253</td>
<td>-.340</td>
<td>.215</td>
</tr>
</tbody>
</table>
4.5.1) GISTM group (Table 21)

Algometer, ultrasonography and ankle dorsiflexion were not correlated with any other measurements.

Change in NRS-101 was positively correlated with change in FFI-1 (worst pain) (rho = 0.698, p=0.004). NRS-101 was taken as an average between worst pain and least pain, therefore the correlation with FFI-1 (worst pain) would be expected.

The only other correlations within this group were those that were expected between the FFI total score and FFI components and between the various FFI components themselves.

The correlation between FFI-1 (worst pain) and FFI-2 (morning pain) would be expected as for the majority of participants their morning pain equaled their worst pain. Morning pain also correlated with FFI-11 (pain getting up from a chair), which would also be expected as both involve weight-bearing after a prolonged period of rest.
Table 22: Spearman correlation between changes in subjective and objective outcomes in the placebo group

| Outcome                  | Change in algometer | Change in ultrasonography | Change in ankle dorsiflexion | Change in NRS-101 | Change in FFI total | Change in FFI-1 | Change in FFI-2 | Change in FFI-11 |
|--------------------------|----------------------|---------------------------|------------------------------|-------------------|---------------------|----------------|---------------|----------------|-----------------|
| Change in algometer     | Correlation Coefficient | 1.000                     | .288                         | -.429             | -.387               | -.573(*)       | -.488         | -.481          | -.407           |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in ultrasonography| Correlation Coefficient | .288                      | 1.000                        | -.384             | .213                | .036           | .023          | .260           | .100            |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in ankle dorsiflexion | Correlation Coefficient | -.429                     | -.384                        | 1.000             | -.061               | .225           | .007          | .043           | .255            |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in NRS-101        | Correlation Coefficient | -.387                     | .213                         | -.061             | 1.000               | .667(**)       | .896(**)      | .892(**)       | .654(**)        |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in FFI total      | Correlation Coefficient | -.573(*)                  | .036                         | .225              | .667(**)            | .775(**)       | .810(**)      | .862(**)       |                 |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in FFI-1         | Correlation Coefficient | .026                      | .899                         | .419              | .007                | .001           | .000          | .000           | .000            |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in FFI-2         | Correlation Coefficient | -.488                     | .023                         | .007              | .896(**)            | .775(**)       | 1.000         | .905(**)       | .748(**)        |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |
| Change in FFI-11        | Correlation Coefficient | -.407                     | .100                         | .255              | .654(**)            | .862(**)       | .748(**)      | .853(**)       | 1.000           |
| Sig. (2-tailed)          |                      |                            |                              |                   |                     |                |               |                |                 |

* Correlation is significant at the 0.05 level (2-tailed).
** Correlation is significant at the 0.01 level (2-tailed).
4.5.2) Placebo group (Table 22)

Change in algometer was negatively correlated with change in FFI total score (rho = -0.573, p=0.026). Thus as algometer reading increased, score decreased and vice-versa. This would be expected as an increase in algometer readings represents a lower local pain level, which would correspond with the decrease in FFI score.

Change in NRS-101 was highly significantly correlated with FFI total score, worst pain, morning pain, and getting up from a chair. Certain of the FFI items were highly correlated with other FFI items.

Ultrasonography and ankle dorsiflexion were not correlated with any other outcome.
4.6) **Summary and Conclusions**

This study showed evidence for a beneficial effect of the GISTM on the plantar fascia, but only demonstrated on ultrasonography. The other outcomes measured were not significantly affected by the technique, which showed no benefit over and above that of the placebo.

GISTM is used to detect and treat soft tissue lesions (Carey-Loghmani, 2003:12; Hammer, 2004). Therefore GISTM, through the breakdown of scar tissue, prevention of adhesion formation and aid in the correct orientation of the repair collagen (Carey-Loghmani, 2003:31; Vizniak, 2003:165) can result in a decrease in the thickness of the plantar fascia.

Breaking down the fibrotic tissue and remodeling the collagen to the correct alignment assists in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996:134). Increasing the pliability of the plantar fascia can result in decreased tension and therefore pain (Cheung *et al.*, 2004), as well as improved function.

GISTM, similarly to frictions, aids the inflammatory process. GISTM initiates inflammation allowing the process to complete itself and for healing and the tissue remodelling to occur, ultimately decreasing the patient’s pain and improving function (Carey-Loghmani, 2003:31, 51-62; Hammer; 2001).

Transverse friction massage, and so GISTM, can also improve pain levels by increasing large-fibre input and, therefore, decrease nociceptive transmission through the ‘Gate Control Theory’ put forward by Melzack and Wall (Lynch and Kessler, 1990:48; Melzack and Wall, 1965:971).

There was not a lot of correlation between intra-group outcomes. The GISTM group showed no correlation between any of the objective outcomes (ultrasonography, algometer, ankle dorsiflexion) with the subjective outcomes.
(NRS, FFI). However, correlations between NRS-101, FFI-1 (worst pain), FFI-2 (morning pain) and FFI-11 (pain getting up from a chair) were found.

4.6.1) The first hypothesis

The first hypothesis was that GISTM would result in a decrease in the thickness of the plantar fascia, seen on the ultrasonography findings of the plantar fascia post treatment.

The above hypothesis is accepted as the GISTM group demonstrated a sharp decrease in thickness of the plantar fascia on ultrasonography.

4.6.2) The second hypothesis

The second hypothesis was that GISTM would be effective in the treatment of PF in terms of subjective and objective clinical findings.

The above hypothesis was rejected as all subjective and objective outcomes measured, apart from ultrasonography, were not significantly affected by the technique, which showed no benefit over and above that of the placebo.

4.6.3) The third hypothesis

The third hypothesis was that the ultrasonography findings would correlate with the subjective and objective clinical findings.

The above hypothesis is rejected as there was no correlation between ultrasonography and the remainder of the subjective and objective clinical findings.
Chapter Four: Results

Image 1: Ultrasonography of the plantar fascia (week 1), thickness of 5mm.

Image 2: Ultrasonography of the plantar fascia (week 8), thickness of 3mm.
Chapter Five: Conclusions and Recommendations

5.1) Introduction

This chapter will discuss the outcomes of this research and make recommendations with regards to further research.

5.2) Conclusions

The purpose of this study was to look at the effectiveness of the GISTM in the management of PF in runners, and to determine any correlations between scar tissue reduction and clinical findings.

In terms of objective data:
1) GISTM was effective in decreasing the thickness of the plantar fascia seen on ultrasonography, indicating scar tissue reduction.
2) GISTM provided no significant treatment benefit compared to placebo, in terms of algometer readings.
3) GISTM provided no treatment benefit in terms of weight-bearing ankle dorsiflexion.

In terms of subjective data:
1) GISTM provided no significant treatment benefit compared to placebo, in terms of NRS-101 readings.
2) GISTM provided no significant treatment benefit compared to placebo, in terms of FFI readings.

In terms of correlation:
The ultrasonography findings were not found to significantly correlate with any of the other objective or subjective outcomes.
In conclusion, Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM) was found to be beneficial in certain aspects of the treatment of PF in runners. GISTM can be effectively used by practitioners to break down scar tissue and prevent the formation of further adhesions.

5.3) Recommendations

1) Ultrasonography measurements were taken at the point of maximum thickness of the fascia. If measurements were taken at a specific distance from the point of attachment, it may provide more accurate measures of the change in thickness over time.

2) Weight-bearing ankle dorsiflexion was used as a measure of the flexibility of the plantar fascia. However, this is an indirect measure and too greatly affected by stiffness in the triceps surae muscles. In future studies into the effect of GISTM on PF, it is recommended to include GISTM to the posterior calf muscles. In doing so ankle dorsiflexion may then be a more reliable measure.

3) A critical measure to reflect the effect of GISTM on the plantar fascia is great toe extension. This measure was not used in this study and is recommended for subsequent studies.

4) The FFI was used as a subjective measure of pain and function. However, this instrument was initially developed for use in RA patients and is therefore not specific for PF. A more specific questionnaire may yield more relevant information.

5) Lack of blinding could have resulted in researcher bias. Having a peer intern or clinician to take objective and subjective measures may result in more reliable readings.

6) A larger sample size is suggested to improve the validity of the study and for the results to be more statistically significant. Furthermore the
small sample size could have allowed for an outlier to change the average significantly, further emphasising the benefits a larger sample size would provide.

7) A future study could include a third control group to rule out the effect of possible effect from the placebo treatment.

8) Bilateral PF generally has a worse prognosis, and does not respond as well to treatment, including GISTM. In future studies, the researcher should possibly consider excluding individuals exhibiting bilateral symptoms.

9) Participants in the study were asked not to change their exercise programs. As a result post exercise stiffness may have affected some of the outcomes. To avoid this a period of rest could be enforced, however this is still not ideal as rest is already a proven form of treatment of PF.

10) The Graston Technique (GT) = GISTM + exercise. For the purposes of this research, participants did not receive the full GT protocol. It is important to note, that GISTM should be used in conjunction with a cardio warm-up, targeted stretching and strengthening exercises and post treatment cryotherapy. By following this comprehensive treatment approach, the full benefit/effect of GISTM can be realized.
References


Blake, T.L. 2003. The effectiveness of manipulation of the subtalar joint combined with static stretching of the triceps surae muscles compared to manipulation alone in the treatment of plantar fasciitis. M Tech: Chiropractic, Durban Institute of Technology, Durban. Pg 81, 86.


de Villiers, P. 2005. Personal communication with S. Dunn.


ATTENTION RUNNERS
Age: 25 - 50

Do you suffer from pain in the soles of your feet, heel pain and arch pain?
FREE TREATMENT available to those who qualify to take part in this study.

For further information on the research being conducted with respect to this condition, please contact Shoshanna Dunn or Kirsten Maartens on (031) 204 2205/2512.

DURBAN INSTITUTE OF TECHNOLOGY - CHIROPRACTIC DAY CLINIC
Appendix B

Dear Participant

Welcome to this study.

Title of Research: The evaluation of ultrasonographic findings in the management of plantar fasciitis in runners and the association with clinical findings.

Name of Research Student: Shoshanna Dunn Contact number: (031) 204-2205

Name of Supervisor: Dr C Korporaal Contact number: (031) 204-2611

Introduction and Purpose of Study:
You have the opportunity to take part in this study which is an evaluation into the ultrasonographic findings in the management of plantar fasciitis in runners and the association with clinical findings (objective and subjective).

Reasons why you may be withdrawn from the study:
If you are taking any medication, or undergoing any other form of treatment for your foot pain, you may be excluded from the study. Please try not to alter your normal lifestyle or daily activities in any way as this could interfere with the results of this study.

Procedure:
At the initial consultation you will be screened for suitability. Clinical measurements will be taken and you will be sent for diagnostic ultrasound of the plantar fascia before the commencement of treatment. A total of 10 treatments will take place over a period of eight weeks. A second and third ultrasound evaluation will take place in week 5 and week 8 of the research period.

Benefits:
You will receive treatment for your plantar fasciitis, which you are expected to benefit from according to current hypotheses.

Risks:
Slight bruising, pain and/or discomfort of the area being treated may be experienced, however all these effects are temporary in nature.

Confidentiality:
All patient information is confidential and the results of the study will be made available in the Durban Institute of Technology Library in the form of a mini-dissertation.

You are free to withdraw from the study at any stage. Please don’t hesitate to ask questions on any aspect of the study. Your full co-operation will assist the Chiropractic profession in expanding it’s knowledge of this condition.

Thank you.

Yours sincerely,

Shoshanna Dunn
(Research Student)

Dr C Korporaal
(Supervisor)
INFORMED CONSENT FORM
(To be completed by patient / subject)

Date:

Title of research project:
The evaluation of ultrasonographic findings in the management of plantar fasciitis in runners and the association with clinical findings.

Name of supervisor:
Tel:

Name of research student:
Tel:

Please circle the appropriate answer: YES / NO

1. Have you read the research information sheet? Yes No
2. Have you had an opportunity to ask questions regarding this study? Yes No
3. Have you received satisfactory answers to your questions? Yes No
4. Have you had an opportunity to discuss this study? Yes No
5. Have you received enough information about this study? Yes No
6. Do you understand the implications of your involvement in this study? Yes No
7. Do you understand that you are free to withdraw from this study at any time without having to give any a reason for withdrawing, and without affecting your future health care. Yes No
8. Do you agree to voluntarily participate in this study? Yes No
9. Who have you spoken to?

Please ensure that the researcher completes each section with you
If you have answered NO to any of the above, please obtain the necessary information before signing

Please Print in block letters:

Patient/Subject Name: ___________________________ Signature: ______________

Parent/Guardian: _______________________________ Signature: ______________

Witness Name: _________________________________ Signature: ______________

Research Student Name: _________________________ Signature: ___
DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: ___________________________ Date: __________
File # : ____________ Age: __________
Sex : ________ Occupation: ____________________________
Intern : __________________________ Signature __________________________
FOR CLINICIANS USE ONLY:
Initial visit
Clinician: __________________________ Signature :

Case History:

Examination:
Previous: __________________________ Current: __________________________

X-Ray Studies:
Previous: __________________________ Current: __________________________

Clinical Path. lab:
Previous: __________________________ Current: __________________________

CASE STATUS:
PTT: __________________________ Signature: __________________________ Date: __________

CONDITIONAL:
Reason for Conditional:

Signature: __________________________ Date: __________

Conditions met in Visit No: __________________________ Signed into PTT: __________________________ Date: __________

Case Summary signed off: __________________________ Date: __________
**Intern’s Case History:**

1. **Source of History:**

2. **Chief Complaint : (patient’s own words):**

3. **Present Illness:**

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<th>Complaint 1</th>
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<tbody>
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<td></td>
</tr>
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4. **Other Complaints:**

5. **Past Medical History:**
   - General Health Status
   - Childhood Illnesses
   - Adult Illnesses
   - Psychiatric Illnesses
   - Accidents/Injuries
   - Surgery
   - Hospitalizations
6. **Current health status and life-style:**

- Allergies
- Immunizations
- Screening Tests incl. x-rays
- Environmental Hazards (Home, School, Work)
- Exercise and Leisure
- Sleep Patterns
- Diet
- Current Medication
  - Analgesics/week:
- Tobacco
- Alcohol
- Social Drugs

7. **Immediate Family Medical History:**

- Age
- Health
- Cause of Death
- DM
- Heart Disease
- TB
- Stroke
- Kidney Disease
- CA
- Arthritis
- Anaemia
- Headaches
- Thyroid Disease
- Epilepsy
- Mental Illness
- Alcoholism
- Drug Addiction
- Other

8. **Psychosocial history:**

- Home Situation and daily life
- Important experiences
- Religious Beliefs
9. **Review of Systems:**

- General
- Skin
- Head
- Eyes
- Ears
- Nose/Sinuses
- Mouth/Throat
- Neck
- Breasts
- Respiratory
- Cardiac
- Gastro-intestinal
- Urinary
- Genital
- Vascular
- Musculoskeletal
- Neurologic
- Haematologic
- Endocrine
- Psychiatric
Appendix E

DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
PHYSICAL EXAMINATION

Patient: ___________________________ File#: ___________ Date: _______

Clinician: _________________________ Signature: _______________________

Student: ___________________________ Signature: _______________________

1. VITALS

Pulse rate:
Respiratory rate:
Blood pressure: R L Medication if hypertensive:
Temperature:
Height:
Weight: Any change Y/N If Yes: how much gain/loss Over what period

2. GENERAL EXAMINATION

General Impression:
Skin:
Jaundice:
Pallor:
Clubbing:
Cyanosis (Central/Peripheral):
Oedema:
Lymph nodes - Head and neck:
  - Axillary:
  - Epitrochlear:
  - Inguinal:
Urinalysis:

3. CARDIOVASCULAR EXAMINATION

1) Is this patient in Cardiac Failure?
2) Does this patient have signs of Infective Endocarditis?
3) Does this patient have Rheumatic Heart Disease?

Inspection - Scars
  - Chest deformity:
  - Precordial bulge:
  - Neck -JVP:

Palpation: - Apex Beat (character + location):
  - Right or left ventricular heave:
  - Epigastric Pulsations:
  - Palpable P2:
  - Palpable A2:

Percussion: - borders of heart

Auscultation: - heart valves (mitral, aortic, tricuspid, pulmonary) - Murmurs (timing, systolic/diastolic, site, radiation, grade).

4. **RESPIRATORY EXAMINATION**

1) Is this patient in **Respiratory Distress**?

**Inspection** - Barrel chest: 
- Pectus carinatum/caviumatum: 
- Left precordial bulge: 
- Symmetry of movement: 
- Scars:

**Palpation** - Tracheal symmetry: 
- Tracheal tug: 
- Thyroid Gland: 
- Symmetry of movement (ant + post) 
- Tactile fremitus:

**Percussion** - Percussion note: 
- Cardiac dullness: 
- Liver dullness:

**Auscultation** - Normal breath sounds bilat.: 
- Adventitious sounds (crackles, wheezes, crepitations) 
- Pleural frictional rub: 
- Vocal resonance - Whispering pectoriloquy: 
- Bronchophony: 
- Egophony:

5. **ABDOMINAL EXAMINATION**

1) Is this patient in **Liver Failure**?

**Inspection** - Shape: 
- Scars: 
- Hernias:

**Palpation** - Superficial: 
- Deep = Organomegally: 
- Masses (intra- or extramural) 
- Aorta:

**Percussion** - Rebound tenderness: 
- Ascites: 
- Masses:

**Auscultation** - Bowel sounds: 
- Arteries (aortic, renal, iliac, femoral, hepatic)
Rectal Examination - Perianal skin:
- Sphincter tone & S4 Dermatome:
- Obvious masses:
- Prostate:
- Appendix:

6. G.U.T EXAMINATION

External genitalia:
Hernias:
Masses:
Discharges:

7. NEUROLOGICAL EXAMINATION

Gait and Posture - Abnormalities in gait:
- Walking on heels (L4-L5):
- Walking on toes (S1-S2):
- Romberg's test (Pronator Drift):

Higher Mental Function - Information and Vocabulary:
- Calculating ability:
- Abstract Thinking:

G.C.S.: - Eyes:
- Motor:
- Verbal:

Evidence of head trauma:

Evidence of Meningism: - Neck mobility and Brudzinski's sign:
- Kernig's sign:

Cranial Nerves:

I Any loss of smell/taste:
Nose examination:

II External examination of eye: - Visual Acuity:
- Visual fields by confrontation:
- Pupillary light reflexes = Direct:
  = Consensual:
- Fundoscopy findings:

III Ocular Muscles:
Eye opening strength:

IV Inferior and Medial movement of eye:

V a. Sensory - Ophthalmic:
- Maxillary:
- Mandibular:

b. Motor - Masseter:
- Jaw lateral movement:

c. Reflexes - Corneal reflex
- Jaw jerk

VI Lateral movement of eyes
VII  a. Motor  
- Raise eyebrows:
  - Frown:
  - Close eyes against resistance:
  - Show teeth:
  - Blow out cheeks:

b. Taste  
- Anterior two-thirds of tongue:

VIII  General Hearing:
  Rinnes = L:  
  R:  
  Webers lateralisation:  
  Vestibular function  
  - Nystagmus:  
  - Rombergs:  
  - Wallenbergs:  

  Otoscope examination:

IX &  Gag reflex:

X  Uvula deviation:

  Speech quality:

XI  Shoulder lift:

  S.C.M. strength:

XII  Inspection of tongue (deviation):

Motor System:
  a. Power  
  - Shoulder  
    = Abduction & Adduction:
    = Flexion & Extension:
  - Elbow  
    = Flexion & Extension:
  - Wrist  
    = Flexion & Extension:
  - Forearm  
    = Supination & Pronation:
  - Fingers  
    = Extension (Interphalangeals & M.C.P's):
  - Thumb  
    = Opposition:
  - Hip  
    = Flexion & Extension:
    = Adduction & Abduction:
  - Knee  
    = Flexion & Extension:
  - Foot  
    = Dorsiflexion & Plantar flexion:
    = Inversion & Eversion:
    = Toe (Plantarflexion & Dorsiflexion):

b. Tone  
  - Shoulder:
  - Elbow:
  - Wrist:
  - Lower limb - Int. & Ext. rotation:
  - Knee clonus:
  - ankle clonus:

c. Reflexes  
  - Biceps:
  - Triceps:
  - Supinator:
  - Knee:
  - Ankle:
  - Abdominal:
  - Plantar:
Sensory System:

a. Dermatomes
   - Light touch:
   - Crude touch:
   - Pain:
   - Temperature:
   - Two point discrimination:

b. Joint position sense
   - Finger:
   - Toe:

c. Vibration:
   - Big toe:
   - Tibial tuberosity:
   - ASIS:
   - Interphalangeal Joint:
   - Sternum:

Cerebellar function:

Obvious signs of cerebellar dysfunction:
   = Intention Tremor:
   = Nystagmus:
   = Truncal Ataxia:

Finger-nose test (Dysmetria):
Rapid alternating movements (Dysdiadochokinesia):
Heel-shin test:
Heel-toe gait:
Reflexes:
Signs of Parkinsons:

8. **SPINAL EXAMINATION**:(See Regional examination)

Obvious Abnormalities:
Spinous Percussion:
R.O.M:
Other:

9. **BREAST EXAMINATION**:

Summon female chaperon.

**Inspection**
   - Hands rested in lap:
   - Hands pressed on hips:
   - Arms above head:
   - Leaning forward:

**Palpation**
   - masses:
   - tenderness:
   - axillary tail:
   - nipple:
   - regional lymph nodes
Foot and ankle regional examination

Patient: ___________________________ File no: ______ Date: ______

Intern / Resident: ___________________ Signature: _______________________________________

Clinician: __________________________ Signature: _______________________________________

Observation
Gait analysis (antalgic limp, toe off, arch, foot alignment, tibial alignment).

Swelling
Heloma dura / molle
Skin
Nails
Shoes
Contours (achilles tendon, bony prominences)

Active movements

<table>
<thead>
<tr>
<th>Weight bearing:</th>
<th>R</th>
<th>L</th>
<th>Non weight bearing:</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plantar flexion</td>
<td></td>
<td></td>
<td>50°</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td></td>
<td></td>
<td>20°</td>
<td></td>
<td></td>
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<tr>
<td>Supination</td>
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<tr>
<td>Pronation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe dorsiflexion</td>
<td></td>
<td></td>
<td>40°(mtp)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe plantar flexion</td>
<td></td>
<td></td>
<td>40° (mtp)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Big toe dorsiflexion (mtp) (65-70°)
Big toe plantar flexion (mtp) 45°
Toe abduction + adduction
5° first ray dorsiflexion
5° first ray plantar flexion

Passive movement motion palpation (Passive ROM quality, ROM overpressure, joint play)

<table>
<thead>
<tr>
<th>Ankle joint: Plantarfexion</th>
<th>R</th>
<th>L</th>
<th>Subtalar joint: Varus</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plantarfexion</td>
<td></td>
<td></td>
<td>Plantarfexion</td>
<td></td>
<td></td>
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<tr>
<td>Long axis distraction</td>
<td></td>
<td></td>
<td>Midtarsal: A-P glide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First ray: Dorsiflexion</td>
<td></td>
<td></td>
<td>P-A glide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantarfexion</td>
<td></td>
<td></td>
<td>Plantarfexion rotation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circumduction of forefoot on fixed rearfoot</td>
<td></td>
<td></td>
<td>Intermetatarsal glide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interphalangeal joints: L→A dist</td>
<td></td>
<td></td>
<td>Tarso metatarsal joints: A-P</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A-P glide</td>
<td></td>
<td></td>
<td>Metatarsophalangeal dorsiflexion (with associated plantar flexion of each toe)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
### Resisted Isometric movements

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>L</th>
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</thead>
<tbody>
<tr>
<td>Knee flexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantar flexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsiflexion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Supination (inversion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pronation (eversion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe extension (dorsiflexion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Toe flexion (plantar flexion)</td>
<td></td>
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</tbody>
</table>

### Neurological

<table>
<thead>
<tr>
<th></th>
<th>R</th>
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<tbody>
<tr>
<td>Dermatomes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myotomes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reflexes</td>
<td></td>
<td></td>
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<tr>
<td>Balance/proprioception</td>
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</tr>
</tbody>
</table>

### Special tests

<table>
<thead>
<tr>
<th></th>
<th>R</th>
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</thead>
<tbody>
<tr>
<td>Anterior drawer test</td>
<td></td>
<td></td>
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<tr>
<td>Talar tilt</td>
<td></td>
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<tr>
<td>Thompson test</td>
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<tr>
<td>Homan sign</td>
<td></td>
<td></td>
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<tr>
<td>Tinel's sign</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test for rigid/flexible flatfoot</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kleiger test (med. deltoid)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Alignment

<table>
<thead>
<tr>
<th></th>
<th>R</th>
<th>L</th>
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</thead>
<tbody>
<tr>
<td>Heel to ground</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Feiss line</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tibial torsion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heel to leg (subtalar neutral)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subtalar neutral position:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forefoot to heel (subtalar &amp; Midtarsal neutral)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First ray alignment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Digital deformities</td>
<td></td>
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<tr>
<td>Digital deformity flexible</td>
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</tr>
</tbody>
</table>

### Palpation

#### Anteriorly

<table>
<thead>
<tr>
<th></th>
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<th>L</th>
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</thead>
<tbody>
<tr>
<td>Medial maleoli</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Med tarsal bones, tibial (post) artery</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lat.malleolous, calcaneus, sinus tarsi, and cuboid bones</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inferior tib/fib joint, tibia, mm of leg</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior tibia, neck of talus, dorsalis pedis artery</td>
<td></td>
<td></td>
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</tbody>
</table>

#### Posteriorly

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Calcaneus, Achilles tendon, Musculotendinous junction</td>
<td></td>
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</tr>
</tbody>
</table>

#### Plantarily

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<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Plantar muscles and fascia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sesamoids</td>
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</tr>
</tbody>
</table>

21/10/2002
Appendix G

Data Recording Sheet

Ultrasound Evaluation of the Plantar Fascia

Patient: ________________________________  Sign: _________
Doctor: ________________________________  Sign: _________
Date: __________________
Visit Number: 1 / 2 / 3

Increased thickness

Measurement: ____________ mm
Comment: e.g. inflammation (hypoechoicity), tears
__________________________________________________________
__________________________________________________________
**Data Recording Sheet**

**Algometer Reading**

<table>
<thead>
<tr>
<th>Patient: __________________________</th>
<th>Date: ________</th>
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</thead>
<tbody>
<tr>
<td>1)</td>
<td>2)</td>
</tr>
<tr>
<td>Assessment 1 (wk1):</td>
<td></td>
</tr>
<tr>
<td>Assessment 2 (wk5):</td>
<td></td>
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<tr>
<td>Assessment 3 (wk8):</td>
<td></td>
</tr>
</tbody>
</table>

**Data Recording Sheet**

**Weight-bearing Ankle Dorsiflexion**

<table>
<thead>
<tr>
<th>Patient: __________________________</th>
<th>Date: ________</th>
</tr>
</thead>
<tbody>
<tr>
<td>1)</td>
<td>2)</td>
</tr>
<tr>
<td>Assessment 1 (wk1):</td>
<td></td>
</tr>
<tr>
<td>Assessment 2 (wk5):</td>
<td></td>
</tr>
<tr>
<td>Assessment 3 (wk8):</td>
<td></td>
</tr>
</tbody>
</table>
Participants stood on the involved leg and dorsiflexed the ankle while flexing the knee up to a point where no further dorsiflexion took place, without lifting the heel from the ground. A set square was used to measure the horizontal distance \((x)\) from the back of the heel to the front of the knee. The vertical distance \((y)\) from the ground to the front of the knee was measured similarly. The degree of ankle dorsiflexion was calculated using: \(\tan \theta = \frac{y}{x}\).
**FOOT FUNCTION INDEX**

**INSTRUCTIONS:** Please fill in a value somewhere between 0 and 10 describing your pain
0 indicates no pain and 10 indicates the worst pain
If the question is not applicable then indicate this by writing N/A next to it

<table>
<thead>
<tr>
<th>Section A:</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Worst pain</td>
<td></td>
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<tr>
<td>Morning Pain</td>
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<tr>
<td>Pain walking barefoot</td>
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<tr>
<td>Pain walking with shoes</td>
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<td>Pain standing with shoes</td>
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</table>

<table>
<thead>
<tr>
<th>Section B: Can you</th>
<th>0</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Walk in the house</td>
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<tr>
<td>Walk outside</td>
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<tr>
<td>Climb stairs</td>
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<tr>
<td>Descend stairs</td>
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<tr>
<td>Stand on tip toe</td>
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</tr>
<tr>
<td>Get up from a chair</td>
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</tr>
<tr>
<td>Climb curbs</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Section C: Do you have to?</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stay inside all day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stay in bed all day</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix K

Numerical Rating Scale - 101 Questionnaire

Date:__________  File no:__________  Visit no:

Patient name: __________________________________________

Please indicate on the line below, the number between 0 and 100 that best describes the pain you experience when it is at its worst. A zero (0) would mean “no pain at all”, and one hundred (100) would mean “pain as bad as it could be”.

Please write only one number.

0 ________________________________ 100

Please indicate on the line below, the number between 0 and 100 that best describes the pain you experience when it is at its least. A zero (0) would mean “no pain at all” and one hundred (100) would mean “pain as bad as it could be”.

Please write only one number.

0 ________________________________ 100