

DEDICATION

To Jesus, my lifeline.

To my husband, Michael,
who has kept me motivated with his love.

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ABSTRACT

According to research, continuing symptoms of pain, instability, crepitus, weakness, stiffness (Pellow and Brantingham, 2001) and swelling (Patel and Warren, 1999:332) commonly follow an acute ankle sprain. The cause of these symptoms is often attributed to the development of a tight sensitive scar (Reid, 1992:251) within the injured ligament.

The treatment options available include scar tissue debridement (Bassewitz and Shapiro, 1997), manipulation (Edmond, 1993:164), mobilization, (Hockenbury and Sammarco, 2001) and ultrasound (Thomson, Skinner & Piercy, 1991:43-44).

Transverse friction massage could also be used to reduce adhesions (Kessler, 1990:85) and improve mobility of the tissues (Kessler, 1990:140). The Graston Technique Instrument Assisted Soft Tissue Mobilization (GTIASTM) comprises a set of stainless steel instruments (Carey 2003:2) designed to detect and reduce scar tissue and adhesions (Carey 2003:7) by bringing about an area of controlled microtrauma (Hammer, 2003(b):1) and inflammation (Carey 2003:32) through a mechanism similar to that of friction massage.

The aim of this study was to determine the efficacy of the GTIASTM in the reduction of scar tissue in the anterior talofibular ligament for the management of chronic ankle instability syndrome (a clinical presentation that develops as a result of prior ankle sprains (Hertling and Kessler 1996:421-424)) following a Grade I or Grade II ankle inversion sprain. The study was also designed to establish any changes in pain and edema levels and to identify any correlation between scar tissue reduction, pain and edema.

This study included a total of 30 ankles among 24 people who had experienced an ankle inversion sprain and had subsequently developed chronic ankle instability syndrome. These patients were systematically assigned to receive detuned ultrasound (placebo) or GTIASTM.

The two groups then received 8 treatments over 4 weeks. A diagnostic ultrasound scan was taken before the first consultation, to determine the presence and amount of scar tissue within the anterior talofibular ligament, and after the final treatment, to identify any changes in thickness of the scar tissue. In addition to this algometer, edema measures and a foot function index were also repeated at set intervals.

The SPSS version 11.5 was used to analyse the data (SPSS Inc, Chicago, Ill, USA). Repeated measures ANOVA was used to examine changes in quantitative outcomes over the time points (intragroup analysis) and a treatment effect (intergroup analysis). To control for the partial pairing in the intergroup analysis, a variable which classified each subject as paired (both left and right ankle used in study) or non-paired (only used once in study) was used as a factor in the model. Correlations between the intragroup changes in the various outcome variables were assessed using Pearson's correlation coefficients.

Statistical analysis of the subjective and objective data revealed significantly improved pain levels and also improved proprioceptive skills in the GTIASTM group. Both groups demonstrated edema reduction and while both groups displayed reduction in scar tissue, there appeared to be more clinically significant reduction within the proximal portion of the ligament in those subjects who received GTIASTM.

The findings imply that the GTIASTM is effective in the treatment of chronic ankle instability syndrome particularly with regards to pain and proprioception. It was not found in any way to be detrimental to the patients' well being as outcomes never proved worse than those who had received detuned ultrasound.

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CHAPTER ONE - INTRODUCTION

1.1 Introduction

The talocrural joint is a synovial hinge type of joint located between the inferior ends of the fibula and tibia and the superior part of the talus (Moore, 1992: 487). Although the ankle joint is usually called a uniaxial joint, it is dynamic in character taking up different positions during dorsiflexion and plantarflexion (Moore, 1992: 488; Gray 1980:491). During dorsiflexion the ankle joint is extremely strong, being supported by powerful ligaments (anterior and posterior talofibular ligaments and the calcaneofibular ligament) and crossed by several tendons (peroneus longus and brevis). Stability is further enhanced during dorsiflexion by the fact that the trochlea of the talus fills the mortise (Moore, 1992: 489). During plantarflexion the ankle is considered to be fairly unstable as it is in this position that the trochlea of the talus moves anterior in the mortise. In full plantarflexion of the foot some side movement can be demonstrated (Moore, 1992: 489). The lateral ankle ligaments are involved in limiting varus movements and the medial ankle ligaments limit valgus movements of the foot (Cailliet, 1997:202).

The ankle is one of the most commonly injured joints in the body (Fallat *et al.*, 1998) and the most common sports-incurred injury is injury to the lateral ankle ligaments during an ankle inversion sprain (Cailliet, 1997:205).

Continuing symptoms of pain, instability, crepitus, weakness, stiffness (Pellow and Brantingham, 2001) and swelling, following an ankle inversion sprain are frequently reported (Patel and Warren, 1999:332) and are thought to be related to untreated ligamentous damage (Pellow and Brantingham, 2001 and Reid, 1992:250) or can be attributed to healing of the injured ligament with adhesion formation (Hertling and Kessler, 1996:424-425). Symptoms persisting for more than 6 weeks after an acute sprain are termed chronic ankle instability syndrome^a.

The stability of the ankle is dependent on having intact ligaments as the capsule of the ankle joint is fairly thin and weak (Norkin and Levangie, 1992: 384). The lateral collateral ligaments are generally weaker than the medial collateral ligaments (Norkin and Levangie, 1992: 384). The anterior talofibular ligament (ATFL) is the most vulnerable of the lateral ankle ligaments

^a Chronic ankle instability syndrome is a clinical presentation that presents as a result of prior ankle sprains. This term is necessary in order to avoid confusion that the term "chronic ankle sprain" implies as this is a different clinical entity (Wolfe *et al* 2001, Hertel 2002 and Hertling and Kessler 1996:421-424)

(Karlsson *et al.*, 2003:541) with injury most commonly occurring during inversion and internal rotation with the ankle in various degrees of plantarflexion (Bennett, 1994). Treatment of chronic ankle instability syndrome should therefore target these injured ligaments. The literature suggests the following treatment options (Davies and Chu, 1999:389):

- a. Deep transverse friction massage (Cyriax, 1984:8-9)
- b. Joint manipulation (Pellow and Brantingham, 2001)
- c. Balance exercises and muscle strengthening (Reid, 1992: 238)

The Graston Technique Instrument Assisted Soft Tissue Mobilization (GTIASTM) comprises a set of stainless steel instruments that function in a similar way to deep transverse friction massage to break down scar tissue and adhesions (Carey, 2003: 2; 37). Studies are needed in order to investigate the role that GTIASTM may play in the treatment of scar tissue and to see, if any, the effect that this may have on the patients' symptoms (i.e. pain and edema). This study investigated the efficacy of the GTIASTM in breaking down adhesions and reducing scar tissue. It was however also determined if there was any subsequent effect on pain and edema.

It was therefore hypothesized that the use of GTIASTM for the treatment of grade I and grade II chronic ankle instability syndrome would prove to be an effective treatment in terms of subjective and objective findings as compared to placebo. Following Pellow and Brantingham's (2001) use of detuned ultrasound, this study has used detuned ultrasound as the placebo^b.

^b Placebo is an "indispensable tool for study of the reaction or processing component of suffering." It is merely an "aid to therapeutic suggestion" given as a device for eliminating bias". (Beecher 1955: 1602)

1.2 Aims/objectives of the study

The purpose of this systematic placebo controlled study was to fulfil the following objectives:

1.2.1 The first objective

To measure the efficacy of the Graston Technique Instrument Assisted Soft Tissue Mobilization in the reduction of scar tissue (diagnostic ultrasound findings) in patients experiencing chronic ankle instability syndrome following an ankle inversion sprain.

1.2.2 The second objective

To measure the efficacy of the Graston Technique Instrument Assisted Soft Tissue Mobilization on clinical measures (viz. the reduction of pain, edema and other clinical outcomes observed from the Foot Function Index) in patients experiencing chronic ankle instability syndrome following an ankle inversion sprain.

1.2.3 The third objective

To determine if there is any degree of correlation between scar tissue, pain and edema.

1.3 Statement of the hypothesis

1.3.1 The first hypothesis

It is hypothesized that the use of the GTIASTM for the reduction of scar tissue as a treatment of chronic ankle grade I or II ankle sprains will prove to be an effective treatment in terms of subjective and objective findings.

1.3.2 The second hypothesis

It is hypothesized that the GTIASTM will prove to be an effective treatment for concomitant clinical measures (viz. the reduction of pain, edema and other clinical outcomes observed from the Foot Function Index).

1.3.3 The third hypothesis

It is hypothesized that there will be some degree of correlation between scar tissue, pain and edema findings.

1.4 Delimitation

These research findings, although directed at the rehabilitation of ligaments may not be generalised to all ligaments. This is because of their inherent anatomical position, function and/or physiological make up of individual ligaments. Therefore any conclusions drawn will be relevant to the ankle ligaments only.

CHAPTER TWO - LITERATURE REVIEW

2.1 Introduction

The ankle joint sustains the highest incidence of sports injuries (Jerosch and Bischoff, 1996) with the most common injury to this area being a lateral ankle inversion sprain (Mack, 1982). This results in damage to the lateral ankle ligaments, and of particular concern, the anterior talofibular ligament (ATFL) which is considered to be the most important stabilizing ligament at the lateral ankle (Reid, 1992:220). The injury may produce scarring of the ATFL (Hockenbury and Sammarco, 2001) and according to Reid (1992:251) a tight sensitive scar may be the cause of chronic symptoms following an ankle inversion sprain.

The author of this study chose to compare the GTIASTM with placebo (detuned ultrasound) in order to determine the effects that the GTIASTM may or may not have on any scar tissue present within the ATFL and to assess whether this has any subsequent effects on the symptoms of chronic ankle instability syndrome (pain and edema).

2.2 Incidence and Prevalence

The ankle is one of the most commonly injured joints in the body (Fallat *et al.*, 1998 and Jerosch and Bischof, 1996) and lateral ankle sprains are among the most common injuries (Hertel, 2002).

A 1-year retrospective study on injuries of the foot and ankle was conducted by Grimm and Fallat (1999). Of the 3851 new injuries that presented to the Oakwood Hospital Downriver Centre Occupational Medicine Clinic, 245 (6.4%) were due to foot and ankle injuries. Upon analysis, the most commonly injured region was found to be the ankle (46.9%) and of those patients 40.8% were diagnosed with “ankle sprain”.

Bridgman *et al.* (2003) estimated that in the UK there were 302 000 new ankle sprains and 42 000 new, severe, ankle sprains reported every year. In congruence with this and according to Kannus and Renstrom (1991 in Hertel, 2002) it is estimated that in the United States more than 23 000 ankle sprains occur per day which equates to one sprain per 10 000 people daily. In the South African context, studies have shown that the incidence and prevalence of ankle sprains also accounts for a large number of injuries within the population groups under study (Needham, 2001:78 and Pellow and Brantingham, 2001).

Garrick (1977) reported that there was no sex predilection for experiencing an ankle sprain. Beynnon *et al.* (2002) also found that gender does not appear to be a risk factor for suffering an ankle sprain.

The different profile of sporting activities within a population results in a varying incidence of ankle ligament injuries (Karlsson *et al.*, 2003:540). Running and jumping sports e.g.: soccer, basketball and volleyball, are high risk activities for ankle ligament injuries (Karlsson *et al.*, 2003:540).

In a study carried out by Lysens *et al.* (1984) students with a previous history of injury were at a higher risk of re-injury and certain injuries, especially sprains, tended to recur. Lysens *et al.* (1984) found that out of 162 reported ankle sprains, 72 (44.5%) were re-injured within the 4 year study.

In a study, conducted by Woods *et al.* (2003) over two competitive football seasons it was found that ankle ligament sprains accounted for 11% of total injuries with 77% of these involving the lateral ankle ligaments, the anterior talofibular ligament being involved in 73% of cases. A total of 12 138 days and 2033 matches were missed as a result of ankle sprains, this equates to an average of 18 days and three games missed per sprain. Furthermore it was found by Woods *et al.* (2003) that the incidence rather than the severity of the ankle sprain made them more problematic injuries. They reported that those players experiencing re-injury missed on average more training days and matches than those with first time injuries and suggested that the rehabilitation period may have been too short resulting in a high re-injury rate (83% had less than one month rehabilitation) (Woods *et al.*, 2003).

2.3 Anatomy and Biomechanics of the ankle joint

The ankle complex is made up of 3 articulations: the ankle joint or more specifically the talocrural joint (Norkin and Levangie, 1992:381), the subtalar joint and the distal tibiofibular syndesmosis (Hertel, 2002).

According to Denegar and Miller (2002), the talocrural joint is a highly congruent joint located between the talus and the mortise created by the distal tibiofibular joint. It is a synovial hinge type of joint. The mortise is a deep three sided socket (formed by the two malleoli and the inferior end of the tibia) into which the trochlea of the talus fits (Moore, 1992:488). The trochlea is wider anterior than it is posterior (Mack, 1982), convex from anterior to posterior and slightly concave from side to side (Moore, 1992:488).

The stability of the talocrural joint depends on joint congruency; supporting ligaments (Hockenbury and Sammarco, 2001) and musculotendinous support (Hertel, 2002). During dorsiflexion of the foot the talus moves from anterior to posterior and becomes “locked” into the mortise (Baker and Todd, 1995). In this position the ankle joint is considered stable (Baker and Todd, 1995) and very strong (Moore, 1992:489). When the foot is plantarflexed the talus is “unlocked” (Baker and Todd, 1995) as the trochlea of the talus moves anterior in the mortise (Moore, 1992:489). In this position the ankle is considered to be fairly unstable and in full plantarflexion of the foot some side movement can be demonstrated (Moore, 1992:489).

Strong collateral ligaments on either side of the ankle joint support the thin fibrous capsule of the ankle joint (Gray, 1984:491-492). The lateral ligaments, although strong, are weaker than the medial deltoid ligaments (Moore, 1992:489). Lateral stability to the ankle joint is provided for by three ligaments that attach the lateral malleolus to the talus and calcaneus (Moore, 1992:489). They are the anterior talofibular ligament (ATFL), posterior talofibular ligament (PTFL) and the calcaneofibular ligament (CFL) (Hockenbury and Sammarco, 2001). These ligaments provide resistance against inversion and internal rotation stress (Hockenbury and Sammarco, 2001).

The ATFL is a weak flat ligament extending from the lateral malleolus to the neck of the talus (Moore, 1992:488-489) and is a thickening of the lateral capsule (Bassewitz and Shapiro, 1997). A round cord, the CFL, runs postero-inferiorly from the tip of the lateral malleolus to the lateral surface of the calcaneus (Moore, 1992:488-489). The ATFL and CFL work together to support the lateral ankle (Bassewitz and Shapiro, 1997). In the plantarflexed position the ATFL is aligned vertically and under tension, and in the dorsiflexed position the CFL is vertically aligned and under tension (Bassewitz and Shapiro, 1997).

The PTFL is the strongest of the lateral ligaments and resists forward dislocation of the leg and the foot (Mack, 1982). The PTFL runs horizontally medially and slightly posteriorly from the malleolar fossa to the lateral tubercle of the posterior process of the talus (Moore, 1992:489).

Crossing the CFL superficially are the tendons of the fibularis longus and brevis muscles (Moore, 1992:489) which pass distal and inferior to the lateral malleolus (Mack, 1982). These tendons further contribute to the ankle stability by being tightly bound down by thickenings of

the deep fascia called the retinacula (Moore, 1992:489) and function as pronators and everters of the foot (Mack, 1982). The tendon sheath of these muscles covers the posterior and lateral aspect of the CFL, hence when this ligament is injured the inner wall of the tendon sheath is also injured. There is an intricate relationship between these tendons and the stabilizing ligaments where the tendons are capable of absorbing stress and protecting the ligaments from injury (Mack, 1982).

The subtalar joint, a synovial joint (Moore, 1992:490), is located between the talus and the calcaneus (Moore, 1992:490 and Hertel, 2002). Also known as the talocalcaneal joint, the function of this joint is to dampen rotational forces imposed by body weight in the weight-bearing position (Norkin and Levangie, 1992:388). This joint has ample strong ligaments that make it a very stable joint that very rarely dislocates (Norkin and Levangie, 1992:388).

The distal tibiofibular joint is a fibrous syndesmotomic joint (Moore, 1992:487) located between the concave facet of the tibia and the convex facet of the fibula (Norkin and Levangie, 1992:383). The distal tibiofibular joint ligaments are responsible for primarily restricting movement at both the proximal and distal tibiofibular joints and maintain mortise stability (Norkin and Levangie, 1992:383). According to Norkin and Levangie (1992:383) the function of the talocrural joint is dependent on the tibiofibular mortise. If the tibia and fibula were allowed to separate or if one side of the mortise was missing the tibia and fibula would be unable to grasp and hold onto the distal joint segment (Norkin and Levangie, 1992:381).

According to Reid (2002:220) the bony configuration of the ankle joint, fibular movement and the peroneal muscles all play an important synergistic role with the ankle ligaments. Reid (1992:220) concludes that if these biomechanical and anatomic relations are considered then the ATFL is probably one of the most important stabilizing constituents of the lateral ligamentous apparatus. This implies that an isolated tear of the ATFL is not a minor injury and can lead to marked chronic rotational instability if the best possible treatment is not received (Reid, 1992:220)

2.4 Mechanism of injury

According to Mack (1982) bony stability in the neutral position is greater laterally than medially therefore predisposing an individual's ankle towards inversion rather than eversion. Once inversion has begun the ankle loses its bony stability, and the medial malleolus instead of functioning to stabilize the foot becomes a fulcrum for further inversion (Mack, 1982 and Karlsson *et al.*, 2003:541). If the peroneal muscles are not strong enough, the tensile strength

of the lateral ligaments may be exceeded (Mack, 1982). These lateral ligaments are responsible for providing stability in this position of reduced bony stability (Anderson, 2002).

If, at the time of stress, the foot is in the plantarflexed position then the ATFL is affected (Cailliet, 1988:357). In this position the ATFL becomes vertically aligned, parallel to the fibula and therefore under tension (Bassewitz and Shapiro, 1997). In the dorsiflexed or neutral position the CFL sustains injury (Cailliet, 1988:356) as in this position the CFL is in line with the fibula and under tension (Bassewitz and Shapiro, 1997). Hence these ligaments are variably injured depending on ankle position at the time of stress (Bassewitz and Shapiro, 1997).

The ATFL is the most vulnerable of the lateral ankle ligaments (Karlsson *et al.*, 2003:541). Lateral ankle ligament injury most commonly occurs when the ankle undergoes inversion and internal rotation with the ankle in various degrees of plantarflexion (Bennett, 1994), while the tibia simultaneously rotates outward (Karlsson *et al.*, 2003:541). Hence the ATFL, being more exposed, and under greater tension during plantarflexion is the most commonly injured ligament (Bassewitz and Shapiro, 1997). While injury frequently affects the ATFL, the CFL may also be injured, and occasionally the PTFL may also be involved (Brantingham *et al.*, 1993).

According to Reid (1992:220) a major mechanism of injury is landing from a jump, particularly when landing on another player's foot. Other mechanisms of injury include abrupt changing of direction especially if associated with deceleration (Reid, 1992:220), walking on uneven surfaces or stepping in a hole (Kuwada, 1995).

Risk factors for ankle sprains include the following:

- Previous ankle sprain: According to Beynnon *et al.* (2002) ankle ligament sprains result in disruption of a ligament, compromising an important biomechanical stabilizer. Tropp *et al.* (1985) found that players with a history of ankle problems experienced more sprains than those with no previous history.
- Height and weight: These 2 factors have been implicated as risk factors (Beynnon, *et al.*, 2002) as an increase in either factor proportionally increases the amount of inversion torque that needs to be resisted by the ligaments and muscles that span the ankle complex.
- Tight Achilles tendon: The gastrocnemius and soleus tendons exert a pull on the ankle joint via the calcaneus (Reid, 1992:221). This has the tendency to pull the

calcaneus into slight inversion thus increasing the likelihood of landing on the outside of the foot during jumping (Reid, 1992:221).

- Crossover gait: In this position the foot is excessively supinated during heel strike (Forcum, 1997:484).
- Proprioception and muscle strength: Proprioception is the ability to feel or determine the ankle's position in space and the capabilities of the muscles to react to changes (Forcum, 1997:484). According to Hintermann (1999 in Anderson, 2002) impaired proprioception is associated with functional instability and recurrent sprains. In a study by Jerosch and Bischof (1996) a correlation between functional instability and proprioceptive deficit was also found following an ankle sprain. When a joint moves impulses arise from three areas: muscle, fascial tendon and articular receptors, and injury to any of these areas results in sensory deficit (Mattacola and Dwyer, 2002).
- Muscle imbalances: Weak everters (Fibularis muscles) or tight inverters (tibialis anterior and posterior) may also contribute to an ankle sprain (Vizniak and Carnes, 2004: 191).
- Anatomical variation: A genu varum, tibia varum or rearfoot varum can also contribute to improper foot landing (Vizniak and Carnes, 2004: 191). Also a varus heel or tarsal coalition may predispose to ankle injuries (Reid, 1992:221).
- Generalized ligament laxity may contribute to ankle instability (Reid, 1992:221).
- Shoes: According Forcum (1997:484) shoes that are worn out postero-laterally predispose the foot towards inversion sprains. In addition to this shoes that are too narrow so that the lateral aspect of the foot bulges over the lateral aspect of the shoe can also position the foot in an inverted position (Forcum, 1997:484)
- Playing surfaces: Uneven surfaces or potholes increase the risk of having an ankle sprain (Reid, 1992:220-221).
- Bracing and taping: There is a decrease in ankle injury potential for athletes who have ankle taping and bracing (Reid, 1992:220 and Beynnon *et al.*, 2002)
- Limb dominance: The literature is divided with regards to limb dominance as a risk factor for ankle sprains, but perhaps it could be implicated as a risk factor as most athletes tend to place a greater demand on their dominant limb (Beynnon *et al.*, 2002).

Chronic ankle sprains can occur in activities, like ballet, which result in gradual stretching of the ligaments because of poor postural habits and weak muscles controlling the subtalar joint (Reid, 1992:222). Some sports (football and soccer) can result in chronic bone and joint changes due to the stresses and forces placed on the players' ankle (Reid, 1992:220).

2.5 Grading of ankle injuries and the clinical presentation of Chronic Ankle Instability Syndrome

In terms of the inversion ankle sprain, three grades exist (Reid, 1992:226):

- Grade I (mild): no haemorrhage, minimal swelling, point tenderness, negative anterior drawer sign and no varus laxity.
- Grade II (moderate): some haemorrhage, localized swelling, less defined margins of the Achilles tendon, anterior drawer sign may be positive, and no varus laxity.
- Grade III (severe): diffuse swelling on both sides of the Achilles tendon, early haemorrhage, tenderness may occur medially and laterally, positive anterior drawer sign and positive varus laxity.

Grade I injuries result in a mild stretch of a single ligament (Forcum, 1997:485) and these patients can bear weight on the ankle immediately after the injury (Garrick and Schelkun, 1997). Some tearing of the ligaments occurs in a grade II injury, with more swelling and the patient can generally bear some weight (Garrick and Schelkun, 1997). Forcum (1997:485) mentions that a complete tear of the ATFL or a partial tear to both the ATFL and the CFL can occur in a grade II injury. Generally a grade III injury involves the complete tearing of one or more of the ligaments and the patient presents with significant swelling and bruising and may show functional and clinical instability of the ankle (Garrick and Schelkun, 1997). The patient is unable to bear full body weight, and often bearing even partial body weight is painful (Forcum, 1997:485). Forcum (1997:485) believes that in a grade III injury there exists tearing of both the ATFL and the CFL, complete tears to the anterior capsule and tibiofibular ligament. From the above it can be noted that differences exist in the grading of ankle sprains and sometimes the margins that delineate different grades can be blurred.

Hertling and Kessler (1996:424) reported that following the initial injury of the ankle some patients will suffer recurrent “giving way “of the ankle followed by pain and swelling. Three possible causes of this were considered; healing of the ligament with adherence to adjacent tissues, loss of protective reflex muscle stabilization and gross mechanical instability (Hertling and Kessler, 1996:424-425). The adherence to adjacent tissues has been hypothesised to result in limited hypomobility of the joint (dependant on the direction and area of scar tissue development), whereas hypermobility would result in those areas not tethered by the scar tissue or controlled by the ligaments normally presenting with this function (viz. they have been torn by the injury – e.g. ATFL). This results in instability that could contribute to an unstable ankle (Hertling and Kessler, 1996:424-425).

In clinical practice, ongoing symptoms following an ankle ligamentous injury are frequently seen (Patel and Warren, 1999:332). Most of these continuing symptoms include pain, instability, crepitus, weakness and stiffness (Pellow and Brantingham, 2001). Swelling is another residual symptom (Patel and Warren, 1999:332) and according to Esterson (1979) in Tatro-Adams *et al.* (1995) typically occurs around the ATFL, CFL and the anterior tibiofibular ligament.

These continuing symptoms are thought to be related to untreated ligament damage (Pellow and Brantingham, 2001 and Reid, 1992:250). The main causes of these symptoms are functional instability, a tight, sensitive scar, incomplete rehabilitation and stiffness from a loss of fibula and subtalar motion (Reid, 1992:250).

Functional instability is a subjective description of symptoms experienced by the patient (Karlsson *et al.*, 2003:543) The patient may describe a feeling of “looseness” or “giving way” of the ankle more commonly than pain and they may have a history of recurring inversion injuries to the ankle (Patel and Warren, 1999:332). Stepping off curbs or sudden stops and starts may cause the ankle to give way and result in pain (Baker and Todd, 1995). A patient who presents with chronic functional ankle instability clinically presents differently if compared to a patient experiencing an acute sprain (Baker and Todd, 1995). The swelling in chronic functional instability is generally more diffuse and ecchymosis is absent (Baker and Todd, 1995). Functional instability is a complex syndrome in which the exact etiologic factors are unknown. It is thought to arise from increased ligamentous laxity, inhibition of proprioceptive function and peroneal muscle weakness (Karlsson *et al.*, 2003:543). Lateral instability can present with swelling, lateral pain, tenderness and recurrent feelings of giving way (Bassewitz and Shapiro, 1997).

Following an ankle sprain fibrous connective tissue can form in the antero-lateral and antero-medial ankle which can be a source of chronic antero-lateral pain when the fibrosis becomes pinched during dorsiflexion (Bassewitz and Shapiro, 1997). Impingement syndrome should be considered if the pain persists longer than 6 months despite appropriate conservative care and if the pain is worse on dorsiflexion (Bassewitz and Shapiro, 1997). Scar tissue debridement is a recommended form of treatment in this case (Bassewitz and Shapiro, 1997).

When the ligament heals with adherence to surrounding tissues it becomes tightened and with repetitive stress, pain and swelling subsequently develop. Occasionally a forceful stress

placed on the ankle, ruptures these adhesions and the person experiences another sprain. Treatment for this type of patient presentation consists of deep transverse friction massage to the ligament in the direction of the adhesions, thus gradually restoring normal mobility (Hertling and Kessler, 1996: 424).

Therefore in this study only patients who have experienced grade I or II acute ankle inversion sprains and who have subsequently developed any continuing symptoms more than 6 weeks later shall be included. This clinical presentation that presents as a result of the prior ankle sprains will for the purposes of this study be termed a chronic ankle instability syndrome. This term is necessary in order to avoid confusion that the term “chronic ankle sprain” implies as this is a different clinical entity.

In this respect and for purposes of clarity “chronic ankle sprains” are generally interpreted as “repeated spraining of the ankle to such a degree that it is common to have patients presenting with an acute on chronic sprain”, thus making it difficult to delineate clinically when a sprain is chronic, acute or acute on chronic. Chronic ankle sprain is one symptom (instability) of chronic ankle instability syndrome. Therefore chronic ankle instability syndrome is a more accurate description as the results of the above clinical phenomena allow for more of an accurate clinical diagnosis and syndrome identification and therefore greater relevance of study outcomes. The results of chronic ankle instability syndrome are recurrent “giving way” of the ankle followed by pain and swelling, (Hertling and Kessler, 1996:424), instability, crepitus, weakness and stiffness (Pellow and Brantingham, 2001).

2.6 Differential diagnosis

Although common, an ankle inversion sprain and its resulting chronic ankle instability syndrome, needs to be differentiated from a number of other conditions that occur at the lateral ankle joint. Specific injuries that can occur at the lateral ankle joint and tests which can be performed in order to rule them out include:

1. Osteochondritis dessicans

- Mortise view ankle x-rays (Trojian and McKeag, 1998 and Stokes and Western, 2001)
- If the original radiographs are negative and joint pain continues, a second set 6-12 weeks after injury should be taken (Bassewitz and Shapiro, 1997)
- The patients may also complain of night pain and swelling (Stokes and Western, 2001).

2. Peroneal tendon subluxation or dislocation. Caused by disruption of the retinaculum or fracture of the distal fibula
 - Palpate over the tendon with resisted dorsiflexion and eversion. Tendon will be felt to subluxate or it will cause pain. The patient reports pain with walking and with walking on the balls of the feet (Trojian and McKeag, 1998). Pain and tenderness is generally posterior to the lateral malleolus (Anderson, 2002).
3. Lateral malleolar fracture
 - X-rays and Ottawa ankle rules (Trojian and McKeag, 1998).
4. Bifurcate ligament injury or avulsion fracture
 - The point of maximal tenderness is found midway on a line connecting the tuberosity of the fifth metatarsal and the distal tip of the lateral malleolus (Trojian and McKeag, 1998).
5. Tibiofibular syndesmosis sprain
 - Palpation over the anterior inferior tibiofibular ligament produces pain (Alonso *et al.*, 1998).
 - Squeeze test: by squeezing tibia and fibula together above midcalf produces pain distally at the syndesmosis (Alonso *et al.*, 1998).
 - External rotation stress: with the knee flexed to 90° and the ankle in neutral, stabilize the leg and externally rotate the ankle. This produces pain over the syndesmosis (Trojian and McKeag, 1998).
 - Side to side test: place ankle in neutral, stabilize tibia and fibula with one hand, hold heel with the other and apply lateral and medial forces. A positive test is confirmed with an audible thud or pain (Trojian and McKeag, 1998).
 - Dorsiflexion compression test: patient is standing and dorsiflexes once unassisted and once with the therapist applying a manual compressive support around the malleoli. A positive is indicated by either an increase in the ankle range of motion or a decrease in the end of range pain with added compression (Alonso *et al.*, 1998).
 - External rotation stress x-rays - >5mm widening of the tibiofibular clear space indicates complete rupture (Trojian and McKeag, 1998).
6. Flexor hallucis longus injury
 - Palpation of the sheath with active and passive ranges of motion of the hallux will reproduce symptoms (Trojian and McKeag, 1998).
7. Lateral periostitis
 - Lateral talus palpation with the foot in plantarflexion and inversion can elicit pain. Symptoms are similar to a lateral sprain but there is no ATFL tenderness (Trojian and McKeag, 1998).

8. Os trigonum injury

- Forceful passive plantarflexion causes lateral posterior triangle pain. Resisted eversion will be pain free (Trojian and McKeag, 1998).

9. Anterior tibialis injury

- Pain on the dorsum of the foot with foot drop if complete avulsion occurs and resisted dorsiflexion is weak or tender (Trojian and McKeag, 1998).

10. Sinus tarsi syndrome

- Pressure placed over the sinus tarsi can elicit exquisite pain (Klausner and McKeigue, 2000).
- Symptomatic relief on injection of 2-3ml of local anaesthetic into the tarsal canal is diagnostic (Klausner and McKeigue, 2000).
- Demonstrated ankle instability using the anterior drawer test or excessive talar tilt on inversion will rule out sinus tarsi syndrome (Klausner and McKeigue, 2000).

11. Synovial impingement

- Impingement sign: patient supine. To examine the left foot the examiner's right hand grasps the patient's calcaneus with fingers placed around the calcaneal tuberosity and the thumb over the antero-lateral part of the ankle. The left hand then grasps the forefoot to control flexion at the ankle. The foot is initially held in plantarflexion and an antero-lateral pressure is applied with the examining thumb. This may produce some pain because of synovial hypertrophy, but is not sensitive or specific. With continued thumb pressure the foot is moved from plantarflexion to dorsiflexion. Any hypertrophic synovium present will be forced into the joint by the examiner's thumb and be impinged. Dorsiflexion with no thumb pressure should not be painful. A positive result is pain on dorsiflexion with thumb pressure (Molloy *et al.*, 2003).

12. Achilles tendon rupture

- Patients would have felt a sharp pain in their Achilles and often state that it sounded like someone shot them (Trojian and McKeag, 1998).
- Thompson's test: The patient flexes their knee to 90°, and doctor squeezes middle third of the calf. A lack of plantarflexion indicates tendon rupture (Trojian and McKeag, 1998).

According to Reid (1992:224) the location of tenderness and swelling, obvious deformity, excessive edema, or increasing pain and swelling with weight-bearing should raise ones suspicions of a more complex diagnosis.

2.7 Treatment options available with elaboration on the treatment application in this study.

Failure to adequately treat an acute ankle injury can result in chronic ankle joint instability. Acute management involves the application of the RICE principle (rest, ice, compression and elevation), however the importance of rehabilitation cannot be overemphasized, as a lack of it could lead to persistent pain and swelling, decreased range of motion and chronic joint instability (Wolf *et al.* ,2003).

Various treatment regimes and modalities can be employed to achieve appropriate care of a patient with chronic ankle instability syndrome, these include manipulation (Pellow and Brantingham, 2001; Needham, 2001:109; Edmond, 1993:164), mobilization, (Hockenbury and Sammarco, 2001), ultrasound (Thomson, Skinner & Piercy, 1991:43-44), cryotherapy (Hockenbury and Sammarco, 2001 and Ogilvie-Harris and Gilbert, 1995), range of motion and strengthening exercises (Anderson, 2002; Reid, 1992:238) and deep transverse friction (Hertling and Kessler, 1996:424).

The GTIASTM is also a treatment that can be used in chronic ankle sprains^c. It is hypothesized by Hammer (2003(b):1) that the Graston Technique instruments enhance ones ability to detect and treat soft tissue lesions. It is believed that the treatment results in an induced yet controlled microtrauma, causing fibroblastic proliferation (Hammer, 2003(c):1). Thus starting and promoting the healing cascade.

According to Cyriax and Coldham (1984 as cited by Hammer(a), 2003:1) there is a four-fold effect of deep transverse friction: traumatic hyperemia, therapeutic movement, increased tissue perfusion and mechanoreceptor stimulation. This ultimately results in an inflammatory response and new collagen formation (Hammer, 2003(b):1). The rationale of deep transverse friction to induce inflammation is to bring about a fibroblastic proliferation which is necessary for the repair and regeneration of collagen (Hammer (2003(c):1). The friction massage results in microtrauma to an area of excessive soft tissue fibrosis or scar tissue (Hammer 2003(c):1).

^c “Chronic ankle sprains” are utilized here instead of “chronic ankle instability syndrome” as past research has been directed towards that of chronic ankle sprains (as delineated by the respective researchers). This unclear terminology and the use of varied definitions of “chronic ankle sprains” have made it almost impossible to interpret the findings of this research based on the fact that no one diagnostic parameter is present and consistent in the definitions applied.

Friction massage (Hammer 2003(c):1) can also be achieved by augmented soft tissue mobilization using special instruments.

As a result of Hammer's (2003) hypotheses, the focus of this study was on the treatment of scar tissue and adhesions within the ligament. Following injury to the lateral ankle ligaments scar tissue may develop in the ATFL and joint capsule which leads to the formation of "meniscoid tissue" (Hockenbury and Sammarco, 2001). Hertling and Kessler (1996:424) also mention that the injured ligaments could heal with adherence to adjacent tissues. Scar tissue within the ligaments or joint capsule could cause chronic ankle pain (Hockenbury and Sammarco, 2001). Repetitive stress to the tightened structure will result in pain and swelling (Hertling and Kessler, 1996:424).

In chronic conditions the GTIASTM can be used more aggressively to promote tissue mobility and to control the amount of tissue inflammation (Carey, 2003:32). The GTIASTM is believed to bring about improved recovery, patient satisfaction and improved quality of life for the patient. For the clinician it is said to provide enhanced patient care and improved ability to detect soft tissue lesions. (Carey, 2003:9).

In order to critically and objectively evaluate the effect that the GTIASTM may or may not have on a patient's chronic ankle instability syndrome symptoms it is necessary for a placebo to be used, as was used by Pellow and Brantingham (2001). According to Roberts *et al.* (1993, as cited by Dogan, 2003:53) the effective outcome of placebo treatment could be "high" or "excellent" in 40% of the participants in the placebo group under study, with an "average outcome" in 30% and "poor outcome" in the remaining 30% of placebo participants. In effect, therefore, the placebo treatment within the placebo group is not biased to either the "excellent" or "poor". Keating (1987) goes on to mention that the use of placebo treatments in Chiropractic research indicates that all healing methods involve a placebo effect. This allows placebo to be another effective tool in comparing another unproved treatment.

2.8 Conclusion

In conclusion, scar tissue and adhesions could contribute to a patient's chronic ankle instability syndrome symptoms (particularly instability and pain). The GTIASTM claims to break down scar tissue and adhesions. It is therefore the purpose of this research to compare the relative effectiveness of the GTIASTM against a placebo (detuned ultrasound) in order to determine whether the GTIASTM does break down scar tissue and adhesions and to correlate these findings, if any, with changes in the patients' pain and edema levels

CHAPTER THREE

METHODOLOGY

3.1 Introduction

Reproducibility of results is a requirement of modern science. It is therefore important to describe the methodology of this study, so that it can be repeated if need be. This chapter focuses on the design of the study, the sampling procedure, the interventions that were applied and the data collected, as well as the statistical analysis applied to the data.

This study was a quantitative, parallel group placebo controlled clinical trial conducted in order to determine the effectiveness of the Graston Technique instrument assisted soft tissue mobilization (GTIASTM) in the reduction of scar tissue for the management of chronic ankle instability syndrome following an ankle inversion sprain. This study compared two groups of patients. One group received GTIASTM while the other group received placebo treatment in the form of detuned ultrasound.

3.2 Study Design

3.2.1 Patient recruitment and Advertising

Patients were selected from those who respond to advertisements (Appendix 11) placed in public places, sport clubs, notice boards around the Durban Institute of Technology, pamphlet distribution and word of mouth. The study was available to any person who could arrive for treatments at the Chiropractic Day Clinic at the Durban Institute of Technology and who was prepared to travel to Pietermaritzburg (PMB) twice for measurement purposes. The patient profile was not limited to a specific group of people (gender, race or occupation).

Initially the patients were screened via a telephonic interview to determine their suitability for the research in terms of the following criteria:

- If the patient was currently on any medication or treatment for their ankle symptoms and wanted to continue that treatment then they were excluded from the study.
- How long ago did the patient injure their ankle? The patient must have injured their ankle at least 6 weeks earlier.
- Was the patient diagnosed as having had an ankle inversion sprain?
- Age of the patient. Only patients between 18-50 years of age were included in this study

- Location of any pain in the patients' foot. The patient should have initially and periodically following the sprain have experienced pain on the lateral aspect of their foot around the lateral malleolus.
- Do they have any contraindications to treatment as stipulated below?
- How did the patient injure their ankle? Were they able to weight bear immediately after the injury or not? Did they hear a pop or snapping sound? Majority of ankle inversion sprains occur in the plantarflexed and inverted foot position (Patel and Warren, 1999:325). Landing from a jump (Anderson, 2002) or changing directions while running, especially on uneven surfaces may also be another mechanism of injury (Reid, 1992:220). If the patient was unable to weight bear immediately after the injury then a more serious injury and possible fracture was suspected (Trojian and McKeag, 1998). An audible sound or popping, snapping or cracking sensation often signifies a significant tear (Reid, 1992:222). In both these circumstances where a more serious tear was suspected the patients were excluded from the study.
- Rate of onset and amount of swelling. Although not a reliable sign, rapid onset of swelling may indicate a severe injury (Reid, 1992:222). In a grade I injury the swelling is mild and located to one side of the Achilles tendon. Following a grade II injury there is more localized swelling causing blurring to the margins of the Achilles tendon. Diffuse swelling on either side of the Achilles tendon with loss of definition indicates a grade III injury (Vizniak and Carnes, 2004: 190). Grade III injuries were excluded from the study.
- Does the person ever feel as if their foot is going to "give way"? What movements cause this to happen? One of the residual symptoms following an ankle sprain is the sensation of "giving way" (Louwerens and Snijders, 1999:343). This indicates functional ankle instability (Reid, 1992:252) and the person becomes apprehensive when walking on rough ground (Garrick and Schelkun, 1997). Patients with functional ankle instability were included in the study.
- Where the patient lives. Only patients who lived in and around Durban were selected for the study. Patients who lived in PMB were also allowed to participate in this study provided they travelled to Durban at least twice a week for their appointments.
- Do they have access to transport to and from the DIT Chiropractic Clinic? No transport was provided to appointments scheduled at the Chiropractic Clinic. Transport was, however, provided to and from St Anne's X-ray Department in Pietermaritzburg if the need arose.

3.2.2 Sampling technique

A non-probability purposive sampling technique was used to recruit patients. The study was limited to those patients who experienced chronic ankle instability syndrome and any other continuing symptoms, fulfilled the necessary inclusion criteria as set out below, and resided in the KwaZulu-Natal Province.

The study was limited to 30 ankles with chronic ankle instability syndrome.

3.2.3 Patient procedure:

Patients that were accepted post the telephonic interview, were sent to PMB for a diagnostic ultrasound scan of their injured ankle. Once the presence and amount of scar tissue had been determined (Appendix 10) the patients were then given a letter of information (Appendix 1) and an informed consent form (Appendix 2) prior to the commencement of the first consultation. If the patient did not wish to further participate in the study at this stage then they could withdraw immediately.

Patient suitability was then further determined at the initial consultation with the researcher during which a full history (Appendix 3), physical examination (Appendix 4), foot and ankle regional examination (Appendix 5), and the inclusion and exclusion criteria set out below were conducted.

3.2.4 Inclusion and exclusion criteria

3.2.4.1 The following inclusion criteria were used in the study:

- The patient must be between 18 and 50 years of age (Pellow and Brantingham, 2001).
- The patient was required to give informed consent before any treatment was to be administered and they must have agreed by signing the informed consent form. (Appendix 2) (Pellow and Brantingham, 2001).
- No restrictions were placed with respect to race or gender, patient occupation, or income bracket (Pellow and Brantingham, 2001).
- All patients were required to have been diagnosed with previous ankle inversion sprain (Pellow and Brantingham, 2001).
- The diagnosis for this study was based on the history of the most recent sprain and any continuing symptoms of (Pellow and Brantingham, 2001):
 1. Instability
 2. Pain

3. Crepitus
4. Weakness
5. Stiffness
6. Edema (Patel and Warren, 1999:332)

If 4 out of 6 of the above symptoms are experienced, then it was taken/ understood that the patient has chronic ankle instability syndrome.

- The patient must have experienced the following clinical symptoms.

Subjectively all these should have been present:

1. A history of an acute sprain (Hertling and Kessler, 1996: 743); usually after landing awkwardly on their own or an opponent's foot, they may also have felt or heard a snap or pop and experienced pain along the lateral side of the ankle (Bassewitz and Shapiro, 1997).
2. One or more episodes of "giving way" during activities involving jumping or quick lateral movements (Hertling and Kessler, 1996: 743). Difficulty and apprehension with walking on rough ground may also have been experienced (Garrick and Schelkun, 1997).
3. Subsequent ankle sprains resulted in pain, swelling and dysfunction that were not as severe as that occurring with the initial injury (Hertling and Kessler, 1996: 743).

Variable objective findings may have been found depending on the causative factors and may not necessarily all have been present (Hertling and Kessler, 1996: 743):

1. Hypermobility of anterior glide of the talus in the mortise indicating true structural instability,
 2. Hypomobility or pain on the anterior glide of the talus in the mortise may indicate residual ligamentous adhesions, and
 3. Poor balance reactions on one-legged standing may indicate an alteration in proprioceptive neuromuscular protective response.
- Objectively the patient must also have been found to have scar tissue on the initial diagnostic ultrasound.

3.2.4.2 *The following exclusion criteria were used in the study:*

- Patients who experienced an acute injury or acute re-injury were excluded from the study (Pellow and Brantingham, 2001).
- Patients taking any medications (NSAIDS, other analgesics, anticoagulant therapy, muscle relaxants, anti-inflammatory or any drugs for arthritides) or undergoing any other modes of treatment for their ankle injury were excluded. Patients were instructed

not to initiate any other forms of treatment while taking part in the study (Pellow and Brantingham, 2001).

- Patients showing signs of gross mechanical ankle instability (grade III ankle sprain) and syndesmosis injury were excluded from the study (Pellow and Brantingham, 2001).
- Patients who demonstrated any allergies to the ultrasound therapy gel or contraindications to the Graston technique on the basis of case history, physical examination, foot and ankle examination were excluded.

Absolute and relative contraindications for use of the Graston Technique tools:

- Absolute contraindications:
 - open wounds, unhealed fractures, thrombophlebitis, uncontrolled hypertension, patient intolerance/non-compliance/hypersensitivity, hematoma, osteomyelitis, myositis ossificans
- Relative contraindications:
 - anti-coagulant medication, cancer, varicose veins, burn scars, acute inflammatory conditions (eg: synovitis), kidney dysfunction, inflammatory condition secondary to infection, rheumatoid arthritis, pregnancy or osteoporosis

(Carey, 2003:11)

- Diabetics were excluded from this study as faulty wound healing in this patient population occurs as a result of abnormal inflammatory pathways, peripheral neuropathy and vascular disease (Sharman, 2003:1).
- Any patient who has Peripheral Vascular Disease (Longmore *et al.*, 2001: 478) or Peripheral Neuropathy Diseases (Tener, 2004:1) for any reason were excluded from the study as these conditions are associated with sensory disturbances.
- Any patient who was not naïve to ultrasound therapy. This indicates that any patient included in the study should not have received either modality within 12 months preceding the study (Assendelft *et al.*, 1992).
- Any patient not prepared to sign the indemnity form from the Durban Institute of Technology (Appendix 2) or if the permission to be treated (PTT) section of the appropriate paperwork was not signed by the consulting clinician.

3.2.5 Sample selection and group allocation

Once chosen, the patients were systematically allocated into one of 2 groups. All even numbered patients were allocated to group 1 and all odd numbered patients were allocated

to group 2. Group 1 received detuned ultrasound (placebo) to the lateral ankle ligaments and Group 2 received GTIASTM (instruments number 3 and 6) (Appendix 7A).

3.3 Treatment Application

3.3.1 Placebo treatment

Patients in Group 1 received placebo treatment, using detuned ultrasound over the lateral ankle, with the patient facing away from the machine. As described by Pellow and Brantingham (2001) patients in the placebo group received detuned ultrasound for 5 minutes per treatment session. This was done so as to remove any possible direct mechanical changes to the patients injured ankle (Pellow and Brantingham, 2001). Placebo treatment is a form of treatment often given as an aid to therapeutic suggestion and for eliminating bias. It does not matter what the placebo is made of, how much is used or for what length of time. What is important is that the patient does not become aware at any stage that a placebo is being used (Beecher, 1955).

The advantage of using detuned ultrasound as the placebo treatment is that there is a form of metallic contact with the skin much the same as the Graston instruments would provide. Ultrasound is also similar in that it uses a gel and Graston uses an emollient to decrease friction between the skin and the instruments (Carey, 2003:27). Further to the above, other treatment methods have the potential to bring about changes to scar tissue whereas detuned ultrasound, being a placebo should have no affect on scar tissue and therefore is an appropriate placebo treatment that would not interfere with any results and therefore obscure the changes in scar tissue that are being measured in response to the GTIASTM instruments (Mouton, 2001:106).

3.3.2 Graston Technique Instrument assisted soft tissue mobilization treatment

Patients allocated to Group 2 received GTIASTM. The Graston Technique tools (instruments number 3 and 6 (Appendix 7A)) were applied to the site of the lesion and applied in multiple directions maintaining an application angle of 30-60 degrees to allow for maximum fibre separation and contouring of the instrument to the body. The patient was in a relaxed and comfortable position with their ankle being fully exposed and supported. Having the ankle fully supported reduced muscle activity and allowed for more effective treatment. The stroke rate over this small area was fairly quick, but it was reduced if the patient experienced higher pain levels. The ideal treatment duration is between 4-8 sessions allowing approximately 3-5

minutes for each treatment. In this research the patients each received a total of 8 treatments (Carey, 2003:27-29).

3.3.3 Intervention frequency

Initially the patients travelled to Pietermaritzburg for a diagnostic ultrasound scan. The patients, once accepted into the study, then each received a total of 8 treatments with two treatments per week over 4 week's duration. Following these 8 treatments the patients then made one final trip to Pietermaritzburg for a repeat scan.

3.4 Measurements

Subjective measurements were recorded using the Foot Function Index (Appendix 8) at the first and final consultations. Objective measurements were recorded using the Algometer (Appendix 9A), Figure-of-Eight edema measures (Appendix 9B) and the Diagnostic ultrasound scans (Appendix 10). Algometer readings were taken at the first treatment and at the start of the fifth and final treatments. Figure-of-Eight edema measures were recorded at the first and final treatments. Diagnostic ultrasound scans were taken before the initial consultation and repeated within one week of the final consultation.

The data was collated and used for inter and intra group analysis to determine the effect that detuned ultrasound and the GTIASTM had on any scar tissue present. It was then necessary to correlate this with the patients' pain levels and ankle edema.

3.4.1 Objective measurements

3.4.1.1 Diagnostic ultrasound measurements

Diagnostic ultrasound can be used to evaluate the physiological state of soft tissues like tendons and ligaments in the foot (Cohen, 2004:1; Musculoskeletal / Spinal Ultrasound, 2004:1). It also allows the patients to understand the source of their discomfort, can establish baseline pathology, and can be useful when reassessment of the pathology is necessary to establish any improvement (Musculoskeletal / Spinal Ultrasound, 2004:1).

Diagnostic ultrasound is considered an "excellent tool" when evaluating the foot and ankle, allowing dynamic imaging in multiple planes and positions (Fessel and van Holsbeeck, 1999). A high frequency transducer is needed (7.5 MHz or higher) for evaluating the foot and ankle (Fessel and van Holsbeeck, 1999).

The thickness and integrity of the injured ligament (in this case the ATFL) was assessed by a radiologist at St Anne's X-ray department. If the ligament was found to have scar tissue then its thickness was measured. The radiologist was blinded with respect to which treatment the patients had received. The diagnostic ultrasound machine that was used was an APLIO ultrasound machine from Toshiba. A high resolution 14 megahertz probe was used in this study

The limitations of diagnostic ultrasound for soft tissue evaluation include operator dependence (Boyse *et al.*, 2000 and Fessel and van Holsbeeck, 1999). In this study the same radiologist, who was blinded to the study, examined patients before and post treatment. This assisted with consistency and aided in the elimination of operator bias.

3.4.1.2 Figure-of-eight measure for ankle swelling

The figure-of-eight method is a commonly used and reliable method of measuring ankle swelling and was reproduced using bony landmarks. A tape measure was used to span each of the anatomical sites where swelling in ankle sprains typically occurs: the ATFL, CFL and the anterior tibiofibular ligament (Tatro-Adams *et al.*, 1995).

A tape measure was wrapped around the patients' ankle joint in the following manner:

1. The patient was seated comfortably in a long sitting position with both feet extended beyond the end of the bed to the level of the patients mid calf.
2. The foot to be measured was maintained in a neutral dorsiflexed position.
3. The beginning of the tape was placed midway between the tibialis anterior tendon and the lateral malleolus.
4. The tape was drawn medially across the instep and placed just distal to the tuberosity of the navicular.
5. The tape was pulled across the arch and up just proximal to the base of the 5th metatarsal.
6. The tape was then pulled across the tibialis anterior tendon.
7. The tape was continued around the ankle joint just distal to the distal tip of the medial malleolus,
8. pulled across the Achilles tendon and
9. placed just distal to the distal tip of the lateral malleolus.
10. The measurement was then ended at the start of the tape.

(Tatro-Adams *et al.*, 1995)

3.4.1.3 Algometer measurements

Algometer pain measurements (Appendix 9A) were conducted at the first consultation, before the 5th treatment and before the 8th treatment in order to determine if there were any changes in the patients' pain levels. The algometer used in this study was a force dial manufactured by Wagner Instruments: P.O Box 1217, Greenwich, CT 06836, USA.

The algometer was used in order to determine pressure threshold (the minimum pressure inducing pain) and was carried out in a similar manner to Dogan (2003:62) and Blake (2003:34):

1. The procedure of increasing pressure over the area was explained to the patient and the patient was instructed to inform the examiner as soon as pain was experienced.
2. The examiner located the area of maximal tenderness over the anterior talofibular ligament by palpation.
3. The algometer was set to zero and placed at a 90° angle vertical to the skin. Pressure was slowly and continuously applied at an even rate until the patient indicated pain.
4. The reading on the algometer was then taken and recorded in kg/cm². The higher the reading, the less the tenderness of the tissue (Fischer, 1986). A higher reading therefore shows an improvement (Fischer, 1986).

3.4.2 Subjective measurements

3.4.2.1 Foot Function Index (FFI)

The FFI (Appendix 8) was used to gather information on the impact that the patients' foot pain had had on their daily activities. It was originally developed for use among the elderly with rheumatoid arthritis to measure pain, disability and activity restriction (Budiman-Mak *et al.*, 1991). The FFI used in this study comprised 12 questions that were scored from 0 (equivalent to no pain at all) to 10 (equivalent to the worst pain imaginable). Two more questions were qualitative and answered with a yes or no answer.

3.5 Statistical analysis

SPSS version 11.5 was used to analyse the data (SPSS Inc, Chicago, Ill, USA).

Demographics were compared between treatment groups using chi squared tests for categorical variables and independent samples t-tests for quantitative variables.

Repeated measures ANOVA was used to examine changes in quantitative outcomes over the time points (intragroup analysis) and a treatment effect (intergroup analysis). To control for the partial pairing in the intergroup analysis, a variable which classified each subject as paired (both left and right ankle used in study) or non-paired (only used once in study) was used as a factor in the model. Profile plots were generated to visually assess group and time changes.

Correlations between the intragroup changes in the various outcome variables were assessed using Pearson's correlation coefficients.

A two-tailed p value of <0.05 was considered as statistically significant.

CHAPTER FOUR

RESULTS AND DISCUSSION

4.0 Introduction

This Chapter describes the results of the study and evaluates their meaning through the discussion. Intra-group evaluation will reflect any indication of significant differences in the subjective and objective findings within each treatment group (that is GTIASTM and placebo). The data was obtained at the beginning of the first, fifth and final consultations. Inter-group analysis will indicate any significant differences between the two treatment groups with respect to data obtained at the beginning of the first, fifth and final consultations.

Objective data was obtained from the:

- Diagnostic ultrasound
- Algometer readings
- Figure-of-eight method for ankle swelling

Subjective data was obtained from the:

- Foot function index (FFI)

Key

Treatment groups: Both the GTIASTM group and the placebo group.

df: degrees of freedom

n: no. of people in sample group

P: probability that the null hypothesis is correct. The lower the value of p the greater the chance of rejection. Therefore if the p value is <0.05 the null hypothesis is rejected and the test is significant.

Sig: Significance

Std: Standard

T: Time

4.1 Demographics by treatment group

Although subjects were systematically assigned to treatment groups, there was no significant difference in demographic variables between the two groups (Tables 1b and 2). Table 1a shows that the proportions were similar in each treatment group. Table 2a shows the mean ages of participants in the two groups were not significantly higher in the GTIASTM group than the placebo group. Table 2 b shows that the number of years since initial injury was not significantly different between the two groups ($p = 0.534$). Similarly there was no significant difference in mean number of years since the latest injury ($p = 0.308$) (Table 2c).

Table 1a: Demographic variables by treatment group

		Treatment group			
		GTIASTM		Placebo	
		Count	Column %	Count	Column %
SEX	Female	7	46.7%	5	33.3%
	Male	8	53.3%	10	66.7%
RACE	African	1	6.7%	1	6.7%
	Indian	0	.0%	1	6.7%
	White	14	93.3%	13	86.7%
Left or Right ankle	Left	7	46.7%	7	46.7%
	Right	8	53.3%	8	53.3%

According to Louwerens and Snijders (1999: 341) the incidence of ankle injuries in the young male population is higher, but after 40 years of age the incidence for females increases more than males. Other literature, however, suggests that gender predilection does not appear to be a risk factor for experiencing an ankle sprain (Beynon *et al.*, 2002 and Garrick, 1977). In a study by Fallat *et al.* (1998) 54.8% of the patients were male and 45.2% were female. In the present study there was no significant difference demonstrated (Table 1b) between the treatment groups and in total 60% of the patients were male and 40% were female, which were equally distributed between the groups. Therefore although not totally in congruence with the literature, these data imply that the trends are similar.

Ethnic distribution did also not appear to be significantly different between treatment groups. There were a high percentage of white patients (90%) within the study that were fairly evenly distributed between the two groups. Indian patients accounted for only 3.33% of patients and African patients made up the remaining 6.67%. Needham's study (2001:78) similarly had a large percentage of white patients (68%) compared to Indians (17%), Africans (12%) and

Coloured patients (3%). Even though these studies indicate a similar ethnic distribution, this is not consistent with the general population of the South African province of KwaZulu-Natal (<http://www.statssa.gov.za/census2001/digiAtlas/index.html>). Reasons for this could include the lack of understanding by non English speaking populations as to the scope and practice of Chiropractic which is limited to the English speaking population that is familiar with western culture and has been exposed to health care disciplines outside of the traditional medicine or allopathic medicines that have dominated in South Africa, and thus in KwaZulu-Natal. In addition to this the need to travel between two centers (Durban and Pietermaritzburg) for purposes of the readings required in this study, could have resulted in a sample that is representative only of the highly mobile portion of the population, which does not necessarily reflect the general population of KwaZulu-Natal.

Table 1b: Pearson Chi-Square Tests

		Treatment group
SEX	Chi-square	.556
	df	1
	Sig.	.456
RACE	Chi-square	1.037
	df	2
	Sig.	.595(a,b)
Left/Right ankle	Chi-square	.000
	df	1
	Sig.	1.000

Results are based on non empty rows and columns in each innermost subtable.

a More than 20% of cells in this subtable have expected cell counts less than 5. Chi-square results may be invalid.

b The minimum expected cell count in this subtable is less than one. Chi-square results may be invalid.

Table 2a: Independent samples t-test for comparison of mean age of participants between treatment groups

Treatment group	N	Mean	Std. Deviation	Std. Error Mean	P
GTIASTM	15	31.80	9.112	2.353	0.340
Placebo	15	28.80	7.757	2.003	

From the tables above it can be seen that there was no significant difference of mean age between the two treatment groups. This speaks well to the homogeneity achieved between the two groups which is significant in terms of the healing time that different ages have in respect of ligament damage (Longmore *et al.*, 2001:522). It has been noted that most age-related biologic functions peak at < 30 years of age (The Merck Manual, 1999:2503). If, as in

this case, the ages in the groups were homogenous, it would rule out differences due to rate of healing as a result of age in the improvement or lack of improvement shown by one or either group and thus limit the distortion of the results, especially in view of the fact that age is one factor which affects the mechanical properties of ligaments (Akeson *et al.*, 1995:23).

Fallat *et al.* (1998) found the average age of acute injuries to be 34 years. In contrast to this and according to Boruta *et al.* (1990, as cited by Louwerens and Snijders, 1999:341) most ankle sprains occur within the 15-35 year age group. This contrast may be due to the fact that the 2 references cited above looked at different aspects of ankle sprains (e.g. acute or chronic). This is supported by Dogan (2003:75), who in his study of chronic ankle sprains had a mean age of 26.75 and 26.15 between his two treatment groups. Thus it would seem that chronic sprains occur at an older age and acute sprains at a younger age.

In this light and considering that this study looked at “a chronic ankle instability syndrome”, which was not defined as an acute, acute on chronic or chronic sprain, but indicated a chronic presence of signs and symptoms post the event of recurrent ankle sprains, it stands to reason that the patients would need to be older than those experiencing the initial acute injuries. In addition a current re-injury to the ankle, for purposes of this study, would have had to occur at least 6 weeks prior to having been accepted onto this study.

Thus the mean age of this study can therefore be considered to be fairly representative of the general ankle injury age, with a tendency towards the age range for chronic ankle sprains (Dogan, 2003:75 and Boruta *et al.*, (1990) as cited by Louwerens and Snijders, 1999:341).

Table 2b: Independent samples t-test for comparison of mean years since initial injury of participants between treatment groups

Treatment group	N	Mean	Std. Deviation	Std. Error Mean	P
GTIASTM	15	8.8000	5.73461	1.48067	0.534
Placebo	15	7.5333	5.27618	1.36230	

The significance of having had similar time elapsed since the original injury is important as there has therefore been a similar degree of resolution that has taken place within the injured ligament and thus the possibility of the distortion of the results would have been limited . A torn ligament takes 4 months to heal and will strengthen to only 70% of the original strength

(Lachmann and Jenner, 1994:28) as the remodelling as part of the healing of the ligament occurs, which Kellet (1986, in Norris, 1998:37) indicates may last up to 12 months.

Table 2c: Independent samples t-test for comparison of mean years since latest injury of participants between treatment groups

Treatment group	N	Mean	Std. Deviation	Std. Error Mean	P
GTIASTM	10	1.4000	1.34990	.42687	0.308
Placebo	11	2.5455	3.20511	.96638	

Although there is a slight difference in mean years since the most recent injury it was not found to be significant (Table 2c). This phenomenon was however noted as a possible factor that could have contributed to a difference in the improvement rate between the two groups – favouring the GTIASTM group, where active healing was more likely still to be occurring and at a faster rate than the placebo group (Lachmann and Jenner, 1994:28 Kellet (1986) in Norris, 1998:37). This effect would be better seen if the sample sizes where larger. Thus it is recommended that a study with a larger sample size would be beneficial for the purposes of elucidating this trend more accurately.

4.2 Comparison of outcomes over time and by treatment group

4.2.1 Algometer measurements

4.2.1.1 Intragroup analysis

GTIASTM group

There was a significant change over the three time points in the GTIASTM group for algometer readings ($p=0.001$). The profile plot in Figure 1 shows that the mean scores for this group actually decreased between T1 and T2, but increased to a large degree between T2 and T3. Repeated contrasts showed the time effect to only be significant from T2 to T3 ($p<0.001$) compared to T1 to T2 (0.320).

Table 3: Repeated measures ANOVA intragroup time comparison for GTIASTM group for algometer readings.

Time effect Wilk's lambda	p value
0.313	0.001

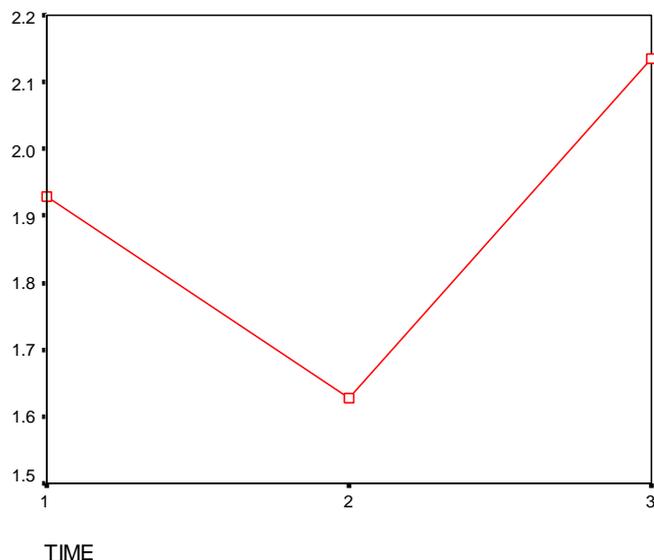


Figure 1: Profile plot of mean algometer readings in the GTIASTM group over time.

In the context of the above results, it must be remembered that the algometer with a 1cm² disc surface, as was used in this study, can be used effectively for the measurement of deep tissue tenderness in ligaments, muscles, tendons and joint capsules (Fischer, 1986).

As noted in figure 1 the mean scores taken between T1 and T2 decreased. This indicates that an increase in the patients' pain levels was experienced by those in the GTIASTM group.

When considering the adhesions present within the anterior talofibular ligament at the start of the research, it must be considered that this was a possible cause of the patients' original pain and inflammation (T1) as the tissue was not "sufficiently deformable enough to attenuate the energy of loading" from use of the ligament (Kessler, 1990:139). In addition to this the inability of the ankle joint to perform its normal function would have been limited (Mrozek and Vernon, 2005) thereby causing changes within the movement often to the detriment of the joint, leading to reactive inflammation, swelling and pain.

The effect of GTISATM is to bring about a controlled micro trauma (Hammer, 2003(c):1) which is thought to reinitiate the inflammatory process to allow for proper healing and tissue remodelling (Carey, 2003:31). This would perhaps account for the increase in the patients pain levels experienced within the first 4 treatments (T2). Also it must be considered that the treatment can result in temporary bruising that is often experienced from GTIASTM which results from localized microtrauma and breakdown of associated scar tissue (Carey, 2003:37). According to Carey (2003:37) soft tissue which has been traumatized is more susceptible to bruising which could possibly account for the development of the measured increase in the original pain. It is thought that this is a sign that scar tissue, adhesions and restrictions have been released (Carey, 2003:37).

After T2 the pain levels experienced by the patients' were seen to improve. Following the release of the adhesions the patients progress often improves markedly as tissue remodelling and strengthening can occur without the constraint of the adhesions, scar tissue and restrictions (Carey, 2003:37, Hammer, 2003(c):1 and Kessler, 1990:85). This is consistent with Wolff's law, which indicates that bones remodel according to their imposed demands (Lawrence and Bergmann, 1993:38). Akeson *et al.* (1995:29) believe that this law should not only be applied to bone but rather generalized to include connective tissue adaptation to applied stress, in other words living tissue responds to chronic changes in stress and strain (Leach, 1994:207). Furthermore Akeson *et al.* (1995:29) states that ligaments are morphologically, biochemically, and biomechanically sensitive to stress. Thus it would stand to reason that following the induced inflammation and adhesion release, the collagen in the ligament is remodelled and laid down along the lines of stress. Between T2 and T3 proper healing of the ligament is now presumed to be occurring.

In addition to this it would also be possible that there is an increased normalization of movement within the joint due to lack of adhesions present (Mrozek and Vernon, 2005). Synovial joints have large mechanoreceptors (Wyke type I, II and III) which, following movement normalization, it could be argued, are maximally stimulated (Leach 1994:90). This stimulation would allow for increased firing of the large mechanoreceptive fibers, which are thought to assist in inhibiting pain cycles through the mechanism of the gate control theory as proposed by Melzack and Wall (1965:971-979).

In this respect the gate control theory of pain as proposed by Melzack and Wall (1965) is used by therapists to decrease pain by increasing large-fiber input into the substantia gelatinosa (SG) (Hertling and Kessler, 1990:47-48). Massage and similarly friction massage

which can be compared to the GTIASTM is said to stimulate large-fiber input into the SG and thereby cause a decrease in pain (Lynch and Kessler, 1990:48).

Placebo group

There was no significant change over time for the placebo group algometer readings ($p = 0.453$ in Table 4). Repeated contrasts showed that neither T1 to T2 ($p = 0.458$) or T2 to T3 ($p=0.273$) showed a significant change. The profile plot in Figure 2 shows that there was an overall small scale increase in this group.

Table 4: Repeated measures ANOVA intragroup time comparison for Placebo group for algometer readings.

Time effect Wilk's lambda	p value
0.885	0.453

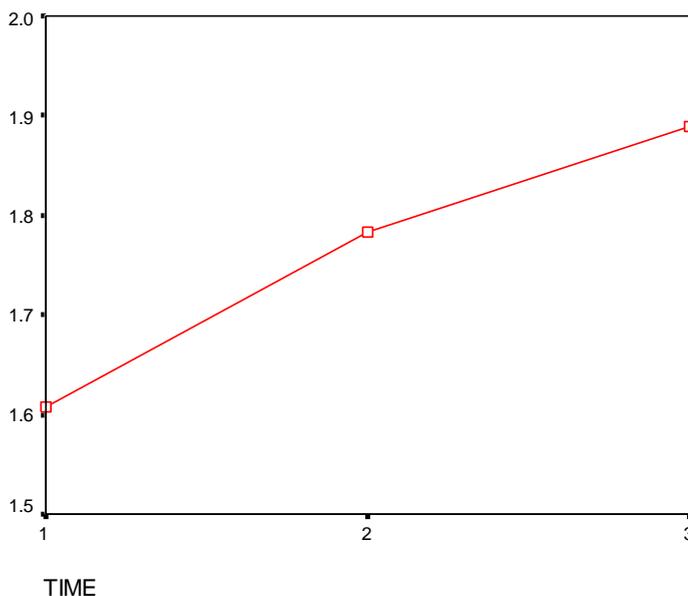


Figure 2: Profile plot of mean algometer readings over time in placebo group

Although a small decrease in the patients' pain levels was experienced in the placebo group it was not significant ($p=0.453$). Therefore the suggested mechanisms for improvement as found in the GTIASTM group is not applicable and thereby supports the mechanisms suggested.

It is however noted that there is a small rise in algometer readings, which could be attributed to the following:

- Natural history dictates that with time the injury will heal and strengthen (Lachmann and Jenner, 1994:28).
- The Hawthorne effect where human beings when they know they are being studied behave in an atypical manner (Mouton, 2002:152).
- As already mentioned the gate control theory could be another aspect responsible for a decrease in pain levels. Mechanoreceptors in the skin over the anterior talofibular ligament may have been stimulated by the touch of the ultrasound machine over the area. Transcutaneous nerve stimulation results in large-fiber input which abolishes or decreases pain levels (Lynch and Kessler, 1990:48).
- The doctor-patient interaction must also be considered as 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).

In the presence of an initiating disorder (in this case the pain from the scar tissue) which is present for a specified length of time or has/continues to have sufficient stimulus of the pain fibers, allows for the development of segmental spinal cord facilitation (Leach, 1994:101). This facilitation remains even if the source of stimulation is removed. This abnormal segmental reflex circuit maintains the symptoms (Leach, 1994:101) as per the syndrome of chronic ankle instability. In context the placebo group does not receive sufficient stimulation to reset the abnormal reflex circuits which would impede the improvement of signs and symptoms (e.g. pain) thereby implying a negligible increase in the tolerance of pain measured by the algometer.

4.2.1.2 Intergroup analysis

Table 5 shows that for algometer readings there was a significant treatment effect ($p = 0.005$). In the presence of an interaction, one cannot interpret the main effects. Examining the profile plot in Figure 3 shows that the GTIASTM group increased at a faster rate overall than the placebo group.

Table 5: Repeated measures ANOVA within and between subjects effects for algometer readings

Effect	Wilks' lambda	p value
Time	0.575	0.001
Group	F= 0.639	0.431
Time*group	0.658	0.005

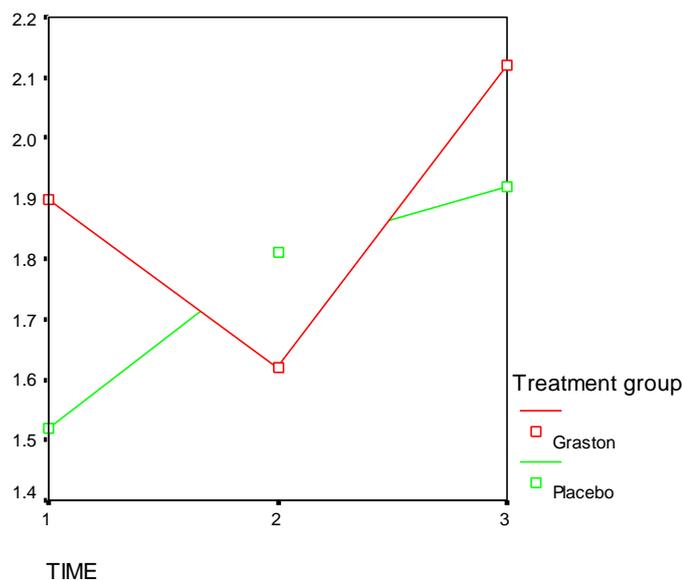


Figure 3: Profile plot of mean algometer readings over time by treatment group

A significant treatment effect ($p = 0.005$) was seen when comparing the two treatment groups. When comparing the two graphs in figure 3 one can see that the inferences made for the intra group analysis for pain measures still hold, but one cannot state that they are absolute. It is therefore recommended by the researcher of this study that more readings are taken on a more frequent basis in order to confirm or refute what has been stated, as increased readings will allow for differentiation of the mechanisms by virtue of time and the effect treatment has on the healing rate.

4.2.2 Edema

4.2.2.1 Intragroup analysis

GTIASTM group

There was a significant change over time for edema in the GTIASTM group ($p = 0.01$). The profile plot in Figure 4 shows that this was a decrease in edema over time.

Table 6: Repeated measures ANOVA intragroup time comparison for GTIASTM group for edema.

Time effect Wilk's lambda	p value
0.616	0.010

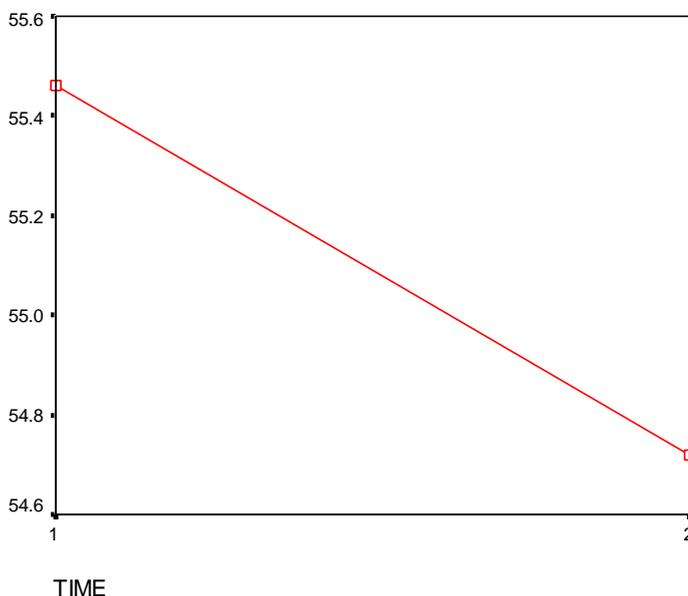


Figure 4: Profile plot of edema readings in the GTIASTM group over time

As a result of previous trauma, the articular nerve fibers within the injured ligament have been damaged which results in ankle instability (Norris, 1998:311). Following the initial injury, the patient may experience repeated episodes of giving way” (Patel and Warren, 1999:332) resulting in mild swelling (Louwerens and Snijders, 1999:344), which is associated with “chronic ankle instability syndrome”.

Treatment in respect of friction massage can be likened to the GTIASTM and so in a similar fashion promotes increased interfiber mobility, reduces fibrous adhesions (Kessler, 1990:85) and aids the normal orientation of fibers as they are produced (Kessler, 1990:140). As a result, the normal mechanics of movement are restored to the ligament and surrounding structures which could cause a reduction in swelling. If fibrosis in ligaments can result in pain and inflammation (Kessler, 1990:139), then a breakdown of this fibrosis should have the effect of decreasing pain and inflammation and therefore a decrease in swelling.

Another aspect that cannot be ignored is resolution due to natural history of the condition. Chronic ankle instability syndrome does result in periodic swelling (Hertling and Kessler, 1996: 424) and depending on the time since the last actual re-injury or episode of “giving

way” this may have had an effect on the amount of swelling that was present at the time of measurement.

With only two readings been taken; one at the first consultation and another at the final consultation, the researcher can only hypothesize what happened with no conclusive confirmation. More frequent readings are recommended to challenge the following hypothesis.

Placebo group

There was no significant change over time for the placebo group ($p = 0.290$). Figure 5 shows a small scale decrease over time.

Table 7: Repeated measures ANOVA intragroup time comparison for placebo group for edema.

Time effect Wilk's lambda	p value
0.920	0.290

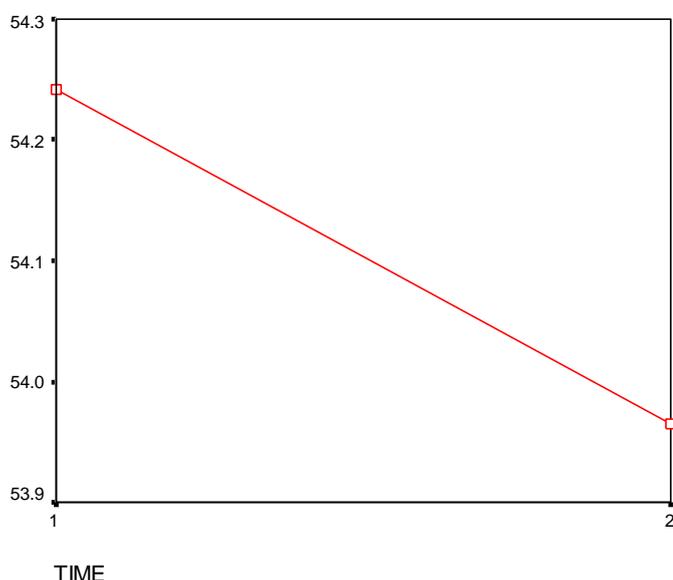


Figure 5: Profile plot of edema readings in the placebo group over time

In the placebo group a decrease in swelling was noticed which was not significant ($p= 0.290$) but perhaps attests to resolution due to natural history playing a role in patient improvement. With time swelling decreases as the injury is given more time to heal and the body compensates for the restrictions within the joint.

4.2.2.2 Intergroup analysis

There was no significant treatment effect ($p = 0.327$) although the overall time effect was significant ($p = 0.007$). This means that both groups changed over time at the same rate (i.e. the profiles were parallel). This is shown in Figure 6. There was a slight non significant trend towards a steeper rate of decline visible in the GTIASTM group relative to the placebo group.

Table 8: Repeated measures ANOVA within and between subjects effects for edema

Effect	Wilks' lambda	p value
Time	0.749	0.007
Group	F=0.678	0.418
Time*group	0.963	0.327

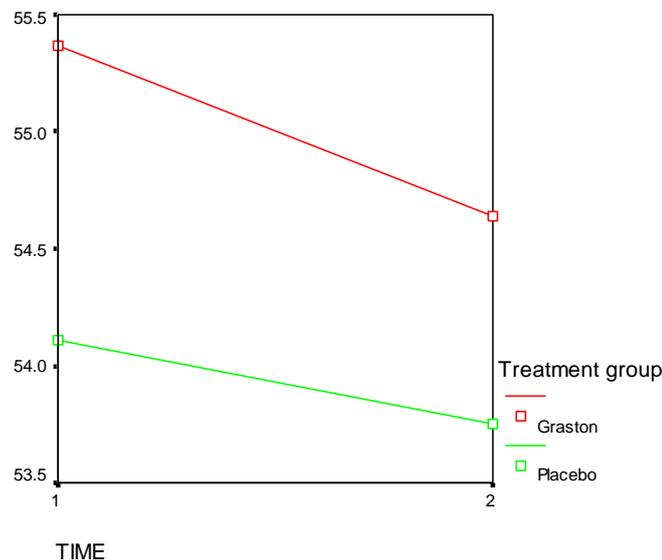


Figure 6: Profile plot of edema readings over time by treatment group

Once again both treatment groups demonstrated a decrease in ankle swelling over time. The GTIASTM group did have a faster rate of decline as already mentioned but this may be attributed to the fact that patients' in that group on average had younger injuries (refer to Table 2c). Consequently natural history dictates that their rate of resolution should decrease more rapidly.

It is recommended by the researcher that there be a decreased time over which treatment is given or an increase in patient numbers to counteract the natural history effect (i.e.: more people at different stages of their natural history would negate the natural history represented in the different groups).

In addition to the natural history of chronic ankle instability syndrome there are a couple of other factors that need to be taken into account when looking at ankle swelling. These factors are:

- Prolonged standing which causes edema in dependent limbs (Costanzo, 1995:89). Some of the research patients may have to stand for prolonged periods at work prior to being measured by the researcher.
- The research was carried out in summer with many hot humid days resulting in high body temperatures, decreased arteriolar resistance and therefore edema (Guyton, 1991:281).

4.2.3 Diagnostic Ultrasound: Proximal anterior talofibular ligament

4.2.3.1 Intragroup analysis

GTIASTM group

There was a significant time effect in the GTIASTM group ($p = 0.001$). Figure 7 shows that there was a steep decrease in scar tissue thickness.

Table 9: Repeated measures ANOVA intragroup time comparison for GTIASTM group for Proximal ultrasound.

Time effect Wilk's lambda	p value
0.425	0.001

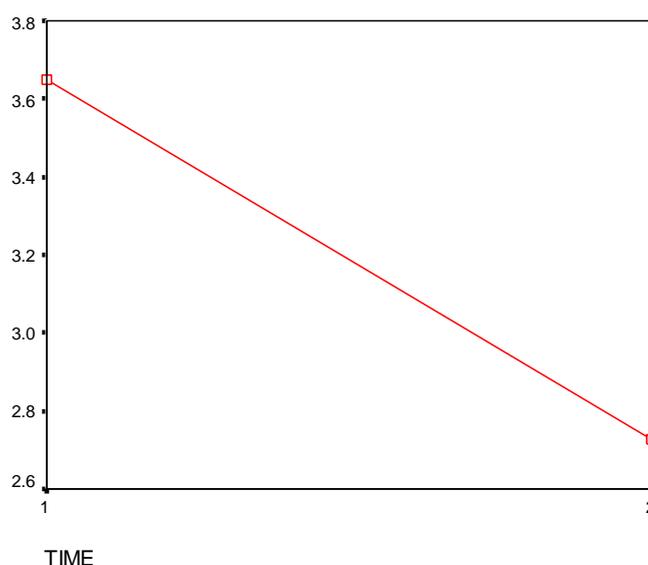


Figure 7: Profile plot of proximal ultrasound readings in the GTIASTM group over time

It was hypothesized that the GTIASTM may be an effective tool in the breakdown of scar tissue and adhesions. According to the above diagnostic ultrasound readings it would appear that this hypothesis holds true. It must be remembered that the diagnostic ultrasound only measures the thickness (in millimetres) of the ligament which would then indicate any changes that may have occurred within the ligament.

According to Hammer (2003(c):1) research conducted in Indiana examined the effects of friction massage. In the study researchers used augmented soft tissue mobilization which involves the use of specialized instruments to bring about a controlled microtrauma resulting in the breakdown of fibrosis (Hammer, 2003(c):1). Augmented soft tissue mobilization was the initial terminology used for the GTIASTM (Carey, 2003:63). Hertling and Kessler (1990:266) noted that the most acceptable method of scar modification is with the application of stress to the scar with one of those stresses being massage. Friction massage or GTIASTM is a form of massage and therefore should modify the scar tissue.

Nociceptors are found in the ligaments and joint capsule (Raja *et al.*, 1999:40) around the ankle. Scar tissue contracts as it matures (Lachmann and Jenner, 1994:28) and hinders normal joint movement (Iocono *et al.*, 1998:16) which has the potential to stimulate nociceptors and become a source of chronic pain. This may explain the improvement in patients' pain levels after the scar tissue was broken down.

Placebo group

There was no significant change over time in the placebo group ($p = 0.703$). Figure 8 below shows that the decrease was very small scale in the placebo group.

Table 10: Repeated measures ANOVA intragroup time comparison for placebo group for Proximal ultrasound.

Time effect Wilk's lambda	p value
0.988	0.703

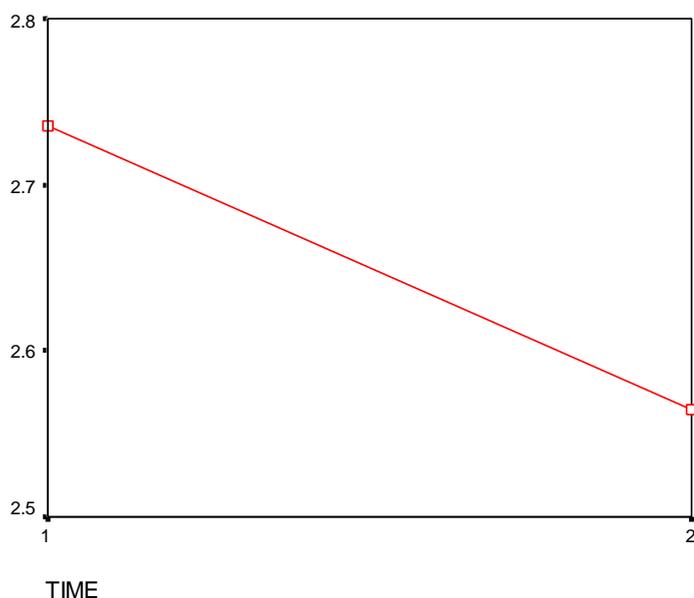


Figure 8: Profile plot of proximal ultrasound readings in the placebo group over time

The only possible explanation why the placebo group improved was that of natural history over time. Scar tissue contracts over time (Lachmann and Jenner, 1994:28), indicating a decrease in size, which could explain the changes in measures that were seen. However these changes occur slowly (Lachmann and Jenner, 1994:28) and therefore over a greater time span than would have been applicable in this study.

4.2.3.2 Intergroup analysis

There was no significant treatment effect ($p = 0.096$) although the profiles of the two groups appear to be non parallel, with the GTIASTM group experiencing a steeper decline over time than the placebo group (Figure 9). The group effect was significant ($p = 0.028$) meaning that the mean for the GTIASTM group was higher than that for the placebo group. This was not a treatment effect as it was due to the huge baseline differences between the groups.

Table 11: Repeated measures ANOVA within and between subjects effects for proximal ultrasound

Effect	Wilks' lambda	p value
Time	0.869	0.069
Group	F=3.137	0.028
Time*group	0.889	0.096

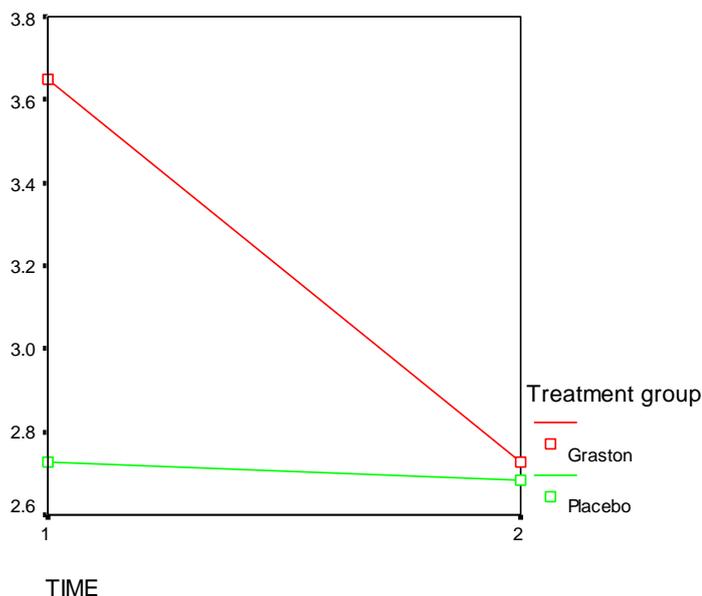


Figure 9: Profile plot of proximal ultrasound readings over time by treatment group

Following the systematic allocation of patients to the different treatment groups it was only later noticed that overall the GTIASTM group had a higher initial starting baseline. Although the GTIASTM group had a higher baseline difference in scar measures there still appeared to be a greater improvement within the GTIASTM group. This re-inforces the fact that scar tissue contraction over time (Lachmann and Jenner, 1994:28) could not have been the sole reason for improvement in the GTIASTM where it could have been in the placebo (as the rates of improvement shown above are completely different). This indicates that GTIASTM has a higher efficacy than the placebo.

4.2.4 Diagnostic Ultrasound: Mid anterior talofibular ligament

4.2.4.1 Intragroup analysis

GTIASTM group

There was a significant decrease over time in the GTIASTM group for this outcome measure ($p < 0.001$) as shown in Figure 10.

Table 12: Repeated measures ANOVA intragroup time comparison for GTIASTM group for mid ultrasound.

Time effect Wilk's lambda	p value
0.368	<0.001

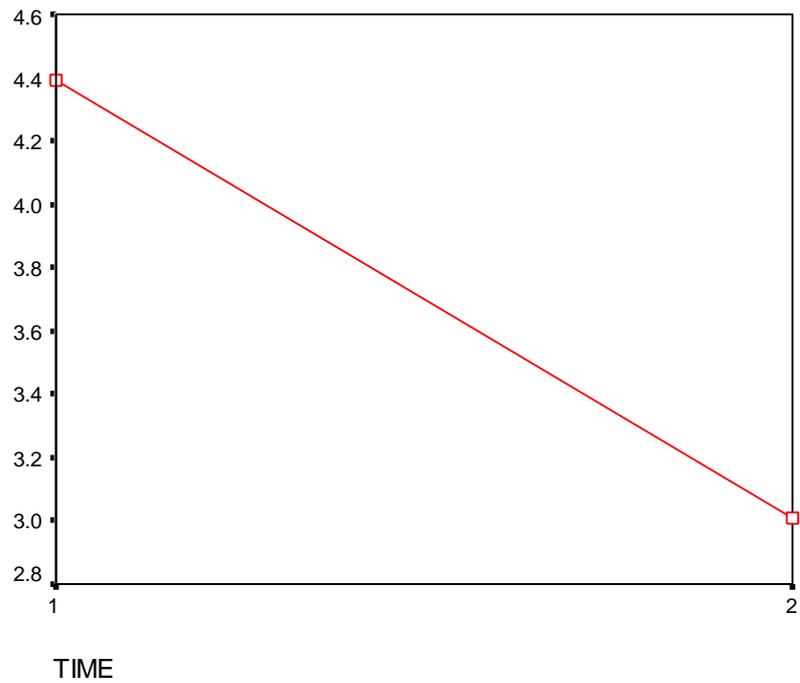


Figure 10: Profile plot of mid ultrasound readings in the GTIASTM group over time

Again there appears to be an improvement in the scar tissue measures which are noted as being significant ($p < 0.001$). This will be assessed when compared to the placebo group (Figure 12).

Placebo group

There was a significant decrease in values over the two time points in the placebo group ($p = 0.004$). This is shown in Figure 11.

Table 13: Repeated measures ANOVA intragroup time comparison for placebo group for Mid ultrasound.

Time effect Wilk's lambda	p value
0.484	0.004

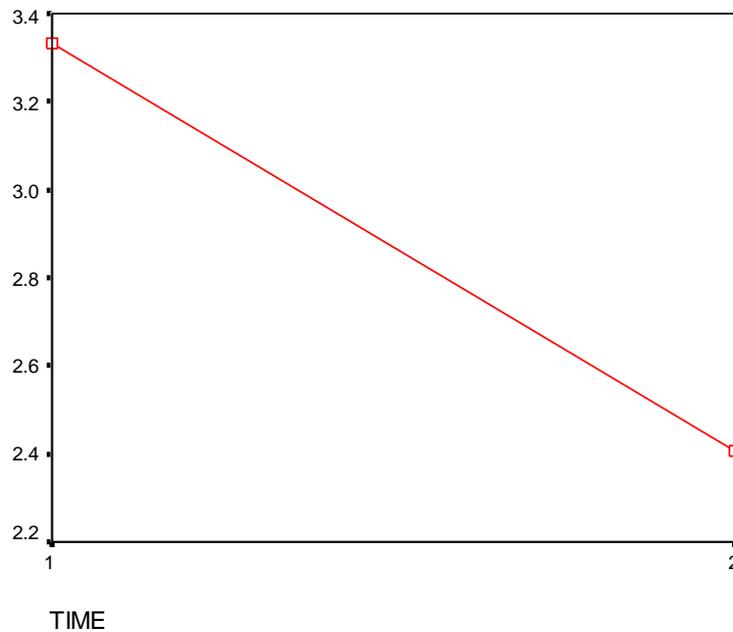


Figure 11: Profile plot of mid ultrasound readings in the Placebo group over time

Natural History of scar tissue contraction may once again have played a role here, as there was no active treatment in this group.

4.2.4.2 Intergroup analysis

There was no evidence of a treatment effect ($p = 0.305$) for mid ultrasound. There was a significant change over time in both groups ($p < 0.001$) and at both time points mean values in the GTIASTM group were higher than the placebo group (not an indication of treatment effect, rather baseline differences). Figure 12 shows that the slopes of the lines of the two groups were relatively parallel.

Table 14: Repeated measures ANOVA within and between subjects effects for Mid ultrasound

Effect	Wilks' lambda	p value
Time	0.393	<0.001
Group	F=8.361	0.010
Time*group	0.956	0.305

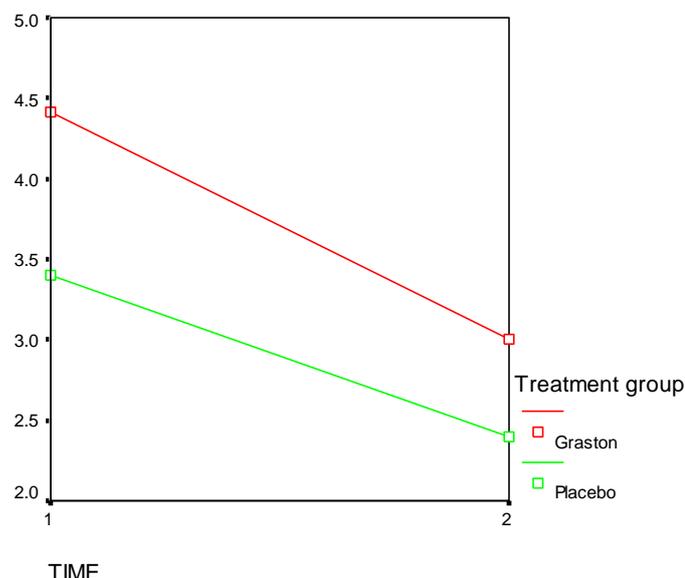


Figure 12: Profile plot of mid ultrasound readings over time by the treatment groups

There appears to be an improvement in the scar tissue measures which are noted as being significant ($p < 0.001$). These changes possibly indicate that there is a response to:

- The Graston Technique instruments.
- The natural history of the condition with the improvement in scar tissue naturally.
- It could also be related to scar tissue contraction over time (Lachmann and Jenner, 1994:28).

Taken together however from Figure 12 it can be seen that although the lines are not parallel the GTIASTM group does decrease marginally more than the placebo. Thus it would seem that the improvement that is greater in the Graston Technique group is as a result of the treatment.

4.2.5 Diagnostic Ultrasound: Distal anterior talofibular ligament

4.2.5.1 Intragroup analysis

GTIASTM group

Table 15 shows that the GTIASTM group showed a significant time effect ($p < 0.001$). Figure 13 shows that this was in the form of a decrease over time.

Table 15: Repeated measures ANOVA intragroup time comparison for GTIASTM group for distal ultrasound.

Time effect Wilk's lambda	p value
0.323	<0.001

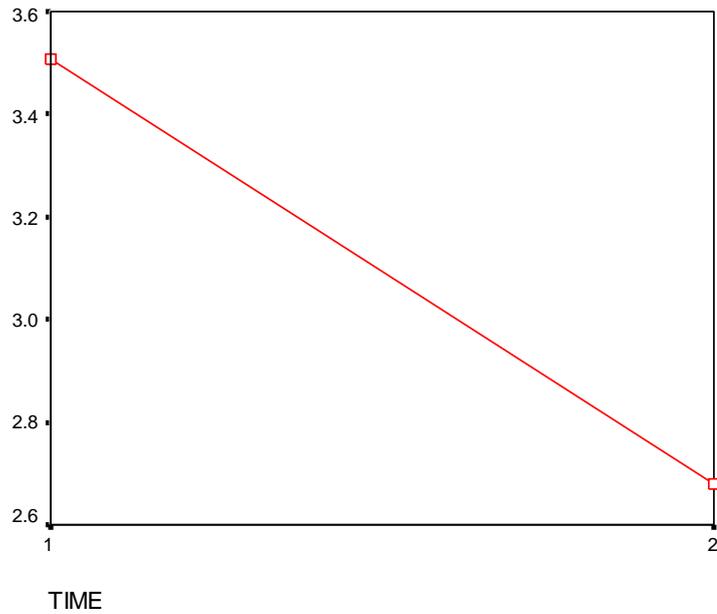


Figure 13: Profile plot of distal ultrasound readings in the GTIASTM group over time

The thickness of the scar tissue was still seen to decrease at the distal ligament. It was frequently noted by the radiologist that although there did appear to be a change in thickness of the ligament what was of greater change was the improvement in fiber quality throughout the ligament (de Villiers, 2005).

Placebo group

The placebo group showed a significant decrease in mean values over time ($p=0.028$) as evident in Table 16 and Figure 14.

Table 16: Repeated measures ANOVA intragroup time comparison for placebo group for distal ultrasound.

Time effect Wilk's lambda	p value
0.658	0.028

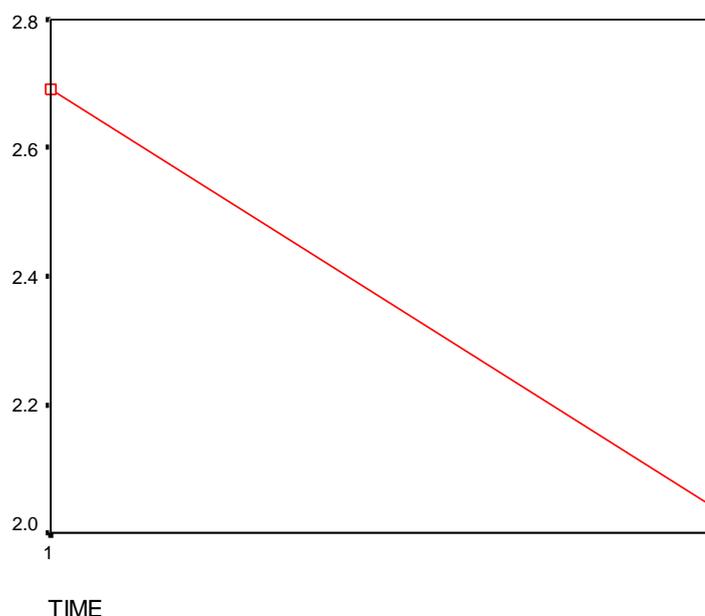


Figure 14: Profile plot of distal ultrasound readings in the Placebo group over time

4.2.5.2 Intergroup analysis

There was no evidence of a treatment effect for distal measurements ($p = 0.741$). However, both groups decreased to the same extent over time, and at both time points the mean values of the GTIASTM group were higher than the placebo group ($p = 0.004$). Figure 15 shows that the profiles of the two groups were parallel.

Table 17: Repeated measures ANOVA within and between subjects effects for distal ultrasound.

Effect	Wilks' lambda	p value
Time	0.488	<0.001
Group	F=10.263	0.004
Time*group	0.995	0.741

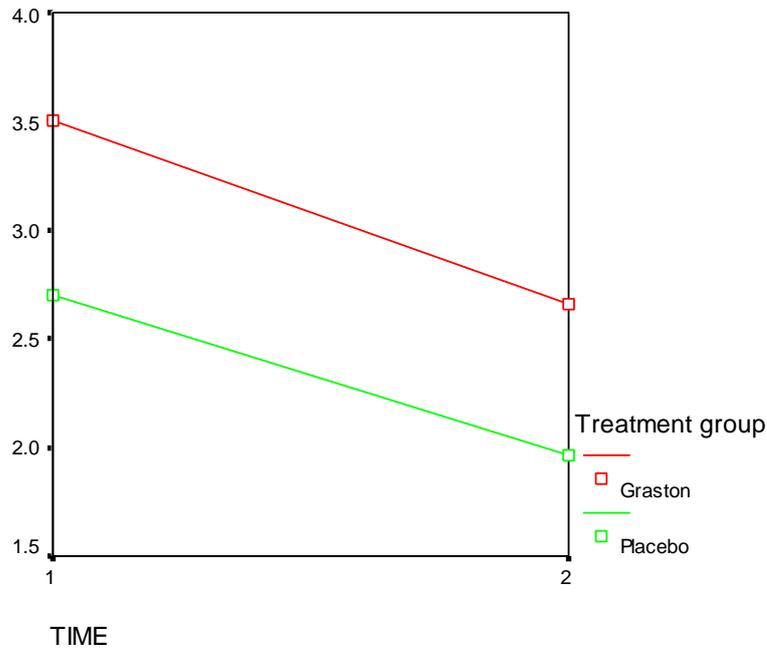


Figure 15: Profile plot of distal ultrasound readings over time by treatment group

4.2.5.3 Comparison of all the intergroup figures

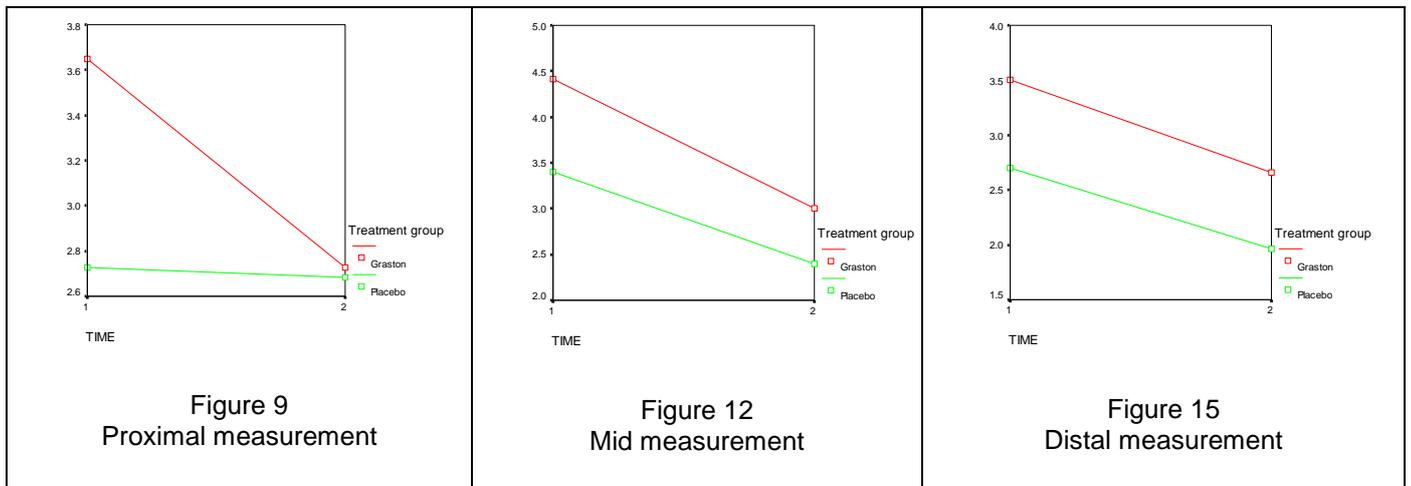


Figure 16: Summary Comparison of treatment groups for changes in the proximal, middle and distal Diagnostic ultrasound measures.

To conclude with regards to the diagnostic ultrasound readings it can be seen from Figure 16 that majority of the change occurred within the proximal portion of the ligament for the GTIASTM group. Although both treatment groups saw a reduction in ligament measures over time the proximal measure resulted in the most marked difference between the treatment groups.

The proximal portion of the ligament is the fibular attachment of the anterior talofibular ligament and the talus is the distal attachment. It is thought that there could be a difference in the amount of movement at the talar portion of the ligament as compared to the more stable fibular portion of the ligament, which accounts for the difference in healing. This is thought to be as a result of the degree of stress being highest at the talar portion of the ligament and therefore increased healing as according to Wolff's law (Lawrence and Bergmann, 1993:38, Akeson *et al.*, 1995:29 and Leach, 1994:207). Thus if the GTIASTM tools are applied one would expect the most change due to external application of treatment to occur at the fibular portion of the ligament.

The continuing symptoms of instability, pain, stiffness, crepitus, weakness (Pellow and Brantingham, 2001) and edema (Patel and Warren, 1999:332) following an acute lateral ankle sprain would indicate a joint or ligament pathology or both, the symptoms being consistent with re-injury. A normal ligament displays properties of a viscoelastic material (Norris, 1998:51) and therefore demonstrates creep (Norris, 1998:9) and hysteresis (Lawrence and Bergmann, 1993:43). Creep is the progressive deformation of a structure placed under a constant load, while hysteresis is the absorption of energy from a distorted structure (Lawrence and Bergmann, 1993:43-44). When structural stiffness occurs (as in scar tissue) the normal stress-strain curve becomes altered with the initial slope being steeper as less deformation can occur as stress increases (Norris, 1998:9). Scar tissue is as strong or even stronger than the ligament but is not elastic and so will not respond to stress in the same manner as the ligament (Norris, 1998:9). These two properties of viscoelastic materials which are therefore not found in scar tissue could be a possible explanation for reinjury (instability) and persistent pain following injury to the anterior talofibular ligament.

Perhaps an inference can be made that a proprioceptive improvement brought about through the reduction in scar tissue and the further normalization of the ligament may result in a decrease of re-injury rate seen in the GTIASTM group as opposed to the placebo group.

4.2.6. The Foot Function Index (FFI)

The Foot Function Index measures the effect that foot pathology has on function in terms of pain, difficulty and activity restriction. It is focused on the foot and should therefore have more precision and sensitivity than existing instruments. Although originally tested on patients with rheumatoid arthritis (RA), nothing in its design was specific for rheumatoid arthritis and therefore it is believed that it could be generalized to a non-rheumatoid arthritis population with foot pain. It is easily completed by the patient and provides a practical method for measuring foot function (Budiman-Mak *et al.*, 1991).

4.2.6 FFI: Worst pain

4.2.6.1 Intragroup analysis

GTIASTM group

The GTIASTM group showed a highly significant decrease in worst pain over the two time points ($p = 0.001$) (Table 18 (Appendix 12)). This is shown graphically in Figure 17 (Appendix 12)

Placebo group

There was a significant decrease in worst pain over the two time points in the placebo group ($p = 0.027$). (Table 19 and Figure 18 in Appendix 12)

4.2.6.2 Intergroup analysis

There was no evidence of a significant treatment effect for worst pain ($p = 0.738$). Both groups experienced significant pain reduction over time ($p < 0.001$). Figure 19 suggests that the rate of pain decrease was slightly faster in the GTIASTM group than in the placebo group, since the slope of the line was slightly steeper in the GTIASTM group.

Table 20: Repeated measures ANOVA within and between subjects effects for worst pain.

Effect	Wilks' lambda	p value
Time	0.636	<0.001
Group	F=0.000	0.986
Time*group	0.998	0.738

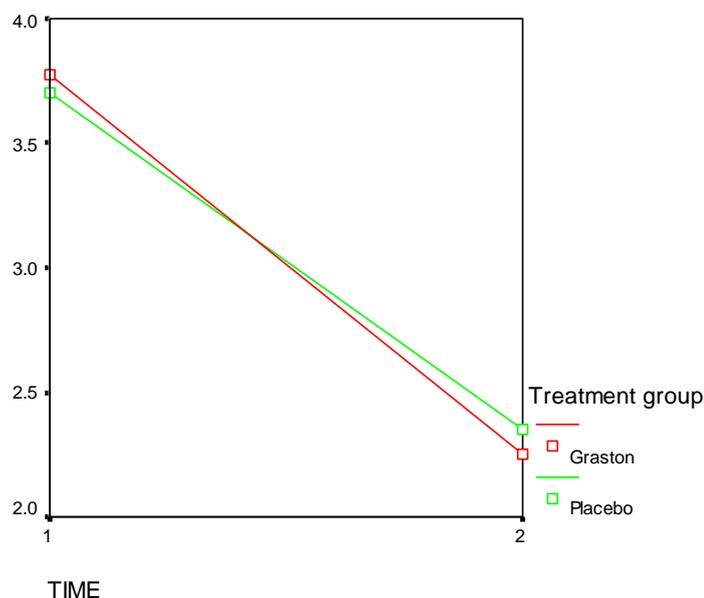


Figure 19: Profile plot of worst pain over time by treatment group

The worst pain readings within both groups were seen to decrease, but slightly more so in the GTIASTM group. This could perhaps be attributed to a breakdown in scar tissue (Carey, 2003:37). The scar tissue would reduce the extensibility of the ligament (Kessler, 1990:85) and with repetitive stress on this tightened structure, pain and swelling result (Hertling and Kessler, 1996:424). Hence a breakdown in scar tissue could result in a reduction of pain. Perhaps the GTIASTM provides a slightly faster alternative to the progress of natural history as indicated by the decline in pain within the placebo group. The Hawthorne effect may have contributed to the improvement seen in the placebo group (Mouton, 2002:152). It is therefore suggested that an increased patient pool may counteract the natural history changes demonstrated.

4.2.7 FFI: Morning pain

4.2.7.1 Intragroup analysis

GTIASTM group

There was a non significant decrease in morning pain over time in the GTIASTM group ($p = 0.103$) shown in Table 21 (Appendix 12). The slope of the decrease is shown in Figure 20 (Appendix 12).

Placebo group

In the placebo group there was a borderline significant decrease in morning pain over time ($p = 0.064$) shown in Table 22 and in Figure 21 (Appendix 12).

4.2.7.2 Intergroup analysis

There was no treatment effect for morning pain ($p = 0.899$). Both groups decreased at the same rate over time. Figure 22 shows that the slopes of the two groups' profiles were parallel.

Table 23: Repeated measures ANOVA within and between subjects effects for morning pain

Effect	Wilks' lambda	p value
Time	0.827	0.028
Group	F=0.997	0.746
Time*group	0.999	0.899

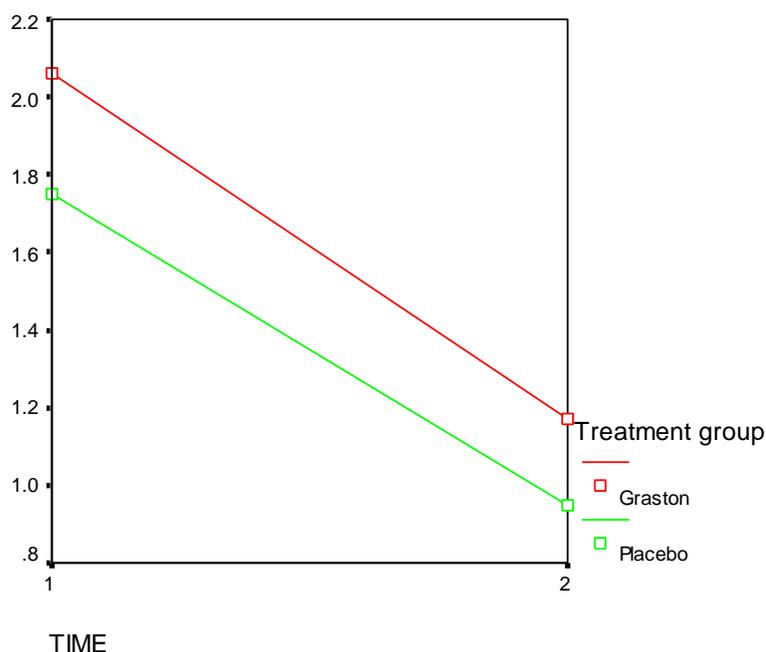


Figure 22: Profile plot of morning pain over time by treatment group

During sleep the ankle joint is fairly immobile which results in fluid accumulation (Guyton and Hall, 1997:209). The joint capsule has many pain fibers that when stretched (by fluid accumulation) can cause pain (Creamer, 1999:498). Following treatment with GTIASTM there is an induced localized inflammation (Hammer, 2003(c):1) which can, according to

Guyton and Hall (1997:209), lead to intracellular edema. This induced inflammation allows for increased blood flow and reduction of the static swelling that has accrued due to lack of movement.

However, both groups demonstrated a decrease in morning pain which indicates that the GTIASTM does not seem to aggravate the patients' morning pain (i.e. increase the swelling to any significant point any worse then the placebo, even though it seems to promote more rapid healing and a decrease in pain).

4.2.8 FFI: Pain walking barefoot

4.2.8.1 Intragroup analysis

GTIASTM group

There was a non significant decrease in pain walking barefoot in the GTIASTM group ($p = 0.423$). This is shown in Table 24 and Figure 23 (Appendix 12).

Placebo group

Pain associated with walking barefoot did not change significantly in the placebo group ($p = 0.132$). See Table 25 and Figure 24 (Appendix 12)

4.2.8.2 Intergroup analysis

Table 26 shows that pain from walking barefoot did not decrease significantly over the two time points in any of the groups and the rate of decrease was the same in both the groups, i.e.: no treatment effect. Figure 25 shows that the slopes of the lines were parallel.

Table 26: Repeated measures ANOVA within and between subjects effects for pain walking barefoot.

Effect	Wilks' lambda	p value
Time	0.936	0.192
Group	F=1.072	0.664
Time*group	1.000	0.966

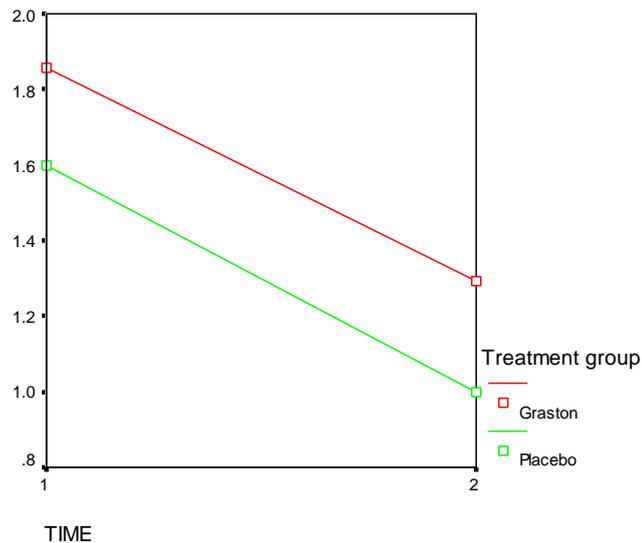


Figure 25: Profile plot of pain walking barefoot over time by treatment group

No differences are noted between the two treatment groups and both decreased over time indicating that the GTIASTM does not cause any aggravation of symptoms when compared to the placebo group, especially when one considers that the GTIASTM is responsible for increased localized inflammation (as hypothesized).

One could however also argue that GTIASTM is not any better than placebo in changing the rate of decrease of discomfort noted when the patient walks barefoot. This argument is counteracted when one assesses other parameters of clinical improvement (as discussed previously in this chapter). Thus it would seem reasonable to infer that the parameter of discomfort in the foot is not an accurate measure of improvement when assessing the patient for clinical improvement of ligamentous changes. Budiman-Mak *et al.* (1991) state that some of their findings, (activity limitation subscale) may be unique to RA patients who frequently have other lower extremity joint involvement other than just the foot. This could have contributed to their pain and disability thereby influencing the outcomes of the FFI. Other than the activity limitation subscale the authors of the study (Budiman-Mak *et al.*, 1991) still felt that it could be generalized to the non-RA population.

Furthermore it must also be considered that the patients that did not receive treatment may be representing the pain that they have from their chronic ankle instability syndrome, whereas those receiving GTIASTM may be experiencing pain due to the treatment that they are receiving. However in the GTIASTM group the pain was seen to decrease after the fourth treatment if one looks at the algometer readings previously discussed (Figure 3). As no FFI was taken after the fourth treatment and only at the end of the study one can only make

assumptions at what was occurring in this respect and further more defined research would be required.

4.2.9 FFI: Pain walking with shoes

4.2.9.1 Intragroup analysis

GTIASTM group

Pain walking with shoes did not significantly decrease in the GTIASTM group over time ($p = 0.090$). See Table 27 and Figure 26 (Appendix 12).

Placebo group

There was a non significant decrease over time in the placebo group for pain from walking with shoes ($p = 0.079$). This is shown in Table 28 and graphically in Figure 27 (Appendix 12).

4.2.9.2 Intergroup analysis

Although both groups decreased significantly over time, there was no difference in the rate of decrease by group ($p = 0.978$). Figure 28 shows that the slopes of the lines for the groups were parallel.

Table 29: Repeated measures ANOVA within and between subjects effects for pain walking with shoes.

Effect	Wilks' lambda	P value
Time	0.815	0.022
Group	F=5.806	0.211
Time*group	1.000	0.978

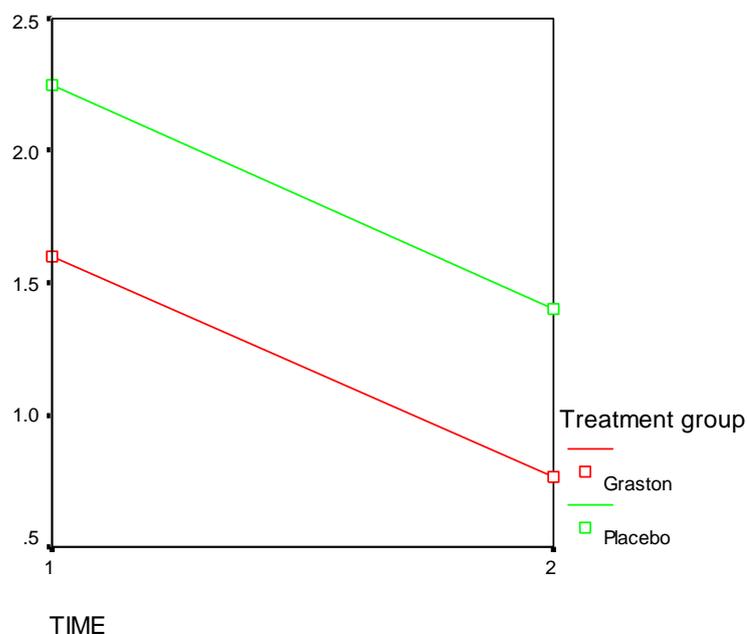


Figure 28: Profile plot of pain walking with shoes over time by treatment group

The GTIASTM caused no more discomfort to the patient than placebo. The reasoning applied in the barefoot walking (4.2.8) above may be applicable for this result as well. In the RA patient, the support of the shoe may have made a difference to joint weight bearing and decreased the patients symptoms, whereas here with the “chronic ankle instability syndrome” the joint is essentially normal and the ligament that is abnormal (or deranged) does not weight bear.

In contrast to this, it must also be remembered that these patients would be walking on both even and uneven ground, therefore the degree of pain/discomfort that is felt by the patients could be directly linked to the degree of stress placed on the ligament during ambulation. In patients that had the placebo they would have had a persistence of the chronic symptoms resulting in discomfort, whereas those receiving GTIASTM may have had discomfort from the swelling and inflammation induced by the treatment.

4.2.10 FFI: Pain standing with shoes

4.2.10.1 Intragroup analysis

GTIASTM group

There was a non significant decrease in the GTIASTM group over time. See Table 30 and Figure 29 (Appendix 12).

Placebo group

There was a non significant decrease in the placebo group over time.

See Table 31 and Figure 30 (Appendix 12).

4.2.10.2 Intergroup analysis

Both groups decreased significantly over time at the same rate. There was no evidence of treatment effect ($p = 0.738$).

Table 32: Repeated measures ANOVA within and between subjects effects for pain standing with shoes.

Effect	Wilks' lambda	p value
Time	0.636	<0.001
Group	F=2.002	0.986
Time*group	0.998	0.738

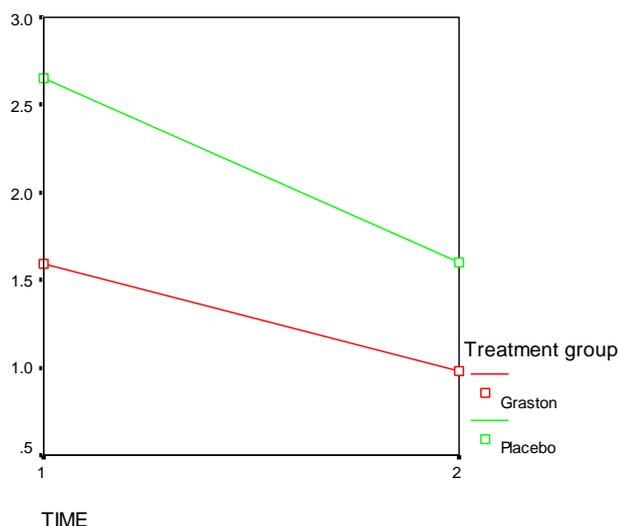


Figure 31: Profile plot of pain standing with shoes over time by treatment group

It can be observed from Figure 31 that the placebo group experienced a slightly more rapid rate of decreasing pain while standing with shoes as the lines are not parallel. This could imply that, for this measure, placebo is no better – and probably worse than natural history; however it still demonstrates that the GTIASTM did not aggravate the patients' pain and was not more effective than the placebo.

In furtherance to the above it could also be seen that the shoes, when standing, place pressure on the outside of the ankle (around the lateral malleolus). This pressure could decrease the rate of improvement seen in the treatment groups whereby the shoe pressure on newly inflamed tissue as a result of treatment could result in shoe pressure tenderness.

4.2.11 FFI: Walking in the house

4.2.11.1 Intragroup analysis

GTIASTM group

There was a non significant decrease in the GTIASTM group over time. See Table 33 and Figure 32 (Appendix 12).

Placebo group

There was a small scale non significant decrease in the placebo group ($p = 0.220$). See Table 34 and Figure 33 (Appendix 12).

4.2.11.2 Intergroup analysis

Overall there was a borderline significant decrease over time for both groups ($p = 0.054$). There was no statistical evidence of a treatment effect ($p = 0.227$), however, when one examines Figure 34 there is a clear trend towards the GTIASTM group showing a steeper rate of decline than the placebo group.

Table 35: Repeated measures ANOVA within and between subjects effects for walking in the house pain.

Effect	Wilks' lambda	p value
Time	0.865	0.054
Group	F=0.899	0.352
Time*group	0.944	0.227

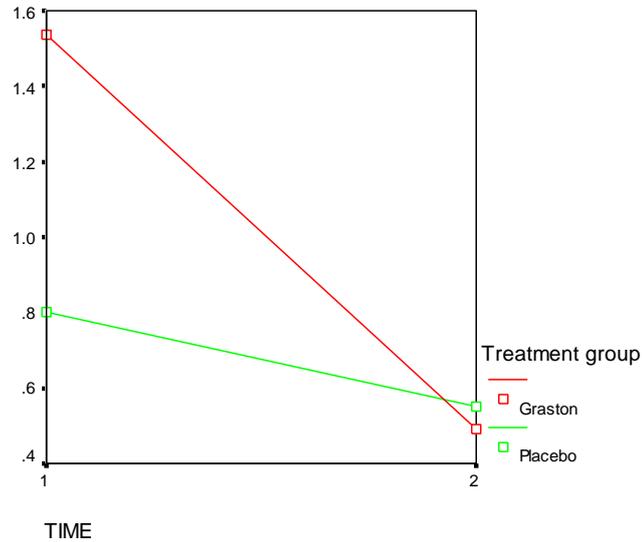


Figure 34: Profile plot of walking in the house over time by group

Walking inside a house can be comparable to walking on flat, hard surfaces.

The GTIASTM demonstrated a more marked improvement with walking on hard surfaces. This could perhaps be attributed to an improvement in both proprioception and improved range of motion from having fewer restrictions.

Proprioception is brought about through unconscious sensations of the skin, joints and muscles (Magnusson and McHugh, 1995:191). According to Freeman *et al.*, (1965, in Magnusson and McHugh, 1995:191) functional ankle instability could be attributed to the disruption of afferent input from mechanoreceptors within the injured joint capsule and ligament. Kaikkonen *et al.*, (1994) in their study also found that ankle proprioception is impaired as a result of ankle ligament injury and that peripheral sensation is important in maintaining static postural stability. Therefore walking inside a house would be much more easily accomplished than walking outside for a person with proprioceptive deficit. Any improvement would more likely be seen first while walking on flat hard surfaces (inside a house) then on a soft uneven surface (outside).

4.2.12 FFI: Walking outside

4.2.12.1 Intragroup analysis

GTIASTM group

There was a significant decrease in the GTIASTM group over time. See Table 36 and Figure 35 (Appendix 12).

Placebo group

The placebo group showed a non significant decrease over time ($p = 0.097$).

See Table 37 and Figure 36 (Appendix 12).

4.2.12.2 Intergroup analysis

According to Table 38, there was a non significant interaction between treatment group and time ($p = 0.168$), however, when one examines Figure 37 there is a visible trend towards the GTIASTM group decreasing at a faster rate than the placebo group. Both groups decreased significantly over time ($p = 0.003$).

Table 38: Repeated measures ANOVA within and between subjects effects for pain walking outside.

Effect	Wilks' lambda	p value
Time	0.715	0.003
Group	F=0.590	0.450
Time*group	0.928	0.168

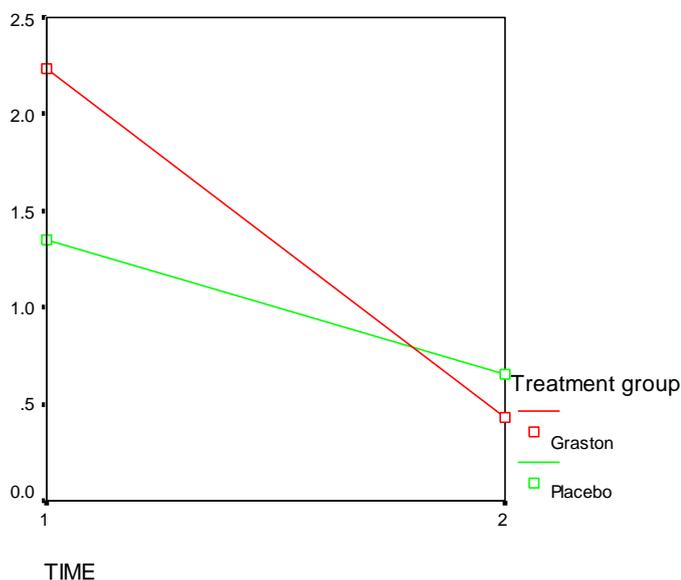


Figure 37: Profile plot of pain walking outside over time by group

The outside environment is an unstable surface as there is soft grass, divots and ruts. Good proprioception is therefore required in order to negotiate this terrain. Misstepping on a small stone or walking on an uneven surface causes the ankle to readily give way resulting in more pain and swelling (Louwerens and Snijders, 1999:344). The significant decrease in the GTIASTM group suggests that following the treatment of the scar tissue within the ligament

there appears to also be an improvement in the proprioceptive ability of the ligament to detect changes in terrain.

It is interesting to note that an improvement was seen for both hard and soft terrain irrespective of the absence or presence of shoe support (refer to figures 25 and 28). This could indicate an improvement in the GTIASTM group relative to:

- Proprioception
- Biomechanical function of the ankle joint
- Or both

Climbing and descending stairs

For the following two measures (climbing and descending stairs) it is important to remember that functional instability is best evaluated walking up and down stairs, especially when descending stairs (Kaikkonen *et al.*, 1994). These tasks require repeated, low-intensity performance (Kaikkonen *et al.*, 1994). In addition these tasks require that the ankle moves into and out of its closed packed position of maximal dorsiflexion (Magee, 2002: 766) which requires the use of the ligaments for stability (Moore, 1992:489).

4.2.13 FFI: Climbing stairs

4.2.13.1 Intragroup analysis

GTIASTM group

There was a borderline non-significant decrease over time in the GTIASTM group ($p = 0.094$). This is shown in Table 39 and Figure 38 (Appendix 12).

Placebo group

There was a non significant decrease over time in the placebo group ($p = 0.142$). This is shown in Table 40 and Figure 39 (Appendix 12).

4.2.13.2 Intergroup analysis

Both groups decreased significantly over time ($p = 0.037$). There was no significant treatment effect however the profile plot shows evidence of a trend towards the GTIASTM group decreasing at a faster rate than the placebo group.

Table 41: Repeated measures ANOVA within and between subjects effects for pain climbing stairs.

Effect	Wilks' lambda	p value
Time	0.844	0.037
Group	F=0.108	0.745
Time*group	0.989	0.595

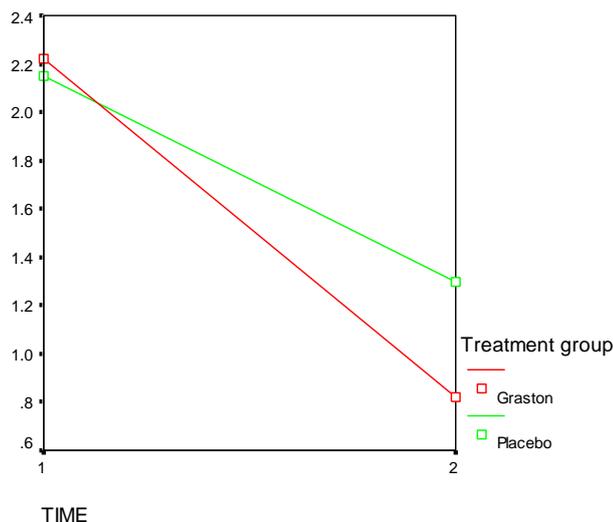


Figure 40: Profile plot of pain climbing stairs over time by group

4.2.14 FFI: Descending stairs

4.2.14.1 Intragroup analysis

GTIASTM group

There was a borderline non significant decrease over time in the GTIASTM group. See Table 42 and Figure 41 (Appendix 12).

Placebo group

There was no significant change over time in the placebo group. See Table 43 and Figure 42 (Appendix 12).

4.2.14.2 Intergroup analysis

The treatment effect was not statistically significant ($p = 0.595$), but there was a trend towards a steeper rate of decline in the GTIASTM group relative to the placebo group, as seen in Figure 43.

Table 44: Repeated measures ANOVA within and between subjects effects for pain descending stairs.

Effect	Wilks' lambda	p value
Time	0.852	0.043
Group	F=0.147	0.704
Time*group	0.989	0.595

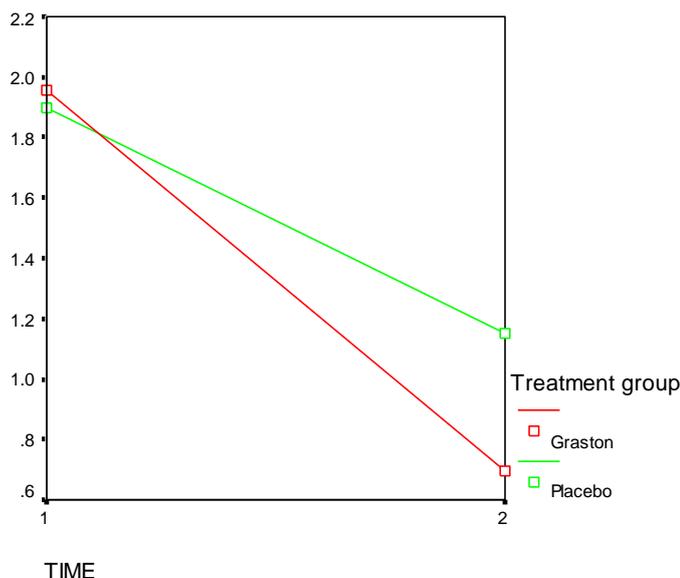


Figure 43: Profile plot of pain descending stairs over time by group

The results for climbing and descending stairs are examined below.

When climbing stairs the foot moves into plantarflexion and therefore since the talus becomes "unlocked" from the mortise it assumes the loose pack position (Baker and Todd, 1995) of 10° of plantarflexion midway between full inversion and eversion (Norris, 1998:21). In this position the ankle joint receives static support from the surrounding ligaments (Baker and Todd, 1995).

In order to descend stairs full dorsiflexion of 20° is necessary (Magee, 2002:797). With fewer adhesions and restrictions to joint movement following treatment with the GTIASTM this would be possible and less pain would result when the patient descended stairs.

An improvement was seen in both treatment groups with the GTIASTM group improving more than the placebo group. This perhaps supports the above theory (4.2.11.2 and 4.2.12.2) pertaining to an improvement in the proprioceptive abilities of the ankle. The

improvement in these readings would suggest that the proposal put forward in respect of the function of various factors within this scale being more related to joint pathology seem appropriate, as those measures that assess the joint in the closed pack position (standing) seem to infer that the GTIASTM group do not improve, whereas those stressing the ligaments (going up and down stairs) seem to infer an improvement of the GTIASTM group as opposed to the placebo.

4.2.15 FFI: Standing on tip toe

4.2.15.1 Intragroup analysis

GTIASTM group

There was a significant decrease over time in the GTIASTM group. This is evident in Table 45 and Figure 44 (Appendix 12).

Placebo group

There was no significant change over time in the placebo group. This is evident in Table 46 and Figure 45 (Appendix 12).

4.2.15.2 Intergroup analysis

There was a borderline non significant treatment effect for standing on tip toes ($p = 0.090$). However, the trend is evident from Figure 46 that those subjects in the GTIASTM group improved faster and to a greater extent than those in the placebo group.

Table 47: Repeated measures ANOVA within and between subjects effects for pain standing on tip toe.

Effect	Wilks' lambda	p value
Time	0.859	0.049
Group	F=0.000	0.989
Time*group	0.894	0.090

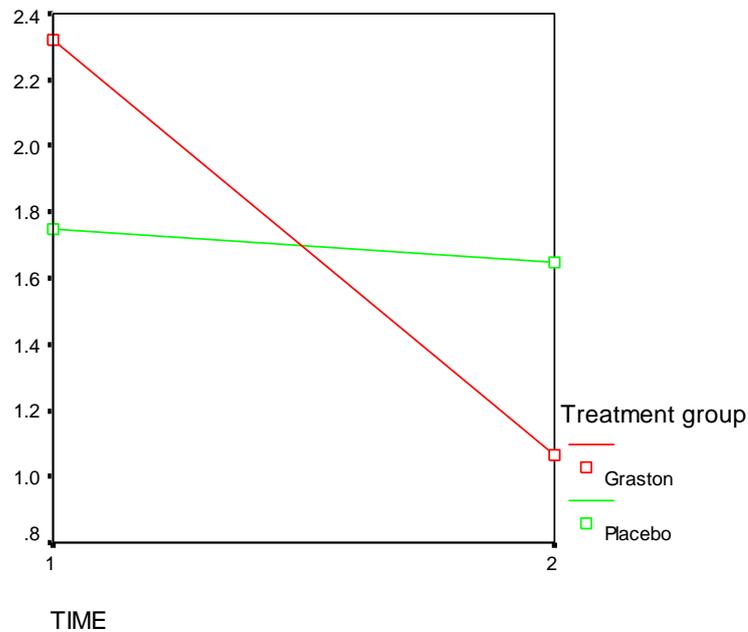


Figure 46: Profile plot of pain standing on tip toe over time by group

This again indicates an improvement in respect of a ligament reliance test, where the ankle is in the loose packed position (Baker and Todd, 1995), which would infer the need for good proprioceptive feed back from the lateral ankle ligaments. Thus it is argued that the significant improvement seen within the GTIASTM group could only be attributed to an improvement in proprioceptive and biomechanical control of the foot and ankle.

4.2.16 FFI: Getting up from a chair

4.2.16.1 Intragroup analysis

GTIASTM group

As evident in Table 48 and Figure 47 (Appendix 12) there was a non significant decrease over time in the GTIASTM group.

Placebo group

There was a non significant time effect in the placebo group. This is seen in Table 49 and Figure 48 (Appendix 12).

4.2.16.2 Intergroup analysis

Although there was no evidence of a treatment effect for this outcome, Figure 49 shows that the GTIASTM group decreased at a steeper rate than the placebo group. This was not statistically significant though ($p=0.760$).

Table 50: Repeated measures ANOVA within and between subjects effects for pain getting up from a chair.

Effect	Wilks' lambda	p value
Time	0.913	0.127
Group	F=0.049	0.826
Time*group	0.996	0.760

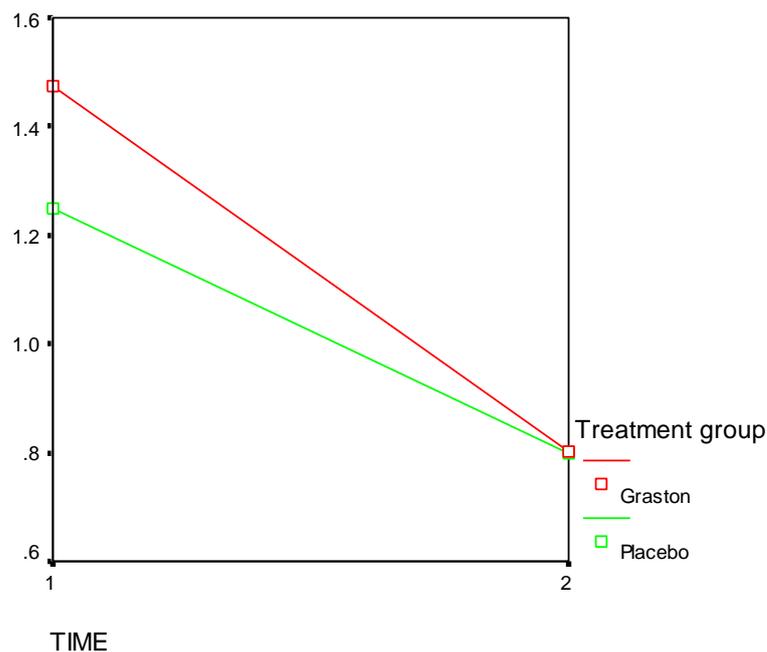


Figure 49: Profile plot of pain getting up from a chair over time by group

Pain getting up from a chair could be likened to morning pain. Immobility of the ankle joint after sitting for prolonged periods could lead to fluid accumulation within the ankle joint resulting in distension and pain. The GTIASTM group appeared to improve at a steeper rate perhaps because of the improved biomechanics resulting in:

- Improved circulation decreasing the swelling whilst seated
- Improved ligament function as stabilizer in this movement
- On standing up there is a slight increase in ankle dorsiflexion and with greater freedom of movement within the ankle joint a reduction in pain could result.

4.2.17 FFI: Climbing curbs

4.2.17.1 Intragroup analysis

GTIASTM group

There was a non significant decrease over time in the GTIASTM group. This is shown in Table 51 and graphically in Figure 50 (Appendix 12).

Placebo group

There was a non significant difference over time in the placebo group. This is shown in Table 52 and graphically in Figure 51 (Appendix 12).

4.2.17.2 Intergroup analysis

Although there was no statistical evidence of a treatment effect, Figure 52 shows that the GTIASTM group decreased at a faster rate than the placebo group.

Table 53: Repeated measures ANOVA within and between subjects effects for pain climbing curbs.

Effect	Wilks' lambda	p value
Time	0.891	0.086
Group	F=0.077	0.783
Time*group	0.964	0.312

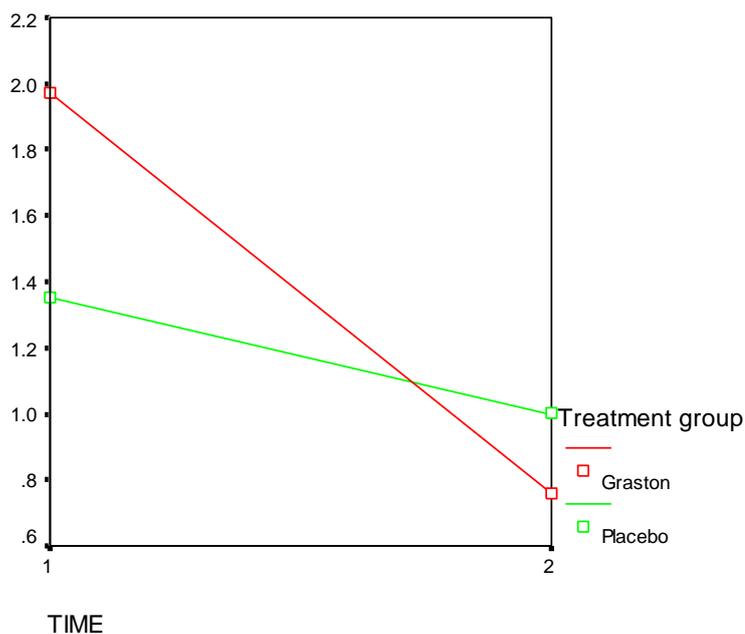


Figure 52: Profile plot of pain climbing curbs over time by group

This situation is analogous to climbing stairs. The loose pack position is assumed and curbs are often uneven causing the ankle to rely on good proprioceptive feedback in order to not have the ankle “give way”. The GTIASTM group has again proved better than the placebo group and this can confirm that there does appear to be an improvement in ankle proprioception.

It must be mentioned with reference to the final two questions in the FFI (Appendix 8) that no patients stayed inside all day and no patients stayed in bed all day, therefore the readings taken will have been relative.

To conclude, the FFI has provided a thorough analysis of the patients’ ability levels before and post treatment. Improvements in pain levels were seen in both treatment groups. Worst pain levels showed a decline in favour of GTIASTM and there appeared to be a marked improvement with ankle proprioception in the GTIASTM group as demonstrated with walking outside, climbing curbs, negotiating stairs and standing on tip toe.

4.3 Intra-group correlations between changes in outcome measures

GTIASTM group

In the GTIASTM group there was a significant positive correlation between change in edema and change in proximal ultrasound measurement ($r=0.575$, $p = 0.031$ (Table 54)). This relationship is shown graphically in Figure 53. Change in distal and mid ultrasound measurement were also significantly positively correlated ($r=0.547$, $p = 0.043$ (Table 54)). Table 55 discusses the various positive and negative significant correlations found within the GTIASTM group.

Table 54: Correlation matrix of change in outcome variables in GTIASTM group.

		Change in edema	Change in algometer from T1 to T3	Change in proximal	Change in mid	Change in distal
Change in edema	Pearson Correlation	1	-.046	.575(*)	-.413	.071
	Sig. (2-tailed)	.	.872	.031	.126	.809
	N	15	15	14	15	14
Change in algometer from T1 to T3	Pearson Correlation	-.046	1	-.251	.015	.038
	Sig. (2-tailed)	.872	.	.387	.959	.898
	N	15	15	14	15	14
Change in proximal	Pearson Correlation	.575(*)	-.251	1	-.177	-.070
	Sig. (2-tailed)	.031	.387	.	.545	.821
	N	14	14	14	14	13
Change in mid	Pearson Correlation	-.413	.015	-.177	1	.547(*)
	Sig. (2-tailed)	.126	.959	.545	.	.043
	N	15	15	14	15	14
Change in distal	Pearson Correlation	.071	.038	-.070	.547(*)	1
	Sig. (2-tailed)	.809	.898	.821	.043	.
	N	14	14	13	14	14

* Correlation is significant at the 0.05 level (2-tailed).

Table 55: A discussion on the positive and negative significant correlations found within the GTIASTM group

<i>Reading 1</i>	<i>Relationship</i>	<i>Reading 2</i>	<i>Discussion</i>
Edema	Negative	Algometer	An increase in edema would result in decreased algometer readings, which is associated with increased pain.
	Negative	Mid ligament changes	An increase in edema is associated with a decrease in the mid ligament diameter, indicating that there is a decrease in scar tissue.
	Positive and significant	Proximal ligament changes	A decrease in edema is associated with a decrease in proximal ligament diameter indicating a decrease in scar tissue. This is in contrast to the above where an increase in edema is associated with a decrease in mid ligament diameter.
Proximal ligament changes	Negative	Mid ligament changes	As the scar tissue decreased in the proximal ligament, so it increased in the mid ligament.
	Negative	Distal ligament changes	As the scar tissue decreased in the proximal ligament, so it increased in the distal ligament.
Mid ligament changes	Positive and significant	Distal ligament changes	As the scar tissue decreased in the mid ligament, so it decreased in the distal ligament.
Algometer	Negative	Proximal ligament changes	As the pain decreased, the algometer increased and the proximal portion of the ligament decreased. Indicating that a reduction in scar tissue brought about pain reduction.

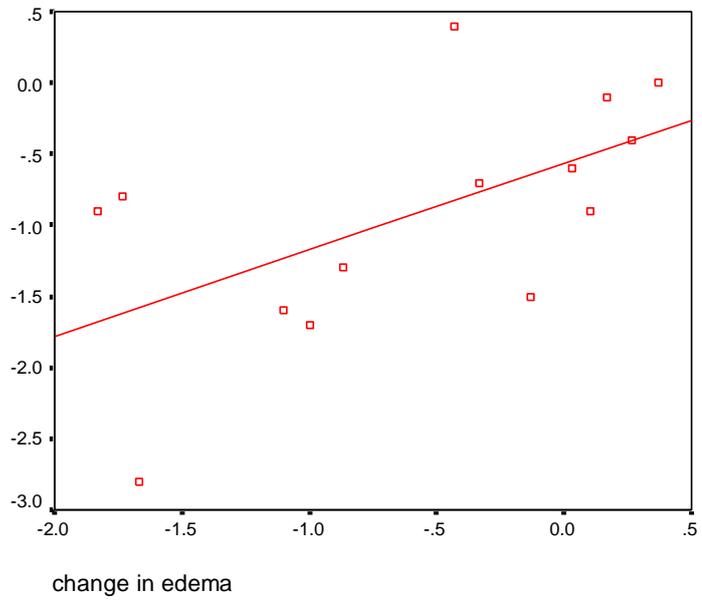


Figure 53: Scatterplot of change in edema by change in proximal ultrasound measurement in GTIASTM group

Placebo group

In the placebo group there was a significant positive correlation between change in algometer measurement and change in proximal ultrasound measurement ($r=0.625$, $p = 0.017$) (See Table 56 and figure 54). Changes in proximal and mid, and mid and distal were also significantly correlated in this group. Table 57 discusses the positive and negative significant correlations found within the placebo group.

Table 56: Correlation matrix of change in outcome variables in Placebo group.

		Change in edema	Change in algometer from T1 to T3	Change in proximal	Change in mid	Change in distal
Change in edema	Pearson Correlation	1	.435	.309	.503	.409
	Sig. (2-tailed)	.	.105	.283	.080	.166
	N	15	15	14	13	13
Change in algometer from T1 to T3	Pearson Correlation	.435	1	.625(*)	.498	.457
	Sig. (2-tailed)	.105	.	.017	.083	.116
	N	15	15	14	13	13
Change in proximal	Pearson Correlation	.309	.625(*)	1	.735(**)	.481
	Sig. (2-tailed)	.283	.017	.	.004	.096
	N	14	14	14	13	13
Change in mid	Pearson Correlation	.503	.498	.735(**)	1	.716(**)
	Sig. (2-tailed)	.080	.083	.004	.	.006
	N	13	13	13	13	13
Change in distal	Pearson Correlation	.409	.457	.481	.716(**)	1
	Sig. (2-tailed)	.166	.116	.096	.006	.
	N	13	13	13	13	13

* Correlation is significant at the 0.05 level (2-tailed).

** Correlation is significant at the 0.01 level (2-tailed).

Table 57: A discussion on the positive and negative significant correlations found within the placebo group

<i>Reading 1</i>	<i>Relationship</i>	<i>Reading 2</i>	<i>Discussion</i>
Algometer	Positive and significant	Proximal ligament changes	It would seem that as the pain decreased due to natural history, the algometer increased resulting in increased diameter of the ligament, therefore demoting increased scar tissue.
Proximal ligament changes	Positive and significant	Mid ligament changes	As the scar tissue decreased in the proximal ligament, so too did it decrease in the mid ligament.
Mid ligament changes	Positive and significant	Distal ligament changes	As the scar tissue decreased in the mid ligament, so too did it decrease in the distal ligament.

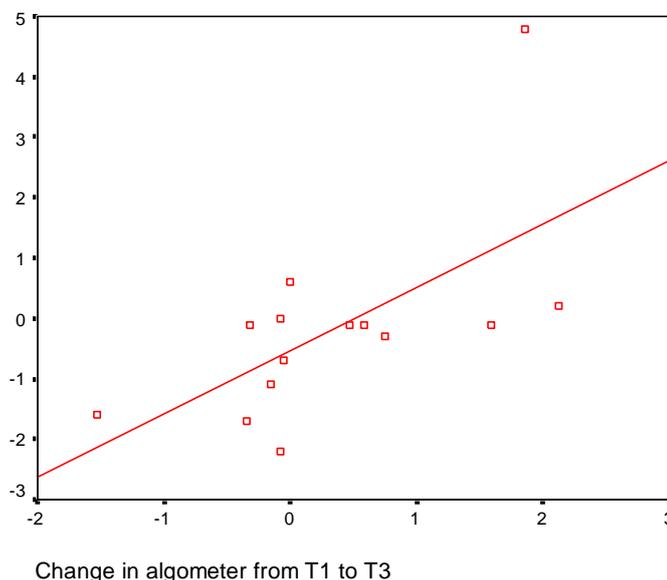


Figure 54: Scatterplot of change in algometer by change in proximal ultrasound measurement in placebo group

4.4 Summary and recommendations

For one outcome (algometer measurements) there was a significant improvement in the treatment group over and above that in the placebo group. For other outcomes, there was a suggestive trend towards a faster rate of improvement in the GTIASTM group than the placebo group, although the treatment effect was non-significant. These outcomes were edema and proximal ultrasound measurement, as well as several of the FFI measurements.

There was a significant positive correlation between improvement in proximal ultrasound measurement and improvement in edema in the GTIASTM group. Thus those participants who received the GTIASTM technique would improve in both these outcomes in a linear fashion.

The sample size was not large enough to show a significant interaction between treatment groups and time (treatment effect) even where it was evident that one existed. Thus a larger study powered to detect such an interaction would be recommended if definitive evidence is required. However, this study showed many trends of a treatment effect and in none of the outcomes was there any suggestion of a harmful or less effective effect of the treatment relative to the placebo. Thus the treatment under study is at least as good as the placebo for many outcomes, and significantly better than the placebo for algometer.

4.4.1 The first hypothesis

It is hypothesized that the use of the GTIASTM for the reduction of scar tissue as a treatment of chronic ankle grade I or II ankle sprains will prove to be an effective treatment in terms of subjective and objective findings.

The above hypothesis is accepted as a reduction in scar tissue was brought about by the GTIASTM which did result in reduction of pain and improved proprioception.

4.4.2 The second hypothesis

It is hypothesized that the GTIASTM will prove to be an effective treatment for concomitant clinical measures (viz. the reduction of pain, edema and other clinical outcomes observed from the Foot Function Index)

The above hypothesis is accepted for pain reduction but rejected for edema reduction. Pain levels were significantly reduced under treatment with the GTIASTM. However the GTIASTM

tools did not appear to reduce edema any more than the placebo. It is noted that the treatment was not any worse than the placebo.

The above hypothesis is also accepted with regards to improved proprioception that was demonstrated in the Foot Function Index.

4.4.3 The third hypothesis

It is hypothesized that there will be some degree of correlation between scar tissue, pain and edema findings.

The hypothesis stated above is rejected with regards to a correlation between scar tissue and pain within the GTIASTM group as no significant positive correlation was found between scar tissue reduction and reduction in pain.

With regards to a correlation between scar tissue and edema within the GTIASTM group the hypothesis is accepted as a decrease in edema was associated with a decrease in proximal ligament diameter which indicated a decrease in scar tissue.

Acceptance of the hypothesis is found when looking at pain and edema as a negative correlation existed between edema and pain: an increase in edema resulted in a decrease in algometer readings, which is associated with increased pain.

CHAPTER FIVE

CONCLUSIONS AND RECOMMENDATIONS

5.1 Conclusions

The purpose of this study was to look at the effectiveness of the GTIASTM in the breakdown of scar tissue and adhesions for the treatment of chronic ankle instability syndrome and to determine any correlations between scar tissue reduction, pain and edema.

In terms of objective data:

- There appeared to be scar tissue reduction within both treatment groups. The proximal portion of the ligament, however demonstrated the most clinically significant response to the GTIASTM.
- A statistically significant improvement in terms of patients' pain threshold was seen within the GTIASTM group.
- Both groups showed a decrease in edema levels indicating that GTIASTM is no worse than placebo treatment.

In terms of the subjective data, clinically significant improvement was evident with regards to proprioception especially within the GTIASTM group.

In conclusion, the results of this study demonstrate that the Graston Technique Instrument Assisted Soft Tissue Mobilization tools have a place within the treatment of chronic ankle instability syndrome following a grade I or II ankle inversion sprain. This role needs to be further examined in terms of chronic ankle instability syndrome and perhaps in the treatment of scar tissue within other ligaments.

5.2 Recommendations

1. A larger sample size would have strengthened the results of the study.
2. Researcher bias could have resulted from a lack of blinding in this study. Although the Diagnostic ultrasound measures were recorded by a person blinded to the study further blinding could have been used by having someone else assess and record the other objective measurements and aid in the completion of the subjective questionnaire. The results of the study could have been strengthened had more blinding been used.

3. The researcher recommends that a follow up consultation could have been done at 1 and 3 months after the final consultation to analyse the long term benefits of using the GTIASTM for the treatment of chronic ankle instability syndrome.
4. As this research was not representative of the general ethnic population within the KwaZulu-Natal region perhaps there should be further studies conducted into the effects that the GTIASTM may have on other population groups.
5. Eight treatments were given over a four week period, however the time intervals between treatments were not equal. Perhaps a more structured time interval set up between treatments would have been more beneficial in allowing healing of the ligament and aided in research consistency.
6. It is recommended that further studies be conducted to look at the optimum number of treatments necessary to bring about scar tissue resolution, as the current study parameters were based on anecdotal reports.
7. The diagnostic ultrasound measured thickness of the ligament in which scar tissue was found. A change in thickness could perhaps indicate that scar tissue and adhesions had indeed broken down but without actual tissue sampling there is no definite indication of this having occurred.
8. As it was frequently noted by the radiologist that the fibre quality of the ligament had improved, perhaps another study could look further into this aspect of ligamentous change.
9. Perhaps a future study could assess the effect that the GTIASTM had on ligament length which may have become altered (lengthened) following an acute ankle sprain.
10. Increased number of measurements taken at more frequent intervals would have strengthened the hypotheses made in this study.
11. A study comparing edema reduction in acute ankle sprains may aid in the ability to detect any improved outcomes since the degree of swelling would be greater initially.
12. The inferences made with respect to the proprioceptive improvement could be further substantiated by additional research that directly compares differences in proprioceptive ability.
13. A future study could include a third control group to rule out the effect of possible "friction" by the ultrasound head.

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APPENDIX 1

Letter of information

Dear Patient. Welcome to my research study.

Title of study:

The efficacy of the Graston Technique Instrument Assisted Soft Tissue Mobilization in the reduction of scar tissue in the management of chronic ankle instability syndrome following an ankle inversion sprain

Supervisors:

Dr. A. Docrat (031) 2042094

Research Student:

Alexandra Parker (031) 7015780 / 084 444 8465

Institution:

Durban Institute of Technology

Purpose of the study:

The purpose of this study is to determine the effectiveness of the Graston Technique instrument assisted soft tissue mobilization tools in the reduction of scar tissue in the management of chronic ankle instability syndrome following an ankle inversion sprain. Thirty patients with chronic ankle instability syndrome will be included into this study.

Procedures:

At the first consultation diagnostic ultrasound will be performed on all patients in order to assess the amount and size of scar tissue present in the patients lateral ankle ligaments. This will be done in Pietermaritzburg at St Anne's X-ray Department . The patient will also at the first and final consultations, be asked to fill in a questionnaire. Algometer readings will be taken at the first, fifth and eighth treatments. Edema measures will be taken at the first and final treatment. Diagnostic ultrasound will be repeated within one week following the final (eighth) treatment.

Risks/ Discomforts:

The application of the Graston tools may produce some discomfort, but does not cause any harm. In this case the applied pressure can be modified to patient tolerance, but some discomfort may be necessary to release tough scar tissue and restrictions. Bruising may result from the release of scar tissue and breakout may also be experienced by the patient.

Benefits:

Your contribution to this study, by volunteering to partake, will help us as Chiropractors to build on our knowledge. This will benefit you as a patient in the long run, as we will be able to provide you with more effective health care in the future.

New findings:

You will be made aware of any new findings during the course of this study.

Reasons why you may be withdrawn from this study without your consent:

You may be removed from participation in this study without your consent for the following reasons:

- If you are unable to attend all your appointments.
- If you have changed any lifestyle habits during your participation in this study that may effect the outcome of this research (e.g. medication, supplements or treatment)

AS A VOLUNTARY PARTICIPANT IN THIS RESEARCH STUDY, YOU ARE FREE TO WITHDRAW FROM THE STUDY AT ANY TIME, WITHOUT GIVING A REASON.

Remuneration:

You will not be receiving a travel allowance in order to attend your appointment at the Chiropractic Day clinic at the Durban Institute of Technology. But depending on certain criteria (as stipulated below) you may receive petrol money for travelling to Pietermaritzburg (PMB) twice. But this will only be given at the completion of the research.

- No travel allowance will be given if you live in PMB and work in Durban.
- No travel allowance will be given if a group trip to PMB can be arranged with the researcher

Cost of the study:

All the treatments and diagnostic ultrasound will be done free of charge and your participation in this study is voluntary.

Study breakdown

Date	Time	Venue	Treatment plan
		DIT – Initial consultation	History, Physical, Ankle regional
		St Anne’s –PMB	Ultrasound
		DIT – visit 1	FFI, Algometer, edema measure
		DIT – visit 2	
		DIT – visit 3	
		DIT – visit 4	
		DIT – visit 5	Algometer readings
		DIT – visit 6	
		DIT – visit 7	
		DIT – visit 8	Algometer, FFI, Edema readings
		St Anne’s – PMB	Ultrasound

Confidentiality:

All patient information is confidential. The results from this study will be used for research purposes only. Only individuals who are directly involved in this study will be allowed to access these records.

Persons to contact should you have any problems or questions:

Should you have any questions that you would prefer being answered by an independent individual, feel free to contact my supervisors on the above numbers. If you are not satisfied with a particular area of this study, please feel free to forward any concerns to the Durban Institute of Technology Research and ethics committee.

Thank you for participating in my research study.

Alexandra Parker
(Research Student)

Dr. A Docrat
(Supervisor)

Ms Carey-Loghmani
(Co-Supervisor)

APPENDIX 2

INFORMED CONSENT FORM

(To be completed by patient / subject)

Date :

Title of research project :

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: _____

APPENDIX 3
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:

	Complaint 1	Complaint 2
<ul style="list-style-type: none">▶ Location▶ Onset : Initial: Recent:1. Cause:▶ Duration▶ Frequency▶ Pain (Character)▶ Progression▶ Aggravating Factors▶ Relieving Factors▶ Associated S & S▶ Previous Occurrences▶ Past Treatment▶ Outcome:		

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. xrays
- ▶ 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 4

Durban Institute of Technology

PHYSICAL EXAMINATION: SENIOR

Patient Name : _____ **File no :** _____ **Date :** _____

Student : _____ **Signature :** _____

VITALS:

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

GENERAL EXAMINATION:

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

SYSTEM SPECIFIC EXAMINATION:

CARDIOVASCULAR EXAMINATION

RESPIRATORY EXAMINATION

ABDOMINAL EXAMINATION

NEUROLOGICAL EXAMINATION

COMMENTS

Clinician: _____ **Signature :** _____

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

Weight bearing:	Ⓡ	Ⓛ	Non weight bearing:	Ⓡ	Ⓛ
Plantar flexion			50°		
Dorsiflexion			20°		
Supination					
Pronation					
Toe dorsiflexion			40°(mtp)		
Toe plantar flexion			40° (mtp)		
			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)

	Ⓡ	Ⓛ		Ⓡ	Ⓛ
Ankle joint: <i>Plantarflexion</i>			Subtalar joint: <i>Varus</i>		
<i>Dorsiflexion</i>			<i>Valgus</i>		
Talocrural: <i>Long axis distraction</i>			Midtarsal: <i>A-P glide</i>		
First ray: <i>Dorsiflexion</i>			<i>P-A glide</i>		
<i>Plantarflexion</i>			<i>rotation</i>		
Circumduction of forefoot on fixed rearfoot			Intermetatarsal glide		
			Tarso metatarsal joints: <i>A-P</i>		
Interphalangeal joints: <i>L → A dist</i>			Metatarsophalangeal dorsiflexion (with associated plantar flexion of each toe)		
<i>A-P glide</i>					
<i>lat and med glide</i>					
<i>rotation</i>					

Resisted Isometric movements

	(R)	(L)		(R)	(L)
Knee flexion			Pronation (eversion)		
Plantar flexion			Toe extension (dorsiflexion)		
Dorsiflexion			Toe flexion (plantar flexion)		
Supination (inversion)					

Neurological

	(R)	(L)
Dermatomes		
Myotomes		
Reflexes		
Balance/proprioception		

Special tests

	(R)	(L)
Anterior drawer test		
Talar tilt		
Thompson test		
Homan sign		
Tinel's sign		
Test for rigid/flexible flatfoot		
Kleiger test (med. deltoid)		

Alignment

	(R)	(L)
Heel to ground		
Feiss line		
Tibial torsion		
Heel to leg (subtalar neutral)		
Subtalar neutral position:		
Forefoot to heel (subtalar & Midtarsal neutral)		
First ray alignment		
Digital deformities		
Digital deformity flexible		

Palpation

	(R)	(L)
<i>Anteriorly</i>		
Medial malleoli		
Med tarsal bones, tibial (post) artery		
Lat.malleolous, calcaneus, sinus tarsi, and cuboid bones		
Inferior tib/fib joint, tibia, mm of leg		
Anterior tibia, neck of talus, dorsalis pedis artery		
<i>Posteriorly</i>		
Calcaneus, Achilles tendon, Musculotendinous junction		
<i>Plantarily</i>		
Plantar muscles and fascia		
Sesamoids		

21/10/2002

APPENDIX 6
DURBAN INSTITUTE OF TECHNOLOGY

<i>Patient Name:</i>		<i>File #:</i>		<i>Page:</i>
<i>Date:</i>	<i>Visit:</i>	<i>Intern:</i>		
<i>Attending Clinician:</i>		<i>Signature:</i>		
<i>S: Numerical Pain Rating Scale (Patient)</i> <i>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</i>		<i>Intern Rating</i> <input type="text"/>	<i>A:</i>	
<i>O:</i>		<i>P:</i>		
		<i>E:</i>		
<i>Special attention to:</i>		<i>Next appointment:</i>		
<i>Date:</i>	<i>Visit:</i>	<i>Intern:</i>		
<i>Attending Clinician:</i>		<i>Signature:</i>		
<i>S: Numerical Pain Rating Scale (Patient)</i> <i>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</i>		<i>Intern Rating</i> <input type="text"/>	<i>A:</i>	
<i>O:</i>		<i>P:</i>		
		<i>E:</i>		
<i>Special attention to:</i>		<i>Next appointment:</i>		
<i>Date:</i>	<i>Visit:</i>	<i>Intern:</i>		
<i>Attending Clinician:</i>		<i>Signature</i>		
<i>S: Numerical Pain Rating Scale (Patient)</i> <i>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</i>		<i>Intern Rating</i> <input type="text"/>	<i>A:</i>	
<i>O:</i>		<i>P:</i>		
		<i>E:</i>		
<i>Special attention to:</i>		<i>Next appointment:</i>		

Patient Name: _____ **File #:** _____ **Page:** _____

Date: _____ **Visit:** _____ **Intern:** _____
Attending Clinician: _____ **Signature:** _____

S: Numerical Pain Rating Scale (Patient)
Least **0 1 2 3 4 5 6 7 8 9 10** Worst

Intern Rating

A:

O: _____ **P:** _____

E: _____

Special attention to: _____ **Next appointment:** _____

Date: _____ **Visit:** _____ **Intern:** _____
Attending Clinician: _____ **Signature:** _____

S: Numerical Pain Rating Scale (Patient)
Least **0 1 2 3 4 5 6 7 8 9 10** Worst

Intern Rating

A:

O: _____ **P:** _____

E: _____

Special attention to: _____ **Next appointment:** _____

Date: _____ **Visit:** _____ **Intern:** _____
Attending Clinician: _____ **Signature:** _____

S: Numerical Pain Rating Scale (Patient)
Least **0 1 2 3 4 5 6 7 8 9 10** Worst

Intern Rating

A:

O: _____ **P:** _____

E: _____

Special attention to: _____ **Next appointment:** _____

APPENDIX 7 A

GRASTON INSTRUMENT ASSISTED SOFT TISSUE MOBILIZATION

Graston tools (GT) numbers 3 and 6 will be used in the treatment of patients suffering with chronic ankle pain.

GT – 3

Also known as the tongue depressor, is used to localized restrictions and treat small areas.

GT – 6

The carpal tunnel tool is used to release fascial restrictions

Carey (2003:20-25).

The Graston tools will be applied to the site of the lesion and applied in multiple directions maintaining an application angle of 30-60 degrees for maximum fibre separation and contouring of the instrument to the body. The patient should be in a relaxed and comfortable position with their ankle being fully exposed and supported. Having the ankle fully supported reduces muscle activity and allows for more effective treatment. Stroke rate over this small area is fairly quick, but this rate should be reduced if the patient experiences higher pain levels. The ideal treatment duration is between 4-8 sessions allowing approximately 3-5 minutes for each treatment. In this research the patients will each receive a total of 8 treatments (Carey-Loghmani 2003:27-29).

APPENDIX 7 B

Patents

The Graston Technique has been awarded five patents. Please refer to the chart below for U.S. Patent Numbers and descriptions:

NUMBER	TITLE
5,231,977	Tools and method for performing Soft-Tissue Massage
5,366,437	Tools for performing Soft-Tissue massage
5,441,478	Tools and method for performing Soft-Tissue Massage
5,707,346	Systems and method for performing Soft-Tissue Massage
6,126,620	Systems and method for performing Soft-Tissue Massage

APPENDIX 8

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

Section A:	0	1	2	3	4	5	6	7	8	9	10	
Worst pain												
Morning Pain												
Pain walking barefoot												
Pain walking with shoes												
Pain standing with shoes												

Section B :Can you	0	1	2	3	4	5	6	7	8	9	10	
Walk in the house												
Walk outside												
Climb stairs												
Descend stairs												
Stand on tip toe												
Get up from a chair												
Climb curbs												

Section C: Do you have to?	Yes	No
Stay inside all day		
Stay in bed all day		

Appendix 9A

Algometer readings.

	FIRST			BEGINNING OF FIFTH			FINAL		
DATE									
READING									

Appendix 9B

Edema measurements

	First consultation			Within one week of final consultation		
Date						
Readings						

Appendix 10
Diagnostic ultrasound

PATIENT NAME:

DATE	First consultation	Final consultation
Scar length (in mm)		

Radiologist signature: _____ (First Consultation)
Dr. P. de Villiers

Radiologist signature: _____ (Final Consultation)
Dr. P. de Villiers

**Are you between the ages of
18 and 50
and**

**Do you suffer with
Chronic ankle sprains /
sprain your
ankle repeatedly?**

**Research is currently being carried out at
the
Durban Institute of Technology
Chiropractic Day Clinic**

FREE TREATMENT

**Is available to those who qualify to take
part in this study**

**For more information contact
Alexandra Parker on 204 2205 / 2512**

APPENDIX 12
FFI: Statistical results

Worst pain:

Table 18: Repeated measures ANOVA intragroup time comparison for GTIASTM group for Worst pain.

Time effect Wilk's lambda	P value
0.457	0.001

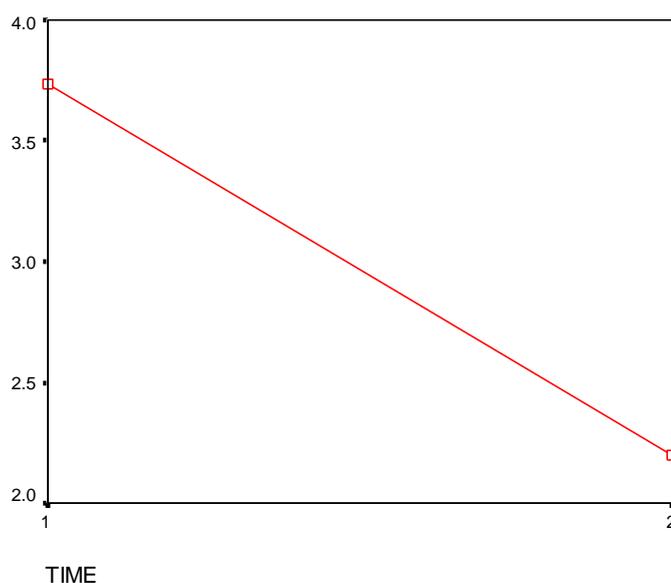


Figure 17: Profile plot of worst pain in the GTIASTM group over time

Table 19: Repeated measures ANOVA intragroup time comparison for placebo group for worst pain.

Time effect Wilk's lambda	P value
0.696	0.027

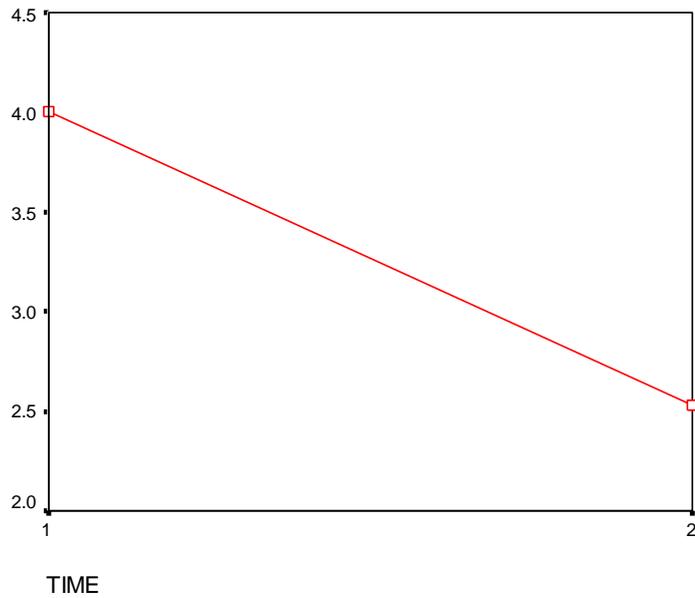


Figure 18: Profile plot of worst pain in the placebo group over time

Morning Pain:

Table 21: Repeated measures ANOVA intragroup time comparison for GTIASTM group for morning pain.

Time effect Wilk's lambda	p value
0.821	0.103

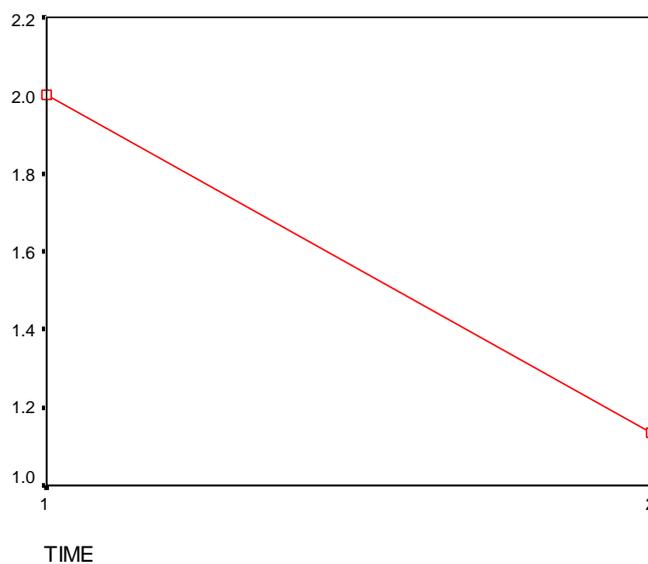


Figure 20: Profile plot of morning pain in the GTIASTM group over time

Table 22: Repeated measures ANOVA intragroup time comparison for placebo group for morning pain.

Time effect Wilk's lambda	p value
0.776	0.064

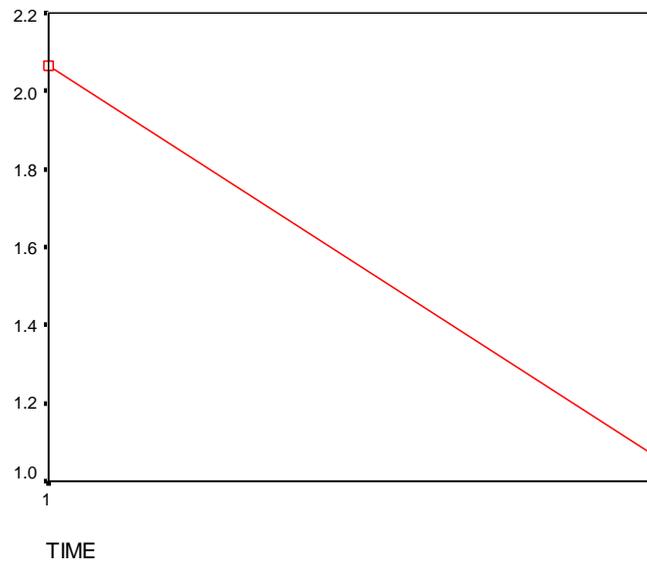


Figure 21: Profile plot of morning pain in the placebo group over time

Pain walking barefoot:

Table 24: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain walking barefoot.

Time effect Wilk's lambda	p value
0.954	0.423

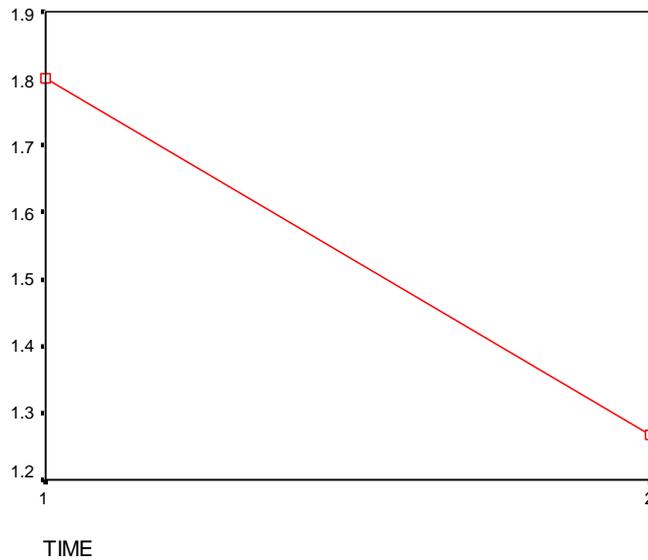


Figure 23: Profile plot of pain walking barefoot in the GTIASTM group over time

Table 25: Repeated measures ANOVA intragroup time comparison for placebo group for pain walking barefoot.

Time effect Wilk's lambda	p value
0.846	0.132

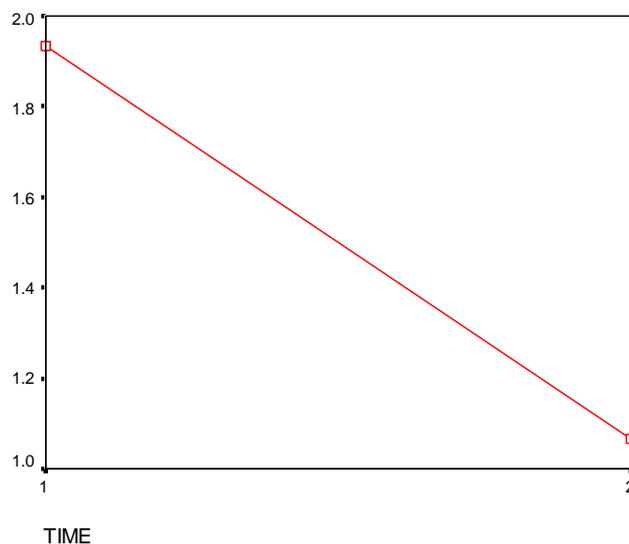


Figure 24: Profile plot of pain walking barefoot in the placebo group over time

Pain walking with shoes:

Table 27: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain walking with shoes.

Time effect Wilk's lambda	p value
0.808	0.090

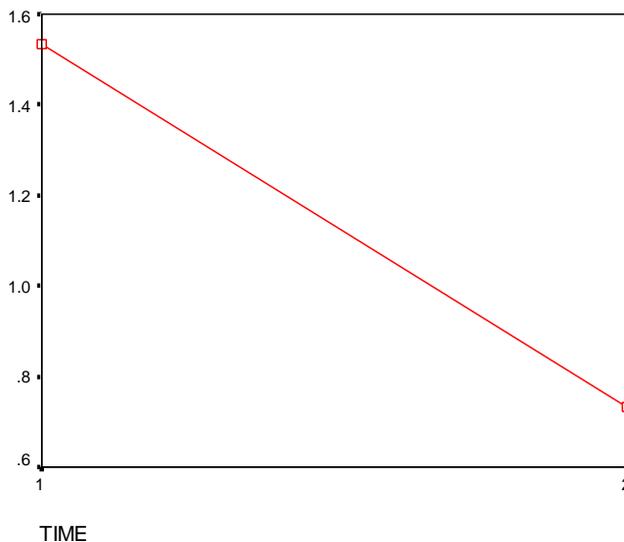


Figure 26: Profile plot of pain walking with shoes in the GTIASTM group over time

Table 28: Repeated measures ANOVA intragroup time comparison for placebo group for pain walking with shoes.

Time effect Wilk's lambda	p value
0.796	0.079

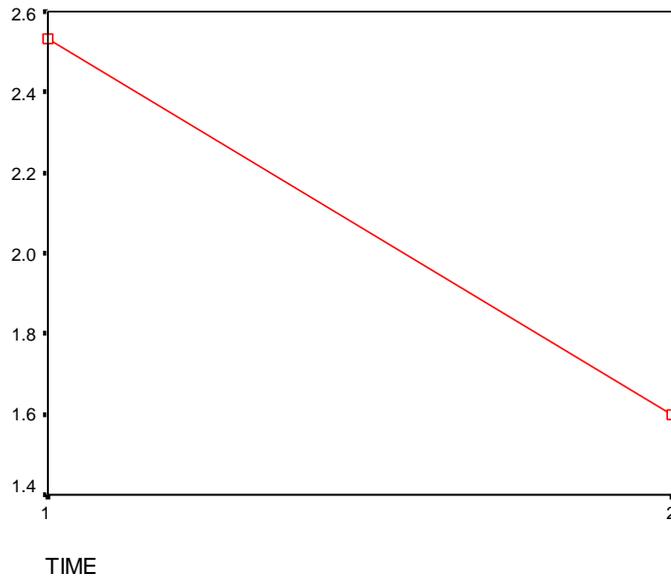


Figure 27: Profile plot of pain walking with shoes in the placebo group over time

Pain standing with shoes:

Table 30: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain standing with shoes.

Time effect Wilk's lambda	p value
0.989	0.228

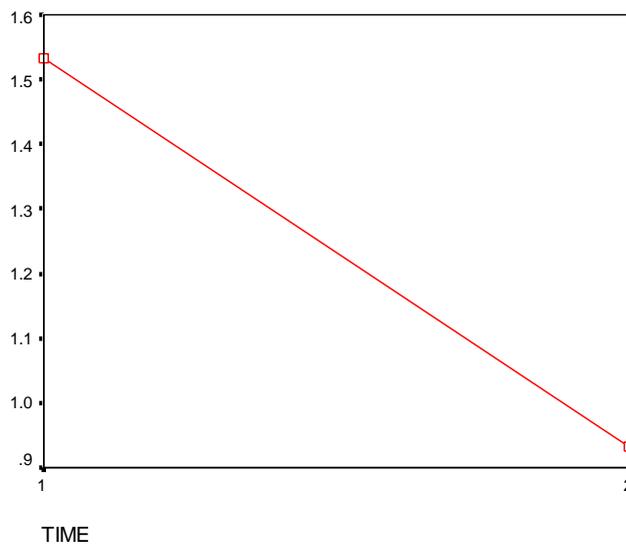


Figure 29: Profile plot of pain standing with shoes in the GTIASTM group over time

Table 31: Repeated measures ANOVA intragroup time comparison for placebo group for pain standing with shoes.

Time effect Wilk's lambda	p value
0.819	0.100

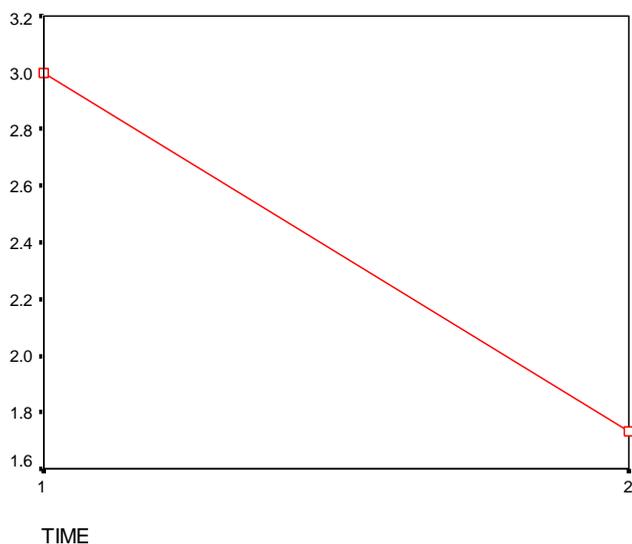


Figure 30: Profile plot of pain standing with shoes in the placebo group over time

Pain walking in the house:

Table 33: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain walking in the house.

Time effect Wilk's lambda	p value
0.800	0.082

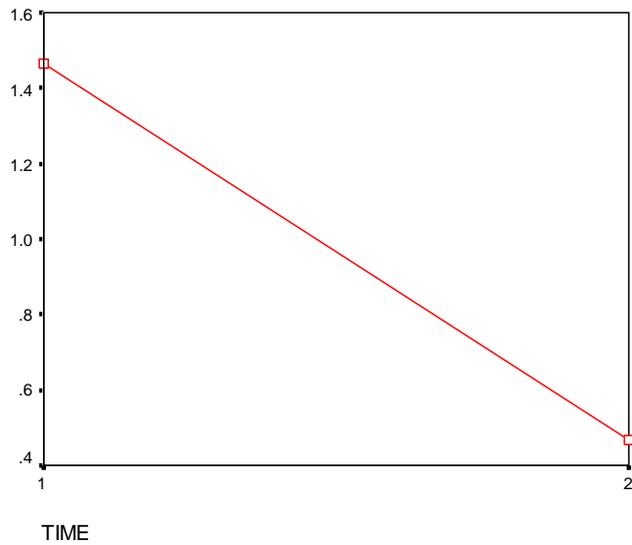


Figure 32: Profile plot of pain walking in the house over time in the GTIASTM group

Table 34: Repeated measures ANOVA intragroup time comparison for placebo group for pain walking in the house.

Time effect Wilk's lambda	p value
0.895	0.220

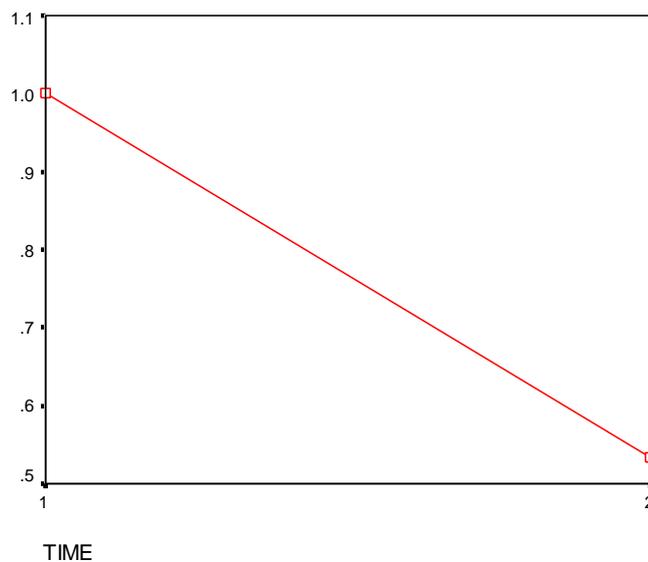


Figure 33: Profile plot of pain walking in the house over time in the placebo group

Pain walking outside:

Table 36: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain walking outside.

Time effect Wilk's lambda	p value
0.642	0.014

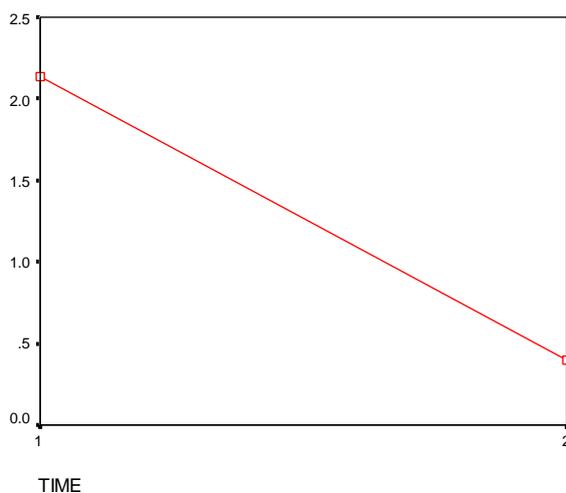


Figure 35: Profile plot of pain walking outside over time in the GTIASTM group

Table 37: Repeated measures ANOVA intragroup time comparison for placebo group for pain walking outside.

Time effect Wilk's lambda	p value
0.815	0.097

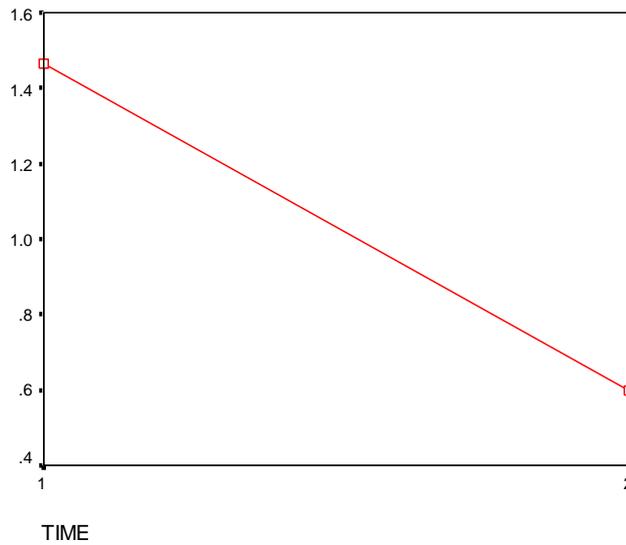


Figure 36: Profile plot of pain walking outside over time in the placebo group

Pain climbing stairs:

Table 39: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain climbing stairs.

Time effect Wilk's lambda	p value
0.812	0.094

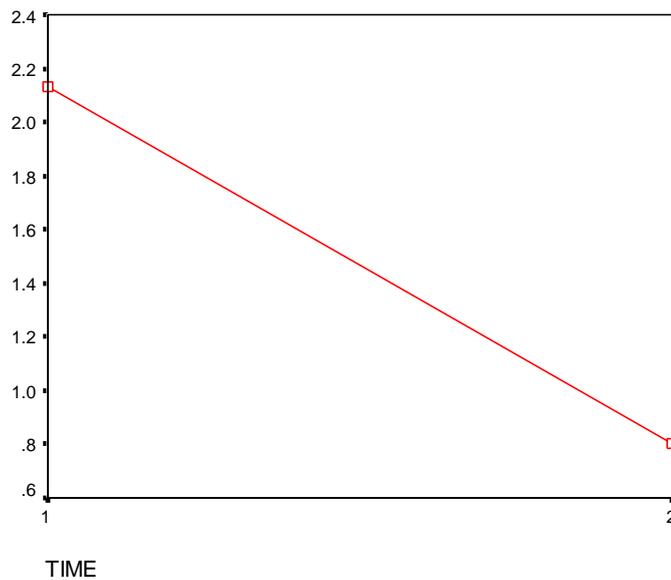


Figure 38: Profile plot of pain climbing stairs over time in the GTIASTM group

Table 40: Repeated measures ANOVA intragroup time comparison for placebo group for pain climbing stairs.

Time effect Wilk's lambda	p value
0.853	0.142

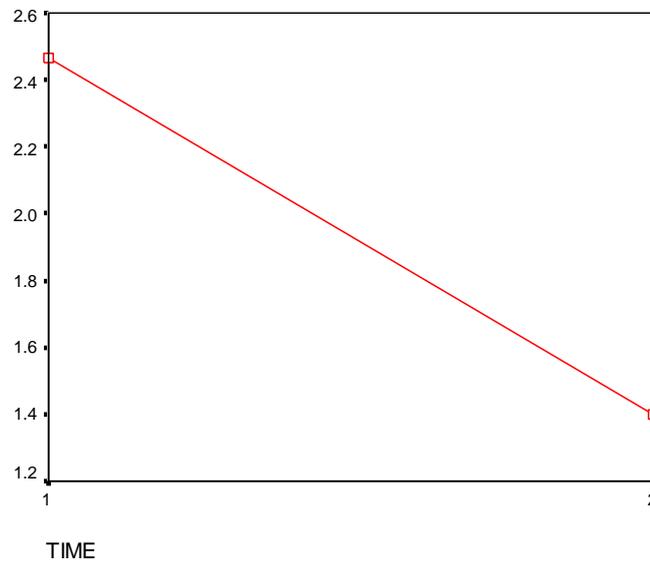


Figure 39: Profile plot of pain climbing stairs over time in the placebo group

Pain descending stairs:

Table 42: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain descending stairs.

Time effect Wilk's lambda	p value
0.796	0.079

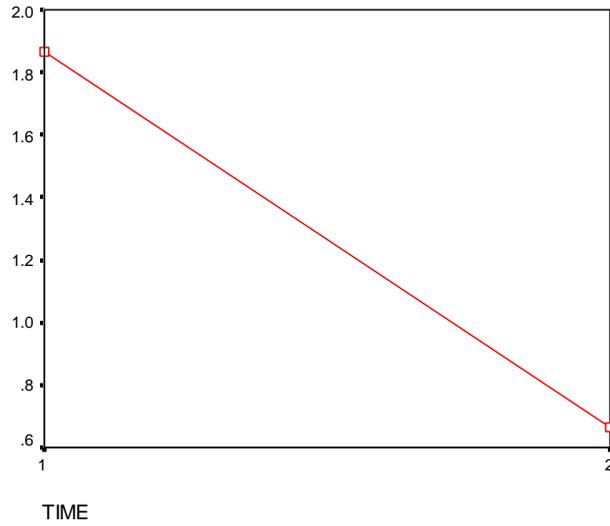


Figure 41: Profile plot of pain descending stairs over time in GTIASTM group

Table 43: Repeated measures ANOVA intragroup time comparison for placebo group for pain descending stairs.

Time effect Wilk's lambda	p value
0.879	0.187

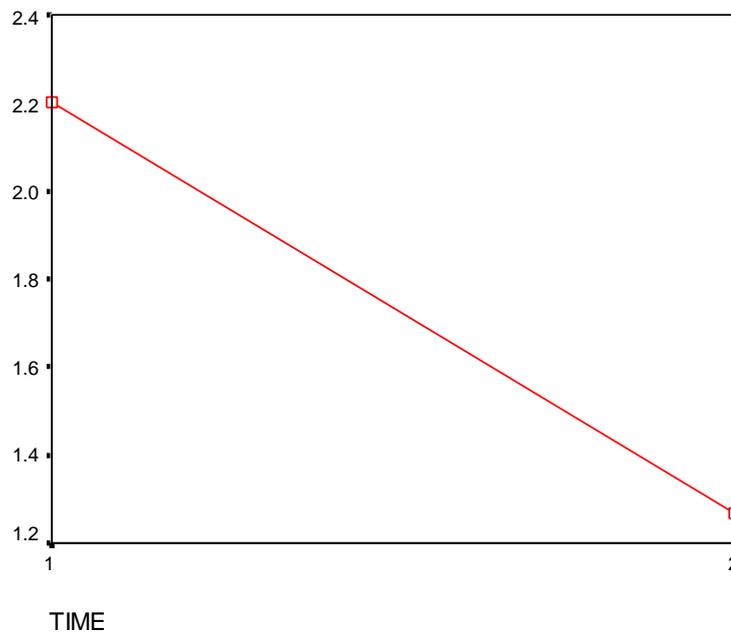


Figure 42: Profile plot of pain descending stairs over time in placebo group

Standing on tip toe:

Table 45: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain standing on tip toe.

Time effect Wilk's lambda	p value
0.716	0.033

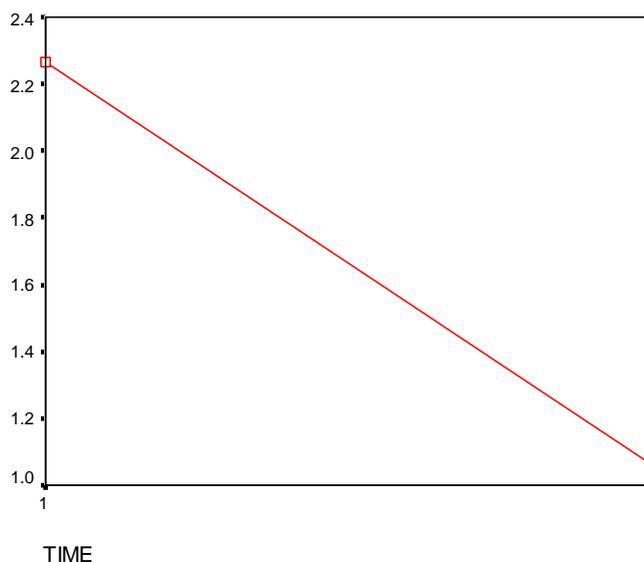


Figure 44: Profile plot of pain standing on tip toe over time in the GTIASTM group

Table 46: Repeated measures ANOVA intragroup time comparison for placebo group for pain standing on tip toe.

Time effect Wilk's lambda	p value
0.998	0.876

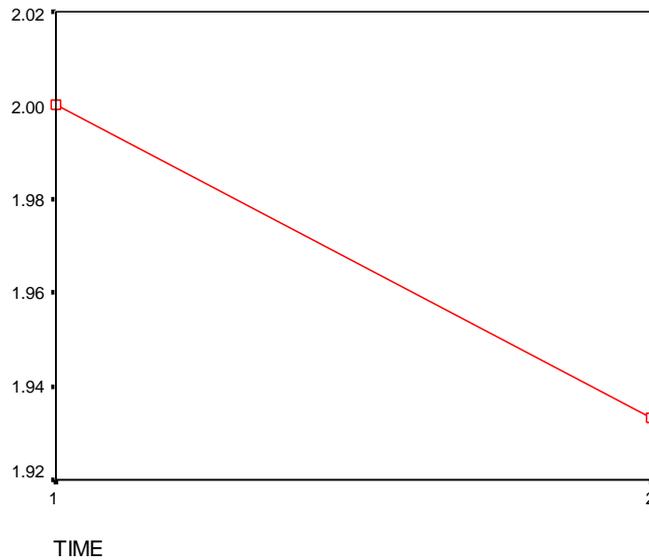


Figure 45: Profile plot of pain standing on tip toe over time in the placebo group

Getting up from a chair:

Table 48: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain getting up from a chair.

Time effect Wilk's lambda	p value
0.933	0.334

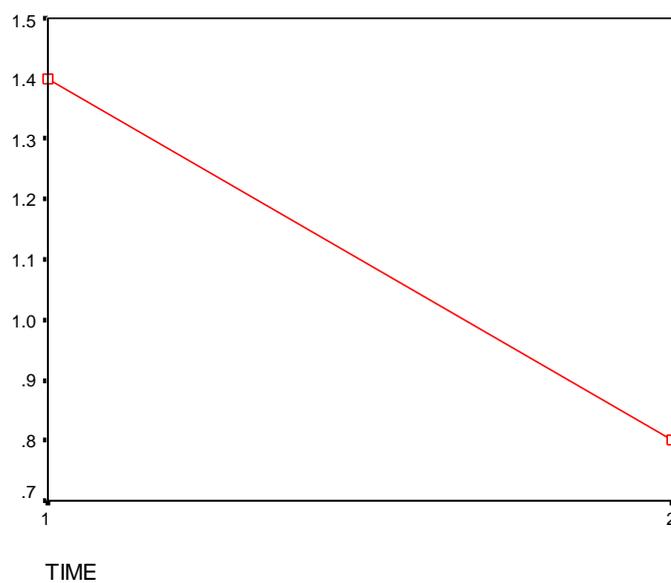


Figure 47: Profile plot of pain getting up from a chair over time in the GTIASTM group

Table 49: Repeated measures ANOVA intragroup time comparison for placebo group for pain getting up from a chair.

Time effect Wilk's lambda	p value
0.933	0.334

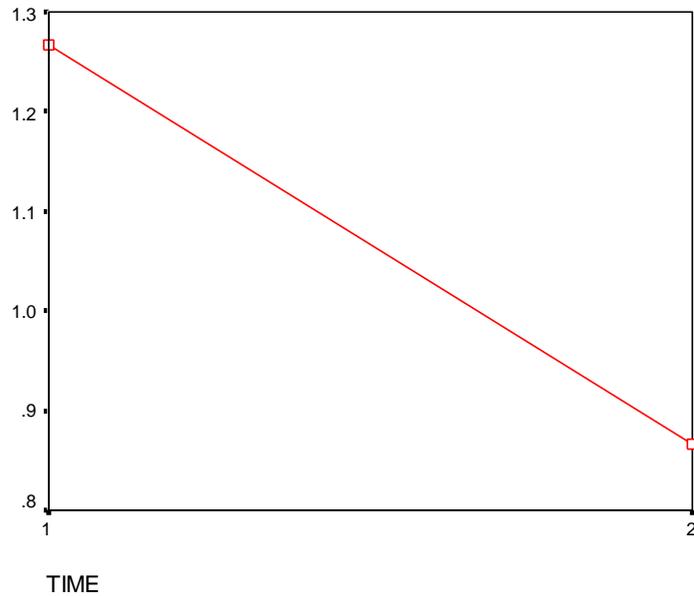


Figure 48: Profile plot of pain getting up from a chair over time in the placebo group

Climb curbs:

Table 51: Repeated measures ANOVA intragroup time comparison for GTIASTM group for pain climbing curbs.

Time effect Wilk's lambda	p value
0.859	0.152

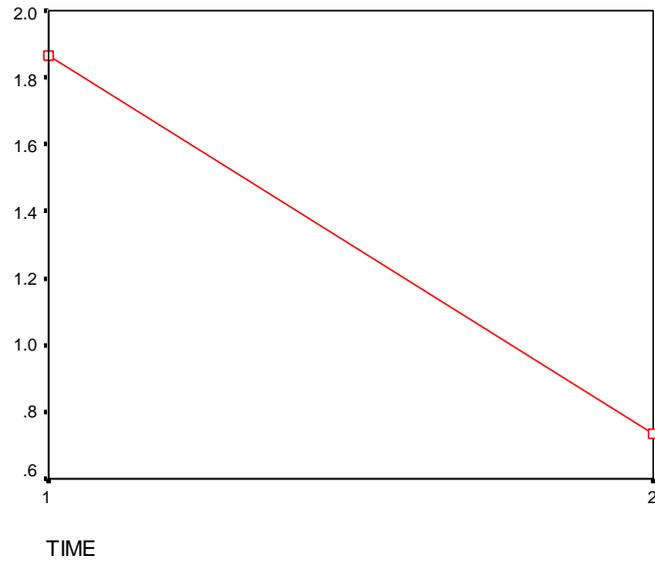


Figure 50: Profile plot of pain climbing curbs over time in GTIASTM group

Table 52: Repeated measures ANOVA intragroup time comparison for placebo group for pain climbing curbs.

Time effect Wilk's lambda	p value
0.950	0.405

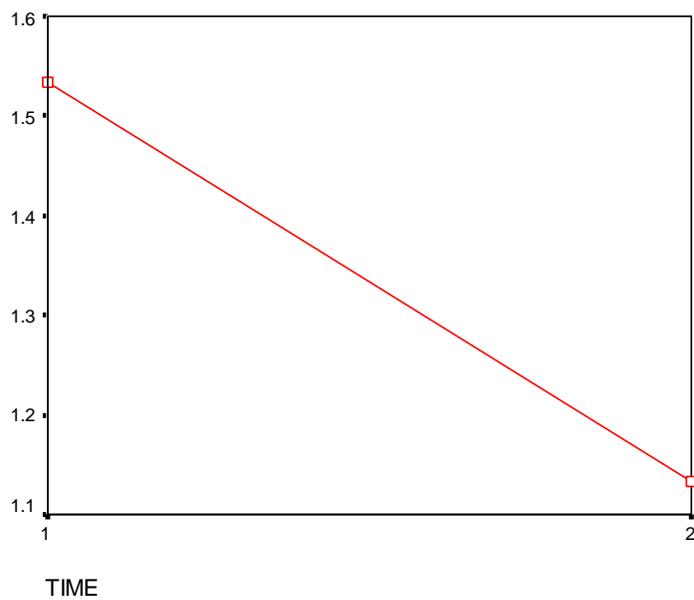


Figure 51: Profile plot of pain climbing curbs over time in placebo group