THE PREVALENCE AND CLINICAL PRESENTATION OF FIBULARIS MYOFASCIAL TRIGGER POINTS IN THE ASSESSMENT AND TREATMENT OF INVERSION ANKLE SPRAINS.

By
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A dissertation submitted in partial compliance with the requirements for the Master’s degree in Technology:
Chiropractic at Durban University of Technology

I, Ingrid van der Toorn, do declare that this dissertation is representative of my own work.

_________________                      ___________
Ingrid van der Toorn                      Date

APPROVED FOR FINAL EXAMINATION BY:

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Supervisor                      Date
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DEDICATION:

To the VDT's
ACKNOWLEDGEMENTS:

My parents, Joop and Stephne, who have supported me from the other side of the world. Thank you for making the last six years possible! I love you both and I am blessed to have you in my life.

Rajdu, all your interest, all your listening, all your help and humour. Thank you.
  Rashida.

Dr. White, my supervisor, thank you for all the time you put into this research. Your encouragement and realistic outlook on the research process helped me get through it, I am very grateful!

Class of 2006, you guys made Durban home!

And a big thank you to the patients that participated in this study, you made this study possible.

My Lord Jesus, thank you for the gift of talents, and for opportunities to further develop them.
ABSTRACT:

Ankle sprains account for 85% of all injuries to the ankle (Garrick, 1997). Inversion sprains result from a twisting of a weight-bearing foot into a plantarflexed and inverted position leading to lateral ankle ligament injury.

Louwerens and Snijders (1999) state that there are multiple factors involved in ankle sprains or lateral ankle instability. These include injury to the lateral ankle ligaments, proprioceptive dysfunction and decrease of central motor control. Other factors that still need further research include the role of the fibularis muscles, the influence of foot geometry and the role of subtalar instability in ankle sprains (Louwerens and Snijders, 1999). This study focused on the fibularis muscles.

Fibularis longus and brevis muscles are found in the lateral compartment of the leg and function to evert/pronate the foot and plantarflex the ankle. Fibularis tertius is found in the anterior compartment and its function is to evert and dorsiflex the foot. Myofascial trigger points in these three muscles refer pain primarily over the lateral malleolus of the ankle, above, behind and below it (Travell and Simons, 1993 2: 371). This is the exact area where ankle sprain patients experience pain.

Travel and Simons (1993 2:110) state that a once off traumatic occurrence can activate myofascial trigger points. When considering the mechanism of injury of a lateral ankle sprain, the importance of the fibularis muscles becomes obvious. When the ankle inverts during a lateral ankle sprain, these muscles are forcefully stretched whilst trying to contract to bring about their normal action. Therefore these muscles are often injured from traction when the foot inverts (Karageanes, 2004). It stands to reason that as a result of this mechanism of injury myofascial trigger points may develop in the fibularis muscles.

It was hypothesised that fibularis muscle trigger points would prove to be more prevalent in the injured leg when compared to the uninjured leg. To further
investigate this hypothesis, an analytical, cross sectional study (phase 1) was done on 44 participants between the ages of 15 and 50. Consecutive convenience sampling was used and participants were screened according to phase 1’s inclusion and exclusion criteria.

According to Travel et al. (1999: 19) myofascial trigger points (whether active or latent) can cause significant motor dysfunction. Trevino, et al. (1994) stated that fibularis muscle weakness is thought to be a source of symptoms after an inversion sprain.

Treatment for ankle sprains involves minimising swelling and bruising and encouraging adequate ankle protection in the acute phase. The patient is advised to rest for up to 72 hours to allow the ligaments to heal (Ivins, 2006). After the acute phase has passed, rehabilitation is focused on. This includes improving the ankle range of motion and proprioception. Attention is also given to strengthen the muscles, ligaments and tendons around the ankle joint. In the recommended treatment protocol however, no mention is made of evaluating the musculature around the ankle joint for myofascial trigger points and or treating these points. McGrew and Schenck (2003) noted that if the musculature and neural structures surrounding the ankle joint were affected during an ankle sprain injury, and were left unresolved, they would lead to chronic instability.

It was hypothesised that lateral ankle pain due to inversion ankle sprain injuries may be due to referred pain from the fibularis muscle trigger points. Patients treated with dry needling of the fibularis muscle trigger points would therefore show a greater improvement in terms of subjective and objective clinical findings when compared to a placebo treatment (detuned ultrasound) applied to the fibularis muscle trigger points.

Therefore phase 2 of this study was a randomised controlled trial that involved 40 participants, between the ages of 15 and 50, who were screened according to
phase 2’s inclusion and exclusion criteria. Participants were randomly divided into two groups of 20 participants; one received dry needling of the fibularis myofascial trigger points in the injured leg, the other detuned ultrasound (placebo treatment) applied to the fibularis myofascial trigger points in the injured leg. Each participant received two treatments and measurements were taken before each treatment, followed by a re-evaluation three days after the last treatment.

Data were entered into a MS Excel spreadsheet and imported into SPSS version 13 (SPSS Inc., Chicago, Illinois, USA) for analysis. A p value of <0.05 was considered as statistically significant.

For phase 1 the groups were compared with regard to the various quantitative outcomes using paired t-tests, and comparisons with categorical outcomes were done using McNemar’s chi square tests. Associations between presence/number of trigger points and clinical outcomes were done by means of one-way ANOVA in the injured ankles. Correlations between baseline subjective and objective outcome measurements were done for the injured ankles using Pearson’s correlation coefficients.

For phase 2 Repeated measures ANOVA was used to compare treatment groups over time, with profile plots of means by group over time. A significant time by group (time*group) interaction indicated a significant treatment effect. The direction of the treatment effect was assessed from the profile plots. This was done separately for each outcome measurement.

The results of phase 1 showed a statistically significant prevalence of fibularis longus and brevis myofascial trigger points in the injured leg compared to the uninjured leg. Fibularis tertius trigger points were found to be more prevalent in the injured leg, but in a statistically non-significant manner.

In the injured ankle, subjective pain measured by the NRS was not correlated with any of the severity measurements (Myofascial Diagnostic Scale, Goniometer
readings and Ankle Functional Evaluation Scale) at baseline. This indicates that lateral ankle pain experienced by ankle sprain patients had no correlation to the severity of the myofascial trigger points in the fibularis muscles.

The results of phase 2 showed that only one outcome measurement, namely the Myofascial Diagnostic Scale score (p=0.030) could statistically support the hypothesis that dry needling of the fibularis muscles is a more effective treatment method than the placebo treatment in the relieving of lateral ankle pain experienced by ankle sprain patients. Subjective pain measurement (Numerical Pain Rating Scale), objective pain measurement (Algometer), dorsiflexion and eversion range of motion (Goniometer) showed no difference in the two groups although a statistically insignificant trend was noted towards a more beneficial effect in the dry needling group. Plantarflexion and inversion range of motion decreased in the treatment group compared to the placebo group and indicated a non-significant treatment effect.

The results of this study indicate that although fibularis muscle trigger points are more prevalent in the injured leg than in the uninjured leg (as shown in hypothesis 1), the lateral ankle pain experienced after an inversion ankle sprain cannot solely be attributed to referred pain from the fibularis muscle trigger points. There are many other factors involved in this injury, which amongst others include, lateral ankle ligament and capsular tears and the resulting oedema and haemorrhage (Cailliet, 1997). All these factors need to be considered in an ankle sprain treatment protocol, so as to ensure timely recovery and return to activity.
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CHAPTER ONE

Introduction

1.1. The problem:
Ankle sprains account for 85% of all injuries to the ankle (Garrick, 1997).
Inversion ankle sprains are more common than eversion sprains (Moore and
Agur, 1995: 276) due to the lateral ankle ligaments being much weaker than the
medial ligaments (Shapiro, et al. 1994). Inversion sprains result from a twisting of
a weight-bearing foot into a plantarflexed and inverted position leading to lateral
ankle ligament injury. Often this injury occurs due to running on uneven terrain,
trauma and overload of the fibularis muscles have also been suggested
(Rimando, 2005). Following an inversion sprain, the patient may complain of
tenderness over the lateral ankle, associated with swelling and bruising
(Myerson, 1995).

Diagnosis is based on the patients’ medical history and mechanism of injury,
physical examination and an ankle examination. This involves comparing the
injured ankle with the uninjured ankle in terms of observation (during gait and at
rest), palpation for tenderness, range of motion testing, muscle strength
evaluation, neurological examination, vascular examination and specific
ligamentous examinations (Reid, 1992: 22; McGrew and Schenck, 2003).

Suggested treatment for inversion ankle sprains (grade I and II) includes
protection, rest, ice, compression and elevation, weight bearing as tolerated and
nonsteroidal anti-inflammatory drugs to control pain and swelling. Once pain-free,
the patient should start range of motion and strengthening exercises (Wexler,
1998). In the literature, although strengthening exercises for the ankle
musculature is recommended, no mention is made of evaluating the musculature
for myofascial trigger points and or treating these points (in any way, including dry needling) prior to strengthening.

The lateral compartment of the leg consists of the fibularis longus and fibularis brevis muscles. This compartment’s function is to evert/pronate the foot and plantarflex the ankle. Fibularis tertius forms part of the anterior compartment of the leg and assists with eversion and dorsiflexion of the foot (Moore and Agur, 1995: 254).

Myofascial trigger points in fibularis longus and brevis refer pain and tenderness primarily over the lateral malleolus of the ankle, above, behind and below it. Pain is also felt along the lateral aspect of the foot. Fibularis tertius trigger points refer pain along the anterolateral aspect of the ankle (Travell and Simons, 1993 2:371). This pain distribution is the exact area in which inversion ankle sprain patients experience pain.

When considering the mechanism of injury of a lateral ankle sprain, the importance of the fibularis muscles becomes obvious. During the gait cycle the fibularis muscles contract to allow plantar flexion and pronation. When the ankle inverts during a lateral ankle sprain, these muscles are forcefully stretched whilst trying to contract to bring about their normal action. Therefore these muscles are often injured from traction when the foot inverts (Karageanes, 2004). It stands to reason that as a result of this mechanism of injury myofascial trigger points may develop in the fibularis muscles.

1.2. Aims and Objectives of the study:
This study aims to investigate the prevalence and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains.
1.2.1. **Objective 1:**
Determine the prevalence of fibularis muscle trigger points in ankle sprain patients.

*Hypothesis:* Fibularis muscle trigger points will prove to be more prevalent in the injured leg when compared to the uninjured leg.

1.2.2. **Objective 2:**
To evaluate the role of myofascial trigger points of the fibularis muscle on the clinical presentation of inversion ankle sprains.

*Hypothesis:* Lateral ankle pain due to inversion ankle sprain injuries may be due to referred pain from the fibularis muscle trigger points.

1.2.3. **Objective 3:**
Determine whether dry needling of the fibularis muscle should be considered in the treatment protocol for inversion ankle sprains.

*Hypothesis:* Patients treated with dry needling of the fibularis muscle trigger points would show a greater improvement in terms of subjective and objective clinical findings when compared to a placebo treatment (detuned ultrasound) applied to the fibularis muscle trigger points.

1.3. **Benefits of this study:**
Phase 1 of this research aims to provide information regarding the prevalence of fibularis muscle trigger points in ankle sprain patients as there is presently a lack of literature concerning this topic. It is important to know the prevalence of these trigger points as their presence or absence will affect the recommended treatment protocol for inversion ankle sprains. McGrew and Schenck (2003) stated that the musculature and neural structures surrounding the ankle joint may be affected during an ankle sprain injury, and if left unresolved, these deficits will
lead to chronic instability, which may affect future athletic ability and may increase risk of re-injury.

Phase 2 will compare the clinical outcomes of a treatment method namely dry needling with a placebo treatment (detuned ultrasound). The purpose of the treatment is not to determine the therapeutic effects of dry needling (as this is already known) but rather to affect the trigger points and then monitor for any change in the clinical presentation of the ankle sprain. This phase may give us a clearer picture as to whether the lateral ankle pain experienced by ankle sprain patients is referred pain from the fibularis muscles or whether it is true ankle joint pain.
CHAPTER TWO
Literature review

2.1. Introduction:
This chapter will discuss the following:
- Incidence and prevalence of ankle sprains.
- Anatomy and biomechanics of the ankle and relevant structures.
- Mechanism of injury and grading of sprains
- Treatment methods
- Differential diagnosis
- Conclusion

2.2. Incidence and prevalence of ankle sprains:
One of the most commonly injured joints in the body is the ankle (Fallat, *et al.* 1998 and Jerosch and Bischof, 1996). Ankle sprains are one of the most common musculoskeletal injuries that primary care physicians will come across in their practices (McGrew and Schenck, 2003) and they account for 85% of all injuries to the ankle (Garrick, 1997). Inversion ankle sprains are more common than eversion sprains (Moore and Agur, 1992: 276) due to the lateral ankle ligaments being much weaker than the medial ligaments (Shapiro, *et al.* 1994).

An epidemiological survey on ankle sprains in Hong Kong Chinese athletes showed as much as 73% of these athletes had recurrent ankle sprains and 59% suffered from residual symptoms, which affected their performance. 51.8% of the participants reported unilateral ankle sprains, and it was also noted that the dominant leg was 2.40 times more likely to be injured than the non-dominant leg (Yeung, *et al.* 1994).
2.3. Anatomy and biomechanics of the ankle:

2.3.1. Talocrural joint:
The ankle, also known as the talocrural joint, is a hinge type synovial joint (Moore and Agur, 1995: 274) and is formed by the talus, the medial malleolus of the tibia, and the lateral malleolus of the fibula (Magee, 1997: 599). The ankle joint has a ‘mortise and tenon’ shape; the talus acts as the tenon and articulates with the distal tibia and fibula, which forms the mortise (McGrew and Schenck, 2003). The talus is approximately 2.4mm wider anteriorly than posteriorly (Magee, 1997: 599).

The talocrural joint allows for dorsiflexion and plantarflexion. During dorsiflexion the anterior talus is wedged between the malleoli and therefore allows little or no inversion or eversion of the ankle joint (Magee, 1997: 599). This is the closed pack position of the ankle. During plantarflexion however the posterior talus lies within the mortise, and due to its smaller diameter, allows more mobility to the ankle joint (Magee, 1997: 599).

The articular fibrous capsule attaches superiorly to the borders of the tibia and the malleoli and inferiorly to the talus. The capsule is thin anteriorly and posteriorly, but is supported laterally and medially by collateral ligaments. The lateral ligament consists of three parts:

- The anterior talofibular ligament,
- The posterior talofibular ligament,
- The calcaneofibular ligament.

The medial (deltoid) ligament is stronger than the lateral ligament and consists of four parts:

- The tibionavicular ligament,
- The anterior tibiotalar ligament,
- The posterior tibiotalar ligament,
- The tibiocalcaneal ligament (Moore and Agur, 1995: 275).
**2.3.2. Subtalar joint:**
The functional unit of the ankle has to include the subtalar joint, as this is where inversion and eversion of the ankle occurs (Reid, 1992: 215). The subtalar joint (talocalcaneal) is a plain synovial joint and is formed by the articulation of the inferior surface of the talus and the superior surface of the calcaneus (Moore and Agur, 1995: 277).

**2.3.3. Distal tibiofibular joint:**
This fibrous joint (syndesmosis) is formed by a triangular area on the medial surface of the inferior part of the fibula that articulates with a facet on the inferior end of the tibia. A strong interosseous ligament (continuation of the interosseus membrane) connects the tibia and fibula. The anterior and posterior inferior tibiofibular ligaments provide stability for the joint (Moore and Agur, 1995: 273).

**2.3.4. Nerve supply of the ankle joint:**
Innervation of the ankle joint is derived from the tibial and deep fibular nerve (Moore and Agur, 1995: 275). The distal tibiofibular joint receives innervation from the tibial, deep fibular and the saphenous nerve (Moore and Agur, 1995: 274).

**2.3.5. Muscles related to the ankle joint:**
The leg is divided into an anterior, lateral and posterior compartment. A brief overview will be given of the muscles found in these three compartments (Moore and Agur, 1995: 254-259). The muscles that are of particular relevance to this study will be discussed, in detail, later in this chapter.

<table>
<thead>
<tr>
<th>Anterior compartment:</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle:</td>
<td>Innervation:</td>
<td>Main action:</td>
</tr>
<tr>
<td>Tibialis anterior</td>
<td>Deep fibular nerve</td>
<td>Dorsiflexes ankle and</td>
</tr>
<tr>
<td></td>
<td>(L4 and 5)</td>
<td>inverts foot</td>
</tr>
<tr>
<td>Extensor hallucis</td>
<td>Deep fibular nerve</td>
<td>Extends great toe and</td>
</tr>
<tr>
<td>longus</td>
<td>(L5 and S1)</td>
<td>dorsiflexes ankle</td>
</tr>
<tr>
<td>Muscle</td>
<td>Innervation</td>
<td>Main actions</td>
</tr>
<tr>
<td>------------------------------</td>
<td>----------------------------------</td>
<td>---------------------------------------------------</td>
</tr>
<tr>
<td>Extensor digitorum longus</td>
<td>Deep fibular nerve (L5 and S1)</td>
<td>Extends lateral four digits and dorsiflexes ankle</td>
</tr>
<tr>
<td>Fibularis (fibularis) tertius</td>
<td>Deep fibular nerve (L5 and S1)</td>
<td>Dorsiflexes ankle and aids in eversion of foot</td>
</tr>
</tbody>
</table>

### Lateral compartment:

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Innervation</th>
<th>Main actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibularis (fibularis) longus</td>
<td>Superficial fibular (peroneal) nerve (L5, S1 and S2)</td>
<td>Evert foot and weakly plantarflex ankle</td>
</tr>
<tr>
<td>Fibularis (fibularis) brevis</td>
<td>Superficial fibular (peroneal) nerve (L5, S1 and S2)</td>
<td>Evert foot and weakly plantarflex ankle</td>
</tr>
</tbody>
</table>

### Posterior compartment:

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Innervation</th>
<th>Main actions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Superficial muscles:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrocnemius</td>
<td>Tibial nerve (S1 and S2)</td>
<td>Plantarflexes ankle, raises heel during gait, and flexes leg at knee joint</td>
</tr>
<tr>
<td>Soleus</td>
<td>Tibial nerve (S1 and S2)</td>
<td>Plantarflexes ankle and steadies leg on foot</td>
</tr>
<tr>
<td>Plantaritis</td>
<td>Tibial nerve (S1 and S2)</td>
<td>Weakly assists gastrocnemius in plantarflexing ankle and flexing knee</td>
</tr>
<tr>
<td><strong>Deep muscles:</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Popliteus</td>
<td>Tibial nerve (L4, L5 and S1)</td>
<td>Weakly flexes and unlocks knee</td>
</tr>
<tr>
<td>Flexor hallucis longus</td>
<td>Tibial nerve (S2 and S3)</td>
<td>Flexes great toe at all joints and plantarflexes ankle; supports medial longitudinal arch of foot</td>
</tr>
</tbody>
</table>
Flexor digitorum longus  
Tibial nerve (S2 and S3)  
Flexes lateral four digits and plantarflexes ankle; supports longitudinal arches of foot

Tibialis posterior  
Tibial nerve (L4 and L5)  
Plantarflexes ankle and inverts foot

Of particular importance in this study is the fibularis longus, brevis and tertius muscle. The following table details their proximal and distal attachments (Moore and Agur, 1995: 254-259).

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Proximal attachment:</th>
<th>Distal attachment:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fibularis longus</td>
<td>Head and superior two thirds of lateral surface of the fibula</td>
<td>Base of the first metatarsal and medial cuneiform</td>
</tr>
<tr>
<td>Fibularis brevis</td>
<td>Inferior two-thirds of the lateral surface of the fibula</td>
<td>Dorsal surface of the tuberosity on the lateral side of the base of the fifth metatarsal</td>
</tr>
<tr>
<td>Fibularis tertius</td>
<td>Inferior third of the anterior surface of the fibula and interosseous membrane</td>
<td>Dorsum of the base of fifth metatarsal</td>
</tr>
</tbody>
</table>

2.4. Mechanism of injury and grading of sprains:

When the foot strikes the ground during the normal gait cycle, the foot is plantarflexed and supinated. In this position the talus is moveable within the mortise joint, and so the ankle relies on the ligaments for stability. If there is rotational or lateral stress while weight bearing, the lateral ligaments can be overwhelmed causing an inversion ankle sprain (Calliet, 1997). Often this injury occurs due to direct trauma (Rimando, 2005) or sporting activities e.g. running on uneven terrain, stepping in a hole or landing from a jump in an unbalanced position (Hockenbury and Sammarco, 2001). Overload of the fibularis muscles has also been suggested (Rimando, 2005).
Ankle sprain injuries can be classified into different grades according to the severity of the injury. For this study the classification system as described by Reid (1992: 226) will be used.

<table>
<thead>
<tr>
<th>Severity:</th>
<th>Pathology:</th>
<th>Signs and Symptoms:</th>
<th>Disability:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1 stable</td>
<td>-Mild stretch -No instability -Singular ligament involved -Often ATFL</td>
<td>-No haemorrhage -Minimal swelling -Point tenderness -No anterior drawer -No varus laxity</td>
<td>-No or little limp -Minimal functional loss -Difficulty hopping -Recovery 2-10 days</td>
</tr>
<tr>
<td>Grade 2 stable</td>
<td>-Large spectrum of injury -Mild to moderate instability -Complete tearing of ATFL or partial tearing of ATFL plus CFL</td>
<td>-Some haemorrhage -Localized swelling -Margins of Achilles less defined -May be anterior drawer -No varus laxity</td>
<td>-Limp with walking -Inability to toe raise -Inability to hop -Unable to run -Recovery 10-30 days</td>
</tr>
<tr>
<td>Grade 3 unstable</td>
<td>-Significant instability -Complete tear of anterior capsule and talofibular ligament and associated tear of ATFL and CFL</td>
<td>-Diffuse swelling both sides of Achilles tendon -Early haemorrhage -Medial and lateral tenderness -Positive anterior drawer -Positive varus laxity</td>
<td>-Unable to fully weight bear -Significant pain Inhibition -Almost complete loss of range of motion initially -Recovery 30-90 days</td>
</tr>
</tbody>
</table>

Louwerens and Snijders (1999) state that there are multiple factors involved in ankle sprains or lateral ankle instability. These include injury to the lateral ankle ligaments, proprioceptive dysfunction and decrease of central motor control. Other factors that still need further research include the role of the fibularis muscles, the influence of foot geometry and the role of subtalar instability in ankle sprains (Louwerens and Snijders, 1999). The factors relevant to this study
will be discussed further; they include proprioception and the role of fibularis muscles.

2.5. Proprioception:

Louwerens and Snijders (1999) state: “Proprioception is the sensory feedback to the central nervous system for conscious appreciation of the position and movement of the limbs.” Proprioception is monitored by proprioceptors, which are specialised sensory nerve endings located in muscles and tendons (Martin, 2003). Apart from proprioception, the proprioceptors in the ligaments are said to control muscle tone and coordination around a joint, thereby increasing stability (Freeman and Wyke, 1967). Freeman (1964) suggested proprioceptive deficit is caused by ligamentous and capsular injury (as seen in ankle sprains) that damages these articular nerve endings in the joint capsule and ligaments. This deficit affects the muscles of the injured leg causing the ankle to be more susceptible to the symptom ‘giving way’ (also known as functional instability). Karlsson’s (1989) research substantiates Freeman’s claim. He used a trap door to elicit and simulate the ankle sprain injury, and then measured the time from tilting of the plate to the first response of the fibularis longus or brevis muscle. He compared the symptomatic to the asymptomatic leg and found the mean reaction time in the stable ankle to be less than in the unstable ankle. Konradsen and Ravn (1990) compared the reaction time of the first muscular response of the fibularis muscles to the first muscular response of the upper leg muscles in patients with stable and unstable ankles. They noted a prolonged fibularis reaction time (comparable with results from Karlsson (1989)) in the unstable ankle, but no difference in the reaction time of the upper leg muscles. This suggests the possibility that functional instability is not associated with a central processing disturbance, but rather with a proprioceptive deafferentation (Konradsen and Ravn, 1990).

Travell, et al. (1999 1:19) stated that myofascial trigger points could cause significant motor dysfunction. It is therefore necessary to further investigate the
importance of the fibularis muscle trigger points in the clinical presentation of ankle sprains, so as not to over emphasise proprioception deafferentation when the cause of the delayed fibularis reaction time may in fact be due to myofascial trigger points.

2.6. The role of the fibularis muscles:
The fibularis muscles play a vital role in the movement of the ankle. During the gait cycle the fibularis longus and brevis muscle’s role is to evert and plantarflex the foot, and fibularis tertius assists with eversion and dorsiflexion rather than plantarflexion of the foot (Moore and Agur, 1995: 254). When the ankle inverts during a lateral ankle sprain, these muscles are forcefully stretched whilst trying to contract to bring about their normal action. Therefore these muscles are often injured from traction when the foot inverts (Karageanes, 2004). Travel and Simons (1993 2: 110) state that a once off traumatic occurrence can activate myofascial trigger points. It stands to reason that as a result of the ankle sprain mechanism of injury, myofascial trigger points may develop in the fibularis muscles.

Travell, et al. (1999 1: 35) recommended the following criteria for identifying active or latent trigger points:

**Essential criteria:**
- A palpable taut band.
- Tender nodule in taut band.
- Patients’ recognition of current pain complaint by pressure on the tender nodule (identifies active trigger points).
- Painful limit to full stretch range of motion.

**Confirmatory observations:**
- Visual or tactile identification of local twitch response.
- Pain or altered sensation on compression of the tender nodule.
To diagnose myofascial trigger points all 4 essential criteria must be present, and the presence of the confirmatory signs serve to reinforce the diagnosis (Travell and Simons, 1993 2:35).

Myofascial trigger points in fibularis longus and brevis refer pain and tenderness primarily over the lateral malleolus of the ankle, above, behind and below it. Pain is also felt along the lateral aspect of the foot. Occasionally a spill over pattern may be felt over the lateral aspect of the middle third of the leg. Fibularis tertius trigger points refer pain along the anterolateral aspect of the ankle, mainly anterior to the lateral malleolus, with a spill over pattern to the outer side of the heel (Travell and Simons, 1993 2: 371). The pain distribution of the fibularis muscles correlate with the area where inversion ankle sprain patients experience pain.

Diagram 1: Location and referral pattern of fibularis myofascial trigger points
2.7. **Treatment methods:**

The standard recommended treatment protocol (Hockenbury and Sammarco, 2001) following an acute grade 1/2 ankle sprain could be simplified with a mnemonic: PRICE (protection, rest, ice, compression and elevation).

Protection of the ankle during initial healing is vital. This involves functional bracing or taping (Louwerens and Snijders, 1999) to promote weight bearing and normalise gait (McGrew and Schenck, 2003). Protected range of motion is superior to rigid immobilization with a cast due to early mobilisation of the injured ankle being encouraged (Hockenbury and Sammarco, 2001). On return to activity it is recommended that taping and bracing be continued to prevent reinjury (Wexler, 1998). Ankle taping has also been shown to have positive effects on proprioceptive function (Karlsson and Andreasson, 1992).

Rest involves activity as tolerated. Crutches may initially be needed until weight bearing is pain free (Hockenbury and Sammarco, 2001).

Ice or cryotherapy limits the amount of swelling (effusion) and bleeding (haematoma formation) around the capsule of the ankle joint (Garrick, 1997), and helps to reduce pain (Rimando, 2005). Generally it is recommended that patients apply ice for 15-20 minutes, 3 times daily (Rimando, 2005).

Compression can be any form of pressure placed around the ankle that will limit oedema and haemorrhage (Reid, 1992). Often an ice pack with a wrap is used on the sports field (Reid, 1992). Other forms of compression include an elastic ankle sleeve or taping (Rimando, 2005).

Elevation encourages reduction of swelling. Advise the patient to keep the ankle above the level of the heart (Rimando, 2005).
In the acutely sprained ankle NSAIDs could also be used to reduce pain and limit inflammation (Rimando, 2005). Once the patient is pain free, a rehabilitation program should be started focusing on range of motion, fibularis strengthening exercises and proprioception (McGrew and Schenck, 2003; Calliet, 1997; Hockenbury and Sammarco, 2001; Wexler, 1998). In the literature, although strengthening exercises for the ankle musculature is recommended, no mention is made of evaluating for or treating any myofascial trigger points (in any way, including dry needling) prior to strengthening.

Myofascial trigger points, active or latent, can cause significant motor dysfunction (Travell, et al. 1999 1: 19). Fibularis muscle weakness is thought to be a source of symptoms after an inversion ankle sprain (Trevino, et al. 1994). McGrew and Schenck (2003) stated that the musculature and neural structures surrounding the ankle joint may be affected during an ankle sprain injury, and if left unresolved, these deficits will lead to chronic instability, which may affect future athletic ability and may increase risk of re-injury.

The treatment methods used in this study were dry needling of fibularis muscle trigger points and a placebo treatment (detuned ultrasound) applied to the fibularis trigger points. A short discussion of these treatment methods follows.

Myofascial trigger points can be treated in many different ways; the common denominator in all treatment modalities is the release of contractures in the taut bands of skeletal muscle (Schneider, 1995). Dry needling studies done by Garvey, et al. (1989) and Lewit (1978) found dry needling to be highly effective in the treatment of chronic myofascial pain. Garvey, et al. (1989) concluded that the critical factor in relieving pain was not the injected substance, but rather the mechanical stimulus to the trigger point.

Placebo is defined as a medicine/treatment that is ineffective but may help to relieve a condition because the patient believes in its therapeutic powers (Martin,
2003). Placebo and its effect on patients depends on the environment where the experiment takes place, the tools being used, the patient’s receptivity and the manner and the intent of the doctor (Brom, 1992). To objectively investigate the effect that dry needling of the fibularis muscle trigger points may or may not have on the lateral ankle pain experienced by inversion ankle sprain patients, it is necessary for a placebo to be used (as previously done by Pellow and Brantingham (2001)).

2.8. Differential diagnosis:
Many patients suffer from residual symptoms after sustaining an acute lateral ankle ligament injury, despite having received adequate treatment. The symptoms range from recurrent sprains, pain, swelling, stiffness and sensations of ‘giving way’ (Louwerens and Snijders, 1999). Braun (1999) noted residual symptoms in 72.6% of ankle sprain patients 6 to 18 months post-injury. A careful review of the history, a current physical examination and appropriate plain radiographs is then required to rule out any missed diagnosis (McGrew and Schenck, 2003). Specific injuries that can occur at the lateral ankle joint include:

2.8.1. Chronic lateral instability
Patients complain of a ‘giving way’ sensation or instability in the ankle, pain, swelling and actual re-injury during sports, walking on uneven surfaces or activities of daily life (Louwerens and Snijders, 1999). The most obvious cause would be post traumatic laxity, although other factors also come into play e.g. muscle weakness, poor proprioceptive control, and pain inhibition secondary to impingement or peroneal/ fibularis tendon subluxation (Reid, 1992).

2.8.2. Fractures
Following an ankle sprain, the anterior process of the calcaneus, the lateral process and the dome of the talus, the base of the fifth metatarsal (Myerson, 1995), the navicular and distal fibula (Rimando, 2005) as well as the proximal
fibula (Maisonneuve fracture) should be evaluated for fractures (Wolfe et al. 2001). The patient will complain of pain, swelling, inability to bear weight and gross deformity is often present. Ankle radiographs, digital imaging and bone scans can be utilised to confirm the location of the fracture. In the skeletally immature, epiphyseal separation should not be overlooked (Reid, 1992) and stress views may be needed to rule out Salter Harris fractures.

2.8.3. Osteochondritis dissecans of the dome of the
During an inversion ankle sprain (or any other trauma) a small part of the dome of the talus may lose its blood supply. With time this area slowly deteriorates and forms a rough degenerative surface (Osteochondritis Dissecans-Talus, 1999). As a result a fragment of bone and cartilage may separate from the surface of a joint (Martin, 2003). The usual sites of OCD of the talar dome are the posteromedial aspect (56%) and the anterolateral aspect (44%) of the talus (Osteochondritis Dissecans-Talus, 1999). Symptoms include a deep ache in the ankle joint (aggravated by exercise), ankle swelling, a catching sensation or even joint locking. There may also be joint line tenderness and loss of range of motion. Routine ankle radiographs can detect this injury (Reid, 1992).

2.8.4. Tarsal Tunnel Syndrome
This is a compression neuropathy of the tibial nerve or its terminal branches, the medial and lateral plantar nerves, as it passes through the fibro-osseous tarsal tunnel (Hollis and Lemay, 2005). Patients will complain of a burning pain and paraesthesia in the plantar aspect of the foot. The definitive test to confirm this diagnosis is the nerve conduction test (Reid, 1992).

2.8.5. Tibiofibular synostosis
Secondary to an ankle fracture or sprain there may be a bony fusion between two adjacent bones, namely the tibia and fibula (Yochum and Rowe, 1996). Chronic pain and swelling is present after activity. A radiograph revealing a bony mass between the tibia and fibula will confirm this diagnosis (Reid, 1992).
2.8.6. **Peroneal/ fibularis tendon subluxation**

The patient will present with pain, swelling, or a sensation of ‘snapping’ around the lateral malleolus. There will also be tenderness to palpation along the peroneal sheath posterior to the lateral malleolus (Reid, 1992).

2.8.7. **Achilles tendon rupture**

Due to the traction on the Achilles tendon during an ankle sprain, the tendon may rupture, sounding like a gunshot and causing pain in the posterior leg (Trojan and McKeag, 1998). A depression just above the calcaneus can often be seen and palpated (Marano, 2006). Thomas test should be considered.

2.8.8. **Peroneal and Tibial nerve injury**

This is a very rare complication, but occasionally an ankle sprain is associated with footdrop. This could occur secondary to traction of the peroneal nerve during the forced inversion, or as a result of compression of the nerve between the fibularis longus and the fibula (Reid, 1992).

2.8.9. **Synovial impingement**

Pain will be felt over the anterior and anterolateral aspect of the ankle. This is due to synovial thickening secondary to trauma (Reid, 1992). Synovial thickening can be palpated along the margins of the joint line as a ‘soft spongy’ texture (Gotlieb, 2005). Synovial thickening may be accompanied by the presence of additional fluid, identified by fluctuant swelling (Gotlieb, 2005).

2.9. **Conclusion:**

The recommended treatment protocol for ankle sprains has been discussed. In the acute phase it places emphasis on minimising swelling and bruising and encourages adequate ankle protection. After the acute phase has passed, rehabilitation is focused on. This includes improving the ankle range of motion and proprioception. Attention is also given to strengthen the muscles around the ankle joint. According to Travel, *et al.* (1999 1: 19) myofascial trigger points
(whether active or latent) can cause significant motor dysfunction. Trevino, et al. (1994) stated that fibularis muscle weakness is thought to be a source of symptoms after an inversion sprain.

In the recommended treatment protocol however, no mention is made of evaluating the musculature around the ankle joint for myofascial trigger points and or treating these points. McGrew and Schenck (2003) noted that if the musculature and neural structures surrounding the ankle joint were affected during an ankle sprain injury, and were left unresolved, they would lead to chronic instability.

It is hypothesised that due to the mechanism of injury of an inversion ankle sprain, myofascial trigger points develop in the fibularis muscles. To investigate this hypothesis, a prevalence study was done to determine the presence of fibularis muscle trigger points in ankle sprain patients (Phase 1). It is important to know the prevalence of these trigger points as their presence or absence will affect the recommended treatment protocol for inversion ankle sprains.

Inversion ankle sprain patients typically complain of pain in the area of the lateral malleolus. Travell and Simons (1993, 2: 371) documented the pain referral pattern of the fibularis muscle to be over the lateral ankle malleolus, above, behind and below it.

It is hypothesised that lateral ankle pain following an inversion ankle sprain injury may be due to referred pain from the fibularis muscle trigger points. This hypothesis was further investigated by selecting inversion ankle sprain patients with fibularis muscle trigger points and randomly dividing them into two groups; one received dry needling of the fibularis muscle trigger points, the other received a placebo treatment (detuned ultrasound) applied to the fibularis muscle trigger points (phase 2).
The purpose of the treatment was not to determine the therapeutic effects of dry needling (as this is already known) but rather to affect the trigger points and then monitor for any change in the clinical presentation of the ankle sprain. This evaluated the role of myofascial trigger points of the fibularis muscle on the clinical presentation of inversion ankle sprains. Phase 2 of this study would give a clearer picture as to whether the lateral ankle pain experienced by ankle sprain patients was referred pain from the fibularis muscles or whether it was true ankle joint pain.
CHAPTER THREE

Methodology

3.1. Introduction:
This chapter includes a detailed description on the design of this study, the sampling procedure, the interventions that were applied and the data collected from the study. The statistical analysis of data collected will also be discussed.

3.2. Design:
This study was an analytical, cross sectional study (phase 1) and randomised controlled trial (phase 2) that was conducted in order to determine the prevalence (phase 1) and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains (phase 2).

3.3. Advertising:
Advertisements (Appendix 1) informing the public about this study were placed in local newspapers, around Durban University of Technology campus, in pharmacies and emergency rooms, at local sporting clubs and sporting events. Word of mouth was also used to inform the general public. Upon reply to the advertisements, the prospective participants underwent a cursory telephonic discussion (Appendix 2) with the researcher to exclude subjects that did not fit the criteria for the study.

3.3.1. Sampling Method:
Consecutive convenience sampling was used for this study.

3.3.2. Sampling Allocation:
Participants who successfully complied with the inclusion criteria were selected for phase 1. For phase 2, the first 40 participants with fibularis myofascial trigger
points were selected from phase 1. For this phase the participants were randomly divided into two equal groups. This was done by placing 20 A’s and 20 B’s in an envelope, the participants were asked to remove a piece of paper from the envelope and without looking at it, hand it to the researcher. The paper removed from the envelope determined which group the participant was allocated to. Group A formed the treatment group (dry needling), and group B formed the placebo group (detuned ultrasound). This study was therefore a single blinded study.

3.3.3. Sample Size:
44 candidates with a history of subacute / chronic unilateral Grade I inversion ankle sprains were assessed in phase 1. Phase 1 continued until 40 participants complied with the inclusion criteria for phase 2 of this study. This population size is consistent to previous research done by Kohne (2005) and Gaines (2005).

3.4. Patient Screening:
The participant evaluation and selection process began with participants undergoing a cursory telephonic discussion with the researcher, to exclude participants that did not fit the criteria for the study (appendix 2). Participants who successfully complied with this interview were evaluated at an initial consultation. This involved a case history, physical and regional ankle/foot examination. Participants diagnosed with grade 1 inversion ankle sprains received a letter of information (appendix 3) and were asked to sign an informed consent form (appendix 4) explaining the study and allowing them to withdraw from this study at any time.

3.4.1. Inclusion Criteria:
This study was divided into two phases and separate criteria applied to them. Phase 1 investigated the prevalence of fibularis muscle trigger points in the injured and uninjured leg.
The criteria for phase 1:
• Participants had to have a history of a subacute / chronic unilateral inversion ankle sprain with persistent lateral ankle pain.

• Only participants diagnosed with grade 1 inversion ankle sprains were accepted into this study. Grade 1 ankle sprains were described by Reid (1992:226) in the following manner:

<table>
<thead>
<tr>
<th>SEVERITY:</th>
<th>PATHOLOGY:</th>
<th>DISABILITY:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 1- Mild (stable)</td>
<td>Mild stretch, no instability, single ligament involved.</td>
<td>No or little limp, minimal functional loss, difficulty hopping.</td>
</tr>
</tbody>
</table>

• Participants had to be between the ages of 15 and 50 as recommended by Pellow and Brantingham (2001). This limited age group facilitated increased population group homogeneity.

Phase 2 investigated the clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains.

The criteria for phase 2 included:

• Presence of fibularis myofascial trigger points in the injured leg, as assessed in phase 1.

• A history of a chronic inversion ankle sprain not exceeding 3 months. This time limit was set so as to increase the sample homogeneity.

• Numerical pain rating of 6 or above to increase sample homogeneity.

3.4.2. Exclusion Criteria:

The exclusion criteria listed below applied for both phase 1 and 2:

• Participants who had received any trigger point therapy for their ankle sprain were excluded from this study. Participants were instructed not to initiate any form of treatment while taking part in the study (Pellow and Brantingham, 2001).

• Participants diagnosed with a grade II and III ankle sprain.
- Participants with a history of bilateral inversion ankle sprains. In this study the uninjured leg acted as the control group.
- A history of foot or ankle fracture, dislocation or surgery.
- Participants with any systemic arthritide that affected the ankle.
- Participants with a neurological deficit of the lower limb.
- Participants who presented with any contra indications to dry needling including skin infections over the leg, allergy to specific metals, blood dyscrasias or local malignancies (Liggins, 2003).

Participants who did not meet phase 1 inclusion criteria or had any of the exclusion criteria were referred to other interns in the Chiropractic Day Clinic for treatment of their presenting condition.

3.5. Intervention Method:

3.5.1. Phase 1:
All participants were examined to determine the presence of fibularis myofascial trigger points in the injured and the uninjured leg prior to an intervention. The trigger points in the injured leg were marked with permanent marker and then later covered with a plaster, so as to ensure that measurements were specific and consistent during this study. At this point subjective and objective measurements (as described below) were taken. The data collected from the injured leg was compared to the uninjured leg to determine the prevalence of myofascial trigger points of the fibularis muscles. This method of comparing results from the injured ankle with the uninjured ankle has previously been used in studies done by Santilli, et al. (2005) and Konradsen, et al. (1998).

Participants in phase 1 with no fibularis myofascial trigger points received one free treatment for their ankle sprain at the Chiropractic Clinic, and were then excluded from the rest of the study.
3.5.2. Phase 2:
Phase 1 participants who fulfilled the following criteria were included into phase 2 of this study:
- A history of a chronic inversion ankle sprain not exceeding 3 months.
- Numerical pain rating of 6 or above.
- Presence of fibularis myofascial trigger points in the injured leg.

Phase 2 participants were then randomly assigned to either Group A or B. The participants in group A received dry needling of the identified fibularis myofascial trigger points. A single needle insertion technique was used whereby the needle was inserted directly into the myofascial trigger point, and manually stimulated using the thumb and forefinger. After five minutes the needle was removed. Group B received detuned ultrasound treatment (placebo) of the identified fibularis trigger points. This was also applied for five minutes. Group A and B received another treatment two days after initial treatment. Mance, et al. (1986) stated that in the acute phase, one injection may relieve pain, but a series of injections should be administered every second, third or fourth day according to the complaints of the patient, for complete resolution.

The third consultation (3 days after second treatment) consisted of only measurements (subjective and objective) as described in measuring instruments below. Travel, et al. (1999 1:165) stated that post-needling soreness lasts at most 3 or 4 days.

After the third consultation, participants in group B (placebo group) were then offered two free treatments for their ankle sprain.

3.6. Intervention frequency:
Phase 1 did not involve an intervention.
40 participants in phase 2 received two treatments and one follow up.
Treatments were no shorter than 2 days apart, and the follow up was conducted
3 days after second treatment. The purpose of the treatment was not to
determine the therapeutic effects of dry needling (as this is already known) but
rather to affect the trigger points and then monitor for any change in the clinical
presentation of the ankle sprain. Therefore only two treatments were given.

3.7. **Data collection instruments:**
The following instruments were used for measurement in this study:

3.7.1. **Subjective measurements:**
- The numerical pain rating scale (appendix 9) was used to measure the
  intensity of pain (lateral ankle pain). This scale has been shown to be
  simple, effective and the recommended choice in a study comparing six

3.7.2. **Objective measurements**
- Presence and location of fibularis trigger points (appendix 12) in the
  injured and uninjured leg were assessed by palpation according to the
  criteria stated by Travell, *et al*. (1999 1:35) which are:
  1. Palpable taut band
  2. Focal tenderness
  3. Referred pain in the zone of reference
  4. Painful limit to full stretch range of motion
- Myofascial diagnostic scale (Chettiar, 2001) was used to objectively
determine the extent to which a participant suffered from myofascial pain
(appendix 9).
- Algometer (appendix 10) was used to determine the tenderness of
myofascial trigger points. This instrument measured the level of pain that
a person could withstand. The pain threshold was determined by the
amount of force per square centimetre required for a person to perceive
pain (Fischer, 1987). The algometer was used in the following manner:
1. It was explained to the patient that a procedure of increasing
pressure over the area was going to be applied and the participant
was instructed to inform the examiner when pain was experienced.
2. The researcher located the area of maximal tenderness over the
fibularis muscle by palpation.
3. The algometer was set to zero and placed at a 90-degree vertical
angle to the skin. Pressure was slowly and continuously applied
until the participant indicated pain.
4. The algometer reading was taken and recorded. The higher the
reading, the less the tenderness of the tissue (Fischer, 1987). A
higher reading therefore shows an improvement (Fischer, 1987).

- Goniometer (appendix 11) was used to measure the ankle range of
motion (focussing on inversion, eversion, dorsiflexion and plantarflexion)
in the following manner (as recommended by goniometric examination of
ankle and foot range of motion, n.d.):

**Dorsiflexion:**
Patient position: seated with the ankle at 90 degrees (neutral).
Axis of goniometer: inferior to lateral malleolus.
Stabilising arm: long axis of the fibula.
Moveable arm: lateral border of the foot.
Movement: from neutral (90 degrees) the ankle was actively dorsiflexed to
limit of motion.
Range of motion: 10-30 degrees.

**Plantarflexion:**
Patient position: supine, knee flexed and gastrocnemius muscle
relaxed.
Axis of Goniometer: inferior to lateral malleolus.
Stabilising arm: long axis of the fibula.
Moveable arm: lateral border of the foot.
Movement: from neutral (90 degrees) the ankle was actively plantarflexed to limit of motion.
Normal range of motion: 45-65 degrees.

**Inversion:**
Patient position: prone, feet off the edge of the table with the superior and inferior aspects of the posterior calcaneus marked by pen.
Axis of Goniometer: midpoint of the superior aspect of calcaneus.
Stabilising arm: long axis of the leg.
Moveable arm: Long axis of the midline of the calcaneus.
Movement: calcaneus actively inverted to limit of motion.
Normal range of motion: 30-50 degrees.

**Eversion:**
Patient position: prone, feet off the edge of the table with the superior and inferior aspects of the posterior calcaneus marked by pen.
Axis of Goniometer: midpoint of the superior aspect of calcaneus.
Stabilising arm: long axis of the leg.
Moveable arm: Long axis of the midline of the calcaneus.
Movement: calcaneus actively everted to limit of motion.
Normal range of motion: 15-30 degrees.

- A Functional evaluation scale developed by Kaikkonen, *et al.* (1994) was used to evaluate the functional disability that resulted from the ankle injury. This scoring scale has been shown to demonstrate excellent reproducibility and can significantly differentiate between subjective, objective and functional evaluation of ankle injuries (appendix 13).
### 3.8. Data collection frequency:

Data was collected on all three visits, prior to the treatment being administered. The table below indicates which measuring instruments were used at each visit:

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<thead>
<tr>
<th>VISIT 1</th>
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<tbody>
<tr>
<td>INJURED LEG</td>
<td>UNINJURED LEG</td>
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<tr>
<td>Numerical pain rating scale</td>
<td>Presence and location of MTrp’s</td>
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<tr>
<td>Functional evaluation scale</td>
<td>Myofascial Diagnostic scale</td>
</tr>
<tr>
<td>Presence and location of MTrp’s</td>
<td>Algometer reading</td>
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<tr>
<td>Myofascial diagnostic scale</td>
<td>Goniometer reading</td>
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<td>Algometer reading</td>
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</tbody>
</table>
All measurements were taken and recorded by the researcher and all questionnaires were completed under the supervision of the researcher.

3.9. Data analysis:
Data were entered into a MS Excel spreadsheet and imported into SPSS version 13 (SPSS Inc., Chicago, Illinois, USA) for analysis. A p value of <0.05 was considered as statistically significant.

Phase I:
The control group for the cross-sectional phase of the study was the uninjured ankle of each participant. The groups were compared with regard to the various quantitative outcomes using paired t-tests, and comparisons with categorical outcomes were done using McNemar’s chi square tests. Associations between presence, number of trigger points and clinical outcomes were done by means of one-way ANOVA in the injured ankles. Correlations between baseline subjective and objective outcome measurements were done for the injured ankles using Pearson’s correlation coefficients.

Phase II:
Participants were randomized into two equal groups, treated and followed up over three visits. Repeated measures ANOVA was used to compare treatment groups over time, with profile plots of means by group over time. A significant time by group (time*group) interaction indicated a significant treatment effect. The direction of the treatment effect was assessed from the profile plots. This was done separately for each outcome measurement.
CHAPTER FOUR

Statistics

4.1. Statistical methodology
Data was entered into a MS Excel spreadsheet and imported into SPSS version 13 (SPSS Inc., Chicago, Illinois, USA) for analysis. A $p$ value of <0.05 was considered as statistically significant.

**Phase I:**
The control group for the cross-sectional phase of the study was the uninjured ankle of each participant. The groups were compared with regard to the various quantitative outcomes using paired t-tests, and comparisons with categorical outcomes were done using McNemar’s chi square tests. Associations between presence, number of trigger points and clinical outcomes were done by means of one-way ANOVA in the injured ankles. Correlations between baseline subjective and objective outcome measurements were done for the injured ankles using Pearson’s correlation coefficients.

**Phase II:**
Participants were randomized into two equal groups, treated and followed up over three visits. Repeated measures ANOVA was used to compare treatment groups over time, with profile plots of means by group over time. A significant time by group (time*group) interaction indicated a significant treatment effect. The direction of the treatment effect was assessed from the profile plots. This was done separately for each outcome measurement.

4.2. RESULTS:

4.2.1. DEMOGRAPHICS
Of the forty-five participants examined, forty-four participants were enrolled into phase 1 of the study. Four participants did not complete phase 2 of this study.
The sample’s (n=44) ages ranged from 18 to 50, with a mean age of 28.5 years (SD 7.8 years). The gender distribution of the sample is shown in Table 1. The majority of the sample (59.1%) was males. This is consistent with the gender distribution of 70% males and 30% females seen by Kohne (2005).

Table 1: Gender distribution of Phase 1 sample (n=44)

<table>
<thead>
<tr>
<th>Gender</th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>26</td>
<td>59.1</td>
</tr>
<tr>
<td>female</td>
<td>18</td>
<td>40.9</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>100.0</td>
</tr>
</tbody>
</table>

The racial distribution of the sample is shown in the pie chart in Figure 1. The majority of the sample (59.1%) was White. Blacks constituted 22.7% and Indian or Coloured participants were 18.2%.

Figure 1: Pie chart showing percentage racial distribution of phase 1 sample (n=44)
The demographics correlate with that of Kohne (2005), who also had a majority Caucasian sample, but are not congruent with current demographic profiles as defined by Statistics South Africa (2006).

In 52.3% of participants the injured ankle was on their dominant side. This is shown in Table 2. This correlates with an epidemiological survey on ankle sprains done by Yeung, et al. (1994). 51.9% of their participants reported unilateral ankle sprains, of which 36% reported the injured ankle to be on the dominant side, while only 15.3% of the participant’s injury involved the non-dominant ankle. Therefore injury to the dominant ankle was 2.4 times higher than injury to the non-dominant ankle (Yeung, et al. 1994).

**Table 2: Distribution of injury side in phase 1 participants (n=44)**

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>dominant</td>
<td>23</td>
<td>52.3</td>
</tr>
<tr>
<td>non dominant</td>
<td>21</td>
<td>47.7</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>100.0</td>
</tr>
</tbody>
</table>

The time since injury in the 44 participants is shown in Table 3. There were similar numbers in each of the groups. 34.1% of participants reported an ankle sprain of no longer than a month ago, 34.1% between one and two months and 31.8% between two and three months.

**Table 3: Distribution of time since injury in phase 1 participants (n=44)**

<table>
<thead>
<tr>
<th></th>
<th>Frequency</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1 month</td>
<td>15</td>
<td>34.1</td>
</tr>
<tr>
<td>1-&lt;2 months</td>
<td>15</td>
<td>34.1</td>
</tr>
<tr>
<td>&gt;= 2 months</td>
<td>14</td>
<td>31.8</td>
</tr>
<tr>
<td>Total</td>
<td>44</td>
<td>100.0</td>
</tr>
</tbody>
</table>
4.2.2. PREVALENCE OF THE FIBULARIS MUSCLE TRIGGER POINTS

The first objective of this study was to determine the prevalence of fibularis muscle trigger points in ankle sprain patients.

Table 4 shows that there was a significantly higher prevalence of fibularis longus trigger points in injured (95.5%) than in uninjured ankles (79.5%) (p=0.039).

Table 4: Cross tabulation of presence of fibularis longus trigger points in injured and uninjured ankles

<table>
<thead>
<tr>
<th>Visit 1 fibularis longus</th>
<th>Visit 1 fibularis longus</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>uninjured</td>
<td>absent</td>
<td>present</td>
</tr>
<tr>
<td>Absent</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Present</td>
<td>8</td>
<td>34</td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
<td>35</td>
</tr>
</tbody>
</table>

McNemar’s chi square *p* value 0.039

Table 5 shows that there was a significantly higher prevalence of fibularis brevis trigger points in injured (81.8%) than in uninjured ankles (59.1%) (p=0.031).

Table 5: Cross tabulation of presence of fibularis brevis trigger points in injured and uninjured ankles

<table>
<thead>
<tr>
<th>Visit 1 fibularis brevis</th>
<th>Visit 1 fibularis brevis</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>uninjured</td>
<td>absent</td>
<td>present</td>
</tr>
<tr>
<td>Absent</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>Present</td>
<td>14</td>
<td>22</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td>26</td>
</tr>
</tbody>
</table>

McNemar’s chi square *p* value 0.031
Table 6 shows that there was a non significantly, slightly higher prevalence of fibularis tertius trigger points in injured (66.7%) than in uninjured ankles (52.4%) (p=0.146).

Table 6: Cross tabulation of presence of fibularis tertius trigger points in injured and uninjured ankles

<table>
<thead>
<tr>
<th>Visit 1 fibularis tertius</th>
<th>Absent</th>
<th>present</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>uninjured</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent</td>
<td>11</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>present</td>
<td>9</td>
<td>19</td>
<td>28</td>
</tr>
<tr>
<td>Total</td>
<td>20</td>
<td>22</td>
<td>42</td>
</tr>
</tbody>
</table>

McNemar’s chi square p value 0.146

Tables 4, 5 and 6 above indicate that there was a significantly higher prevalence of fibularis longus and brevis trigger points in injured compared with uninjured ankles, and a non-significantly higher prevalence of fibularis tertius in injured than uninjured ankles. The most prevalent trigger point in injured ankles was fibularis longus (95.5%) and the least was fibularis tertius (66.7%). In uninjured legs the most prevalent trigger point was also fibularis longus (79.5%) and the least also fibularis tertius (52.4%). This study therefore supports the hypothesis that fibularis muscle trigger points are more prevalent in the injured leg when compared to the uninjured leg.

The reason for the high prevalence of fibularis longus trigger points (79.5%) in the injured leg is unknown. It may possibly be related to an antalgic gait (to protect the injured ankle) causing the uninjured leg’s fibularis muscles to become overloaded. Also, previous injuries to the uninjured leg may not have been reported. A study investigating the prevalence of fibularis muscle trigger points in participants who have never injured an ankle is needed to provide baseline statistics.
The reason for the non-significant higher prevalence of fibularis tertius trigger points in injured (66.7%) than in uninjured ankles (52.4%) could be explained by the function of the muscle. Fibularis tertius dorsiflexes the foot and aids in ankle eversion (Moore and Agur, 1995). With an inversion sprain (which involves plantarflexion and inversion), this muscle may be injured to a lesser degree when compared to the fibularis longus and brevis, which causes ankle eversion and assists with plantarflexion of the foot.

4.2.3. COMPARISON OF THE BASELINE OUTCOME MEASUREMENTS BETWEEN THE INJURED AND THE UNINJURED ANKLES

The second objective of this study was to evaluate the role of myofascial trigger points of the fibularis muscle on the clinical presentation of inversion ankle sprains.

The myofascial diagnostic scale (Chettiar, 2001) was used to objectively determine the extent to which a participant suffered from myofascial pain of the fibularis muscles. The MDS score at baseline showed a highly significant difference between the injured and the uninjured ankles (p<0.001). The injured ankle showed a higher mean score than the uninjured (Table 7). This indicates that the participants objectively suffered from an increased amount of myofascial pain in the injured leg when compared to the uninjured leg.

Dorsiflexion was higher in the uninjured ankle than the injured ankle (p=0.006), as was plantarflexion (p=0.022). This could possibly be due to the effect of residual ankle sprain symptoms, which include ankle instability, pain, stiffness and swelling of the ankle (Yeung, et al. 1994). All other outcome measurements namely inversion and eversion goniometer readings and all the algometer readings showed no difference between the two ankles at baseline.
Table 7: Paired t-tests comparison of mean injured with uninjured ankles

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>t value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visit 1 MDS injured</td>
<td>8.60</td>
<td>40</td>
<td>2.845</td>
<td>.450</td>
<td>5.020</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Visit 1 MDS uninjured</td>
<td>5.55</td>
<td>40</td>
<td>3.250</td>
<td>.514</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 algometer fibularis longus injured</td>
<td>4.041</td>
<td>32</td>
<td>1.8141</td>
<td>.3207</td>
<td>-1.286</td>
<td>0.208</td>
</tr>
<tr>
<td>Visit 1 algometer fibularis longus uninjured</td>
<td>4.316</td>
<td>32</td>
<td>2.0193</td>
<td>.3570</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 algometer fibularis brevis injured</td>
<td>4.633</td>
<td>21</td>
<td>1.8629</td>
<td>.4065</td>
<td>-0.320</td>
<td>0.752</td>
</tr>
<tr>
<td>Visit 1 algometer fibularis brevis uninjured</td>
<td>4.743</td>
<td>21</td>
<td>1.8763</td>
<td>.4094</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 algometer fibularis tertius injured</td>
<td>3.748</td>
<td>21</td>
<td>1.5731</td>
<td>.3433</td>
<td>-0.029</td>
<td>0.769</td>
</tr>
<tr>
<td>Visit 1 algometer fibularis tertius uninjured</td>
<td>3.829</td>
<td>21</td>
<td>1.8813</td>
<td>.4105</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 dorsiflexion injured</td>
<td>9.88</td>
<td>40</td>
<td>6.211</td>
<td>.982</td>
<td>-2.885</td>
<td>0.006</td>
</tr>
<tr>
<td>Visit 1 dorsiflexion uninjured</td>
<td>12.00</td>
<td>40</td>
<td>6.139</td>
<td>.971</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 plantarflexion</td>
<td>62.20</td>
<td>40</td>
<td>11.678</td>
<td>1.846</td>
<td>-2.387</td>
<td>0.022</td>
</tr>
</tbody>
</table>
4.2.4. COMPARISON OF THE BASELINE OUTCOME MEASUREMENTS BETWEEN PAIN AND SEVERITY OF TRIGGER POINTS IN INJURED ANKLES

Expanding on the second objective of this study, baseline outcome measurements in the injured ankles were assessed for correlation between pain (measured both subjectively and objectively) and severity of the trigger points (measured objectively).

Pain measured by the NRS was not correlated with any of the severity measurements at baseline. This indicated that the lateral ankle pain reported by the participants had no correlation to the severity of the fibularis muscle trigger points. Pain measured by the algometer in the fibularis tertius muscle was significantly correlated with dorsiflexion \((r=0.547, p=0.003)\). This meant that as algometer measurements increased (i.e. a decrease in the tenderness of myofascial trigger points), dorsiflexion measurements increased. Thus pain in the fibularis tertius muscle was negatively correlated with dorsiflexion.
This indicated that if the myofascial trigger points in the fibularis tertius muscles could withstand an increased amount of pressure (as exerted by the algometer), the trigger points were less severe, and would therefore allow for a greater dorsiflexion range of motion. It is known that fibularis tertius function is to dorsiflex and evert the ankle (Moore and Agur, 1995:254), and so this result substantiates the literature.

No other significant correlations could be found as shown in Table 8.

Table 8: Pearson's correlation between baseline pain measurements and baseline severity measurements in injured ankles.

<table>
<thead>
<tr>
<th></th>
<th>Visit 1 NRS</th>
<th>Visit 1 algometer peronius longus injured</th>
<th>Visit 1 algometer peronius brevis injured</th>
<th>Visit 1 algometer peronius tertius injured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visit 1 MDS injured</td>
<td>Pearson Correlation</td>
<td>-0.235</td>
<td>-0.130</td>
<td>0.035</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>0.144</td>
<td>0.431</td>
<td>0.849</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>40</td>
<td>39</td>
<td>32</td>
</tr>
<tr>
<td>Visit 1 dorsiflexion injured</td>
<td>Pearson Correlation</td>
<td>0.096</td>
<td>0.302</td>
<td>0.293</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>0.554</td>
<td>0.062</td>
<td>0.104</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>40</td>
<td>39</td>
<td>32</td>
</tr>
<tr>
<td>Visit 1 plantarflexion injured</td>
<td>Pearson Correlation</td>
<td>0.015</td>
<td>-0.133</td>
<td>-0.164</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed)</td>
<td>0.928</td>
<td>0.419</td>
<td>0.369</td>
</tr>
<tr>
<td></td>
<td>N</td>
<td>40</td>
<td>39</td>
<td>32</td>
</tr>
<tr>
<td>Visit 1 inversion</td>
<td>Pearson Correlation</td>
<td>-0.022</td>
<td>-0.258</td>
<td>-0.335</td>
</tr>
</tbody>
</table>
### Table 8

|                             | Pearson Correlation | Sig. (2-tailed) | N  |  |  |  |
|------------------------------|---------------------|-----------------|----|  |  |  |
| Ankle functional evaluation  |                     |                 |    |  |  |  |
| N                           | 0.162               | 0.317           | 40 | 39 | 32 | 27 |
| evaluation scale 1           |                     |                 |    |  |  |  |
| N                           | 0.103               | 0.534           | 40 | 39 | 32 | 27 |
| N                           | 0.137               | 0.486           | 40 | 39 | 32 | 27 |
| Visit eversion injured       |                     |                 |    |  |  |  |
| N                           | 0.061               | 0.966           | 40 | 39 | 32 | 27 |
| N                           | 0.014               | 0.844           | 40 | 39 | 32 | 27 |
| Visit 1 injured              |                     |                 |    |  |  |  |
| N                           | 0.890               | 0.399           | 40 | 39 | 32 | 27 |
| N                           | 0.113               | 0.486           | 40 | 39 | 32 | 27 |
| N                           | 0.613               | 0.574           | 40 | 39 | 32 | 27 |

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

#### 4.2.5. EVALUATION OF THE TREATMENT EFFECT

The third objective of this study was to determine whether dry needling of the fibularis muscle should be considered in the treatment protocol for inversion ankle sprains. This was done by comparing outcome measurements in the injured ankles over three time points between the group who received dry needling and the group who received the placebo treatment (phase 2).

#### 4.2.5.1. Numerical Pain Rating Scale (NRS)

Table 8 shows that there was a highly significant time effect (p<0.001), meaning that both groups showed significant time changes. However, the time by group interaction effect (intervention effect) was not significant, implying that both groups changed over time at the same rate. Thus, the intervention did not significantly lower pain compared with the placebo. This is shown graphically in Figure 2, where the rate of decrease in NRS was very similar in both groups. However, the dry needling group showed a slightly faster rate of decrease over time, which was not significant, compared with the placebo group.

This suggests that over this research studies time frame dry needling of the fibularis muscles proved to be no more therapeutic than the placebo treatment in
terms of subjective pain, in the treatment of lateral ankle pain following an inversion ankle sprain.

**Table 9: Within and between subjects (inter- and intragroup) effects for NRS**

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.208</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.958</td>
<td>0.450</td>
</tr>
<tr>
<td>Group</td>
<td>F=1.180</td>
<td>0.284</td>
</tr>
</tbody>
</table>

**Figure 2: Profile plot of mean NRS score over time by group**
4.2.5.2. Myofascial Diagnostic Scale (MDS)

Table 9 shows that a statistically significant time by group interaction (p=0.030) was found for MDS, meaning that the change over time was dependant on which treatment group the participants were in. Figure 3 shows that the MDS score for the dry needling group decreased at a much faster rate over time than the placebo group. Thus, for this outcome there was a statistically significant treatment effect.

This indicates that dry needling of the fibularis muscles is an effective treatment method in the relieving of objective fibularis myofascial pain.

It is important to note that the NRS was used to specifically determine a change in the severity of subjective pain over the lateral ankle, whereas the MDS was used to determine a change in the severity of the fibularis myofascial trigger points. Therefore the decrease in the MDS score with dry needling and the lack of therapeutic effect noted by the NRS indicated that the severity of the lateral ankle pain and the severity of the myofascial trigger points were not related.

Table 10: Within and between subjects (inter- and intragroup) effects for MDS

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.744</td>
<td>0.004</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.828</td>
<td>0.030</td>
</tr>
<tr>
<td>Group</td>
<td>F=2.233</td>
<td>0.143</td>
</tr>
</tbody>
</table>
Figure 3: Profile plot of mean MDS score over time by group

4.2.5.3. Algometer fibularis longus muscle
Table 10 shows that there were no significant effects for algometer readings of the fibularis longus muscle. The treatment effect was non significant (p=0.706). Figure 4 shows that the profiles of the two groups over time were parallel. Both groups showed a decrease in algometer measurements between the first and second visits followed by an increase up to the third visit. Therefore subjectively in terms of the algometer readings, there was no treatment effect.
Table 11: Within and between subjects (inter- and intragroup) effects for algometer fibularis longus muscle

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.946</td>
<td>0.371</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.981</td>
<td>0.706</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.378</td>
<td>0.542</td>
</tr>
</tbody>
</table>

Figure 4: Profile plot of mean algometer measurement of the fibularis longus muscle over time by group
4.2.5.4. Algometer Fibularis brevis muscle
For this outcome there was no evidence of a treatment effect (p=0.482), although the profile plot in Figure 5 shows a slight trend towards a faster rate of increase in the dry needling group than the placebo group.

Table 12: Within and between subjects (inter- and intragroup) effects for algometer fibularis brevis muscle

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.929</td>
<td>0.345</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.951</td>
<td>0.482</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.622</td>
<td>0.436</td>
</tr>
</tbody>
</table>

Figure 5: Profile plot of mean algometer measurement of the fibularis brevis muscle over time by group

59
4.2.5.5. Algometer fibularis tertius muscle
Table 12 shows no significant treatment effect for the fibularis tertius muscle (p=0.799), although, as with the algometer readings of the fibularis brevis muscle, the profile plot shows a clear trend towards a faster rate of increase in algometer measurements in the dry needling group than in the placebo group.

Table 13: Within and between subjects (inter- and intragroup) effects for algometer fibularis tertius muscle

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.956</td>
<td>0.584</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.982</td>
<td>0.799</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.785</td>
<td>0.384</td>
</tr>
</tbody>
</table>

Figure 6: Profile plot of mean algometer measurement of the fibularis tertius muscle over time by group
4.2.5.6. Goniometer readings for dorsiflexion
Dorsiflexion changed significantly over time (p=0.003) but the rate of change was the same in both groups (p=0.803). Thus no treatment effect could be demonstrated for dorsiflexion. Figure 7 shows that between visit 2 and 3 the measurement for the placebo group decreased while that for the dry needling group continued to increase, which suggests a trend towards a more beneficial effect in the treated group, but this was not significant.

Table 14: Within and between subjects (inter- and intragroup) effects for dorsiflexion

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.736</td>
<td>0.003</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.988</td>
<td>0.803</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.076</td>
<td>0.784</td>
</tr>
</tbody>
</table>

Figure 7: Profile plot of mean dorsiflexion over time by group
4.2.5.7. Goniometer readings for plantarflexion
There was also no statistical evidence of a treatment effect for plantarflexion (p=0.400). However, Figure 8 shows that the two groups behaved very differently over time, with an overall decrease in values in the dry needling group and increase in the placebo group.

Table 15: Within and between subjects (inter- and intragroup) effects for plantarflexion

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.997</td>
<td>0.945</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.952</td>
<td>0.400</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.362</td>
<td>0.551</td>
</tr>
</tbody>
</table>

Figure 8: Profile plot of mean plantarflexion over time by group
4.2.5.8. Goniometer readings for inversion
There was a non-significant treatment effect (p=0.168) for inversion (Table 15). Figure 9 shows that the dry needling group decreased in mean values for inversion over time while the placebo group showed a corresponding increase. The profiles crossed over between visit 2 and 3, but this interaction was not quite significant.

Table 16: Within and between subjects (inter- and intragroup) effects for inversion

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.998</td>
<td>0.956</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.908</td>
<td>0.168</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.576</td>
<td>0.453</td>
</tr>
</tbody>
</table>

Figure 9: Profile plot of mean inversion over time by group
4.2.5.9. Goniometer readings for eversion
For eversion there was no evidence of a treatment effect (p=0.336). Figure 10 shows that overall the dry needling group experienced an increase in mean eversion while the placebo group did not change much from their baseline values.

Table 17: Within and between subjects (inter- and intragroup) effects for eversion

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.990</td>
<td>0.835</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.947</td>
<td>0.336</td>
</tr>
<tr>
<td>Group</td>
<td>F=0.003</td>
<td>0.954</td>
</tr>
</tbody>
</table>

Figure 10: Profile plot of mean eversion over time by group
4.2.5.10. Ankle Functional Evaluation Scale (AFES)

The AFES score showed an almost significant time effect ($p=0.058$), but no treatment effect ($p=0.910$). Figure 11 shows that both groups increased in AFES score over time but the rate of increase was very similar in both groups.

Table 18: Within and between subjects (inter- and intragroup) effects for AFES

<table>
<thead>
<tr>
<th>Effect</th>
<th>statistic</th>
<th>$p$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.857</td>
<td>0.058</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.995</td>
<td>0.910</td>
</tr>
<tr>
<td>Group</td>
<td>$F=0.001$</td>
<td>0.973</td>
</tr>
</tbody>
</table>

![Figure 11: Profile plot of mean AFES score over time by group](image)
4.3. SUMMARY
This study has demonstrated significantly more fibularis longus and brevis trigger points in injured ankles than in paired uninjured ankles at baseline. The MDS score at baseline was significantly higher in the injured than in the uninjured ankles (p<0.001). Dorsiflexion was significantly higher in the uninjured ankle than the injured ankle (p=0.006), as was plantarflexion (p=0.022).

In the injured ankle, subjective pain measured by the NRS was not correlated with any of the severity measurements (Myofascial Diagnostic Scale, Goniometer readings and Ankle Functional Evaluation Scale) at baseline. This indicates that lateral ankle pain experienced by ankle sprain patients had no correlation to the severity of the myofascial trigger points in the fibularis muscles. Objective Pain measured by the algometer in the fibularis tertius muscle was significantly correlated with dorsiflexion (r= 0.547, p=0.003). This meant that as algometer measurements increased (i.e. pain decreased), dorsiflexion measurements increased. Thus pain in the fibularis tertius muscle was negatively correlated with dorsiflexion.

A statistically significant beneficial treatment effect of dry needling over placebo treatment was only demonstrated for the MDS score (p=0.030). Most other outcomes showed that both treatment groups improved to the same extent over time while some outcomes suggested that the dry needling technique was beneficial over the placebo, but failed to show a statistically significant effect. This could have been a type 2 error, where a clinical difference is observed, but due to an underpowered study (low sample size) this effect was not statistically significant. Further studies should be done to confirm these findings with a larger sample size and a longer treatment time.
CHAPTER FIVE

5.1. Conclusion
The results of this study showed a statistically significant prevalence of fibularis longus and brevis myofascial trigger points in the injured leg compared to the uninjured leg. Fibularis tertius trigger points were found to be more prevalent in the injured leg, but in a statistically non-significant manner. MDS indicated that participants suffered from more myofascial pain on the injured side compared to the uninjured side. Also dorsiflexion and plantarflexion was greater on the uninjured side. Therefore my first hypothesis, which stated that fibularis myofascial trigger points would be more prevalent in the injured than the uninjured leg of inversion ankle sprain patients, can be accepted.

Travel and Simons (1993 2: 110) state that a once off traumatic occurrence can activate myofascial trigger points. When the ankle inverts during a lateral ankle sprain, the fibularis muscles are forcefully stretched whilst trying to contract to bring about their normal action. Therefore these muscles are often injured from traction when the foot inverts (Karageanes, 2004). McGrew and Schenck (2003) state that the musculature and neural structures surrounding the ankle joint may be affected during an ankle sprain injury, and if left unresolved, these deficits will lead to chronic instability, which may affect future athletic ability and may increase risk of re-injury.

My second hypothesis stated that the lateral ankle pain following an inversion ankle sprain injury may be due to referred pain from the fibularis muscle trigger points. Subjective lateral ankle pain measured by the NRS was, however, not correlated with any of the objective severity measurements (Myofascial Diagnostic Scale, Goniometer and Functional Evaluation Scale) of fibularis myofascial trigger points at baseline. Therefore this hypothesis cannot be accepted. Pain measured by the algometer in the fibularis tertius muscle was
significantly and negatively correlated with dorsiflexion \((r=0.547, p=0.003)\). This meant that as algometer measurements increased (i.e. a decrease in the tenderness of myofascial trigger points), dorsiflexion measurements increased. It is known that fibularis tertius function is to dorsiflex and evert the ankle (Moore and Agur, 1995: 254), and so this result substantiates the literature in this regard.

My third hypothesis stated that dry needling of the fibularis muscles would be a more effective treatment method than the placebo treatment in the relieving of lateral ankle pain experienced by ankle sprain patients. This hypothesis however, cannot be accepted as only one outcome measurement, namely the Myofascial Diagnostic Scale score \((p=0.030)\), could statistically support it. Subjective pain measurement (Numerical Pain Rating Scale), objective pain measurement (algometer), dorsiflexion and eversion range of motion showed no difference in the two groups although a statistically insignificant trend was noted towards a more beneficial effect in the dry needling group.

The results of this study indicate that the lateral ankle pain experienced after an inversion ankle sprain cannot solely be attributed to referred pain from the fibularis muscle trigger points in the injured leg. Although fibularis muscle trigger points develop as a result of an ankle sprain (as shown in hypothesis one), there are many other factors involved in this injury. These include, amongst others, lateral ankle ligament and capsular tears and the resulting oedema and haemorrhage (Cailliet, 1997). In the researchers opinion, all these factors should be considered when treating an ankle sprain, as it is more likely that a combination of all the structures injured cause the lateral ankle pain. It follows that the second hypothesis of this study, which stated that the lateral ankle pain following an inversion ankle sprain injury may be due to referred pain from the fibularis muscle trigger points, cannot be accepted.

Although fibularis myofascial trigger points have been proven not to be the only source of lateral ankle pain in ankle sprain patients, it is still important to treat
these myofascial trigger points, so as to avoid functional instability as a result of fibularis muscle weakness (Louwerens and Snijders, 1999).

5.2. Limitations of this study and recommendations

5.2.1. Demographics
The ethnic distribution in this study was not representative of the South African population.

**Recommendation:** Follow up studies should attempt to achieve a better representation of the South African population. Placing research advertisements in community clinics in rural areas would encourage this.

No research concerning the incidence and prevalence of ankle sprains in South Africa could be found.

**Recommendation:** Research into the incidence and prevalence of ankle sprains specifically in South Africa is suggested. This would also give a better indication of the prevalence in different ethnic groups.

5.2.2. Methodology
In this study the injured leg was compared to the uninjured leg to determine the prevalence of fibularis myofascial trigger points in ankle sprain patients. Therefore the participant acted as his/her own control group.

A high percentage of fibularis myofascial trigger points were however also found in the uninjured leg. This may possibly be related to an antalgic gait (to protect the injured ankle) causing the uninjured legs fibularis muscles to become overloaded. Also, previous injuries to the uninjured leg may not have been reported. This phenomenon could have altered the prevalence results.
**Recommendation:** To be more specific, further research is needed to compare fibularis myofascial trigger points in sprained ankles to participants who have never sprained ankles. This would rule out the theory of altered gait causing fibularis myofascial trigger points in the uninjured leg, and would also provide baseline information on how prevalent fibularis muscle trigger points are without previous injury.

### 5.2.3 Number of treatments

Each participant received two treatments, either dry needling of the fibularis muscle trigger points or ultrasound applied to the fibularis muscle. The treatments were generally spaced 2 days apart. A follow up appointment took place 3 days after the last treatment.

Due to the natural history of an ankle sprain lasting between 2-10 days, this research time frame may have proven too short.

**Recommendation:** For future studies, I recommend that participants should be monitored for a minimum of 10 days, so as to take the natural history of an ankle sprain into account.

### 5.2.4. Sample size

44 participants were included in phase 1, and 40 participants in phase 2 of this study. In phase 2, some of the results indicated a trend towards a more beneficial effect in the dry needling group. This, however, was statistically insignificant possibly due to a small sample size.

**Recommendation:** Increase the sample size to avoid a type 2 error, where a clinical difference is observed, but due to an underpowered study (low sample size) the effect is not statistically significant.
5.2.5. Measurements
The Goniometer was used as an objective measurement to determine ankle range of motion. This study found that the goniometric readings for inversion showed no difference between the two ankles (injured and uninjured) at baseline. This finding was unexpected, as the presence of fibularis muscle trigger points were expected to limit inversion.

This tool is highly dependant on correct placement and although the Goniometer was placed in reference with the same anatomical structure, inaccuracies can occur due to human error. Therefore the specificity of this tool in this study is questioned.

**Recommendation:** In future studies, consider the use of the Inclinometer in place of the Goniometer. An independent observer should randomly check objective data to ensure accurate readings.
REFERENCES:


Statistics South Africa [online]. 2006. Available from: 


Sprained your ankle? Do you still have ankle pain?

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 Ingrid
031-2042205 or 031-2042512
PATIENT SCREENING:

Questions to be asked during telephonic interview:

**Inclusion Criteria:**
- Have you sprained one of your ankles?
- Are you between 15 and 50 years of age?
- Do you currently have ankle pain? If yes, where is the location of the pain?

**Exclusion Criteria:**
- Have you received any treatment for the ankle sprain? If yes, what treatment have you received?
- Have you sprained the opposite ankle in the past?
- Have you had a history of foot or ankle fracture, dislocation or surgery?
- Have you had any history (or currently suffer from) any of the following:
  - Peripheral neuropathy
  - Nerve root entrapment
  - Arthritis affecting your lower limb?
  - Any blood dyscrasias / clotting disorders?
APPENDIX 3

LETTER OF INFORMATION

Dear participant  Date: __________

Welcome to my research project.

Title of Research:
The prevalence and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains.

Name of supervisor: Dr. R. White (M. Tech: Chiropractic)
Tel: 033-3422649

Name of student: Miss Ingrid van der Toorn
Tel: 031-2042205

Purpose of the study:
To determine the prevalence of fibularis muscle trigger points (hyperirritable spots in the muscles on the outside part of your leg) in ankle sprain patients, and the influence these trigger points have, particularly on lateral ankle pain.

Procedures:
This study will involve research on 40 patients and each patient will have either one/three visits (depending on the findings during examination) to the Chiropractic Clinic. You will be required to undergo an initial consultation, of approximately one and a half hours, during which a case history, physical examination and foot regional examination will be performed. After a diagnosis of inversion ankle sprain has been reached, measurements will be taken of your injured and uninjured leg. This will require filling out pain questionnaires and answering questions regarding your ankle pain.

If, during the examination, it is found that you have myofascial trigger points in the fibularis muscle, you will receive two free treatments for these trigger points in 4 days. The treatment will depend on the group you are placed in, and will either be dry needling of the trigger points in the fibularis muscle or ultrasound applied to these trigger points. You will then be required to return to the Chiropractic Clinic after three days following your last treatment so that more measurements can be taken.
However, if no fibularis myofascial trigger points are found during the examination, you will receive one free treatment and will then be excluded from the rest of the study.

**Risks / Discomforts:**
Although this study includes a placebo treatment, the examination and intervention is unlikely to cause any adverse side effects, other than transient tenderness and stiffness. Very rarely patients may develop bruising following dry needling.

If you received the placebo treatment during this research project, you will be entitled to two free treatments at the Chiropractic Clinic for your ankle sprain, following the completion of this study.

**Benefits:**
Your contribution to this study may help us as Chiropractors, to build on our knowledge on how to best manage lateral ankle pain after an ankle sprain.

**New findings:**
You will as a participant to this study 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 participating in this study without your consent for the following reason:
• If you are unable to attend your follow up appointments.

**Remuneration:**
No remuneration should be expected as a result of participation in this study.

**Cost of the study:**
This study will be free of charge and your participation in this study is voluntary.

**Confidentiality:**
All patient information is confidential. The results of this study will be used for research purposes only. Only individuals who are directly related to this study (Dr. R. White, Ingrid van der Toorn) will be allowed access to these records.

**Persons to contact for problems or questions:**
Should you have any questions that you would prefer being answered by an independent individual, feel free to contact my supervisor on the above number. If you are not completely 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 your interest and participation.
Yours sincerely,

Ingrid van der Toorn  
(Research student)

Dr. Rowan White (M. Tech: Chiropractic)  
(Research supervisor)
APPENDIX 4

INFORMED CONSENT FORM
(To be completed by patient / subject )

Date : 

Title of research project : The prevalence and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains.

Name of supervisor
Tel : Dr. R. White (M.Tech: Chiropractic) (033) 3422649

Name of research student
Tel : Ingrid van der Toorn (031) 2042205

Please circle the appropriate answer

<table>
<thead>
<tr>
<th></th>
<th>YES /NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Have you read the research information sheet?</td>
</tr>
<tr>
<td>2.</td>
<td>Have you had an opportunity to ask questions regarding this study?</td>
</tr>
<tr>
<td>3.</td>
<td>Have you received satisfactory answers to your questions?</td>
</tr>
<tr>
<td>4.</td>
<td>Have you had an opportunity to discuss this study?</td>
</tr>
<tr>
<td>5.</td>
<td>Have you received enough information about this study?</td>
</tr>
<tr>
<td>6.</td>
<td>Do you understand the implications of your involvement in this study?</td>
</tr>
<tr>
<td>7.</td>
<td>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.</td>
</tr>
<tr>
<td>8.</td>
<td>Do you agree to voluntarily participate in this study</td>
</tr>
<tr>
<td>9.</td>
<td>Who have you spoken to ________________________</td>
</tr>
</tbody>
</table>

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 5

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:

Signed off: ................................. Date:
Intern's Case History:
1. Source of History:
2. Chief Complaint: (patient's own words):
3. Present Illness:
   - Location
   - Onset: Initial:
     - Recent:
   - 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
     Anasthesis/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 6

DURBAN INSTITUTE OF TECHNOLOGY

PHYSICAL EXAMINATION
SENIOR & RESEARCH

<table>
<thead>
<tr>
<th>Patient:</th>
<th>File#:</th>
<th>Date:</th>
</tr>
</thead>
<tbody>
<tr>
<td>_Student:</td>
<td>Signature:</td>
<td></td>
</tr>
</tbody>
</table>

**VITALS**

<table>
<thead>
<tr>
<th>Pulse rate</th>
<th>Respiratory rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood pressure</td>
<td>R</td>
</tr>
<tr>
<td>Temperature</td>
<td>Height:</td>
</tr>
<tr>
<td>Weight:</td>
<td>Any recent change</td>
</tr>
</tbody>
</table>

**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

COMMENTS

NEUROLOGICAL EXAMINATION: See regionals

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).

<table>
<thead>
<tr>
<th>Observation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Swelling</td>
</tr>
<tr>
<td>Heloma dura / molle</td>
</tr>
<tr>
<td>Skin</td>
</tr>
<tr>
<td>Nails</td>
</tr>
<tr>
<td>Shoes</td>
</tr>
<tr>
<td>Contours (achilles tendon, bony prominences)</td>
</tr>
</tbody>
</table>

**Active movements**

**weight bearing:**

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

| Big toe dorsiflexion (mtp) (65-70°) |
| Big toe plantar flexion (mtp) 45°  |
| Toe abduction + adduction  |
| 5° first ray dorsiflexion  |
| 6° first ray plantar flexion |

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

<table>
<thead>
<tr>
<th>Ankle joint: Plantarflexion</th>
<th>R</th>
<th>L</th>
<th>Subtalar joint: Varus</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dorsiflexion</td>
<td></td>
<td></td>
<td>Valgus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Talocrural: Long axis distraction</td>
<td></td>
<td></td>
<td>Midtarsal: A-P glide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First ray: Dorsiflexion</td>
<td></td>
<td></td>
<td>P-A glide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Plantarflexion</td>
<td></td>
<td></td>
<td>rotation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Circumduction of forefoot on fixed rearfoot</td>
<td></td>
<td></td>
<td>Intermetatarsal glide</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interphalangeal joints: L-&gt;A dist</td>
<td></td>
<td></td>
<td>Metatarsophalangeal dorsiflexion (with associated plantar flexion of each toe)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>A-P glide</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lat and med glide</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rotation</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>
### Resisted Isometric Movements

<table>
<thead>
<tr>
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<td>Test for rigid/flexible flatfoot</td>
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### Alignment

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<td>Medial maleoli</td>
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</tr>
<tr>
<td>Med tarsal bones, tibial (post) artery</td>
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<tr>
<td>Lat.malleolous, calcaneus, sinus tarsi, and cuboid bones</td>
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<td>Inferior tib/fib joint, tibia, mm of leg</td>
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<tr>
<td>Anterior tibia, neck of talus, dorsalis pedis artery</td>
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<tr>
<td>Posteriorly</td>
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### APPENDIX 8

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<tr>
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<td><strong>A:</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</strong></td>
<td></td>
<td></td>
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<tr>
<td><strong>O:</strong></td>
<td><strong>P:</strong></td>
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<td><strong>E:</strong></td>
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**Special attention to:**

**Next appointment:**

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<td><strong>S:</strong> Numerical Pain Rating Scale (Patient)</td>
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<tr>
<td><strong>Least 0 1 2 3 4 5 6 7 8 9 10 Worst</strong></td>
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<td><strong>O:</strong></td>
<td><strong>P:</strong></td>
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<td><strong>E:</strong></td>
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**Special attention to:**

**Next appointment:**

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APPENDIX 9

MYOFASCIAL DIAGNOSTIC SCALE

File no: __________________________

Muscle: __________________________

Visit No: _____________

SIGNS:

1. Soft tissue tenderness
   1. Grade:

   0  No tenderness
   1  Tenderness to palpation WITHOUT grimace/flinch
   2  Tender WITH grimace and or flinch to palpation
   3  Tenderness with WITHDRAWAL (+" Jump sign")
   4  Withdrawal (+" Jump sign") to non-noxious stimuli
      (i.e. Superficial palpation, pin prick, gentle percussion)

2. Snapping palpation of the trigger point evokes a local twitch response

3. The trigger point is found in a palpable taut band

4. Moderate, sustained pressure on the trigger point causes or intensifies pain in the reference zone

   ________

   Total  ________

NUMERICAL PAIN RATING SCALE:

0  1  2  3  4  5  6  7  8  9  10
### ALGOMETER READINGS:

File no: _________

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<tr>
<td>Fibularis brevis</td>
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APPENDIX 11
GONIOMETER READINGS:

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<tr>
<td>Plantarflexion</td>
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<tr>
<td>Plantarflexion</td>
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<tr>
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</tr>
<tr>
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APPENDIX 12

PRESENCE AND LOCATION OF FIBULARIS MYOFASCIAL TRIGGER POINTS

File no: _________

Questions regarding history:

1. Did the ankle sprain involve the dominant/ non-dominant leg?

2. When did you sprain your ankle? ______________

<table>
<thead>
<tr>
<th>VISIT 1</th>
<th>INJURED</th>
<th>UNINJURED</th>
</tr>
</thead>
</table>
| Fibularis longus  
(Trp tenderness about 2-4cm below the head of fibula) |         |           |
| Fibularis brevis  
(Trp tenderness either side of, and deep to, the fibularis longus tendon near the junction of the middle and lower third of leg) |         |           |
| Fibularis tertius  
(Trp tenderness proximal and anterior to the lateral malleolus, distal and anterior to fibularis brevis Trp) |         |           |

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<td>Fibularis brevis</td>
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<tr>
<td>Fibularis tertius</td>
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File no: __________

SUBJECTIVE ASSESSMENT OF THE INJURED ANKLE:

With reference to your ankle injury, are you currently experiencing:
- Pain
- Tenderness
- Stiffness
- Swelling
- Giving way during activity

1. no symptoms of any kind 15
2. mild symptoms (1 of above symptoms present) 10
3. moderate symptoms 5
4. severe symptoms (4 or more symptoms present) 0

Can you walk normally?
1. yes 15
2. no 0

Can you run normally?
1. yes 10
2. no 0

FUNCTIONAL TESTS:

Climbing down two levels of staircase:

- < 18 seconds 10
- 18-20 seconds 5
- > 20 seconds 0

STRENGTH TESTS:
Rising on heels with injured leg:
- > 40 times: 10
- 30-39 times: 5
- < 30 times: 0

Rising on toes with injured leg:
- > 40 times: 10
- 30-40 times: 5
- < 30 times: 0

**BALANCE TEST:**

Single-limb stance on injured leg:
- > 55 seconds: 10
- 50-55 seconds: 5
- < 50 seconds: 0

**CLINICAL MEASUREMENTS:**

Laxity of the ankle joint: (anterior drawer test)
- stable (< 5mm): 10
- moderate stability (6-10mm): 5
- severe instability (>10mm): 0

Dorsiflexion range of motion:
- > 10 degrees: 10
- 5-9 degrees: 5
- < 5 degrees: 0

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<tr>
<th>Excellent: 85-100</th>
<th>Good: 70-80</th>
<th>Fair: 55-65</th>
<th>Poor: &lt;50</th>
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<td>TOTAL: ________</td>
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</table>
The prevalence and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains.

I. van der Toorn (M.Tech: Chiropractic)
From the Department of Chiropractic, Durban University of Technology, South Africa.

Dr. R. White (M. Tech: Chiropractic, RSA)
Supervisor

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Phone: (031) 204 2611, Facsimile: (031) 202 3632

Article written according to Journal of Chiropractic Medicine’s format.
ABSTRACT:

OBJECTIVES: The aim of this study was to determine the prevalence and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains.

METHODS: This study was an analytical, cross sectional study (phase 1) and randomised controlled trial (phase 2). 44 participants with a history of a unilateral inversion ankle sprain were selected by consecutive convenience sampling for phase 1, and examined to determine the presence of fibularis myofascial trigger points in the injured leg and the uninjured leg (control group). 40 Phase 1 participants with fibularis myofascial trigger points in the injured leg were then accepted into phase 2. The participants were randomly divided into two equal groups: a treatment group (dry needling of fibularis muscle trigger points) and a placebo group (detuned ultrasound applied to fibularis muscle trigger points). Each of the participants received two treatments spaced roughly over 4 days. Subjective data was obtained using the Numerical Pain Rating Scale to measure lateral ankle pain. Objective data was obtained by palpation for presence and location of fibularis muscle trigger points, an Ankle Functional Evaluation Scale to evaluate functional disability, Myofascial diagnostic scale to determine the extent to which the participant suffered from myofascial pain. The Algometer was used to measure pain threshold over the fibularis muscle trigger points, and Goniometer to measure the ankle range of motion (plantarflexion, dorsiflexion, eversion and inversion. Data capture took place prior to the first and second treatment and on a follow up consultation. A p value of <0.05 was considered as statistically significant. For phase 1, the groups were compared with regard to the various quantitative outcomes using paired t-tests, and comparisons with categorical outcomes were done using McNemar’s chi square tests. Associations between presence, number of trigger points and clinical outcomes were done by means of one-way ANOVA in the injured ankles. Correlations between baseline subjective and objective outcome measurements were done for the injured
ankles using Pearson’s correlation coefficients. For phase 2, repeated measures ANOVA were used to compare treatment groups over time, with profile plots of means by group over time. A significant time by group (time*group) interaction indicated a significant treatment effect. The direction of the treatment effect was assessed from the profile plots and was done separately for each outcome measurement.

RESULTS: Phase 1 demonstrated significantly more fibularis longus and brevis trigger points in injured ankles than in paired uninjured ankles at baseline. The MDS score at baseline was significantly higher in the injured than in the uninjured ankles (p<0.001). Dorsiflexion was significantly higher in the uninjured ankle than the injured ankle (p=0.006), as was plantarflexion (p=0.022). Phase 2 demonstrated a statistically significant beneficial treatment effect of dry needling over placebo for the MDS score (p=0.030). Most other outcomes showed that both treatment groups improved to the same extent over time while some outcomes suggested that the dry needling technique was beneficial over the placebo, but failed to show a statistically significant effect.

CONCLUSION: This study indicates that fibularis muscle trigger points are more prevalent in sprained ankles compared to uninjured ankles, but treatment of these trigger points with dry needling is no more effective than a placebo treatment in the relieving of lateral ankle pain. Therefore the lateral ankle pain experienced by ankle sprain patients cannot solely be attributed to referred pain from the fibularis muscles.

Key indexing terms: Inversion ankle sprains, fibularis muscle, referred pain, recommended treatment protocol.
ARTICLE:

INTRODUCTION:

One of the most commonly injured joints in the body is the ankle (Fallat, et al. 1998 and Jerosch and Bischof, 1996). Ankle sprains are one of the most common musculoskeletal injuries that primary care physicians will come across in their practices (McGrew and Schenck, 2003) and they account for 85% of all injuries to the ankle (Garrick, 1997). Inversion ankle sprains are more common than eversion sprains (Moore and Agur, 1995:276) due to the lateral ankle ligaments being much weaker than the medial ligaments (Shapiro, et al. 1994).

When the foot strikes the ground during the normal gait cycle, the foot is plantarflexed and supinated. In this position the talus is moveable within the mortise joint, and so the ankle relies on the ligaments for stability. If there is a rotational or lateral stress while weight bearing, the lateral ligaments can be overwhelmed causing an inversion ankle sprain (Calliet, 1997). Often this injury occurs due to direct trauma (Rimando, 2005) or sporting activities e.g. running on uneven terrain, stepping in a hole or landing from a jump in an unbalanced position (Hockenbury and Sammarco, 2001). Overload of the fibularis muscles has also been suggested (Rimando, 2005).

When considering the mechanism of injury of a lateral ankle sprain, the importance of the fibularis muscles becomes apparent. During the gait cycle the fibularis longus and brevis muscle’s role is to evert and plantarflex the foot, and fibularis tertius assists with eversion and dorsiflexion rather than plantarflexion of the foot (Moore and Agur, 1995: 254). When the ankle inverts during a lateral ankle sprain, these muscles are forcefully stretched whilst trying to contract to bring about their normal action. Therefore these muscles are often injured from traction when the foot inverts (Karageanis, 2004). Travel and Simons (1993 2:110) state that a once off traumatic occurrence can activate myofascial trigger
points. It stands to reason that as a result of the ankle sprain mechanism of injury, myofascial trigger points may develop in the fibularis muscles.

Myofascial trigger points in fibularis longus and brevis refer pain and tenderness primarily over the lateral malleolus of the ankle, above, behind and below it. Pain is also felt along the lateral aspect of the foot. Occasionally a spill over pattern may be felt over the lateral aspect of the middle third of the leg. Fibularis tertius trigger points refer pain along the anterolateral aspect of the ankle, mainly anterior to the lateral malleolus, with a spill over pattern to the outer side of the heel. (Travell and Simons, 1993 2:371). The pain distribution of the fibularis muscles correlate with the area where inversion ankle sprain patients experience pain.

The standard recommended treatment protocol (Hockenbury and Sammarco, 2001) following an acute ankle sprain could be simplified with a mnemonic: PRICE (protection, rest, ice, compression and elevation). In the acutely sprained ankle NSAIDs could also be used to reduce pain and limit inflammation (Rimando, 2005). Once the patient is pain free, a rehabilitation program should be started focusing on range of motion, fibularis strengthening exercises and proprioception (McGrew and Schenck, 2003; Calliet, 1997; Hockenbury and Sammarco, 2001; Wexler, 1998). In the literature, although strengthening exercises for the ankle musculature is recommended, no mention is made of treating any myofascial trigger points (in any way, including dry needling) prior to strengthening.

Myofascial trigger points, active or latent, can cause significant motor dysfunction (Travell, et al. 1999 1: 19). Fibularis muscle weakness is thought to be a source of symptoms after an inversion ankle sprain (Trevino, et al. 1994). McGrew and Schenck (2003) stated that the musculature and neural structures surrounding the ankle joint may be affected during an ankle sprain injury, and if left
unresolved, these deficits will lead to chronic instability, which may affect future athletic ability and may increase risk of re-injury.

Phase 1 of this study aimed at determining the prevalence of fibularis myofascial trigger points in ankle sprain patients. Phase 2 compared a placebo treatment (detuned ultrasound) to dry needling of the fibularis myofascial trigger points to evaluate whether treatment of the myofascial trigger points would relieve lateral ankle pain experienced by ankle sprain patients.

**MATERIALS AND METHODS:**

This study was an analytical, cross sectional study (phase 1) and randomised controlled trial (phase 2) that was conducted in order to determine the prevalence (phase 1) and clinical presentation of fibularis myofascial trigger points in the assessment and treatment of inversion ankle sprains (phase 2). Consecutive convenience sampling was used. In phase 1, the injured leg was compared to the uninjured leg, therefore each participant acted as his/her own control. In phase 2, drawing a letter (either A/B) out of an envelope randomly divided the forty participants into two equal groups. A indicated the dry needling group and B indicated the placebo group (detuned ultrasound)

Inclusion criteria:

This study was divided into two phases and separate criteria applied to them. The criteria for phase 1:

- Participants had to have a history of a subacute / chronic unilateral inversion ankle sprain with persistent lateral ankle pain.
- Only participants diagnosed with grade 1 inversion ankle sprains were accepted into this study.
- Participants had to be between the ages of 15 and 50 as recommended by Pellow and Brantingham (2001). This limited age group facilitated increased population group homogeneity.

The criteria for phase 2 included:

- Presence of fibularis myofascial trigger points in the injured leg, as assessed in phase 1.
- A history of a chronic inversion ankle sprain not exceeding 3 months. This time limit was set so as to increase the group homogeneity.
- Numerical pain rating of 6 or above to increase sample homogeneity.

Exclusion criteria:

The exclusion criteria listed below applied for both phase 1 and 2:

- Participants who had received any trigger point therapy for their ankle sprain were excluded from this study. Participants were instructed not to initiate any form of treatment while taking part in the study (Pellow and Brantingham, 2001).
- Participants diagnosed with grade II and III ankle sprain.
- Participants with a history of bilateral inversion ankle sprains. In this study the uninjured leg will act as the control group.
- A history of foot or ankle fracture, dislocation or surgery.
- Participants with any systemic arthritide that affected the ankle.
- Participants with a neurological deficit of the lower limb.
- Participants who present with any contra indications to dry needling including skin infections over the leg, allergy to specific metals, blood dyscrasias or local malignancies (Liggins, 2003).

Those accepted into this study underwent one consultation for phase 1 and if fibularis myofascial trigger points were found, a further 2 consultations for phase 2. Therefore phase 2 involved two treatments and one follow-up over a seven-day period.
Data collection instruments utilised for subjective measurements included Numerical pain Rating Scale to measure lateral ankle pain. Objective data was obtained by palpation for presence and location of fibularis muscle trigger points, an Ankle Functional Evaluation Scale to evaluate functional disability, Myofascial diagnostic scale to determine the extent to which the participant suffered from myofascial pain, the Algometer was used to measure pain threshold over the fibularis muscle trigger points, and Goniometer to measure the ankle range of motion (plantarflexion, dorsiflexion, eversion and inversion). Data capture took place prior to the first and second treatment and on a follow up consultation.

Data analysis:

Data was entered into a MS Excel spreadsheet and imported into SPSS version 13 (SPSS Inc., Chicago, Illinois, USA) for analysis. A p value of <0.05 was considered as statistically significant.

Phase I:
The groups were compared with regard to the various quantitative outcomes using paired t-tests, and comparisons with categorical outcomes were done using McNemar’s chi square tests. Associations between presence, number of trigger points and clinical outcomes were done by means of one-way ANOVA in the injured ankles. Correlations between baseline subjective and objective outcome measurements were done for the injured ankles using Pearson’s correlation coefficients.

Phase II:
Repeated measures ANOVA was used to compare treatment groups over time, with profile plots of means by group over time. A significant time by group (time*group) interaction indicated a significant treatment effect. The direction of
the treatment effect was assessed from the profile plots. This was done separately for each outcome measurement.

RESULTS:

This study demonstrated significantly more fibularis longus and brevis trigger points in injured ankles than in paired uninjured ankles at baseline.

Table 1 shows that there was a significantly higher prevalence of fibularis longus trigger points in injured (95.5%) than in uninjured ankles (79.5%) (p=0.039).

Table 1: Cross tabulation of presence of fibularis longus trigger points in injured and uninjured ankles

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<th>Visit 1 fibularis longus uninjured</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>abs</td>
<td>pres</td>
</tr>
<tr>
<td>----</td>
<td>-----</td>
</tr>
<tr>
<td>abs</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>pres</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>34</td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
</tr>
</tbody>
</table>

McNemar’s chi square p value 0.039

Table 2 shows that there was a significantly higher prevalence of fibularis brevis trigger points in injured (81.8%) than in uninjured ankles (59.1%) (p=0.031).

Table 2: Cross tabulation of presence of fibularis brevis trigger points in injured and uninjured ankles
Table 3 shows that there was a non significantly slightly higher prevalence of fibularis tertius trigger points in injured (66.7%) than in uninjured ankles (52.4%) (p=0.146).

Table 3: Cross tabulation of presence of fibularis tertius trigger points in injured and uninjured ankles

The MDS (Myofascial Diagnostic Scale) score at baseline was significantly higher in the injured than in the uninjured ankles (p<0.001). Dorsiflexion was significantly higher in the uninjured ankle than the injured ankle (p=0.006), as was plantarflexion (p=0.022).
Table 4: Paired t-tests comparison of mean injured with uninjured ankles

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>t value</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visit 1 MDS injured</td>
<td>8.60</td>
<td>40</td>
<td>2.845</td>
<td>.450</td>
<td>5.020</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Visit 1 MDS uninjured</td>
<td>5.55</td>
<td>40</td>
<td>3.250</td>
<td>.514</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 algometer fibularis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>longus injured</td>
<td>4.041</td>
<td>32</td>
<td>1.8141</td>
<td>.3207</td>
<td>-1.286</td>
<td>0.208</td>
</tr>
<tr>
<td>Visit 1 algometer fibularis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>longus uninjured</td>
<td>4.316</td>
<td>32</td>
<td>2.0193</td>
<td>.3570</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 algometer fibularis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>brevis injured</td>
<td>4.633</td>
<td>21</td>
<td>1.8629</td>
<td>.4065</td>
<td>-0.320</td>
<td>0.752</td>
</tr>
<tr>
<td>Visit 1 algometer fibularis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>brevis uninjured</td>
<td>4.743</td>
<td>21</td>
<td>1.8763</td>
<td>.4094</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 algometer fibularis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tertius injured</td>
<td>3.748</td>
<td>21</td>
<td>1.5731</td>
<td>.3433</td>
<td>-0.297</td>
<td>0.769</td>
</tr>
<tr>
<td>Visit 1 algometer fibularis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tertius uninjured</td>
<td>3.829</td>
<td>21</td>
<td>1.8813</td>
<td>.4105</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 dorsiflexion injured</td>
<td>9.88</td>
<td>40</td>
<td>6.211</td>
<td>.982</td>
<td>-2.885</td>
<td>0.006</td>
</tr>
<tr>
<td>Visit 1 dorsiflexion uninjured</td>
<td>12.00</td>
<td>40</td>
<td>6.139</td>
<td>.971</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 plantarflexion injured</td>
<td>62.20</td>
<td>40</td>
<td>11.678</td>
<td>1.846</td>
<td>-2.387</td>
<td>0.022</td>
</tr>
<tr>
<td>Visit 1 plantarflexion uninjured</td>
<td>65.65</td>
<td>40</td>
<td>8.610</td>
<td>1.361</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 inversion injured</td>
<td>6.30</td>
<td>40</td>
<td>3.360</td>
<td>.531</td>
<td>-1.575</td>
<td>0.123</td>
</tr>
<tr>
<td>Visit 1 inversion uninjured</td>
<td>7.20</td>
<td>40</td>
<td>3.275</td>
<td>.518</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visit 1 eversion injured</td>
<td>5.70</td>
<td>40</td>
<td>3.148</td>
<td>.498</td>
<td>-0.232</td>
<td>0.818</td>
</tr>
<tr>
<td>Visit 1 eversion uninjured</td>
<td>5.83</td>
<td>40</td>
<td>3.161</td>
<td>.500</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Pain measured by the NRS was not correlated with any of the severity measurements at baseline. Pain measured by the Algometer in the fibularis tertius muscle was significantly correlated with dorsiflexion ($r=0.547$, $p=0.003$). This meant that as Algometer measurements increased (i.e. a decrease in the tenderness of myofascial trigger points), dorsiflexion measurements increased. Thus pain in the fibularis tertius muscle was negatively correlated with dorsiflexion.

Table 5: Pearson's correlation between baseline pain measurements and baseline severity measurements in injured ankles.

<table>
<thead>
<tr>
<th></th>
<th>Visit 1 NRS</th>
<th>Visit 1 algometer peronius longus injured</th>
<th>Visit 1 algometer peronius brevis injured</th>
<th>Visit 1 algometer peronius tertius injured</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visit 1 MDS injured</td>
<td>Pearson Correlation - 0.235</td>
<td>-0.130</td>
<td>0.035</td>
<td>-0.087</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed) 0.144</td>
<td>0.431</td>
<td>0.849</td>
<td>0.665</td>
</tr>
<tr>
<td></td>
<td>N 40</td>
<td>39</td>
<td>32</td>
<td>27</td>
</tr>
<tr>
<td>Visit 1 dorsiflexion injured</td>
<td>Pearson Correlation 0.096</td>
<td>0.302</td>
<td>0.293</td>
<td>0.547(**)</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed) 0.554</td>
<td>0.062</td>
<td>0.104</td>
<td>0.003</td>
</tr>
<tr>
<td></td>
<td>N 40</td>
<td>39</td>
<td>32</td>
<td>27</td>
</tr>
<tr>
<td>Visit 1 plantarflexion injured</td>
<td>Pearson Correlation 0.015</td>
<td>-0.133</td>
<td>-0.164</td>
<td>-0.312</td>
</tr>
<tr>
<td></td>
<td>Sig. (2-tailed) 0.928</td>
<td>0.419</td>
<td>0.369</td>
<td>0.113</td>
</tr>
<tr>
<td></td>
<td>N 40</td>
<td>39</td>
<td>32</td>
<td>27</td>
</tr>
<tr>
<td>Visit 1 inversion</td>
<td>Pearson Correlation - 0.022</td>
<td>-0.258</td>
<td>-0.335</td>
<td>-0.102</td>
</tr>
</tbody>
</table>
A statistically significant beneficial treatment effect of dry needling over placebo treatment was only demonstrated for the MDS score. Table 5 shows that a statistically significant time by group interaction (p=0.030) was found for MDS, meaning that the change over time was dependant on which treatment group the participants were in. Figure 1 shows that the MDS score for the dry needling group decreased at a much faster rate over time than the placebo group.

Table 6: Within and between subjects effects for MDS

<table>
<thead>
<tr>
<th>Effect</th>
<th>Statistic</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time</td>
<td>Wilk’s lambda=0.744</td>
<td>0.004</td>
</tr>
<tr>
<td>Time*group</td>
<td>Wilk’s lambda=0.828</td>
<td>0.030</td>
</tr>
<tr>
<td>Group</td>
<td>F=2.233</td>
<td>0.143</td>
</tr>
</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
Figure 1: Profile plot of mean MDS score over time by group

Most other outcomes showed that both treatment groups improved to the same extent over time while some outcomes suggested that the dry needling technique was beneficial over the placebo, but failed to show a statistically significant effect. This could have been a type 2 error, where a clinical difference is observed, but due to an underpowered study (low sample size) this effect was not statistically significant.
Figure 2: Profile plot of mean NRS score over time by group

Figure 3: Profile plot of mean algometer measurement of the fibularis longus muscle over time by group
Figure 4: Profile plot of mean algometer measurement of the fibularis brevis muscle over time by group.

Figure 5: Profile plot of mean algometer measurement of the fibularis tertius muscle over time by group.
Figure 6: Profile plot of mean dorsiflexion over time by group

Figure 7: Profile plot of mean plantarflexion over time by group
Figure 8: Profile plot of mean inversion over time by group

Figure 9: Profile plot of mean eversion over time by group
DISCUSSION:
In terms of racial distribution, the majority of the sample (59.1%) was White. Blacks constituted 22.7% and Indian or Coloured participants were 18.2%. The distribution is not representative of the South-African population and research concerning the incidence and prevalence of ankle sprains in South Africa is scant. More research is needed in this area to determine the prevalence of ankle sprains in the South African population.

The results of this study showed a statistically significant prevalence of fibularis longus and brevis myofascial trigger points in the injured leg compared to the uninjured leg. This supports McGrew and Schenck (2003) statement that the musculature and neural structures surrounding the ankle joint may be affected during an ankle sprain injury.

Figure 10: Profile plot of mean AFES score over time by group
Fibularis tertius trigger points were found to be more prevalent in the injured leg, but in a statistically non-significant manner. It has been documented that there are anatomical variants concerning the fibularis tertius muscle and that this muscle may be absent in a percentage of people.

In this study the injured leg was compared to the uninjured leg to determine the prevalence of fibularis myofascial trigger points in ankle sprain patients. Therefore the participant acted as his/her own control group. A high percentage of fibularis myofascial trigger points were however also found in the uninjured leg. This may possibly be related to an antalgic gait (to protect the injured ankle) causing the uninjured legs fibularis muscles to become overloaded. This phenomenon could have altered the prevalence results.

Comparison of the baseline outcome measurements between lateral ankle pain and severity of trigger points in injured ankles revealed no correlation. Therefore the degree of lateral ankle pain experienced by ankle sprain patients could not be linked to the severity of the fibularis myofascial trigger points found in the injured leg.

Pain measured by the algometer in the fibularis tertius muscle was significantly and negatively correlated with dorsiflexion ($r=0.547$, $p=0.003$). This meant that as algometer measurements increased (i.e. a decrease in the tenderness of myofascial trigger points), dorsiflexion measurements increased. This indicated that if the myofascial trigger points in the fibularis tertius muscles could withstand an increased amount of pressure (as exerted by the algometer), the trigger points were less severe, and would therefore allow for a greater dorsiflexion range of motion. It is known that the function of fibularis tertius is to dorsiflex and evert the ankle (Moore and Agur, 1995:254), and so this result substantiates the literature.
A statistically significant beneficial treatment effect of dry needling over placebo treatment was only demonstrated for the MDS score (p=0.030). Most other outcomes showed that both treatment groups improved to the same extent over time while some outcomes suggested that the dry needling technique was beneficial over the placebo, but failed to show a statistically significant effect. This could have been a type 2 error, where a clinical difference is observed, but due to an underpowered study (low sample size) this effect was not statistically significant. Further studies should be done to confirm these findings with a larger sample size.

CONCLUSION:

Based on this study, it would therefore seem that although fibularis myofascial trigger points are more prevalent in the injured leg compared to the uninjured leg of an ankle sprain patient, the lateral ankle pain experienced after an inversion ankle sprain cannot be solely attributed to referred pain from the fibularis muscle trigger points in the injured leg. However, these trigger points need to be addressed in the treatment protocol so as to ensure timely and maximum recovery, and limit the recurrence of injury and resulting development of ankle functional instability.

ACKNOWLEDGEMENTS:

I would like to thank Dr. White for reviewing this manuscript and the patients for their participation.
REFERENCES:


