A Prospective Clinical Trial To Determine The Relative Effectiveness Of Cross Friction Massage Versus Graston Instrument Assisted Soft Tissue Mobilisation In Treating Patellar Tendinopathy

by

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I, Donna Françoise Fraser, do declare that his dissertation is representative of my own work both in conception and execution.

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DEDICATION

My Heavenly Father, Jesus

To the loving memory of my hero and Dad,

Roy Frederick Fraser

22/05/1950 – 14/08/2005

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ABSTRACT

There are mechanical loads applied to the patella tendon in almost all sporting activities and as a result is commonly injured (Peterson and Renström, 2003:321). Patellar tendinopathy is a common chronic tendinopathy (Hamilton and Purdman, 2004) and occurs commonly in athletes who impose rapid eccentric loading of the knee extensor mechanism (Norris, 2004:246).

Deep Transverse Friction Massage (DTFM) and soft tissue mobilization are the two most common forms of manual therapy used to treat patellar tendinopathy (Rees *et al*., 2006). DTFM is considered the most effective treatment for patellar tendinopathy (Brunker and Khan, 2002:487). It is theorised that DTFM causes the softening of scar tissue and the breakdown of adhesions, promoting the realignment of disrupted connective tissue fibrils within the affected tendon (Stasinopoulos and Johnson, 2007).

Graston Instrument Assisted Soft Tissue Mobilization (GIASTM) consists of a set of stainless steel instruments (Carey 2003:2) and is an advanced form of soft tissue mobilization used in detecting and releasing scar tissue, adhesions and fascial restrictions (Carey, 2003:7). The controlled microtrauma created by these instruments is hypothesised to create a localised inflammatory response (Hammer, 2004) in a similar mechanism to that of DTFM.

The aim of this study was to determine the relative effectiveness of GIASTM versus DTFM in treating patellar tendinopathy.

The study included a total of twenty-six knees among twenty-one patients. Patients were placed randomly into either the GIASTM group or the DTFM group. Each patient received a total of twelve treatments over a three month period. Algometer and inclinometer readings were recorded at set intervals and compromised objective measures. Two questionnaires and a numerical pain rating scale (NRS) were administered at set intervals and compromised subjective measures.

SPSS version 13.0 (SPSS Inc., Chicago, Illinois, USA) was used to analyse the data. Repeated measures ANOVA was used to examine changes in quantitative outcomes over the time points (intragroup analysis) and a treatment effect (intergroup analysis). To control for the partial pairing in the intergroup analysis, a variable which classified each subject as paired (both left and right knee used in study) or non-paired (only used once in study) was used as a factor in the model. Correlations between the intragroup changes in the various outcome variables were assessed using Pearson's correlation coefficients.

Statistical analysis of both objective and subjective data revealed significant improvements for most outcome measures in the study. Findings imply that GIASTM is as effective as DTFM in treating patellar tendinopathy.

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DEFINITIONS

Patellar tendinopathy: For the purposes of this study patellar tendinopathy is defined as tendinopathy of the infra patella tendon.

Soft tissue mobilization: This is the hands on mobilization of soft tissues, (i.e. muscle and associated connective tissue that supports it, tendons and ligaments). It is performed on a patient for the purposes of producing beneficial effects on the nervous, muscular, lymphatic and circulatory systems. DTFM and GTIASTM are both forms of soft tissue mobilization and GTIASTM was developed as an alternative to DTFM (http://www.andoaston.com/mobilization.html, 2007).

Cross Friction Massage: For the purposes of this study will be referred to as Deep Transverse Friction Massage.

Mucoid degeneration: Macroscopically, the patella tendon of patellar tendinopathy patients contain soft, yellow-brown and disorganized tissue in the deep posterior portion of the patella tendon adjacent to the lower pole of the patella (Brunker and khan, 2007:524).

Patient: refers to any individual who does or does not participate in sport, as patellar tendinopathy occurs in both athletic (Norris, 2004:246) and non-athletic individuals (Rolf *et al.,* 2001 and Kountouris and Cook, 2007).

Absolute contraindications: For purposes of this study refers to those disease entities that by virtue of their pathogenesis exclude one or more forms of treatment.

Relative contraindications: For purposes of this study refers to those disease entities that by virtue of their pathogenesis may modify the application of one or more treatment modalities.

kN: kilonewton

CHAPTER ONE

1.1 INTRODUCTION

Anterior knee pain due to patellar tendinopathy is one of the most common presenting symptoms in orthopaedic, sports and occupational practices (Almekinders *et al*., 2002 and Brunker and Khan, 2007:506). The diagnosis of patellar tendinopathy is made clinically. It is considered resistant to treatment as well as recurrent in nature (Kongsgaard *et al*., 2006). Research indicates that patellar tendinopathy has a prevalence of up to fifty percent among elite athletes involved in jumping sports such as basketball, volleyball, netball and high jump (Lian *et al*., 2003 and Brunker and Khan, 2007:508).

Although patellar tendinopathy was uncommon until the 1960's, it is now considered by some authors to have reached epidemic proportions as a result of greater demands placed on athletes in the sporting arena (Ombregt *et al.*, 1996:826 and Panni *et al.*, 2000). Therefore, this condition is commonly seen in athletes whose sport places a high demand on the extensor mechanism of the knee (Taunton *et al.*, 2003 and Peace and Healy, 2006), such as: weight lifters, high jumpers and soccer players (Penn *et al.,* 2006). However patellar tendinopathy can occur in non-athletes, although this occurs to a lesser degree (Rolf *et al.,* 2001 and Kountouris and Cook, 2007). As a result patellar tendinopathy can cause significant morbidity in such athletes (Peace and Healy, 2006) and non-athletes (Rolf *et al.*, 2001).

Nevertheless conservative management of patellar tendinopathy has been based primarily on clinical experience (Cook and Khan, 2001). This has resulted in several treatment options, which have provided various anecdotally effective care options (Cannell *et al.*, 2001). However, studies have shown that only a few of these treatment interventions have a strong evidence base (Rees *et al.*, 2006). Within these treatment options, several forms of manual therapy are used, with the most common interventions being that of DTFM and soft tissue mobilization (Rees *et al.*, 2006).

DTFM has traditionally been one of the conservative treatment interventions used in the treatment of tendinopathies (Cook *et al.* (a), 2000). Its aim being that of localised hyperaemia, therapeutic movement, increased tissue perfusion, mechanoreceptor stimulation (Hammer, 1995) and the prevention of cross link formation between collagen fibres (Norris, 2004:64).

A more recent treatment development has been that of GIASTM, which consists of six stainless steel instruments with bevelled edges. This form of soft tissue mobilization is both similar in concept and application to DTFM (Carey, 2003:7) as well as being able to assist in the detection and releasing of scar tissue, adhesions and fascial restrictions (Carey, 2003:7).

However there are limited clinical trials on the use of GIASTM and therefore this study sought to compare the relative effectiveness of DTFM versus GIASTM in treating patellar tendinopathy.

1.2 AIMS/OBJECTIVES OF THE STUDY

The aim of this pilot study was to compare the relative effectiveness of GIASTM versus DTFM in treating patellar tendinopathy.

Therefore the purpose of this study was to fulfil the following objectives:

1.2.1 The first objective

To measure the relative effectiveness of GIASTM versus DTFM in treating patellar tendinopathy, in terms of objective clinical outcomes.

1.2.2 The second objective

To measure the relative effectiveness of GIASTM versus DTFM in treating patellar tendinopathy, in terms of subjective clinical outcomes.

1.3 THE STATEMENT OF THE HYPOTHESIS

1.3.1 The first null hypothesis

It was hypothesised that the use of GIASTM would be no more effective than DTFM in treating patellar tendinopathy in terms of objective findings.

1.3.2 The second null hypothesis

It was hypothesised that the use of GIASTM would be no more effective than DTFM in treating patellar tendinopathy in terms of subjective findings

1.4 DELIMITATION

It has been reported that successful outcomes of GIASTM cannot be achieved by use of the instruments alone. Instead an overall therapy programme should ideally include soft tissue mobilization; joint mobilization; stretching and strengthening programmes (Carey, 2003:2 and Perle, 2003); neuromuscular and posture re-education; proprioceptive activities; functional progression (Carey, 2003:64 and Hammer and Pfefer, 2005) as well as home exercises (Carey, 2003:2). However, in order to build a scientifically rigorous therapy programme each of the contributing modalities need to be investigated such that their individual significance is known, in order to study whether combinations of these tested therapies produce synergistic or antagonistic effects in terms of the clinical outcomes (Cleland and Durall, 2002). Therefore, this study was limited to use of only the GIASTM instruments and not within a protocol.

Although it is recommended that DTFM be applied for a period of twenty minutes on tendons (Cyriax and Cyriax, 1993:21), the treatment period in this study was ten minutes. It is also acknowledged that this technique may vary amongst conditions but that treatment is not affected as long as principles such as patient position and the amount of pressure used per stroke remain constant (Cyriax and Cyriax, 1993:22).

1.5 RATIONALE

1.5.1 A study investigating beach volleyball concluded that the prevalence of patellar tendinopathy may be due to ineffective treatment received by these athletes and that the prevalence is and maintains at a high level (Bahr *and* Jonathan*,* 2003). This is supported by Panni *et al.,* (2000), who found that more than a third of athletes in their study were unable to return to sport for more than six months due to patellar tendinopathy. Therefore Panni *et al* (2000) stated that adequate nonoperative management should first be attempted in order to prevent surgery as an option. It is further suggested by Bahr and Jonathan (2003) that new technologies and rehabilitative protocols should be investigated and developed in order to maximise treatment potential or minimise the effects of repetitive soft tissue injuries

1.5.2 Symptoms of patellar tendinopathy are not easily quantified (Visentini *et al.*, 1998:22-28) and evidenced based guidelines for management of these symptoms have largely been based on clinical observation (Hammer, 1995). Thus, such anecdotal evidence cannot be utilised as empirical evidence.

1.5.3 Rigorously designed clinical trials comparing the efficacy of different conservative and surgical techniques for the treatment of patellar tendinopathy are necessary (Peers and Lysens, 2005).

1.5.4 Outcomes such as functionality, pain, goal achievement and return to work for a variety of soft tissue conditions were measured on 1004 patients at 51 treatment sites in order to determine the health benefits of GIASTM. These results suggested GIASTM was effective in reducing pain, numbness and functional ability (Perle *et al.*, 2003). This therefore showed

that there is patient improvement and that GIASTM has some clinical effect that is consistent with the anecdotal data available. Therefore there is an anecdotal clinical improvement base, which supports the need for comparison between GIASTM to other modalities such as DTFM.

1.6 BENEFITS

Should the research show GIASTM is indeed more clinically effective than DTFM, the following benefits could be noted by practitioners in the field:

Patient:

- Quicker and improved outcomes (Carey, 2003:9 and Perle, 2003).
- Improvement in activities of daily living and quality of life (Carey, 2003:9).
- Greater depth of penetration of soft tissue lesions (Carey, 2003:9).
- Decrease treatment time (Carey, 2003:9).

Practitioner:

- Increased mechanical advantage and the ability to locate precisely soft tissue lesions (Carey, 2003:9 and Perle, 2003).
- Greater depth of penetration of soft tissue lesions (Carey, 2003:9).
- Joint conservation (Carey, 2003:9; Perle, 2003 and Matthews, 2006:1).

1.7 LIMITATIONS

Although the most common site of presentation in patellar tendinopathy is the inferior pole of the patella (Taunton *et al.*, 2003), the definition of patellar tendinopathy sometimes includes the attachment of the quadriceps tendon to the superior patellar pole (Hyman, 2006). For the purposes of this study, patellar tendinopathy was limited to symptoms at the inferior pole of the patella and did not include symptoms experienced at the superior patellar pole.

1.8 CONCLUSION

GIASTM and DTFM are both interventions used in the treatment of soft tissue disorders. It is the purpose of this study to determine the relative effectiveness of both these interventions (subjectively and objectively) in treating patellar tendinopathy. Therefore, Chapter Two will review the literature of patellar tendinopathy, Chapter Three will define methodology of the study, Chapter Four will interpret and discuss data and Chapter Five will make conclusions and recommendations based on the results of this study.

CHAPTER TWO

2.1 INTRODUCTION

Anterior knee pain due to patellar tendinopathy is one of the most common presenting conditions in orthopaedic, sports and occupational practices (Almekinders *et al*., 2002 and Brunker and Khan, 2007:5) causing significant functional deficit and disability in athletes and non-athletes (Khan *et al*., 1998; Stasinopoulos and Stasinopoulos, 2004 and Peers and Lysens, 2005).

There are mechanical loads applied to the patella tendon in almost all sporting activities and as a result is commonly injured (Peterson and Renström, 2003:321). Patellar tendinopathy commonly occurs in sports such as basketball, volleyball, netball and high jump (Lian *et al*., 2003 and Brunker and Khan, 2007:508) in which rapid eccentric loading of the patella tendon occurs (Norris, 2004:246). However, it also occurs in athletes who don't jump or have a sudden change in direction in their sport (Fritschy and de Gautard, 1988).

There are numerous treatments available for the management of tendon disorders but few are considered to have a strong evidence base (Rees *et al.*, 2006). DTFM and soft tissue mobilization are the two most common forms of manual therapy used to treat patellar tendinopathy (Stasinopoulos and Stasinopoulos, 2004 and Rees *et al.*, 2006).

Therefore this chapter presents:

- The nomenclature of patellar tendinopathy
- An anatomical description of the patella tendon
- The biomechanics of the extensor mechanism of the knee
- The epidemiology of patellar tendinopathy
- The pathoaetiology of patellar tendinopathy
- The clinical features of patellar tendinopathy
- A differential diagnosis of patellar tendinopathy

- Contributing factors of patellar tendinopathy
- Diagnosis of patellar tendinopathy
- Treatment options used in patellar tendinopathy
- DTFM and GIASTM as specific treatment options for patellar tendinopathy

2.2 NOMENCLATURE

Patellar tendinopathy has been described as Jumper's knee or patellar tendinosis, which refer to a tendon disorder of the knee. Despite our growing understanding of this disease process through imaging studies, surgical techniques and histopathology in the past decade (Brunker and Khan, 2007:524), interchanging of these terms still occurs. Patellar tendinopathy was referred to as Jumper's knee due to its frequency in jumping athletes (Panni *et al*., 2000) and was used first by Blazina *et al* in 1973 to describe an insertional tendinopathy seen in skeletally mature athletes (Hyman, 2006).

The exact pathoaetiological mechanism responsible for patellar tendinopathy is not clearly defined (Hamilton and Purdam 2004; Haraldsson *et al*., 2004; Peers and Lysens, 2005; Arnoczky *et al.*, 2007 and Kountouris and Cook, 2007). Therefore, the term tendinopathy is considered by Peers and Lysens (2005) and Brunker and Khan (2007:524) as the most appropriate label for this condition as it does not assume any knowledge of the underlying pathology (Stasinopoulos and Stasinopoulos, 2004 and Lian, 2007:vii).

Lian (2007) and Khan *et al* (1998) have added to this definition of patellar tendinopathy by including both acute and chronic cases.

Notwithstanding the debate around the definition of patellar tendinopathy, the definition used as the basis for this study is in congruence with Taunton *et al.,* (2003) who indicate that patellar tendinopathy is a clinical condition characterised by anterior knee pain and tenderness at the inferior pole of the patella which is aggravated by increased levels of activity.

2.3 THE PATELLA TENDON

The patella tendon consists of a broad band of fibrocartilagenous tissue and is the extension of the common tendon of the quadriceps femoris muscle (Moore and Dalley, 1999:619). It originates from the base of the patella (the anterior surface of the non articulating inferior patella pole), (Peace and Healy, 2006) and extends from the inferior pole to insert onto the tibial tuberosity (Travell and Simons, 1997:254 and Hansen *et al.*, 2006). The bulk attaches to the distal two thirds of the anterior aspect of the patella (Basso *et al.,* 2001) and blends with the medial and lateral patellar retinaculae (Moore and Dalley, 1999:619). Its dimensions are approximately 3cm in the coronal plane and 4.5 mm in the sagital plane (Peers and Lysens, 2005). The average thickness of the patella tendon is three to six millimetres, with variations of ten to fifteen millimetres (Peace and Healy, 2006). The anterior tendon fascicles are longer than the posterior fascicles since the anterior bundles are attached more proximally to the patella as well as more distally to the tibia than their corresponding posterior counterparts (Lian 2007, from Basso *et al.*, 2001).

The patella tendon consists of type one and type two collagen fibres and is surrounded by a paratenon (Peace and Healy, 2006). At the cellular level, the patella tendon is composed predominantly of type one collagen fibre (Kountouris and Cook, 2007) whose mechanical properties allow response to stresses in a viscoelastic manner (Pearson *et al*., 2007). In tendon pathology, type three collagen is produced, which is thinner and inadequate compared to the original type one collagen fibres (Kountouris and Cook, 2007). During the repair phase of tendon healing (which can last from 48 hours to 6 weeks), collagen is not fully oriented in the direction of tensile strength and the quality of newly formed collagen is not as superior as the original (Daniel *et al*., 1990:348 and Vizniak and Carnes, 2004:306). Improper healing during this repair phase results in impairment of the patella tendons' vital role of load absorption and transmission (Kountouris and Cook, 2007) and a weak patella tendon decreases the relative stability of the knee joint (Hall, 1999:125).

The paratenon is a layer of loose areolar connective tissue that surrounds the tendon and allows for the free movement of the tendon against surrounding tissues (Lian 2007 from Kvist *et al* 1985 and Hess *et al*, 1989). Beneath this paratenon is the fine connective tissue sheath of the epitenon, which is contiguous on its inner side with the endotenon. The endotenon surrounds and binds individual tendon fibres, allows individual gliding of the fibre groups and carries nerve, blood and lymphatic vessels to the deep portion of the patella tendon (Lian 2007 from Hess *et al* 1989).

The primary blood supply originates from the patellar fat pad and retinacular structures (Peers and Lysens 2005 and Peace and Healy, 2006) and two anastomotic rings. The blood supply to the proximal portion of the tendon enters precisely around the posterior aspect of the patella tendon and it is this region of the patella tendon that is reported to be most commonly affected by patellar tendinopathy (Khan *et al.*, 1998). These anastomotic rings are fed by the medial and lateral superior genicular arteries, the medial and lateral inferior genicular arteries and the anterior tibial recurrent arteries (Lian, 2007). The proximal and distal enthesial attachments of the patella tendon are relatively avascular (Perle, 2003 and Peace and Healy*,* 2006).

Even with the above-mentioned vascular supply, the patella tendon has a reduced vascularity and elasticity (Penn *et al.*, 2006) at the bone tendon interface compared to other tendons in the body. The stiffer tendon causes an unequal load distribution over the tendon insertion and the reduced vascularity diminishes the tendons recuperative powers (Rippey, 1994:163 and Penn *et al.*, 2006). Both these factors make the patella tendon susceptible to chronic overload injuries. The inferior pole of the patella is the most common site of patellar tendinopathy and may be explained by the distal insertional site of the patella tendon being thinner than the proximal (Penn *et al.*, 2006). As a result a greater load (tension) is borne by the insertion into the tibial tuberosity, especially during eccentric contractions of the quadriceps femoris muscle (Penn *et al.,* 2006 and Pearson *et al.*, 2007).

2.4 BIOMECHANICS

The knee functions as a modified hinge joint, with flexion and extension being its primary motions (Moore and Dalley, 1999:617 and Peterson *et al.*, 2002: 419). The knee joint connects the two longest lever arms in the body, the thigh and leg (Travell and Simons, 1997:254) and withstands large forces generated by the biomechanics of the lower limb (Moore, 1992:373 and Shamus and Shamus, 2001:96).

The patella plays an important role in the biomechanics of the knee (Dutton, 2004:735; Moore, 1992:388 and Pearson *et al*., 2007) as it provides a guide for the patella tendon, decreases friction of the extensor mechanism of the knee (Magee, 2002:663) and lengthens the lever arm for the quadriceps femoris muscle, increasing the effectiveness of knee extension (Souza, 1998: 275).

The extensor mechanism of the leg begins proximally with the quadriceps femoris muscle (Penn *et al.*, 2006). The quadriceps femoris muscle is comprised of the rectus femoris, vastus lateralis, vastus medialis and vastus intermedius muscles (Travell and Simons, 1997:264 and Penn *et al.,* 2006).

All four tendons of the quadriceps femoris muscle unite to form the quadriceps tendon that attaches to and surrounds the patella (Moore, 1992:387). As it passes from the inferior pole of the patella to its distal insertion at the tibial tuberosity, it is known as the patella or infrapatellar tendon. Serving as a connection between the tibia and patella, it is also classified as a ligament by its attachments. However the patella tendon also attaches the quadriceps unit to the tibia and is therefore considered a tendon by function (Dutton, 2004:7).

Static stability of the knee is dependent on the anterior and posterior cruciate ligaments and the medial and lateral collateral ligaments, providing a primary restraint to abnormal knee motion (Magee, 2002:661 and Dutton, 2004:738). Laterally the iliotibial band provides anterolateral reinforcement against

excessive internal rotation of the tibia on the femur. The quadriceps muscle crosses the knee joint anteriorly and is the main extensor of the knee. Medial support is provided by the sartorius and gracilis muscles (Moore and Dalley, 1999:532). The pes anserinius tendon (area where the tendons of the sartorius, gracilis and semitendinosus muscles attach distally) and semimembranous tendon reinforce the joint posteromedially (Moore and Dalley, 1999:563). The prime flexor of the knee joint is the hamstring muscle group with the biceps femoris tendon providing posterolateral support (Moore and Dalley, 1999:563). The gastrocnemius and popliteus muscles provide posterior support (Peterson and Bergmann, 2002:418,419). Internally, stability and control of rotation are provided by the meniscocruciate system (Souza, 1998:275).

2.5 EPIDEMIOLOGY

Patellar tendinopathy is one of the common chronic tendinopathies (Hamilton and Purdman, 2004) and occurs commonly in athletes who impose rapid eccentric loading of the patella tendon (Norris, 2004:246). This condition has also been reported in non-athletic populations (Rolf *et al.,* 2001 and Kountouris and Cook, 2007). Prior to 2003 the prevalence of patellar tendinopathy within different sports was relatively unreported (Peterson and Renström, 2003:2) and the frequency of athletes presenting to treatment clinics with an overuse injury is described as being up to fifty percent (Lian *et al*., 2003 and Brunker and Khan, 2007:508). Cases of patellar tendinopathy have increased over the years as a result of a general increase in the competitive demands placed on athletes (Panni *et al.*, 2000). This has resulted in an increased awareness of tendinopathies within the primary health professions (Panni *et al.*, 2000).

Patellar tendinopathy affects athletes in many different fields of sports and at different levels of participation (Khan *et al*., 1998; Stasinopoulos and Stasinopoulos, 2004 and Peers and Lysens, 2005). Research has indicated a particular affinity for elite jumping athletes (Peace and Healy, 2006) in which there is rapid acceleration, deceleration, jumping and sudden change in

direction of the knee joint (Taunton *et al*., 2003). However, this condition is also reported in athletes who do not perform tasks such as jumping or changing direction (Brunker and Khan, 2007:524). Patellar tendinopathy is most frequently seen in the athletic populations of basketball, volleyball, netball and high jump (Brunker and Khan, 2007:508) and running (Kountouris and Cook, 2007). It is these sports in which there is sudden maximal muscle tendon unit exertion, that place athletes at a significant risk to development of patellar tendinopathy (Peace and Healy, 2006).

Patellar tendinopathy often begins in the second decade and peaks in the third decade (Cook *et al.(*c*),* 2000). Amongst elite athletes, men are reported to have a higher prevalence of patellar tendinopathy (13.55%) compared to women (5.6%) (Lian *et al*., 2005). Unilateral patellar tendinopathy is more common in males with a ratio of 2:1 but males and females are equally affected with regard to bilateral patellar tendinopathy (Hyman, 2006).

2.6 PATHOAETIOLOGY

The exact pathoaetiological mechanism responsible for patellar tendinopathy is not clearly defined despite the increase in literature (Hamilton and Purdam 2004; Haraldsson *et al*., 2004; Peers and Lysens, 2005; Arnoczky *et al.*, 2007 and Kountouris and Cook, 2007). The pathology of patellar tendinopathy is largely considered at a histological level to be precipitated by tendon overload (Peers and Lysens, 2005) with resultant degeneration of the collagen matrix within the tendon (Taunton *et al.*, 2003). However a recent study has also hypothesised that overuse of tendon fibres is not responsible for the degenerative cascade of patellar tendinopathy but that it is a mechanobiological under-stimulation as a result of altered cell matrix interaction (Arnoczky *et al*., 2007).

The disruption that does take place histologically in a symptomatic patella tendon is known as mucoid degeneration (Brunker and Khan, 2007:524). These changes are most commonly seen at the bone tendon interface of the lower pole of the patella but can also occur in the main body of the tendon

(Panni *et al.*, 2000). Ultrasonography and Magnetic Resonance Imaging (MRI) have confirmed these changes clearly (Panni *et al.*, 2000) and include: disorganization of the normal tight collagen bundles, calcification, vascular in-growth (Peace and Healy, 2006), fibrinoid necrosis, pseudocytic changes and the absence of the 'blue line' that demarcates the transition from fibrocartilage to the mineralized fibrocartilage zone (Penn *et al.*, 2006). Clefts in the collagen and fibrinoid necrosis are suggestive of microtears within the patella tendon (Brunker and Khan, 2007:525). The increase in blood vessels and nerves (neovascularisation) is thought to play an important role in patellar tendinopathy. However, as a result of tendon pathology being present in asymptomatic individuals, the exact pathogenesis remains debatable (Peers and Lysens, 2005 and Kountouris and Cook, 2007).

According to Hamilton and Purdam (2004), numerous inconsistencies with regard to this degenerative model of patellar tendinopathy exist and have more recently proposed an adaptive model of response to differential forces within the patella tendon (Hamilton and Purdam, 2004). They postulate that a histological adaptation (at the proximal posterior aspect of the patella tendon) to compressive loading takes place at the expense of the tensile properties of that tendon (Hamilton and Purdam, 2004).

Although patellar tendinopathy has also been reported in non-athletic populations (Kountouris and Cook, 2007), review of the literature for this dissertation did not reveal a proposed pathoaetiological mechanism for this population group. However, the hypothesis that mechanobiological under-stimulation and not overuse leads to patellar tendinopathy (Arnoczky *et al*., 2007) may offer a possible explanation as to its pathoaetiological process.

2.7 CLINICAL FEATURES

The typical presentation of patellar tendinopathy is that of anterior knee pain with associated tenderness at the inferior patella pole (Panni *et al.*, 2000 and Stasinopoulos and Stasinopoulos, 2004). The most common site affected is the deep attachment of the patella tendon to the inferior patella pole (Brunker

and Khan, 2002:481 and Kountouris and Cook, 2007). Occasionally tenderness is experienced at the distal attachment of the tendon to the tibial tuberosity and rarely within the midsubstance of the tendon (Brunker and Khan, 2007:525).

Pain is usually gradual in onset (Brunker and Khan, 2002:481) and can disappear whilst exercising during the early stages (Penn *et al.*, 2006). Onset of pain is often associated with an increased frequency of training sessions in the athlete (Khan *et al*., 1998 and Kountouris and Cook, 2007). Activities such as running and walking up or downstairs can also cause pain (Khan *et al.*, 1998 and Kjaer, 2003:598) and can be exacerbated by prolonged knee flexion (Shamus and Shamus, 2001:98 and Peers and Lysens, 2005). Symptoms are typically provoked by jumping, hopping and bounding (Brunker and Khan, 2002:481). However, any activity that places an eccentric load on the knee (Penn *et al.*, 2006) can cause discomfort (Penn *et al.*, 2006). As the condition progresses, pain may be present before and during participation and may eventually become severe as to stop participation (Shamus and Shamus, 2001:97-98 and Kountouris and Cook, 2007). In the later stages, symptoms may be produced through simple daily activities such as walking and in the athlete participation may be significantly limited (Penn *et al.*, 2006 and Kountouris and Cook, 2007) and could progress to retirement of sport (Kountouris and Cook, 2007). Occasionally pain is experienced at night or during periods of rest (Cook *et al.* (b), 2000).

2.7.1 The Blazina Scale

Patellar tendinopathy has traditionally and clinically been graded on the Blazina Scale (McConnell and Cook, 2007). This scale is empirically based on and takes into account pain as related to activity (Brunker and Khan, 2002:481). Clinically this condition progresses in the following four series of stages (Lian *et al*., 2003):

Stage 1	Pain at the infrapatellar region after practice or after an event.
Stage 2	Pain at the beginning of the activity, disappearing after warm up and reappearing after completion of activity.
Stage 3a	Pain during and after activity, but the patient is able to participate in sports at the same level.
Stage 3b	Pain during and after activity and the patient is unable to participate in sports at the same level.
Stage 4	Complete rupture of the patella tendon.

2.8 DIFFERENTIAL DIAGNOSIS

Anterior knee pain is one of the most common presenting symptoms in clinical sports medicine practice (McConnell and Cook, 2007). The main imitators of patellar disease are: patellofemoral pain syndrome, Hoffa fat pad disease, superolateral fat pad impingement syndrome and infrapatellar plica injury.

2.8.1 Patellofemoral Pain Syndrome:

Patellofemoral pain syndrome is the most prevalent knee disorder seen in clinical practice (Lesher *et al.*, 2006) and is therefore one of the most important differentials to consider when a patient presents with a gradual onset of anterior knee pain (Khan *et al.*, 1998; Cook *et al. (*a*)*, 2000 and Peers and Lysens, 2005). Cases of patellar tendinopathy and patellofemoral pain syndrome can coexist and altered biomechanics of the patellofemoral joint can increase mechanical strain on the patella tendon (Brunker and Khan, 2002:481). However the two tend to differ with regards to onset of pain and related activity. Pain that occurs at the start of activity and disappears after warm up and returns after activity (or the next morning) is more likely to be a patellar tendinopathy. Pain that gradually worsens during activity is more likely to be patellofemoral in origin (McConnell and Cook, 2007). In the majority of cases, the two conditions are difficult to differentiate (Cook *et al. (*a*)*, 2000).

2.8.2 Hoffa Fat Pad Disease:

Hoffa fat pad disease can occur idiopathically or after trauma to the anterior knee but can be reliably diagnosed by findings with MRI (Peace and Healy, 2006) and is considered a very important differential diagnosis for patellar tendinopathy (Peers and Lysens, 2005). A history of trauma (single or recurrent) to the knee may distinguish this clinically from patellar tendinopathy. Idiopathic forms may warrant the use of MRI (Peace and Healy*,* 2006).

2.8.3 Superolateral Fat Pad Impingement Syndrome:

This syndrome presents with localized tenderness at the lateral side of the inferior pole of the patella and is responsible for chronic anterolateral knee pain in an athletic population (Peace and Healy, 2006). In contrast, patellar tendinopathy classically presents with anterior knee pain (Almekinders *et al*., 2002 and Brunker and Khan, 2007:5).

2.8.4 Infrapatellar Plica Injury:

The infrapatellar plica connects the lower pole of the patella to the intercondylar notch or anterior cruciate ligament and though once considered as an incidental finding, is now a recognised cause of anterior knee pain. MRI is useful in diagnosis and reveals a thickened and haemorrhagic patella tendon (Peace and Healy, 2006).

2.8.5 Other conditions that need to be excluded include (Dutton, 2004:757):

2.8.5.1 Meniscal Tears of the Anterior Horns:

In the athlete the onset of symptoms is sudden with pain felt deep within the knee with the injury usually occurring with the knee in a semi flexed and twisted position (Norris, 2004:239 and Goldblatt and Smith, 2005).

2.8.5.2 Osteoarthritis of the Knee:

Osteoarthritis of the knee presents with instability to the knee joint and the knee can be swollen and enlarged (Vizniak and Carnes, 2004:289). Swelling and instability are not common features of patellar tendinopathy (Brunker and Khan, 2007:508).

2.8.5.3 Osteochondritis Desiccans (O.C.D)

This avascular necrosis of subcondral bone occurs more commonly in a younger age group (10-20 years of age) (Vizniak and Carnes, 2004:177) as opposed to patellar tendinopathy which usually only begins in the second decade, usually peaking in the third decade (Cook *et al.* (c)*,* 2000). In contrast to patellar tendinopathy the O.C.D. patient often presents with a chronic history of knee pain, joint locking and recurrent effusions (Vizniak and Carnes, 2004:177).

2.8.5.4 Prepatellar Bursitis

Inflammation of this bursa often occurs in people who are constantly kneeling and presents with a large 'goose egg' swelling on the anterior knee (Vizniak and Carnes, 2004:173).

2.9 CONTRIBUTING FACTORS

Patellar tendinopathy appears to result from multiple intrinsic and extrinsic risk factors (Kjaer, 2003:595 and Gaida *et al*., 2004).

Intrinsic risk factors can contribute to uneven load distribution across the patella tendon (Rees *et al.*, 2006 and Crossley *et al.,* 2007) and those implicated in this susceptibility of the patella tendon, include: an increased Q angle, genu valgum, genu recurvatum, biomechanical alterations of the knee extensor mechanism (Peers and Lysens, 2005 and Norris, 2004:246), patella baja (Hyman, 2006), pes planus, anteversion of the femoral neck, coxa vara, patellar instability, incorrect patella tracking (Peers and Lysens, 2005 and Depalma and Perkins, 2004) and a rigid cavus foot (Shamus and Shamus, 2001:98). Anatomical variants in the hip, foot and ankle joints are considered, in general, to act as possible contributing factors of patellar tendinopathy (Brunker and Khan, 2007:529).

Hamstring tightness (Brunker and Khan, 2007:52) and poor quadriceps femoris muscle flexibility are the only biomechanical factors prospectively linked to patellar tendinopathy (Kountouris and Cook, 2007 and Hyman, 2006). Literature indicates that muscular imbalance or insufficiency can predispose an athlete to patellar tendinopathy (Witvrouw *et al.*, 2001) and weakness of the quadriceps femoris and hamstring muscles can restrict range of motion and increase load on the anterior knee (Depalma and Perkins, 2004).

Extrinsic factors in the development of patellar tendinopathy are more clearly defined (Warden and Brunker, 2003:745) and include: the intensity and frequency of training, footwear and type of training surface (Norris, 2004:246). Hardness of the training surface, especially with regard to volleyball players, is considered an important extrinsic factor (Shamus and Shamus, 2001:96) in the development of patellar tendinopathy.

The mechanism behind which extrinsic and intrinsic factors combine in causing patellar tendinopathy is not well established but is postulated that pathological changes are initiated by changes within the collagen matrix (Warden and Brunker, 2003:746).

2.10 DIAGNOSIS

The criteria that support the diagnosis of patellar tendinopathy are considered restrictive and poorly documented by some authors (Cook and Khan, 2001). Although patellar tendinopathy does not have any pathognomic symptoms (Taunton *et al.,* 2003), tenderness to palpation is always present in patients with patellar tendinopathy (Khan *et al.*, 1998; Cook *et al.*, 2001 and Peers and Lysens, 2005) and it is considered the classic physical examination technique for the detection of patellar tendinopathy (Cook *et al.* (b), 2000). However, such findings during physical examination can be minimal and subtle, as well as dependent on the stage of the condition (Shamus and Shamus, 2001:98). In some cases it may be necessary to perform eccentric activity such as a drop squat or hop to reproduce the pain (Brunker and Khan, 2002:481).

Common symptoms and findings that aid in diagnosis include: the absence of an acute injury to the knee, pain described as a dull ache that has progressed in intensity over a period of time; guarding of the knee during daily activities; modification of sporting technique and pain after lengthy periods of sitting (Penn *et al.*, 2006).

Lian (2007) stated that in addition to the above mentioned clinical findings, the diagnosis of patellar tendinopathy must be supported with and evidenced by structural changes in the patella tendon as per MRI, ultrasound or tendon biopsy (Peers and Lysens, 2005 and Lian 2007:vii). Sonography is considered the investigation of choice in patellar tendinopathy and MRI can be helpful in detecting any conditions that mimic patellar tendinopathy (Peace and Healy, 2006).

The following are common symptoms used to make a clinical diagnosis of patellar tendinopathy (Panni *et al.*, 2000; Shamus and Shamus, 2001:98; Brunker and Khan, 2002:481 and Kjaer, 2003:598):

i. Pain to be located over the inferior pole of the patella and to be gradual/insidious in onset.
ii. Tenderness at the inferior pole of the patella upon palpation.
iii. Symptoms are aggravated / provoked by jumping, hopping and / or bounding as well as prolonged knee flexion.
iv. Pain is required to be present on/during running or walking downstairs.

2.11 TREATMENT

Patellar tendinopathy has a tendency to be recurrent and chronic in nature (Peterson and Renström, 2003:42, Vizniak and Carnes, 2004:176 and Kountouris and Cook, 2007) and is important that the patient be informed that chronic cases of patellar tendinopathy may require lengthy periods of treatment (Brunker and Khan, 2007:527). Patients presenting for the first time with patellar tendinopathy can take between three to six months to recover

and those presenting with a chronic history may take up to six months to one year to return, recurrence free, to sport (Khan *et al.,* 1988 and Brunker and Khan, 2007:528).

There are numerous treatments available for the management of tendon disorders but few are considered to have a strong evidence base (Peers and Lysens, 2005 and Rees *et al.*, 2006). Although several conservative treatments are available in the treatment of patellar tendinopathy few studies have investigated the efficacy of such treatments (Purdam *et al.*, 2004 and Peers and Lysens, 2005). Treatment protocols vary widely between physicians and even countries (Peers and Lysens, 2005). The effects of conservative treatment methods on patellar tendinopathy are not thoroughly understood (Kjaer, 2003:595) but soft tissue techniques are considered an evidence based form of therapy for tendinopathies (Brunker and Khan, 2007:23).

Management within the first six months is largely conservative in nature (Peace and Healy*,* 2006) and can include the following: cryotherapy, soft tissue therapy, load reduction, correction of faulty biomechanics, eccentric strengthening programmes (Brunker and Khan, 2007:527-531). Of these conservative interventions, some investigators believe that the most effective treatment is a strengthening programme of eccentric knee squat exercises. This is supported by Kongsgaad *et al.,* (2006) who reported excellent results in their study when eccentric squats were performed on a twenty-five degree decline board. Surgery is only considered when the outcomes of conservative management are found ineffectual (Peers and Lysens, 2005 and Peace and Healy*,* 2006).

2.11.1 Deep Transverse Friction Massage

According to Brunker and Khan (2002) the most effective treatment for patellar tendinopathy (Brunker and Khan, 2002:487) is DTFM. This widely used manual form of soft tissue massage is considered a specific manipulative technique and was pioneered by Cyriax in 1941

(Norris, 2004:64). DTFM is reported to have the following beneficial effects: treatment induced hyperaemia (Hammer, 1995 and Norris, 2004:38), therapeutic movement, increased tissue perfusion and mechanoreceptor stimulation (Hammer, 1995). One study found that soft tissue mobilization increases fibrobroblastic recruitment and promotes healing in mammalian rat tendons (Gehlsen *et al*., 1999 and Perle, 2003).

It is theorised that scar tissue is formed in an irregular pattern, which decreases flexibility of tissues and acts as a nociceptive foci (Vizniak, 2005:489). DTFM acts by decreasing collagen cross linking which causes the softening of this scar tissue and the breakdown of adhesions, thereby promoting the realignment of the disrupted connective tissue (collagen) fibrils within the affected tendon (Perle, 2003; Vizniak, 2005:489 and Stasinopoulos and Johnson, 2007). Although commonly acknowledged that DTFM has an analgesic effect, the exact reason for this is still unknown (Stasinopoulos and Johnson, 2007).

2.11.2 Graston Instrument Assisted Soft Tissue Mobilization

GIASTM consists of six specially designed stainless steel instruments with bevelled edges and is an advanced form of soft tissue mobilization used both in detecting and releasing scar tissue adhesions and fascial restrictions (Carey, 2003:7). Each instrument is shaped and sized to work on a specific body part (Perle, 2003). GIASTM bases both method and outcome on scientific literature, which describes the effects of manual loading on soft tissue (Perle, 2003 and Hammer, 2004). The literature takes into account the important finding of fibroblast stimulation and the resultant synthesis of proteoglycan as a result of the soft tissue stimulation by these instruments (Perle, 2003 and Hammer, 2004). It has been hypothesised that repeat gliding of the GIASTM instrument over the patella tendon is in itself a form of intermittent loading (Perle, 2003) and that the controlled micro trauma created by these instruments results in micro vascular trauma and

capillary haemorrhage. This in turn creates a localised inflammatory response that stimulates the body's reparative system (Perle, 2003; Hammer, 2004 and Hammer and Pfefer, 2005). It is suggested that GIASTM enhances the proliferation of extra cellular matrix fibroblasts, improves ion transport and decreases cell matrix adhesions (Perle, 2003 and Howitt *et al.,* 2006 from Hammer and Pfefer, 2005).

2.11.3 DTFM AND GIASTM

Although both GIASTM and DTFM are applied differently they produce, through pressure and touch, a mechanical loading of the tendon tissue. It is believed that the subsequent vibrations and rapid changes produced by this loading stimulate mechanoreceptors in the tissues overlying the patella tendon (Rees, 2006). These effects of mechanical loading can be explained in terms of the Gate Control Theory of Pain (Melzack and Wall, 1965:971-979). This theory suggests that pain is not a direct result of stimulation of nociceptors but is rather as a result of modulation in the spinal cord through the interaction of intrinsic neurons and controls emanating from the brain (Dickenson, 2002 and Omoigui, 2007). Large diameter sensory neurons (Type A beta fibres) are stimulated by temperature, touch and pressure (Dickenson, 2002 and Omoigui, 2007). As a result of the increased input by pressure and touch to these large neurons into the substantia gelatinosa, the 'gate' is closed resulting in inhibition of the smaller diameter Type C fibres by the large Type A fibres. It is therefore hypothesised that DTFM and GTIASM may be able to modify pain signals by increasing input into the substantia gelatinosa according to the Gate Control Theory of Pain. This, in terms of the mechanical loading effect of GIASTM and DTFM, causes a decreased viscosity in the proteoglycan gel surrounding the collagen fibres as a result of this autonomic nervous system stimulation (Melzack and Wall, 1965:971-979 and Dickenson, 2002).

A new theory has been proposed by Omoigui (2007). This theory hypothesises that every pain syndrome can be explained in terms of a

unifying law of pain. This theory proposes that the origin of all pain is, at any given level is an inflammatory response. Although it is well established that prostaglandin mediated inflammation is not present on histological examination in patellar tendinopathy (Brunker and Khan, 2007:524 and Kountouris and Cook, 2007), neurogenic inflammatory markers may be present (Kountouris and Cook, 2007 and Omoigui, 2007). DTFM and GIASTM cause structural damage via application, which results in a localised prostaglandin inflammatory response (Hammer, 2004 and Omoigui, 2007). This theory of inflammation (Omoigui, 2007) therefore can be used to explain outcomes of pain in this study.

2.12 CONCLUSION

GIASTM is proposed to release scar tissue, adhesions and fascial restrictions in a similar way to DTFM. The purpose of this study is to investigate the relative effectiveness of GIASTM versus DTFM in treating patellar tendinopathy.

CHAPTER THREE

3.1 INTRODUCTION

This chapter focuses on the design of the study, the sampling procedure, the interventions that were used, the data collected as well as the statistical analysis of this data.

This study was conducted in order to determine the relative effectiveness of GIASTM versus DTFM in treating patellar tendinopathy. The study sample was divided into two groups, the GIASTM group and the DTFM group.

3.2 STUDY DESIGN

This research study was approved by the Faculty of Health Sciences Research and Ethics Committee prior to the study being commenced in 2006. The Institutional Review Board (IRB) approval as received from this committee falls in line with the requirements of the Declaration of Helsinki 1975.

3.2.1 Advertising and patient recruitment

Patients included in this study, were selected from those who responded to advertisements (Appendix 11) placed in public places, sports organizations, social clubs in the greater eThekwini metropolitan areas and on notice boards at the Durban University of Technology. Advertising was also via word of mouth and pamphlet distribution to the greater eThekwini metropolitan areas.

Respondent screening began with a telephonic interview to establish relevant suitability. Criteria for patient suitability were:

1. Respondents must have been between 25-45 years of age.

2. Respondents must have experienced symptoms for longer than four weeks in duration. No acute patients (symptoms less than four weeks) were accepted into this study due to the fact that symptoms of patellar tendinopathy tend to be insidious in nature (Brunker and Khan, 2002:481 and Magee, 2002:5).

3. Pain had to have been located below the knee in the region of the patella tendon (Fredburg and Bolvig, 2002).

4. The respondent could not have had any previous knee surgery.

5. The respondent could not have been taking medication or have received treatment for their symptoms. If they wanted to continue with such treatment, they were excluded from this study. If they wanted to be included in this study they had to undergo a wash out period, which was 48 hours for any non chronic medicinal intervention and 3 months for any chronic medications (Poul *et al*., 1993 and Seth, 1999).

6. The respondent could not have experienced overt trauma to the knee.

3.2.2 SAMPLING TECHNIQUE AND SIZE

The study was limited to patients who fulfilled the inclusion criteria. A non- probability sampling technique (stats.oecd.org/glossary/detail.asp, 2007) was used. Once patients were accepted into the study, the patients were allocated to groups utilising a randomisation table that was drawn up prior to the research commencement, which allocated all even numbers to one group and all odd numbers to the other group. In all, patients were allocated to one of the two treatment groups.

If patients presented with 2 symptomatic knees that fulfilled the criteria for the study and were diagnosed with patellar tendinopathy, then one knee was allocated to one group and the other knee to the other group as both treatments were active interventions.

3.2.3 PATIENT PROCEDURE

Patients that were suitable for the study post the telephonic screening, were asked to come into the Chiropractic Day Clinic in order for further patient screening to take place. In order to ascertain whether the patients met all the inclusion criteria, a full case history (Appendix 3), physical examination (Appendix 4) and regional knee examination (Appendix 5) were conducted. Once suitability was established, the patients were given a letter of information to read (Appendix 1) and an informed consent form (Appendix 2) to sign, in order to facilitate informed inclusion into the study and to allow the patients to understand treatment protocols as set out in this study.

If a respondent was excluded from the study based on the inclusion and exclusion criteria presented below, the patient was referred for out patient treatment at the Chiropractic Day Clinic.

3.2.4 INCLUSION AND EXCLUSION CRITERIA

3.2.4.1 The following inclusion criteria were used in the study:

Patients had to have been between 25 and 45 years of age. Although no particular age predilection has been reported for patellar tendinopathy, it was noted in a two year prospective study that “jumper’s knee” was more common in skeletally mature patients (Witvrouw *et al.*, 2001). Skeletal maturity generally occurs after 24 years of age according to Taylor and Resnick (2000:543). On the other end of the age spectrum, it has been noted that after the age of 45 years, it is common to note degenerative changes

take place in the knee (Laux, 2007). These degenerative changes often present with a similar pain distribution and other clinical characteristics, which could lead either to a misdiagnosis or alternatively complicate the natural history of a concurrent patellar tendinopathy. Therefore the upper age limit was set at 45 years to limit the ability of extraneous variables related to degeneration in affecting the outcomes of this study.

- Signed informed consent was required and obtained from the patient when they agreed to and signed the informed consent form (Appendix 2).

- Both females and males were accepted into the study.

- All race groups were accepted into the study.

- Patients were required to meet the criteria for the diagnosis of patellar tendinopathy. This was based on the following:

    i. Pain to be located over the inferior pole of the patella (Brunker and Khan, 2002:481) and to be gradual / insidious in onset.
    ii. Tenderness at the inferior pole of the patella upon palpation (Panni *et al.*, 2002) was required to be present.
    iii. Symptoms had to be aggravated by jumping or hopping (Brunker and Khan, 2002:481) as well as extended periods of knee flexion (Shamus and Shamus, 2001:98).
    iv. Pain was required to be present during running or walking downstairs (Kjaer, 2003:598).

Clinically patellar tendinopathy can be divided into 4 stages, per the Blazina Scale (2.7.1) and for the purposes of this study the patient was required to fall within stages 1- 3b (Lian *et al.*, 2003 and McConnell and Cook, 2007):

Stage 1	Pain at the infrapatellar region after practice or after an event.
Stage 2	Pain at the beginning of the activity, disappearing after warm up and reappearing after completion of activity.
Stage 3a	Pain during and after activity, but the patient is able to participate in sports at the same level.
Stage 3b	Pain during and after activity and the patient is unable to participate in sports at the same level.
Stage 4	Complete rupture of the patella tendon

3.2.4.2 The following exclusion criteria were used in the study:

Patients with contraindications to the treatment modalities

DTFM:

These contraindications included but were not limited to open wounds, muscle ruptures, tendon ruptures, contusions, burns, fractures, periostitis, rheumatoid arthritis, gout, bursitis, myositis ossificans, skin infections, thrombosis, bleeding disorders and/or tumours (Hertling and Kessler 1996, 139).

GIASTM:

These contraindications included but were not limited to absolute contraindications such as open wounds, unhealed fractures, thrombophlebitis, uncontrolled hypertension, kidney dysfunction, patient hypersensitivity, haematoma formations, osteomyelitis and/or myositis ossificans (Carey, 2003:1 and Perle, 2003).

Relative contraindications such as anticoagulants, cancer, varicose veins, burns, scars, acute inflammatory conditions, rheumatoid arthritis, pregnancy and other ligamentous laxity inducing syndromes and / or osteoporosis (Carey, 2003:1 and Perle, 2003).

- Patients who were receiving any other form of manual or medicinal therapy during the treatment period (Poul *et al*., 1993 and Seth, 1999).

- Patients who had sero negative or positive spondyloathropathies (Dutton, 2004:757) as such syndromes could complicate treatment and /or result in misleading results in this type of research. For similar reasons, patients who had signs and symptoms that indicated possible cancers / malignancies (Dutton, 2004:757) and / or Brodies abscesses (Taylor and Resnick, 2000:657), were excluded from the study.

- Patients with patellofemoral syndrome (Dutton, 2004:757) were excluded from this study as this syndrome is one of the most prevalent knee conditions seen in clinical practice (Lesher *et al.*, 2006). Studies show that patellar tendinopathy and patellofemoral pain syndrome coexist (Brunker and Khan, 2002:481) and therefore could confound the clinical presentation and treatment outcomes.

- Respondents diagnosed with Superolateral Fat Pad Impingement Syndrome were excluded from this study. This syndrome can be confused with patellar tendinopathy (Peace and Healy, 2006) and the possible confounding effects had to be eliminated.

- Respondents who had previous surgery to their knee. The internal scar tissue from surgery may alter the biomechanics of the knee joint and as a result complicate or alter treatment of patellar tendinopathy (Rippey, 1994:163 and Haraldsson *et al*., 2004).

- Respondents diagnosed with ligamentous laxity syndromes. Such syndromes alter normal knee biomechanics and could interfere with the outcomes of the treatment (Dutton, 2004:757)

- Patients not willing to sign the indemnity form (Appendix 13) or if the permission to be treated (PTT) section of relevant paperwork (Appendix 6) was not signed by the consulting clinician. As both these scenarios would be in contravention of the IRB ethics approval and the Declaration of Helsinki 1975.

- Patients changing their daily routine as well as training schedule were excluded from this study. Total rest is contraindicated during the repair stage of healing (Khan *et* al., 1998 and Norris, 2004:38), therefore patients in this study were asked to carry on with their everyday activities and training schedules (Stasinopoulos and Stasinopoulos, 2004). This was done in order to negate the influence that an extended period of rest, with subsequent skeletal muscle atrophy, may have on the patella tendons' susceptibility to further damage (Plaugher, 1993:489-495).

3.3 SAMPLE SELECTION AND GROUP ALLOCATION

The selected patients were allocated into one of two groups. All even numbered patients were allocated to group 1 and all odd numbered patients were allocated to group 2. Group 1 received GIASTM and group 2 received DTFM.

3.4 TREATMENT APPLICATION AND FREQUENCY

3.4.1. GIASTM

Patients allocated to Group 1 received GIASTM. The GIASTM tool number 3 was used in this group (Appendix 7).

The patient lay in the supine position, with the affected leg extended. A towel was placed under their affected knee so as do induce slight flexion of the patella tendon. The instrument was passed over the painful area in multiple directions (usually five or six) to find the usual two or three directions that created the abnormal barrier sensation as defined by Perle (2003) and Hammer (2003). Once the lesion was located, the instrument was moved in a general longitudinal direction, over the patient's lubricated skin (Perle, 2003). The instrument was then held at 30°-60° angles so as to achieve maximum separation of collagen fibres (Carey, 2003:27) and pressure was applied whilst the instrument was passed over the abnormal barrier sensation. The application period was 5 minutes per treatment session.

Ideally treatment duration is 4 to 8 sessions (Carey, 2003:28). However chronic conditions tend to require more treatment sessions, therefore patients received 12 treatments.

3.4.2 DTFM

Patients allocated to Group 2 received DTFM. This procedure was applied transversely across the fibres (Cyriax and Cyriax, 1993:21) for ten minutes. The patient lay in the supine position with the affected leg extended and a small towel placed under the knee so as to induce slight flexion of the knee joint. Short back and forth massage strokes were applied across the tendon with a reinforced index finger (Vizniak, 2005:489). Substantial pressure with the reinforced fingertips was applied to the tendon (Cyriax and Cyriax, 1993:21 and Bloomfield, 1995:252).

It was noted from the literature that six to twelve treatments are normally necessary to achieve maximum clinical benefit (Ombregt *et al.*, 1996). In this study twelve treatments of DTFM were given.

3.4.3 Treatment frequency

Based on the recommended recovery time for patellar tendinopathy (Cook *et al. (*b*),* 2000), the indications for GIASTM (Carey, 2003:10) as well as the indications for DTFM (Perle, 2003; Vizniak, 2005:489 and Stasinopoulos and Johnson, 2007); patients received two treatments per week for four weeks (month one) and one treatment per week for four weeks (month two). This enabled patients to receive 12 treatments in total. A two week and one month follow up took place after treatment twelve. The total duration of the study was therefore over three months.

3.5 MEASUREMENT TOOLS AND MEASUREMENT FREQUENCY

Subjective measures were recorded using:

- Subjective Knee Score Questionnaire (Appendix 11)
- VISA Questionnaire (Appendix 12)
- The NRS (Appendix 9)

The Objective measures included the:

- Algometer (Appendix 8A) and
- Inclinometer (Appendix 8B)

Both the subjective and objective measures were recorded prior to treatment one, prior to treatment nine, prior to visit thirteen, at two week follow up (visit thirteen) and at one month follow up (visit fourteen). However, NRS1 was recorded at every visit, establishing a total of fourteen readings for this outcome.

3.5.1 Objective Measures

3.5.1.1 Algometer measurements

Algometer (Appendix 8A) readings were taken prior to treatment one, prior to treatment nine, prior to two week follow up (visit 13) and prior to one month follow up (visit 14). These measurements were used to determine any changes in the patients' pain levels during the treatment period. The algometer used in this study was a compressive force gauge manufactured by Wagner Instruments: P.O. Box 1217, Greenwich, CT 06836, USA.

The algometer was used to determine pain threshold (threshold tenderness), the point where a sensation such as firm pressure begins to feel unpleasant and painful (Bonci, 2003 and Travell and Simons, 1997:12). The area of discomfort was located and the rubber tip of the algometer was placed on this area. Pressure was then slowly applied. The reading was taken at the point at which the patient was first aware of the sensation of pain or pressure (Bonci, 2003 and Travell and Simons, 1997:12).

3.5.1.2 Inclinometer measurements

This instrument was used to determine the flexibility of the hamstring and quadriceps femoris muscles. Flexibility testing was performed and measured by active hip flexion with the patient in the supine position and the hip flexed to ninety degrees (Livingston, 1992:57). Extension measurements were taken with the patient prone (Vizniak, 2005:509). The leg was extended in the neutral position, the inclinometer set to zero and the patient extended the leg maximally (Livingston, 1992:59). Previous research indicates that these measurements appear to be reliable when taken by the same examiner (Witvrouw, 2001).

3.6. SUBJECTIVE MEASUREMENTS

3.6.1 Numerical Pain Rating Scale

Pain intensity in the quantitative arena is most commonly measured using unidimensional scales such as the NRS (Bolton and Wilkinson, 1998). Two NRS scales were used in this study, NRS1 (Appendix 6) and NRS2 (Appendix 9). For NRS1, pain was assessed as worst and least on a scale of 0 to 10 at each visit, with a total of fourteen readings (NRS1). The NRS2 scale asked patients to mark the intensity of their pain on a 101 point scale, where 0 was no pain and 100 the worst pain possible.

3.6.2 The Subjective Knee Score Questionnaire

This questionnaire rates symptoms such as swelling, pain and stability and sport specific functions such as running, jumping and twisting. It was originally developed to be of benefit to the clinician with regard to monitoring patient progression, altering treatment choice and in making return to sport decisions (Yeomans, 2000:84).

3.6.3 The Victorian Institute of Sport Assessment (VISA) Questionnaire

The Victorian Institute of Sport Assessment (VISA) questionnaire is an index of severity of symptoms in patients with patellar tendinopathy. Visentini (1998) concluded the VISA score a reliable index of the severity for patellar tendinopathy.

This questionnaire assesses: symptoms; simple tests of function in daily activity and the ability to play sport (Visentini *et al.*, 1998 and Kountouris and Cook, 2007) and therefore reflects elements of both sporting success and symptomatic benefit (Coleman *et al.*, 2000). It also measures the domains of pain (Robinson *et al.*, 2001). Six of the eight questions were scored on a pain rating scale from zero to ten, where ten represented optimal health. The maximal VISA score for an asymptomatic patient was 100, with zero representing the minimum score possible (Visentini el al., 1998). The use of this questionnaire at regular time intervals is reported to determine relative effectiveness of treatment programmes. Intervals of at least one month are recommended in order to increase sensitivity to change over time (Kountouris and Cook, 2007).

3.7 STATISTICAL ANALYSIS

SPSS version 13.0 (SPSS Inc., Chicago, Illinois, USA) was used to analyse the data. A p value of <0.05 was considered as statistically significant.

Baseline demographics were compared between the two treatment groups using Pearson's chi square tests or Fisher's exact tests as appropriate for categorical variables, and student's t-test in the case of quantitative variables.

The VISA Questionnaire and the Subjective Knee Score Questionnaire were scored by summing up responses to all scored items at each time point. Both

were scored out of a maximum of 100 points with the lower the score the more severe the symptoms.

Part of the Subjective Knee Score Questionnaire which was not used in the total score was the stiffness, grinding and locking questions which were scored on a scale of 1-4 with higher scores indicating severe symptoms. These scores were summed together at each time point and analysed as “stiffness, grinding and locking score”.

Repeated measures ANOVA tests were used to examine the treatment effect in the GIASTM group compared with the DTFM group for all outcomes separately. A significant ($p<0.05$) time group interaction effect indicated a significant treatment effect. Profile plots were generated to examine the direction of the treatment effect or to assess the existence of trends.

CHAPTER FOUR

4.0 INTRODUCTION

Chapter four of this study describes the results of the data obtained and evaluates and interprets their meaning through discussion. The data was obtained prior to treatment one, after the eighth treatment, after the twelfth treatment, two weeks after visit twelve (two week follow up) and a month after visit twelve (one month follow up) in order to evaluate the following hypotheses:

The first null hypothesis

It was hypothesized that the use of GIASTM would be no more effective than DTFM in treating patellar tendinopathy in terms of objective findings.

The second null hypothesis

It was hypothesized that the use of GIASTM than would be no more effective than DTFM in treating patellar tendinopathy in terms of subjective findings.

To evaluate these two hypotheses, objective data was obtained from:

- Algometer readings
- Inclinometer readings

Subjective data was obtained from:

- Subjective Knee Score Questionnaire
- VISA Score Questionnaire
- Numerical Pain Rating Scale

KEY FOR CHAPTER FOUR:

N: Number of people in sample group

p: Probability that the null hypothesis is correct. The lower the value of p the greater the chance of rejection. If the p value is <0.05 the test is significant and the null hypothesis is rejected, with acceptance of the alternative hypothesis.

Sig: Significance

Std: Standard

T: Time reading. This is represented by numbers 1-14 on the graphs but within the written text, numbers 1-14 are preceded by the letter T, so as to read T1, T2 and so on.

T1: readings taken pre treatment one
T2: readings taken pre treatment nine
T3: readings taken pre visit thirteen
T4: readings taken two weeks after treatment twelve two week follow up)
T5: readings taken one month after treatment twelve (one month follow up)

T1-T14: Specific to NRS1 only (Figure 4); T1 represents treatment one, T2 represents treatment two and similarly for T3 - T12. T13 represents two week follow up and T14 represents one month follow up.

Y axis: Vertical axis of graph

X axis: Horizontal axis of graph

=	Equals
>	Greater than
<	Less than
Graston:	GIASTM
Cross friction massage:	DTFM
Mean:	The average of readings
Time (effect):	The effect over time irrespective of intervention
Time group (effect):	The effect of the intervention over time

4.1 DEMOGRAPHICS

4.1.1 Gender

Although patients were randomly assigned to the two treatment groups, no significant difference between groups (p=0.671) with regards to gender was found (reflected in Table 1).

Nevertheless it is of interest in this study to note that four times more men were affected by bilateral patellar tendinopathy than women but there were equal ratios with regard to unilateral patellar tendinopathy. Current literature states that males and females are equally affected by bilateral patellar tendinopathy, however, men are twice more likely to develop unilateral patellar tendinopathy compared to women (Hyman, 2006). Data from this study was therefore not in congruence with the literature.

Table 1: Cross tabulation of gender by treatment group

			GENDER		
			FEMALE	MALE	TOTAL
GROUP	DTFM	COUNT	5	7	12
		ROW %	41.7%	58.3%	100.0%
	GIASTM	COUNT	7	7	14
		ROW %	50.0%	50.0%	100.0%
TOTAL		COUNT	12	14	26
		ROW %	46.2%	53.8%	100.0%

Pearson's chi square =0.181, p=0.671

4.1.2 Race

There were no differences in racial percentages between treatment groups (p=0.738) this is shown in Table 2. This lack of difference between groups allows for comparison of treatment rather than the

effect that race might have on the outcomes of this study. Overall there were more Whites (42.3%) and Indians (46.2%) but a far lower percentage of Blacks (11.5%). This reflects inconsistencies with regard to the general population of Kwa Zulu Natal, South Africa, where there is a greater Black population compared to both the White and Indian populations (http://www.statssa.gov.za/census2001/digiAtlas/index.html, 2002 and http://mapserver.statssa.gov.za/profiles2006/index.aspx, 2007).

Reasons for the lower distribution of the black population within this study could have been due to the lack of understanding of the term 'Chiropractic' as utilised in the advert (Appendix 10) within this population and the fact that the advertisement for this study was not translated from English into Zulu (http://www.info.gov.za/aboutsa/education.htm, 2007).

Table 2: Cross tabulation of race by treatment group

			RACE			TOTAL
			BLACK	INDIAN	WHITE	
GROUP	DTFM	COUNT	1	5	6	12
		ROW %	8.3%	41.7%	50.0%	100.0%
	GIASTM	COUNT	2	7	5	14
		ROW %	14.3%	50.0%	35.7%	100.0%
TOTAL		COUNT	3	12	11	26
		ROW %	11.5%	46.2%	42.3%	100.0%

Pearson's chi square =0.607, p=0.738

4.1.3 Age

There was no difference in mean age between the two treatment groups (p=0.548). The mean ages of the two groups were very similar

(Table 3) which enables comparison within the groups, without the variances of age confounding the statistical analysis.

Patellar tendinopathy often begins in the second decade, peaking in the third decade (Cook *et al*. (c), 2000) and correlates with the data in this study in which the average age was 35. However, this does not correlate favourably with the statistics available at http://mapserver.statssa.gov.za/profiles2006/index.aspx (2007), in which the 20-24 year age group represents the greatest sector of the population within the eThekwini Metropolitan Municipality (Durban, South Africa), the municipality within which the Chiropractic Day Clinic is located.

Table 3: Comparison of mean age between the two treatment groups

	Group	N	Mean	Std. Deviation	Std. Error Mean	p value
AGE	GIASTM	14	34.36	6.308	1.686	0.548
	DTFM	12	36.00	7.459	2.153	

4.1.4 Sport

Patellar tendinopathy gives rise to considerable functional deficit and disability in professional as well as recreational sport (Khan *et al*., 1998; Stasinopoulos and Stasinopoulos, 2004 and Peers and Lysens, 2005). The level of participation in sport was equal in both groups (Table 4) with regard to professional versus recreational athletes. In terms of numbers, there was a difference in runners but this was not statistically significant. This implies the groups were homogenous (http://www.thefreedictionary.com/homogenous, 2007) and outcomes of this study were not dependent on the sport the patients participated in.

Sporting activities impose stresses high enough to cause fibre failure (Khan *et al.*, 1998) with tendon overloading occurring when a 3-8% strain is applied to the patella tendon (Peers and Lysens, 2005). Jumping activities are reported to impose considerable forces on the

knee extensor mechanism (Haraldsson *et al*., 2004) and forces up to 8kN are reported to occur within the patella tendon during landing from a jump (Khan *et al*., 1998). It is important to note that although research has indicated a particular affinity for elite jumping athletes (Peace and Healy, 2006), no jumping athletes, elite or recreational, participated in this study.

Table 4: Sport or Activity between treatment groups

	GIASTM	DTFM
Runners	3	6
Rugby players	1	1
Cyclists	2	0
Walkers	5	3
Tennis players	1	1
Dancers	0	1
Gym	1	0
No Sport	1	0

Summary of demographic data:

Although there were no significant differences between the groups with respect to the demographic factors noted, the demographic factors were in their presentation different to literature norms. Thus data could be compared between groups based on their homogeneity (Mouton, 1996:91) but comparison to the literature would be more difficult.

4.2 OBJECTIVE OUTCOMES

4.2.1 Algometer

It was found that the effect of GIASTM was not significantly different to DTFM for this outcome (p=0.145), although both groups showed significant changes over time (p<0.001) (Figure 1).

Table 5: Between and within subjects effects for Algometer

Effect	Statistic	p value
Time	Wilk's lambda=0.043	<0.001
Group	F=0.191	0.666
Time*group	Wilk's lambda=0.721	0.145

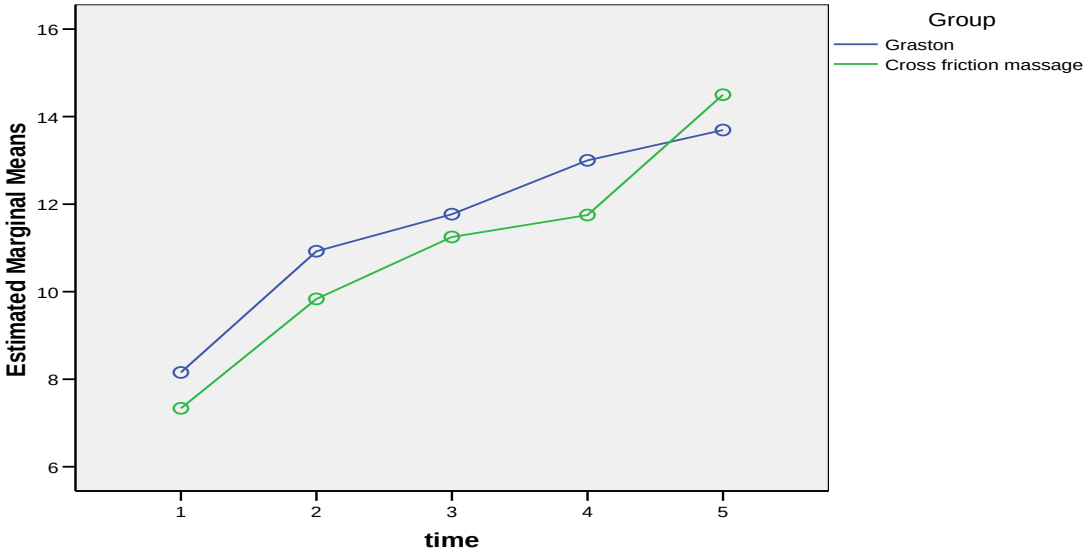

Figure 1: Profile plot of time of mean algometer measurement for each group

GTIASM bases its outcomes on the theory of mechanical loading on soft tissue and similarly one of the therapeutic effects of DTFM is adhesion removal (Hammer, 2004). When applied, DTFM and GTIASM cause a mechanical load on the tendon tissue and it is believed that the subsequent vibrations and rapid changes produced by this loading stimulates mechanoreceptors in the tissues overlying the patella tendon (Rees, 2006). This in turn causes a decreased viscosity in the proteoglycan gel surrounding the collagen fibres as a result of autonomic nervous system stimulation (Melzack and Wall, 1965:971-979 and Dickenson, 2002).

Thus when the patella tendon is exposed to trauma or damage is induced by treatment (treatment induced hyperaemia), the proteoglycan gel in the surrounding tissues increases in density and this in turn leads to an increased stimulation of nociceptors (Hammer, 1995).

Therefore, nociceptors present within tendons are stimulated not only by a chemical agent (i.e. as a result of the treatment induced inflammation) but also by mechanical means (Brunker and Khan, 2007:27). This theory of mechanical loading and stimulation of mechanoreceptors could be used to explain the significant decrease in tenderness over the treatment period for both groups especially with reference to the Gate Control Theory (Melzack and Wall, 1965:971-979 and Omoigui, 2007). This concurs with Brunker and Khan (2007:27) who indicate that mechanical loading stimulates the large Type A beta fibres and reduces the pain and tenderness perceived as conveyed through the smaller Type C fibres (Brunker and Khan, 2007:27).

4.2.2 INCLINOMETER

4.2.2.1 Flexion of hip

With respect to hip flexion neither group showed significant improvement over time (p=0.087) or significant treatment effect in respect of hip flexion (p=0.286). The profiles of the two groups showed only a very slight increase in hip flexion over time and the graphs were almost parallel over time, indicating that the effects of both treatments were the same. This could be related to the fact that younger individuals have inherently stiffer tendons than older individuals (Reeves *et al.,* 2003) which implies that the measurement tool used to measure improvement needs to be highly sensitive to small changes. The inclinometer used in this study only allowed for one decimal point readings and therefore limited its sensitivity to subtle changes as a result of the treatment. Therefore, future research needs to investigate the use of more sensitive measurement tools, with view to the fact that the range of hip flexion with the knee flexed is 120° –130° (Vizniak, 2005:231).

Nevertheless it is noted that decreased flexibility of the quadriceps femoris and hamstring muscles can potentially alter the range of motion of the knee joint thereby increasing the load on the patella tendon (Cook *et al*. (b), 2000). Concurrent hamstring tightness is a functional biomechanical abnormality positively linked with an increased prevalence of patellar tendinopathy (Cook *et al.* (a), 2000*).*

Therefore, the lack of significant inter group change indicates that both treatments are equally effective and that the improvement within the groups is not significantly different. This is congruent with literature that reports increased joint range of motion to be a positive restorative function of both DTFM and GIASTM (Carey, 2003:10 and Norris, 2004:68).

Table 6: Between and within subjects effects for Flexion

Effect	Statistic	p value
Time	Wilk's lambda=0.678	0.087
Group	F=1.192	0.286
Time*group	Wilk's lambda=0.851	0.497

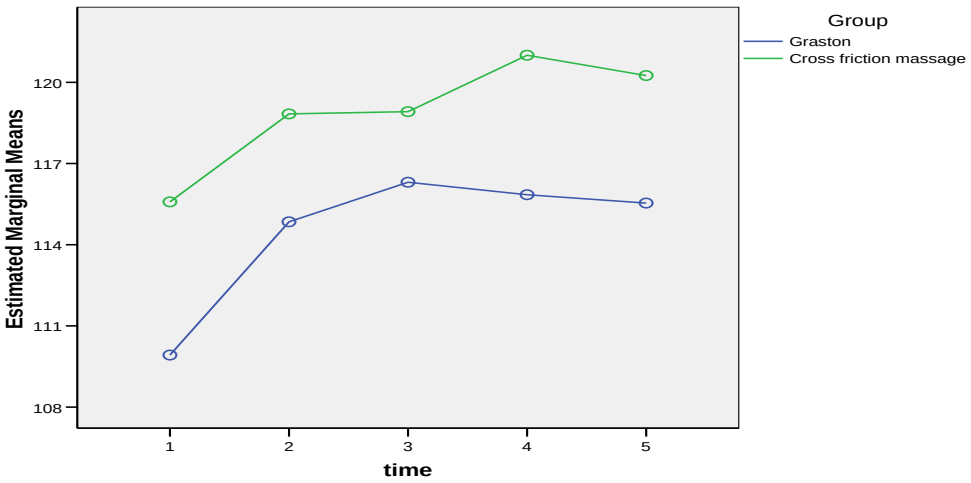

Figure 2: Profile plot of time of mean flexion by group

4.2.2.2 Extension of hip

Overall, hip extension in both groups showed no significant improvement over time (p=0.226) and no significant treatment effect (p=0.659). This result is similar to flexion of the hip for which there were no significant improvements with the exception that the improvement of hip flexion was greater than hip extension. The profiles of the two groups showed that the graphs were almost parallel over time until T4, thereafter they diverged slightly. Thus the effects of both treatments were the same for extension.

Between time intervals T4-T5 in the GIASTM group there is a regression of extension but this plateaus in the DTFM group. It has been reported that the GIASTM instruments allow an advantage to the clinicians' ability to penetrate fibres more deeply than DTFM (Carey, 2003:59 and Hammer and Pfefer, 2005), thus stimulating the level of fibroblastic activity to a greater degree (Perle, 2003). The abovementioned could possibly be used to explain this regression in extension in the GIASTM group. If the GIASTM tools allow for deeper penetration of affected collagen fibres then a more apparent change may have occurred in the beginning stages and is evidenced by time periods T1-T4 in Figure 3. However, such a fast change may not allow for normalisation of the quadriceps to hamstring ratio and subsequent strength changes in these muscle groups. Literature indicates that muscular imbalance or insufficiency can predispose an athlete to patellar tendinopathy (Witvrouw *et al.*, 2001) and weakness of the quadriceps femoris and hamstring muscles can restrict range of motion and increase load on the anterior knee (Depalma and Perkins, 2004). Furthermore, muscle strength in painful limbs is not restricted to one muscle group but seems to affect the whole limb and chronic pain is

significantly related to a reduction of muscle strength of 20-30 percent in a painful knee (Wilgen *et al*., 2003). As a result, more reliance may be placed on ligaments and tendons of the knee thereby increasing the susceptibility of the patella tendon to pathology, as evidenced by time periodsT4-T5 of Figure 3 in the GIASTM group.

Literature hypothesises that the posterior fibres of the patella tendon are subject to higher tensile strains than the anterior fibres in response to quadriceps tension (Basso *et al.*, 2001). With the knee in the flexed position, research has indicated that the posterior proximal tendon fibres are relatively shielded from stress, with low tensile or compressive strains (Almekinders *et al.,* 2002). Therefore in order to stress the posterior proximal tendon fibres, hip extension was performed with the knee in the extended position. This according to Almekinders *et al.,* (2002), would place more of a compressive strain on the posterior proximal portion of the patella tendon. This procedure would therefore increase the sensitivity of the findings showing a decrease in extension of the GIASTM group, particularly in view of the small range of hip extension (25° –35°) (Vizniak, 2005:230).

Few studies have examined the mechanical properties of the anterior and posterior portions of the patella tendon (Haraldsson *et al.,* 2004). Almekinders (2002) state that even though a 45 Newton increase in quadriceps load occurs during full knee extension, particularly on the posterior fibres of the patella tendon, results have suggested that this load is shared equally amongst anterior and posterior fibres of the patella tendon (Almekinders *et al.,* 2002 and Peers and Lysens, 2005:74).

Recovery rates of the components of the bone-tendon-bone complex of the patella tendon are also not equal (Daniel *et al*., 1990:348). The repair processes that take place at the insertion sites of the patella tendon (inferior pole of the patella and tibial tuberosity) are reported to be slower compared to the reparative processes that take place in the tendon substance/tendon proper (Daniel *et al*., 1990:348). Literature also reports that the remodelling phase of soft tissue can last between three weeks to twelve months (Vizniak and Carnes, 2004:306). The combination of a slow reparative rate at the insertional sites of the patella tendon and a remodelling phase that may last up to twelve months could account for the regression seen in time periods T4-T5 in Figure 3 of the GIASTM group.

It has been recommended that a programme of stretching and strengthening be incorporated together with GIASTM (Carey, 2003:2 and Perle, 2003) as strengthening encourages proper realignment of collagen fibres and promotes further release of scar tissue and develops stability within the musculoskeletal system (Carey, 2003:64 and Hammer and Pfefer, 2005). The results evidence the need for a stretching and strengthening programme to be included in patient care.

Table 7: Between and within subjects effects for Extension

Effect	**Statistic**	**p value**
Time	Wilk's lambda=0.763	0.226
Group	F=0.199	0.659
Time*group	Wilk's lambda=0.965	0.946

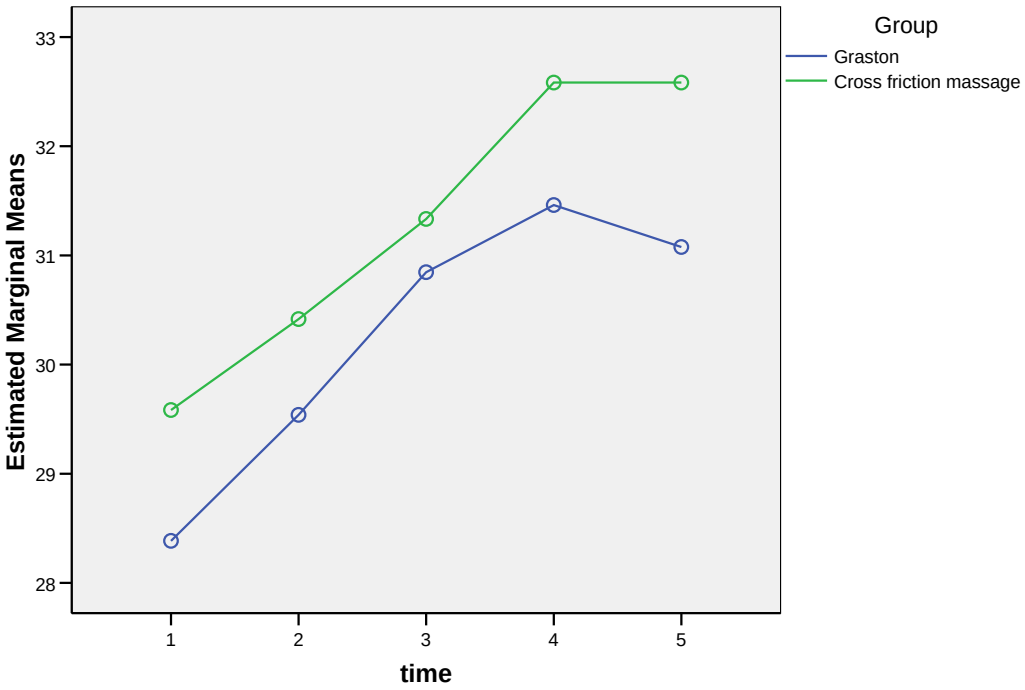

Figure 3: Profile plot of time of mean extension by group

4.3 SUBJECTIVE OUTCOMES

4.3.1 NRS1 and NRS2

It has been shown that consistent use of the same pain rating scale when reassessing a patient increases the consistency of results (Magee, 2002:5). For this reason and the fact that pain is subjective and manifests differently in individuals (Magee, 2002:3), the NRS was measured consistently throughout the study in two ways. For NRS1, pain was assessed at each visit with a total of fourteen readings. Pain was assessed as worst and least on a scale of 0 to 10. For NRS2, measures were recorded prior to treatment one, prior to treatment nine, prior to treatment thirteen, at two week follow up (visit thirteen) and at one month follow up (visit fourteen). Pain was assessed as worst and least on a scale of 0 to 100, with the average of the worst and least pain being used.

4.3.1.1 NRS1

NRS1 showed a significant time effect (p=0.002) but no difference in treatment effect between the groups (p=0.307) was noted. Figure 4 shows that the profiles of the two groups were very similar over time, both groups showed a decrease in pain to the same extent over the 14 visits.

Regression in treatment outcomes occurred at T5, T7 and T12 within the GIASTM group and within T5-T7 in the DTFM group (Figure 4). Literature states that the swelling that occurs as a result of induced inflammation occurs, after application of treatment (Hammer, 1995 and Norris, 2004:38) and the regression noted may therefore represent an expected delayed onset of swelling as a result of previous treatment applications.

When treatment is administered to patients the inflammation induced usually subsides within 24-48 hours, thereby accounting for improvement in outcomes after the regression periods of T5, T7 and T12 within the GIASTM group and T5-T7 within the DTFM group (Guyton, 1987:589-590). The continued improvement during T13 - T14 in both treatment groups' time periods (during which no treatment was given) also correlates with the mechanical theory of loading of soft tissues (Brunker and Khan, 2007:27) as discussed in section 4.2.1. As the patients' pain levels decreased, mobility would have increased and an increased activation of mechanoreceptors would have occurred, thereby improving the treatment outcome because of the decreased reported pain levels (Stojanovic and Abdi, 2002 and Omoigui, 2007).

Table 8: Between and within subjects effects for NRS1

Effect	Statistic	p value
Time	Wilk's lambda=0.075	0.002
Group	F=0.126	0.727
Time*group	Wilk's lambda=0.329	0.307

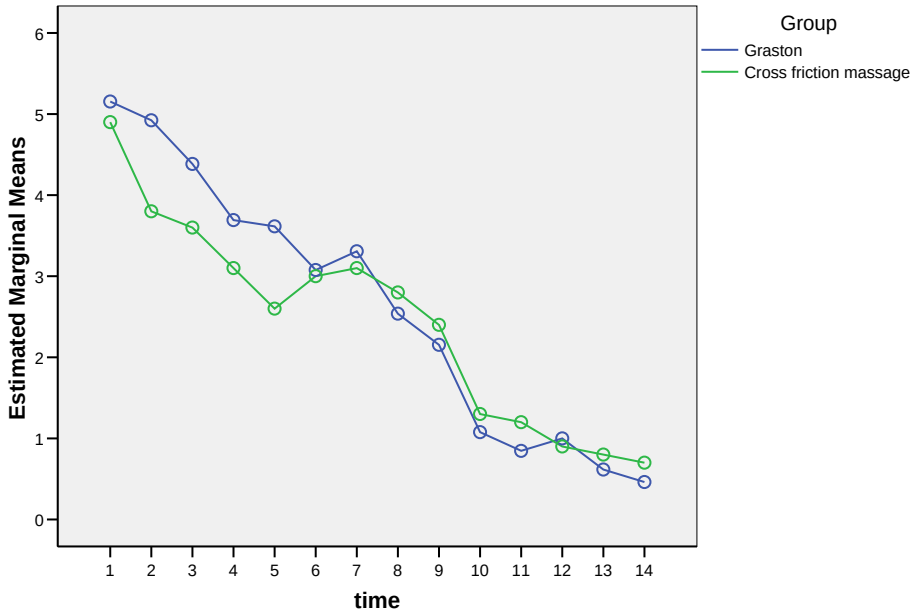

Figure 4: Profile plot of time of mean NRS 1 for each group

4.3.1.2 NRS2

Table 9 shows that NRS 2 also showed a highly significant time effect ($p<0.001$), but there was no evidence of a differential treatment effect ($p=0.998$) since both groups improved to the same extent and at the same rate over time. This is shown by the parallel profiles of the two groups in Figure 5.

The results of NRS2 are the same as NRS1 and would follow the explanation as per 4.3.1

Table 9: Between and within subjects effects for NRS2

Effect	Statistic	p value
Time	Wilk's lambda=0.180	<0.001
Group	F=0.152	0.700
Time*group	Wilk's lambda=0.994	0.998

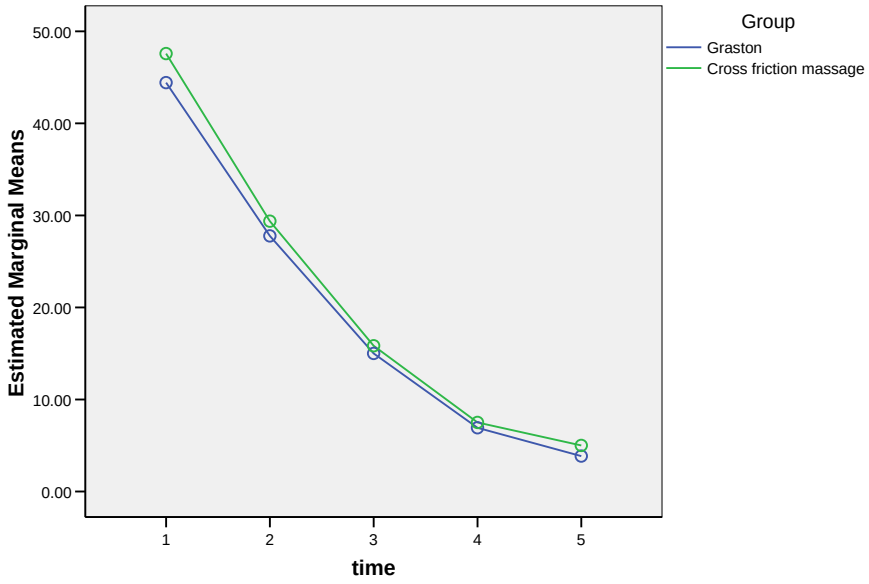

Figure 5: Profile plot of time of mean NRS 2 for each group

4.4 SUBJECTIVE KNEE SCORE QUESTIONNAIRE (Total)

This questionnaire rates symptoms such as swelling, pain and stability and sport specific functions such as running, jumping and twisting. Table 10 shows that the overall time effect in both groups was statistically significant ($p<0.001$), but that there was no difference in treatment effect between the groups ($p=0.518$). This means that both groups improved to the same extent over time, and Figure 6 shows that the profiles of the two groups were almost parallel. However, it does suggest a trend towards the GIASTM group because the profile shows a more linear and steeper increase over time compared to the DTFM group.

The possible reasons explaining the significant time effect in both groups will be discussed in section 4.4.1.

Table 10: Between and within subjects effects for Subjective Knee Score Questionnaire (Total)

Effect	Statistic	p value
Time	Wilk's lambda=0.235	<0.001
Group	F=0.789	0.384
Time*group	Wilk's lambda=0.857	0.518

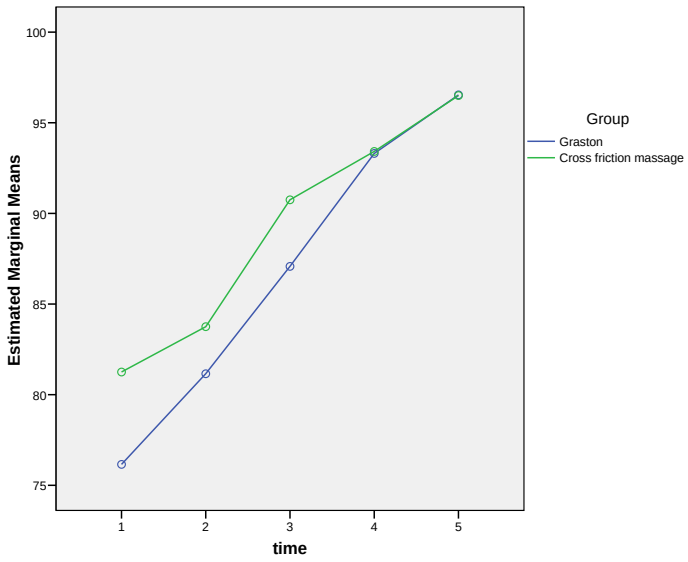

Figure 6: Profile plot of time of mean Subjective Knee Score Questionnaire (Total) by group

4.4.1 REPEATED MEASURES ANOVA ANALYSIS OF THE INDIVIDUAL QUESTIONNAIRE ITEMS

4.4.1.2 Subjective Knee Score Questionnaire

4.4.1.2.1 Pain in relation to activity

There was a highly significant time effect for both treatment groups overall ($p<0.001$). However, the treatment effect was not significant within groups ($p=0.400$) and between the groups as Figure 7 shows the slopes of the profiles of the two groups over time were almost parallel. This indicates that there was no difference in the outcome of pain in relation to activity with respect to treatment groups. These results could be explained in terms of the Gate Control Theory of Pain (Melzack and Wall, 1965:971-979 and Omoigui, 2007) and the mechanical theory of loading (Rees, 2006) (as per 4.2.1).

Table 11: Between and within subjects effects for pain

Effect	Statistic	p value
Time	Wilk's lambda=0.204	<0.001
Group	F=0.052	0.821
Time*group	Wilk's lambda=0.825	0.400

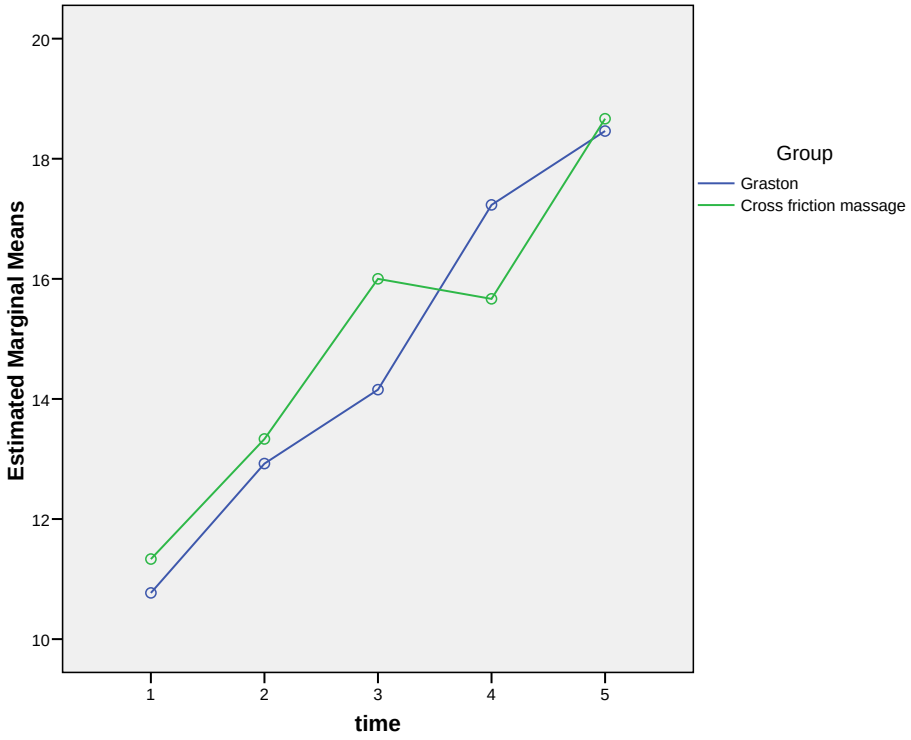

Figure 7: Profile plot of time of mean pain for each group

4.4.1.2.2 Intensity of pain

It is important to note that the numbers on the Y axis (estimated marginal means), are randomly allocated numbers and therefore do not represent any scale. However, they do serve the purpose of allowing comparison between the groups with respect to improvement or regression. Refer to Appendix 11.

For intensity of pain (Table 12), once more a significant time effect is demonstrated ($p<0.001$) but there was no evidence of a differential treatment effect in the two groups ($p=0.766$). Figure 8 shows that the rate of pain intensity was similar in both groups over time; with a general decrease until T 3, after which both groups increased once more. Usually if the frequency and

intensity of pain and/or symptoms increases this is an indicator that the condition is increasing in severity (Magee, 2002:5).

The highly significant time effect experienced in both groups could be due to the Mechanical Theory of Loading (Rees, 2006) and the Gate Control Theory (Melzack and Wall, 1965:971-979) (as previously discussed in 4.2.1).

The intensity of pain increased again in both groups between periods T3 to T4 and T4 to T5. It is important to note that these are the two time periods during which no treatments were given. Possibly if the patients had received treatment during this data collection period then the level of intensity may have continued to have decreased as patellar tendinopathy can take up to twelve months to heal (Visnes *et al*., 2005). It has been recommended that a programme of stretching and strengthening be incorporated together with GIASTM (Carey, 2003:2 and Perle, 2003) as strengthening encourages proper realignment of collagen fibres and promotes further release of scar tissue and develops stability within the musculoskeletal system (Carey, 2003:64 and Hammer and Pfefer, 2005).

The exclusion of a stretching and strengthening programme in this study could therefore also account for the regression seen in both groups. Therefore, future research studies should include either a strengthening or stretching programme or

should be designed in such a way as to compare various combinations of strengthening or stretching protocols with GIASTM and DTFM.

Table 12: Between and within subjects effects for intensity of pain

Effect	Statistic	p value
Time	Wilk's lambda=0.265	<0.001
Group	F=1.382	0.252
Time*group	Wilk's lambda=0.916	0.766

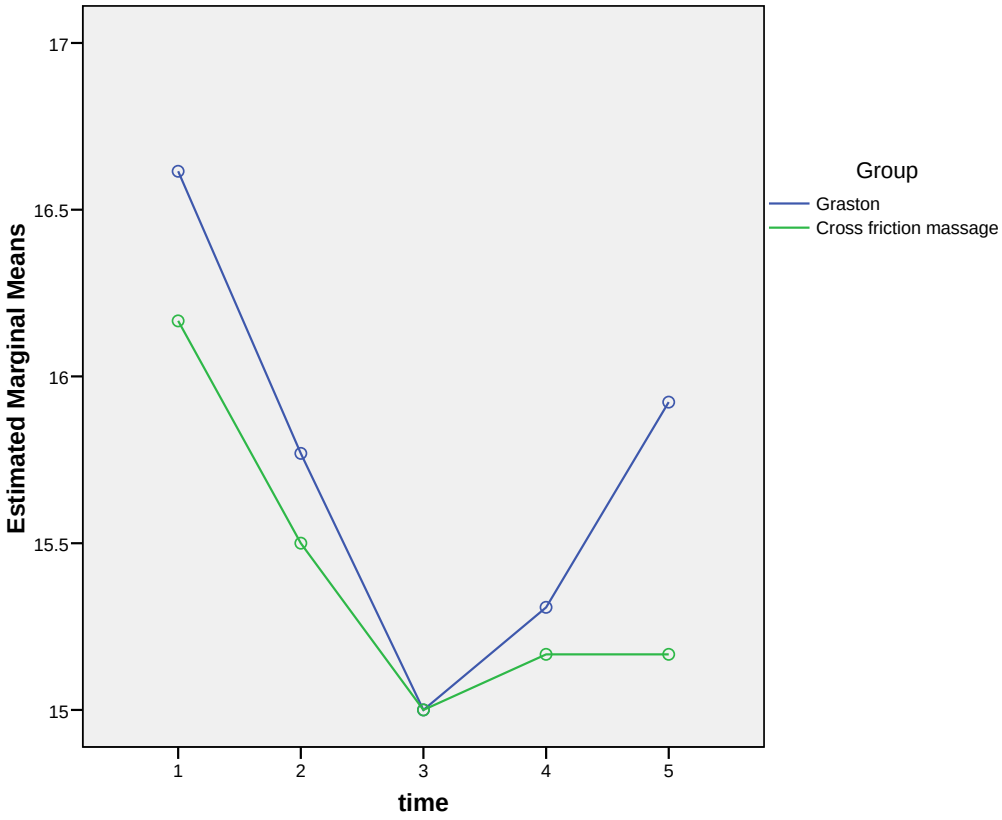

Figure 8: Profile plot of time of mean intensity of pain for each group

4.4.1.2.3 Frequency of pain

Frequency of pain showed no significant overall time effect (p=0.245) or treatment effect (p=0.245) as shown in Table 13. Figure 9 shows that the DTFM group remained constant over the 5 time points, while the GIASTM group improved over time. However, this difference is not statistically significant between the two groups. These results with particular reference to the GIASTM group are in congruence with the Blazina Scale staging 1 to 3a of patellar tendinopathy (Brunker and Khan, 2002:481), as patients entering this study were required to have had a diagnosis of patellar tendinopathy for more than one month, indicating (as per the Blazina scale) (Lian *et al*., 2003), a greater likelihood of a stage 3 patellar tendinopathy. Therefore, it could be expected that the patient's symptoms would have improved from constant to intermittent as shown in Figure 9 during time periods T1 – T4 in the GIASTM group.

The DTFM group did not show as marked improvement as compared to the GIASTM group and recorded a constant level of frequency of pain throughout this study. As previously outlined in section 4.2.2.2, the depth of penetration of DTFM is not as deep as compared to GIASTM (Carey, 2003:59 and Hammer and Pfefer, 2005). A more apparent change in the DTFM group would therefore not be expected and may explain the lack of improvement seen in the DTFM group as seen in Figure 9.

Table 13: Between and within subjects effects for frequency of pain

Effect	Statistic	p value
Time	Wilk's lambda=0.824	0.245
Group	F=4.182	0.052
Time*group	Wilk's lambda=0.824	0.245

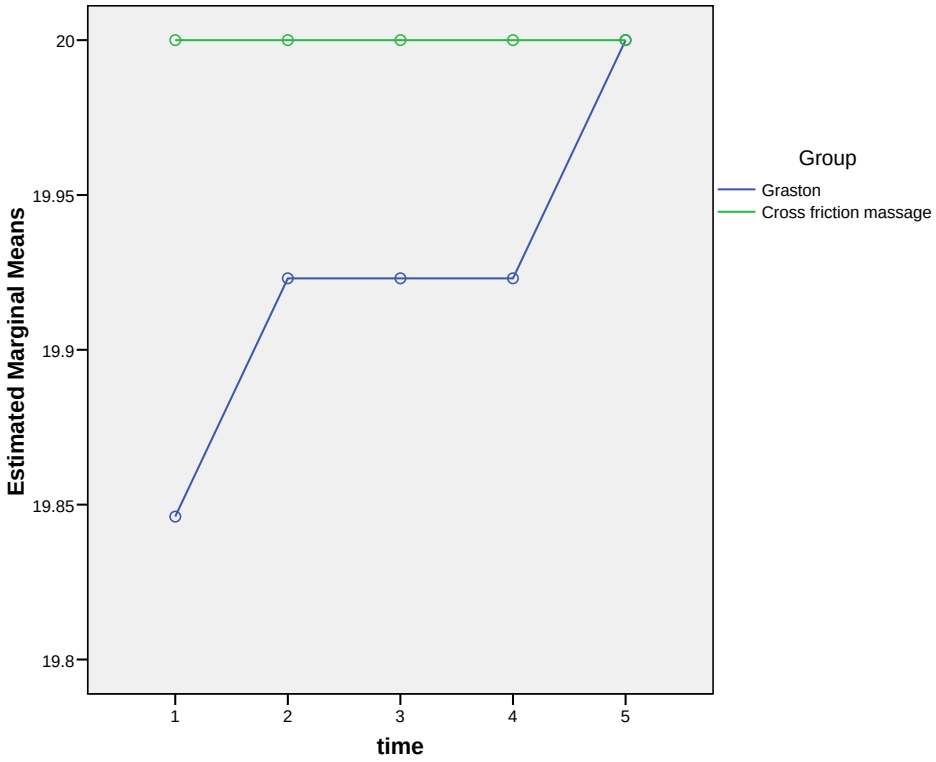

Figure 9: Profile plot of time of mean frequency of pain for each group

4.4.1.2.4 Swelling

Overall there was a significant improvement in swelling scores (decreased swelling) in both groups (p=0.017), but no differential treatment effect (p=0.274) as shown in Table 14. However, Figure 10 shows a trend towards the swelling outcomes improving at a more consistent rate in the GIASTM group compared to the DTFM group.

Swelling is rare in patellar tendinopathy (Vizniak and Carnes, 2004:174 and Brunker and Khan, 2007:508). However, in their study of patellar tendinopathy in athletes, Panni *et al.*, (2002) reported mild swelling in ten of the 42 patients. Although it has been established that the histological picture of a tendinopathy does not contain any inflammatory cells, the degeneration of collagen fibres and subsequent altered biomechanics of the extensor mechanism of the knee would cause swelling to occur around the patella tendon. The bevelled shape of the GIASTM instrument creates a convex to convex contact against the patella tendon which allows for equalisation and dispersion of pressure (Falvey, 2001). This mechanism of pressure dispersion would allow for the movement of fluid (swelling) away from the patella tendon. Similarly but to a lesser degree this explanation is also applicable to DTFM.

Treatment induced hyperaemia (Hammer, 1995 and Norris, 2004:38) and increased tissue perfusion (Hammer, 1995) are reported effects DTFM and this induced inflammatory response aids the subsequent formation of new collagen fibres (Perle, 2003; Vizniak, 2005:489 and Stasinopoulos and Johnson, 2007). Together with the restoration of collagen fibres and therapeutic movement of collagen fibres that occurs with DTFM (Hammer, 1995), the biomechanics of the extensor mechanism of the knee is restored and

therefore swelling around the patella tendon is reduced. The abovementioned would also explain the improvement in swelling (decreased swelling) in the GIASTM group. The lack of differential treatment effect could be accounted for by the similarity in treatment application and mechanism of action of the GTIASM and DTFM.

It has been reported that the GIASTM instruments allow an advantage to the clinician; the ability to penetrate fibres more deeply (Carey, 2003:59; Perle, 2003 and Hammer and Pfefer, 2005). The variance in swelling in the DTFM group during T1-T2 and T4-T5 may therefore be explained in terms of the difference in pressure effect of the two groups as discussed in section 4.2.2.2.

Table 14: Between and within subjects effects for swelling

Effect	Statistic	p value
Time	Wilk's lambda=0.563	0.017
Group	F=2.392	0.136
Time*group	Wilk's lambda=0.783	0.274

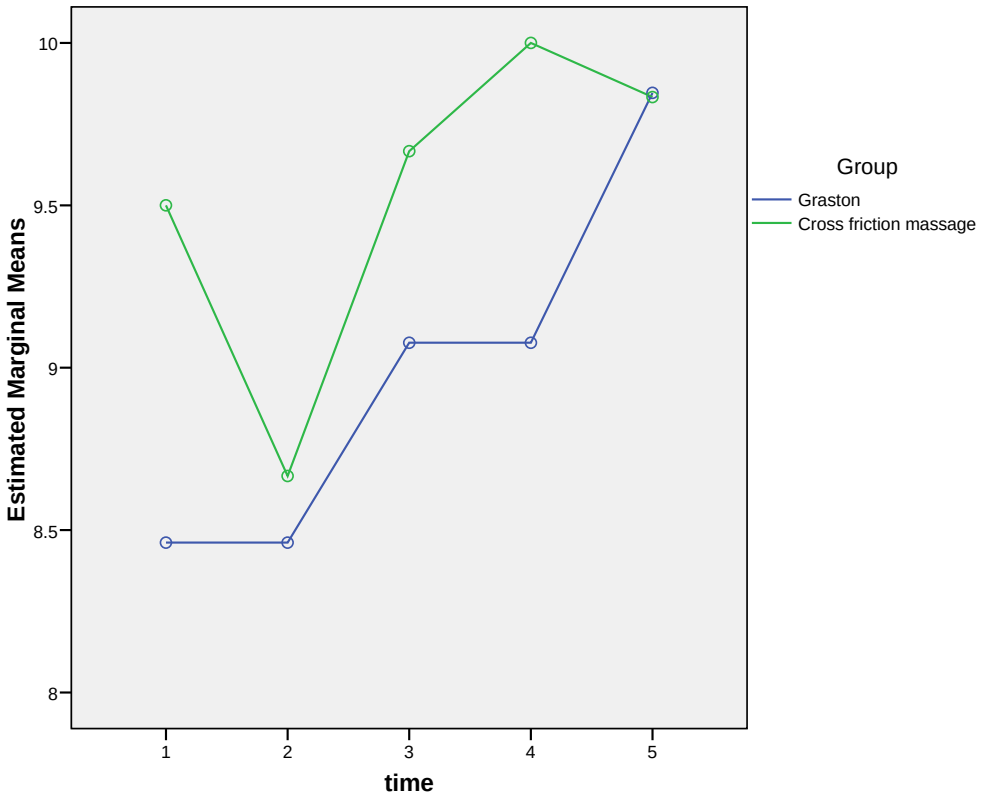

Figure 10: Profile plot of time of mean swelling for each group

4.4.1.2.5 Stability

Occasional instability due to quadriceps inhibition has been reported as a symptom of patellar tendinopathy (Brunker and Khan, 2007:508) and weakness of the quadriceps muscle group decreases the stability of the knee joint (Hall, 1999:125). Both groups showed a significant improvement (p=0.027) in this symptom over the three month study (Table 15) but there was no significant difference between the treatment groups (p=0.318) (Figure 11). This improvement in outcome measure is significant when taking into account that the knee depends more on its surrounding muscles and ligaments for stability

than that of its bony articulation (Magee, 2002:661).

During T4-T5 there is regression of stability in the DTFM group. Regression in swelling (increased swelling) also occurred in this group during this same time period (Figure 10). Also NRS 1 indicated an increase in pain during T5-T7 (Figure 4). The combination of increased swelling and pain in the DTFM group may have caused a reflex inhibition of the quadriceps femoris muscle and this is what was possibly perceived as instability in the DTFM patients (Omoigui, 2007). This regression during T4-T5 may also be related to the superficial depth of penetration of DTFM as opposed to GIASTM (Carey, 2003:59 and Hammer and Pfefer, 2005) as outlined in section 4.2.2.2. DTFM breaks down adhesions and promotes realignment of disrupted collagen fibres (Stasinopoulos and Johnson, 2007 and Vizniak, 2005:489) as evidenced by improvements seen in Figure 11 (T1-T4). However if DTFM does not have as great a depth of penetration as compared to GIASTM (Carey, 2003:59 and Hammer and Pfefer, 2005), any collagen fibres still in a state of remodelling (Vizniak and Carnes, 2004:306) may act as a nociceptive focus (Vizniak, 2005:489) thereby decreasing the perceived joint stability of the DTFM group.

During the repair phase of tendon healing (which can last from 48 hours to 6 weeks), collagen is not fully oriented in the direction of tensile strength and

the quality of newly formed collagen is not as strong as the original (Vizniak and Carnes, 2004:306). Improper healing during this repair phase results in impairment of the patella tendons' vital role of load absorption and transmission (Kountouris and Cook, 2007) and a weak patella tendon decreases the relative stability of the knee joint (Hall, 1999:125) which could also account for the decrease of joint stability seen in the DTFM group during T4-T5.

Table 15: Between and within subjects effects for stability

Effect	Statistic	p value
Time	Wilk's lambda=0.591	0.027
Group	F=0.091	0.766
Time*group	Wilk's lambda=0.798	0.318

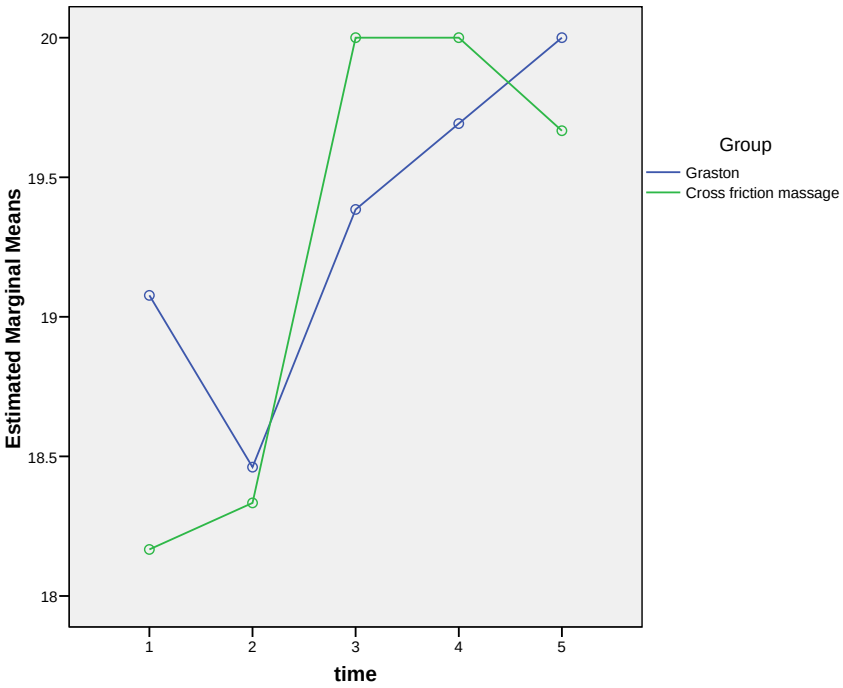

Figure 11: Profile plot of time of mean stability for each group

4.4.1.2.6 Stiffness

Stiffness scores improved significantly over time in both groups (p=0.007) but there was no evidence of a differential treatment effect over time (p=0.446) (Table 16). This is shown in Figure 12, where the rates of change in both groups are similar over time. No difference in treatment effect between the two groups was found and may be explained by the two treatment applications being very similar in nature. A statistical improvement of stiffness over the three month study was found. A possible explanation could be related to the significant decrease in pain levels over time in both groups as seen in Figure 8 and Figure 9. As the patient experienced a decrease in pain level and a decrease in inhibition, the patient became less cautious in bearing weight on the knee and this increased freedom to move the joint during daily activities and this was perceived as a decrease in stiffness.

A caution does need to be raised with respect to the interpretation of stiffness as this may vary from meaning: weakness; fatigue; to a limitation in movement of a joint. It is important that a distinction be made by the physician with regards to the patient's inability to move the joint as well as reluctance to move a joint. In this study, patients were not influenced with regard to their interpretation of the term stiffness. Therefore it is suggested that future studies should quantify the use of the term stiffness to improve accuracy.

Table 16: Between and within subjects effects for stiffness

Effect	Statistic	p value
Time	Wilk's lambda=0.509	0.007
Group	F=0.055	0.817
Time*group	Wilk's lambda=0.838	0.446

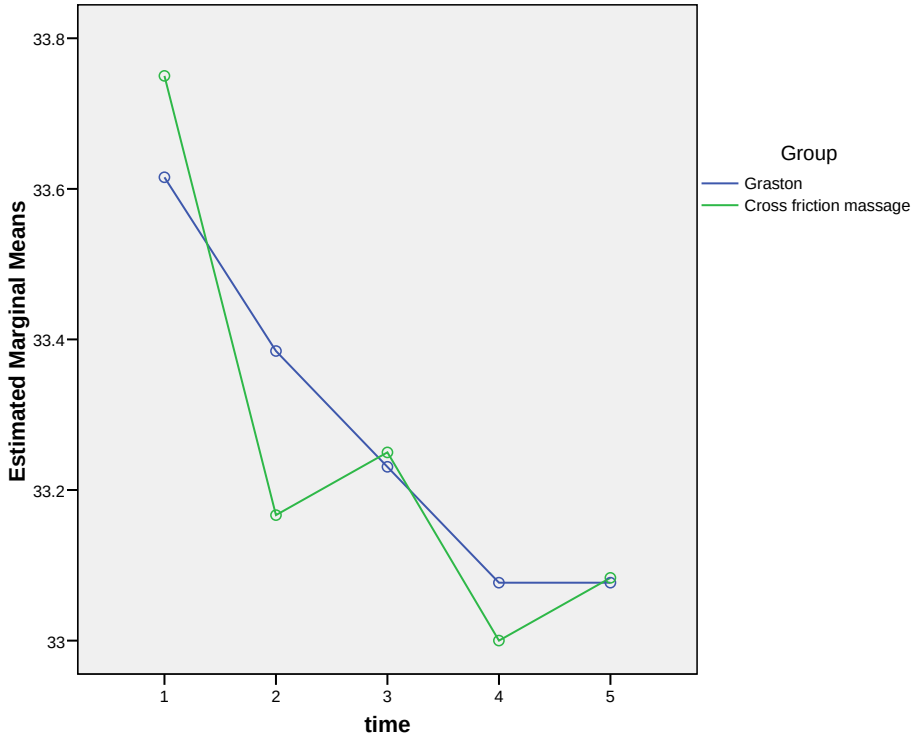

Figure 12: Profile plot of time of mean stiffness for each group

4.4.1.2.7 Grinding

Grinding scores improved significantly over time in both groups (p=0.004) but there was no evidence of a differential treatment effect over time (p=0.636) (Table 17). This is shown in Figure 13, where the rates of change in both groups are similar over time.

Although crepitus is not a reported symptom of patellar tendinopathy (Brunker and Khan,

2007:508) and is reported to be present in 94% of asymptomatic females and 45% percent of asymptomatic males (Johnson *et al*., 1998). No patient in this study started with a reading of zero, thus crepitus was featured in this study. According to Daniel *et al*., (1990:164), the greater the stiffness readings are the greater the grinding readings will be as the patella is held closer to the femoral condyles. Although their statement proposes that these two variables should improve to the same extent over time the grinding scores from this study did not show as great an improvement as the stiffness scores.

Improvement in grinding levels supports the theories that DTFM and GIASTM both address a mechanical component of patellar tendinopathy (Perle, 2003 and Hammer, 2004).

Table 17: Between and within subjects effects for grinding

Effect	Statistic	p value
Time	Wilk's lambda=0.476	0.004
Group	F=1.792	0.194
Time*group	Wilk's lambda=0.886	0.636

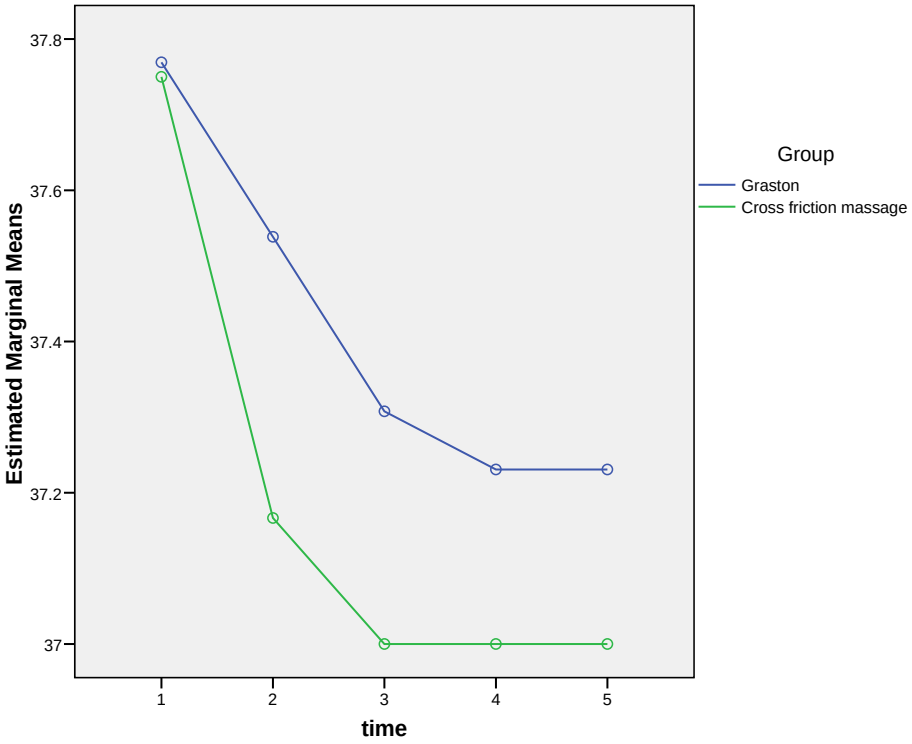

Figure 13: Profile plot of time of mean grinding for each group

4.4.1.2.8 Locking

Locking scores improved significantly over time in both groups (p=0.049) but there was no evidence of a differential treatment effect over time (p=0.577) (Table 18). This is shown in Figure 14, where the rates of change in both groups are similar over time. Figure 14 also shows during T1-T2 there is a sudden significant improvement (decrease) in locking in both groups.

However, in the DTFM group there is a sharp regression between T2-T3 after which a decrease to a plateau level is reached. The sharp regression during T2-T3 in the DTFM group may be as a result of the swelling that is induced as part of the healing cascade (Hammer, 2004 and Hammer and

Pfefer 2005) (Figure11). This treatment induced swelling which may have caused a reflex spasm of the hamstring muscle, which is a known cause of locking in the knee (Magee, 2002:8). In the GIASTM group only a slight regression is seen at T4-T5. Results have shown the same regression (T4-T5) occurs in the GIASTM group with regards to extension indicating the same theoretical constructs offered as an explanation in 4.2.2.2 stand for locking.

Table 18: Between and within subjects effects for locking

Effect	Statistic	p value
Time	Wilk's lambda=0.634	0.049
Group	F=1.047	0.317
Time*group	Wilk's lambda=0.871	0.577

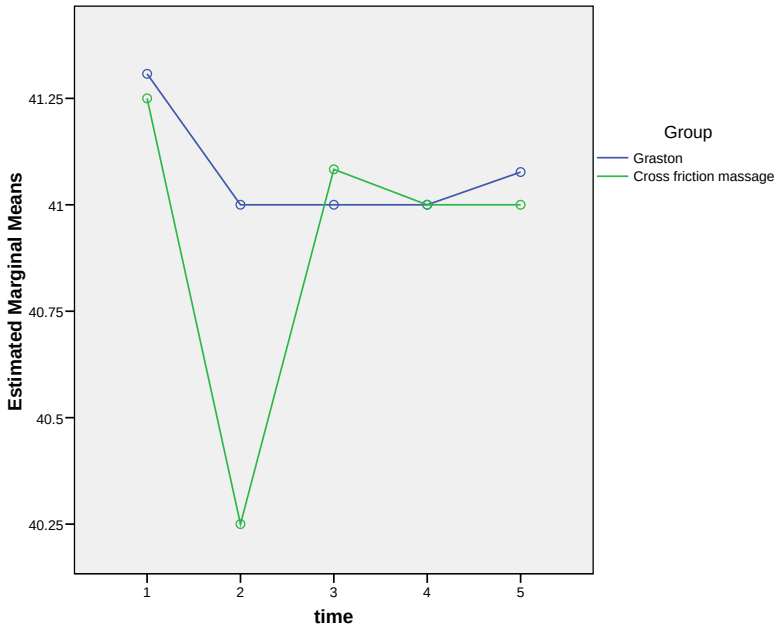

Figure 14: Profile plot of time of mean locking for each group

4.4.1.2.9 Activity

Activity scores improved significantly over time in both groups ($p<0.001$) but there was no evidence of a differential treatment effect over time ($p=0.960$) (Figure 15).

No significant difference in activity levels were found between the two treatment groups. This could be explained as a result of the treatments being very similar in nature and as a result of homogeneity between the groups with regards to the sports played. The significant improvement over time in both groups could possibly be as a result of the improvement in the outcome measures of: pain intensity (Figure 8); frequency of pain (Figure 9); swelling (Figure 10); stability (Figure 11); stiffness (Figure 12); grinding (Figure 13) and locking (Figure 14).

With the improvement of these outcome measures, the patient's functional disability decreases and the patients are therefore able to increase activity in their sports and/or functional daily activities. These results correlate with Carey (2003:9) and Perle (2003) who report that GIASTM is thought to make an improvement in activities of daily living and quality of life (Carey, 2003:9). Based on results achieved in this study, it could be inferred that DTFM and GIASTM have similar effects.

Table 19: Between and within subjects effects for activity

Effect	Statistic	p value
Time	Wilk's lambda=0.298	<0.001
Group	F=0.142	0.710
Time*group	Wilk's lambda=0.970	0.960

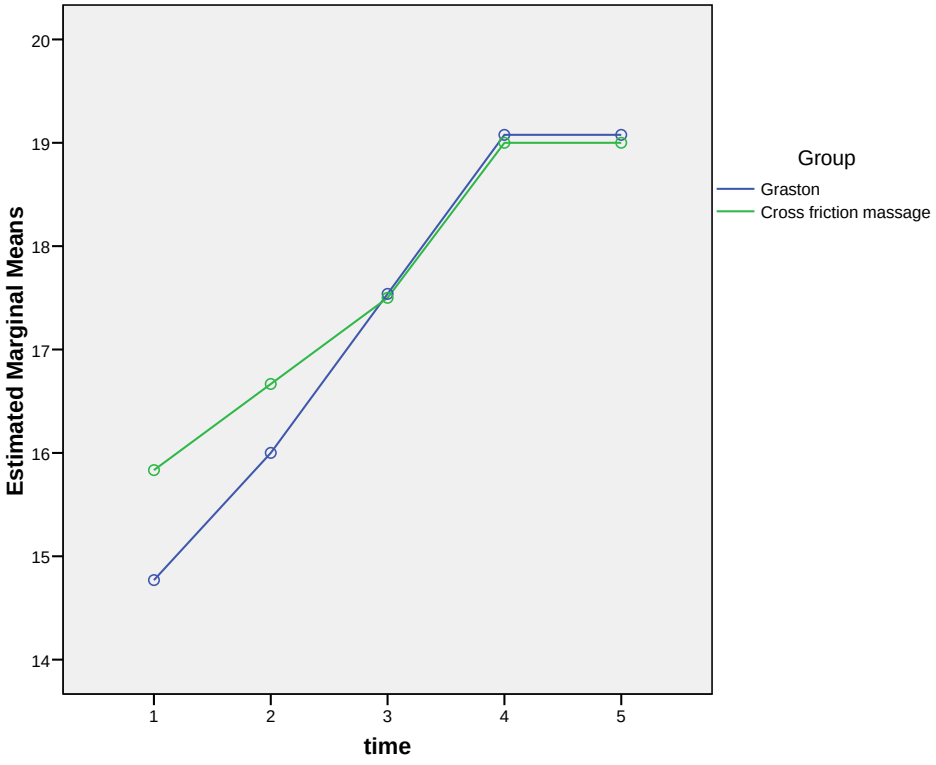

Figure 15: Profile plot of time of mean activity for each group

4.4.1.2.10 Walking

In the later stages of patellar tendinopathy, symptoms may be produced through simple daily activities such as walking (Penn *et al.*, 2006). Walking scores did not improve significantly between groups (p=0.837) (Table 20) and may be explained by the similarities of the two treatment modalities used in this study. However a significant improvement in walking over time did occur (p=0.034) in both groups indicating a good improvement in functions of daily living such as walking. The GIASTM group showed a trend towards a faster rate of improvement in walking compared to the DTFM group (Figure 16). A possible explanation may be that the GTIASM was able to return the components of the patella tendon to a state of controlled inflammation; leading to the resorbtion and thereafter reformation of collagen fibres (Falvey, 2001) at a faster rate compared DTFM due to the greater depth of penetraton of GIASTM (Carey, 2003:59 and Hammer and Pfefer, 2005).

Table 20 Between and within subjects effects for walking

Effect	Statistic	p value
Time	Wilk's lambda=0.609	0.034
Group	F=1.310	0.264
Time*group	Wilk's lambda=0.934	0.837

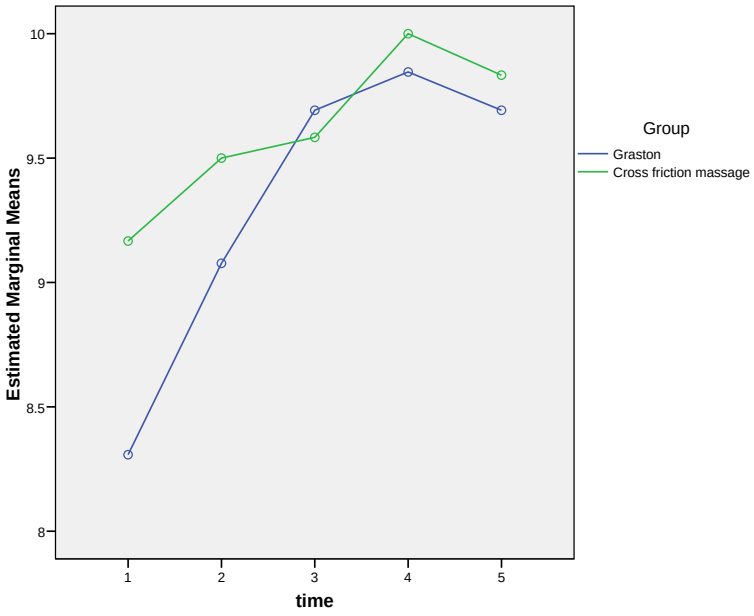

Figure 16: Profile plot of time of mean walking for each group

4.4.1.2.11 Climbing of Stairs

The ability to climb stairs as part of a daily living activity is reported to affect a person's quality of life (Costigan, *et al.,* 2002 and Carey, 2003:9).

Stair climbing scores improved significantly over time in both groups (p=0.001) but there was no evidence of a differential treatment effect over time (p=0.342) (Table 21). This is shown in Figure 17, where the rates of change in both groups are similar over time.

The patella tendon transmits contracting forces from the quadriceps femoris muscle to produce movement of the lower extremity (Moore and Dalley, 1999: 537). It is subjected to great forces (Almekinders *et al*., 2002) during movements such as walking that involve maximal use of the

extensor mechanism of the knee (such as occurs when ascending stairs).

The statistically noted improvement over time in both groups (p=0.001) (Table 21) is significant because research has shown that the extensor mechanism of the knee is reported to constitute a major cause of anterior knee pain in the athletes and overuse injuries are a noted cause of functional deficit in both non-athletes and athletes (Peace and Healy, 2006). In view of the mechanical overload in patellar tendinopathy, the accumulative effects of each loading cycle placed on the knee while ascending stairs and the subsequent disruption of collagen fibres can cause resultant pain and perceived weakness of the knee (Daniel *et al*., 1990:339). It is theorised that DTFM causes the softening of scar tissue and the breakdown of adhesions, thereby promoting the realignment of new (collagen) fibres within the affected tendon (Perle, 2003; Vizniak, 2005:489 and Stasinopoulos and Johnson, 2007).

The GTIASM is also theorised to stimulate a controlled inflammatory response which ultimately results in the formation of stronger new collagen fibres (Falvey, 2001). In view of this literature, both treatment modalities could have increased the rate of collagen formation thereby decreasing any accumulative effects each loading cycle places on the knee during the ascending of stairs. The GIASTM group did have a greater initial improvement (T1-T2) compared to the DTFM

group. This may be explained in terms of depth of penetration of the GIASTM instruments (as discussed in 4.2.2.2).

Table 21: Between and within subjects effects for stair climbing

Effect	Statistic	p value
Time	Wilk's lambda=0.388	0.001
Group	F=1.965	0.174
Time*group	Wilk's lambda=0.807	0.342

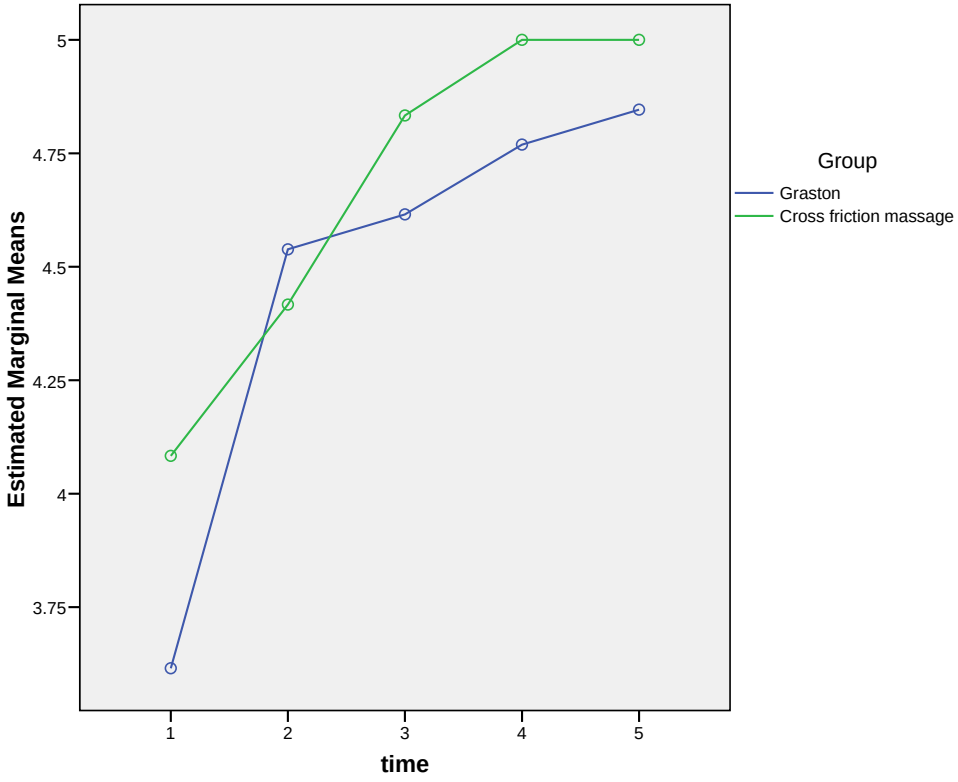

Figure 17: Profile plot of time of mean stair climbing for each group

4.4.1.2.12 Running

Functional deficits during running, ascending or descending stairs, twisting and jumping could indicate instability due to injured ligaments and tendons or could indicate muscle dysfunction (Magee, 2002:5). During running, the patella tendon is subject to forces ranging from six to fourteen times bodyweight (Kountouris and Cook, 2007). Tendon overload occurs when 3-8% strain is applied to the tendon (Peers and Lysens, 2005).

The statistically significant improvement of running scores over time ($P<0.001$) (Table 22) in both groups may be due to the improvement of outcome measures of: intensity of pain (Figure 9); swelling (Figure 9); stability (Figure 12); stiffness (Figure 13); grinding (Figure 14) and locking (Figure 15). As pain levels decrease, reflex inhibition of the quadriceps and hamstring muscles due to anticipated pain (Magee, 2002:8) would also decrease and the patient would therefore find that their ability to run would greatly improve. The lack of differential treatment effect ($p=0.403$) (Table 22) between groups may be due to the similarity of the treatment modalities.

Running scores improved significantly over time in both groups ($p<0.001$) but there was no evidence of a differential treatment effect over time ($p=0.403$) and is shown in Figure 18. However, there was a trend towards the GIASTM group showing a faster rate of improvement than the DTFM group. This is similar to the outcome

measure of stair climbing as shown in Figure 17 and once again may be related to the depth of penetration of the GIASTM instruments (as discussed in 4.2.2.2).

Table 22: Between and within subjects effects for running

Effect	Statistic	p value
Time	Wilk's lambda=0.383	<0.001
Group	F=1.465	0.238
Time*group	Wilk's lambda=0.825	0.403

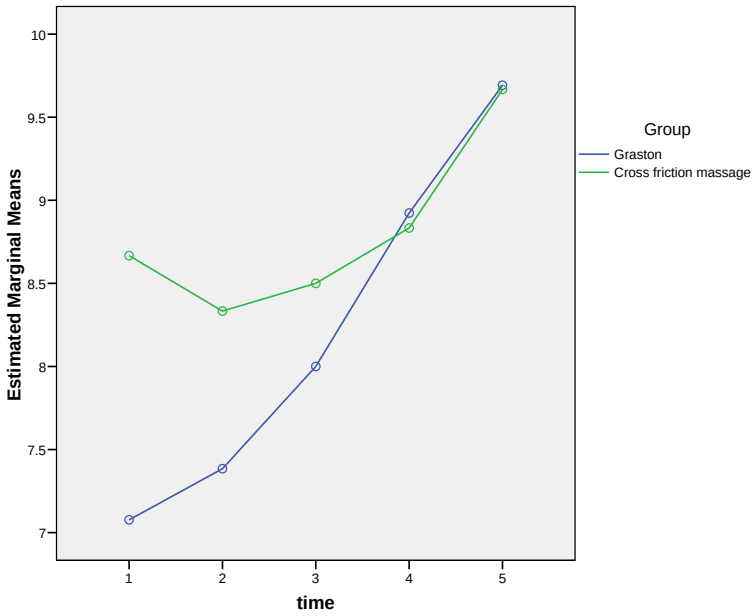

Figure 18: Profile plot of time of mean running score for each group

4.4.1.2.13 Jumping and Twisting

During jumping, the patella tendon is subject to forces ranging from six to fourteen times bodyweight (Kountouris and Cook, 2007). Similarly to jumping and other activities, tendon overload occurs when 3-8% strain is applied to the tendon (Peers and Lysens, 2005). Jumping scores did not improve significantly over time in both groups ($p=0.086$) and there was no evidence of a differential treatment effect over time ($p=0.670$) (Table 23). This is shown in Figure 19, where the rates of change in both groups are similar over time.

Although patellar tendinopathy occurs commonly amongst athletes who repeatedly jump (Penn *et al.*, 2006) the lack of significant time improvement in both treatment groups ($p=0.086$) (Table 23), could be expected as only five of the patients in this study took part in sports that involved jumping and twisting (it is noted that the five participants were equally distributed between the groups). In addition the majority of patients reported either mild or moderate pain at the outset of the study which implies that the reported pain intensity levels (Figure 8) and pain in relation to activity (Figure 15) may not have been sufficient in order to achieve a significant finding with respect to any one particular activity. The GIASTM group had a greater initial improvement compared to the DTFM group and may be explained in terms of depth of penetration of the GIASTM instruments (4.2.2.2).

Table 23: Between and within subjects effects for jumping and twisting

Effect	Statistic	p value
Time	Wilk's lambda=0.678	0.086
Group	F=0.808	0.378
Time*group	Wilk's lambda=0.893	0.670

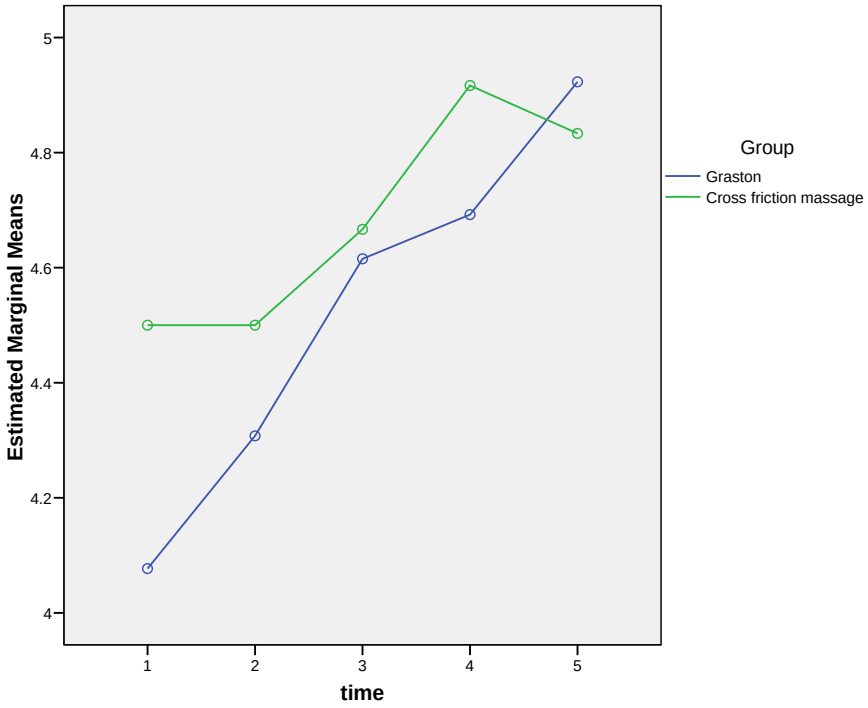

Figure 19: Profile plot of time of mean jumping and twisting score for each group

4.5 VISA SCORE QUESTIONNAIRE (TOTAL)

The Victorian Institute of Sport Assessment (VISA) questionnaire is an index of severity of symptoms in patients with patellar tendinopathy. This questionnaire assesses: symptoms; simple tests of function in daily activity and the ability to play sport (Visentini *et al.*, 1998 and Kountouris and Cook,

2007) and therefore reflects elements of both sporting success and symptomatic benefit (Coleman *et al.*, 2000).

Both treatment groups showed significant improvement with regard to the VISA score over time (p<0.001). No difference in the treatment effect between the groups (p=0.579) was noted (Table 24 and Figure 20).

The possible explanations for the improvement of outcomes over time will be discussed in section 4.5.1.

Table 24: Between and within subjects effects for VISA score

Effect	Statistic	p value
Time	Wilk's lambda=0.248	<0.001
Group	F=1.654	0.211
Time*group	Wilk's lambda=0.872	0.579

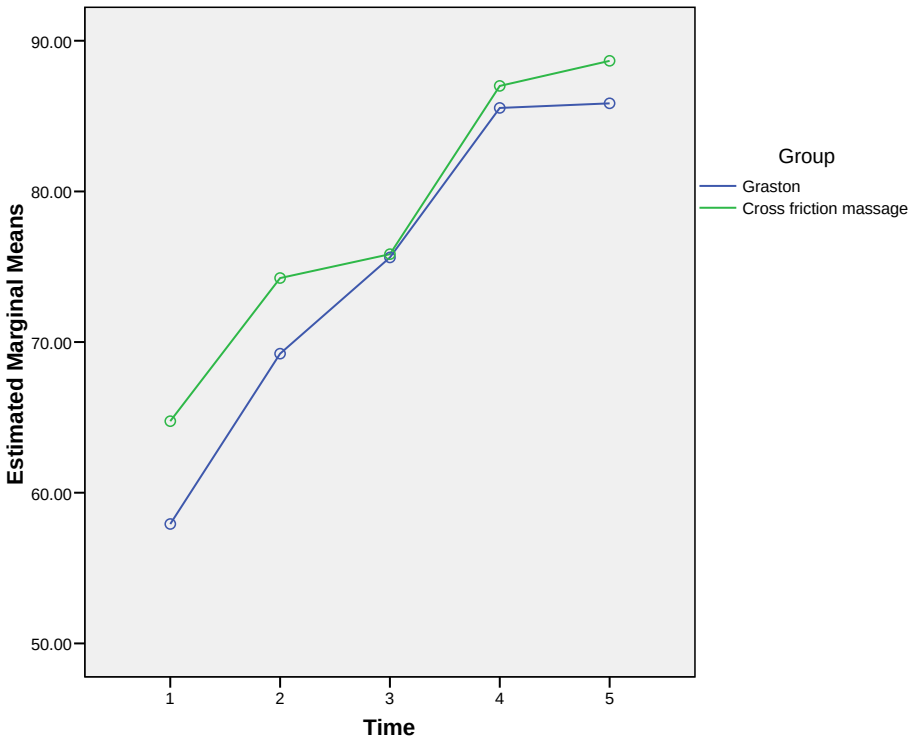

Figure 20: Profile plot of time of mean VISA score by group

4.5.1 VISA Score Questionnaire Individual Items

4.5.1.1 Minutes pain free while sitting

Symptoms of patellar tendinopathy can be exacerbated by prolonged knee flexion (Shamus and Shamus, 2001:98 and Peers and Lysens, 2005). The number of minutes patients could sit pain free, increased significantly over time in both groups (p=0.016) (Table 25). There was no evidence of a differential effect over time (p=0.118) (Table 25). This is shown in Figure 21 as a similar rate in change in both groups over time. The GIASTM group took longer to improve initially compared to the DTFM group as evidenced by time periods T1-T2. This initial slower rate of improvement may be related to the induced treatment effect of swelling by GIASTM (Hammer, 2004 and Hammer and Pfefer 2005). This compared to GIASTM, which allows for deeper penetration of collagen fibres (Carey, 2003:59 and Hammer and Pfefer, 2005), results in a greater amount of swelling in the GIASTM group as opposed to the DTFM initially during treatment.

Knee pain is typically worse after periods of sitting with the knee flexed (Krivickas, 1997:141; Shamus and Shamus, 2001:98 and Peers and Lysens, 2005) and eases with activity (Batt and Tanji, 1995:78). In accordance with the above mentioned literature it is not unexpected to see this slower rate of initial improvement in the GIASTM group as the greater amount of induced swelling by GIASTM would confound knee pain that is already reported to be worse after periods of sitting (Krivicakas, 1997:141). Patients may therefore limit their actual level of physical activity in order to avoid pain or due to their perception that they can no longer perform as effectively, every day activities such as rising from a chair after having been seated (De Groot *et al*., 2007). It is also important to acknowledge that a patient's perception of physical functioning in activities of daily living does not always

correspond to the actual physical activity and that the actual activity (rising from a chair after sitting) remains possible (De Groot *et al.*, 2007).

Table 25: Between and within subjects effects for minutes sitting pain free

Effect	Statistic	p value
Time	Wilk's lambda=0.688	0.016
Group	F=0.757	0.393
Time*group	Wilk's lambda=0.824	0.118

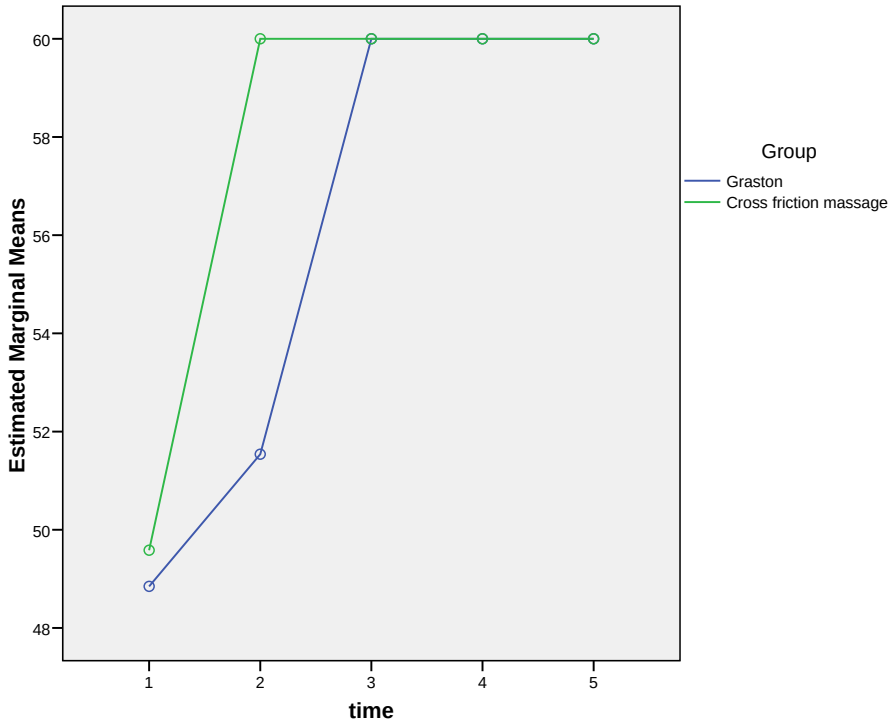

Figure 21: Profile plot of time of mean minutes sitting pain free in each group

4.5.1.2 Pain walking down stairs

Pain levels when walking down stairs improved significantly over time in both groups ($p<0.001$) (Table 26). There was no evidence of a differential treatment effect over time ($p=0.508$) (Table 26). This is shown in Figure 22, where the rates of change in both groups are similar over time. The extensor mechanism of the knee is reported as being important in eccentric contractions, such as walking down stairs and running (Panni *et al*., 2002.). GIASTM is reported to normalise forces within a joint (Carey, 2003:1) and this improvement in proprioception may be used to explain the improvement in walking scores. Similarly this explanation could account for the results obtained in the DTFM group.

This outcome is in agreement with 4.4.1.2.9 (activity), 4.4.1.2.10 (walking), 4.4.1.2.11 (stairs), 4.4.1.2.12 (running) and 4.4.1.2.13 (jumping and twisting), which correlates with the Subjective Knee Score Questionnaire.

Table 26: Between and within subjects effects for pain walking down stairs

Effect	Statistic	p value
Time	Wilk's lambda=0.332	<0.001
Group	F=1.299	0.266
Time*group	Wilk's lambda=0.854	0.508

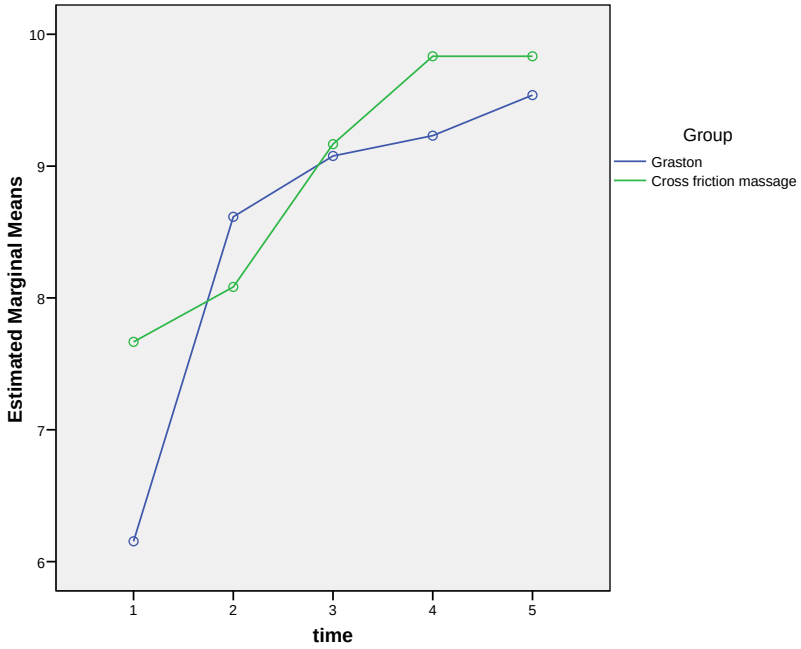

Figure 22: Profile plot of time of mean pain walking down stairs for each group

4.5.1.3 Pain at the knee with full active non weight bearing knee extension

Results of this study showed that active knee extension improved significantly over time in both groups (p=0.013) (Table 27). A possible explanation for this could be linked to the improvement of the outcome measure of pain (threshold tenderness) (Figure 1). As tenderness decreased, reflex inhibition of the quadriceps femoris muscle would have decreased and the reported tightness that can occur in this injury (Peterson and Renström, 2003:321) would also decrease. This improved flexibility would therefore allow eased and improved extension of the knee by the quadriceps femoris muscle.

There was no evidence of a differential treatment effect over time (p=0.316) (Table 27). This is shown in Figure 23, where the rates of change in both groups are similar over time. This is in contrast to (4.2.2.2) in which the extended knee during hip

extension did not allow for significant increases in range of motion, which implies the presence of a limiting factor such as pain. This could be accounted for by the fact that in testing hip extension (during which both the hip and knee are in the extended position) there is a likelihood of increasing patella tendon tension because of the anteroposterior movement of the tibia on the femur (Daniel *et al*., 1990:138), thereby increasing pain and thus reducing the significance of the findings in 4.2.2.2.

Table 27: Between and within subjects effects for pain when knee extended

Effect	Statistic	p value
Time	Wilk's lambda=0.547	0.013
Group	F=0.002	0.962
Time*group	Wilk's lambda=0.798	0.316

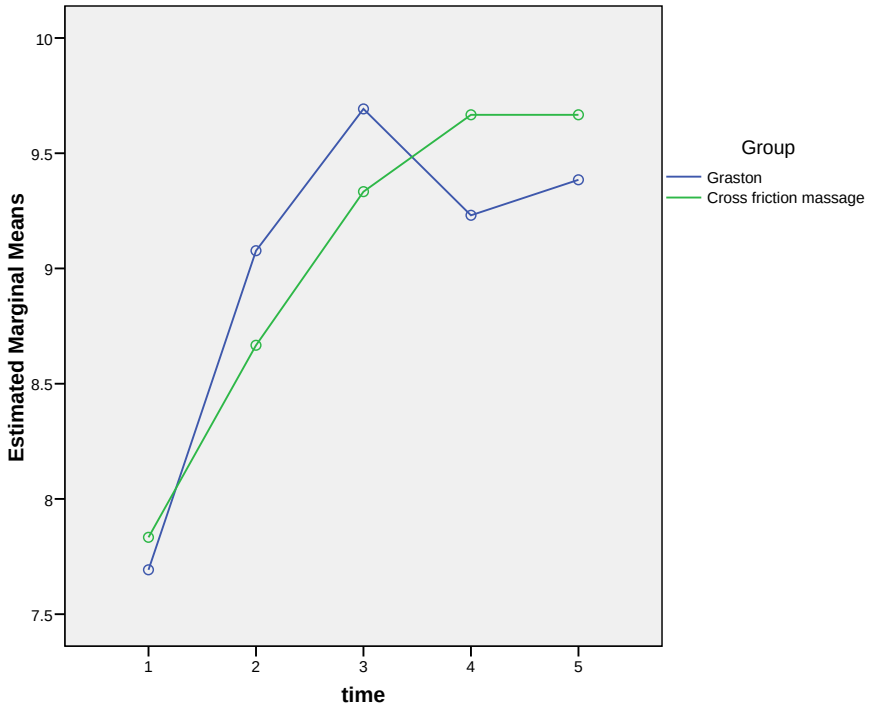

Figure 23: Profile plot of time of mean pain score when knee is extended for each group

4.5.1.4 Pain during weight bearing lunge

The pain when doing a weight bearing lunge improved significantly over time in both groups ($p<0.001$), but there was no evidence of a differential treatment effect over time ($p=0.750$) (Table 28). This is shown in Figure 24, where the rates of change in both groups are similar over time. This is in agreement with the outcome measures of: walking (4.4.1.2.10), climbing of stairs (4.4.1.2.11), running (4.4.1.2.12), jumping and twisting, (4.4.1.2.13), pain walking downstairs (4.5.1.2) and pain at the knee with full active non weight bearing knee extension (4.5.1.3). A weight bearing lunge together with all of the above mentioned outcome measures involved eccentric contraction of the quadriceps femoris muscle and it would therefore be expected that a significant improvement (reduced pain) be seen in the outcome measure of weight bearing lunges (Figure 24).

Table 28: Between and within subjects effects for pain during weight bearing lunge

Effect	Statistic	p value
Time	Wilk's lambda=0.357	<0.001
Group	F=0.385	0.541
Time*group	Wilk's lambda=0.912	0.750

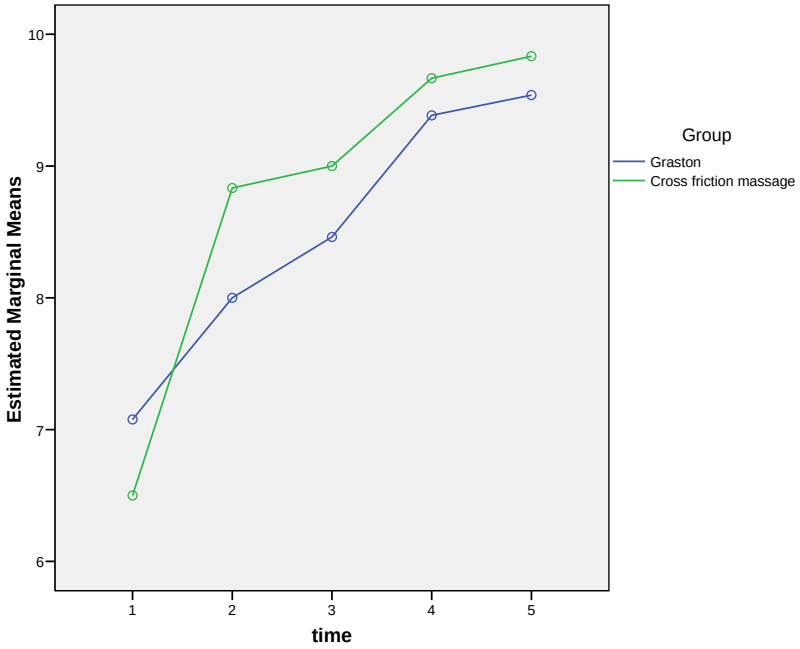

Figure 24: Profile plot of time of mean pain score during weight bearing lunge for each group

4.5.1.5 Pain during or immediately after ten single leg hops

The single leg hop is a functional activity that allows for evaluation of the lower limb kinetic chain (Kountouris and Cook, 2007). As the patella tendon is not an inextensible tissue but rather deforms in response to applied loads which in turn is dependent upon its mechanical properties (Reeves *et al.*, 2003); the lower limb kinetic chain influences the efficacy of function of the knee joint and its muscles during everyday tasks and sporting activities (Kountouris and Cook, 2007). Biomechanical factors that may have lead to altered movement patterns during single leg loading have been linked to patellar tendinopathy (Kountouris and Cook, 2007).

The pain when doing single leg hops improved significantly over time in both groups ($p<0.001$), but there was no evidence of a differential treatment effect over time ($p=0.988$) (Table 29). This is shown in Figure 25, where the rates of change in both groups are similar over time. The movement of a single weight bearing

hop is similar to hip extension but the former relies on a bony lock and the latter is a shear force between the tibia and femur (Daniel *et al*., 1990:138).

Decreased flexibility associated with the quadriceps femoris muscle and hamstring muscle, which is associated with patellar tendinopathy (Kountouris and Cook, 2007 and Hyman, 2006) may have limited the degree of significance attained in this study Therefore future research programmes need to investigate the use of a stretching programme as advocated by Carey (2003:2) and Perle (2003) in addition to GIASTM.

Table 29: Between and within subjects effects for pain during or immediately after ten single leg hops

Effect	**Statistic**	**p value**
Time	Wilk's lambda=0.290	<0.001
Group	F=0.771	0.389
Time*group	Wilk's lambda=0.985	0.988

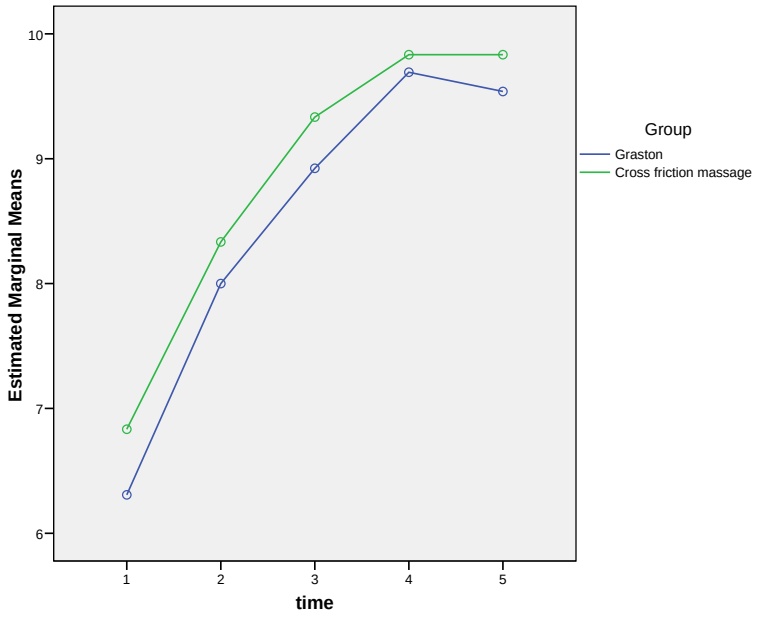

Figure 25: Profile plot of time of mean pain score during or immediately after ten single leg hops for each group

4.5.1.6 Problems trying to squat

Muscle tendon function is defined as the ability of the muscle tendon complex to generate adequate strength and endurance required to perform functional tasks and sporting activities (Kountouris and Cook, 2007). This is of importance as patellar tendinopathy results in the decrease of strength of several muscles. Strength and endurance are important components of daily activities and biomechanics in sports (Kountouris and Cook, 2007). Assessment of muscle-tendon function is considered important to monitor during treatment as poor function is associated with ongoing symptoms (Kountouris and Cook, 2007).

The strength of the quadriceps femoris and gluteal muscles can be assessed by the use of squatting

(Kountouris and Cook, 2007). An improvement in this functional clinical test would indicate improvement in the strength of the quadriceps femoris muscle group. Although the decline squat is reported to be a more sensitive test than conventional squats, the use of conventional squats in this study still enabled loading of the patella tendon and quadriceps activation (Kountouris and Cook, 2007).

Problems when trying to squat improved significantly over time in both groups ($p<0.001$), but there was no evidence of a differential treatment effect over time ($p=0.652$) (Table 30). This is shown in Figure 26, where the rates of change in both groups are similar over time. However, in terms of rate of improvement, results tend to favour the GIASTM group.

Table 30: Between and within subjects effects for problems trying to squat

Effect	Statistic	p value
Time	Wilk's lambda=0.321	<0.001
Group	F=0.111	0.742
Time*group	Wilk's lambda=0.889	0.652

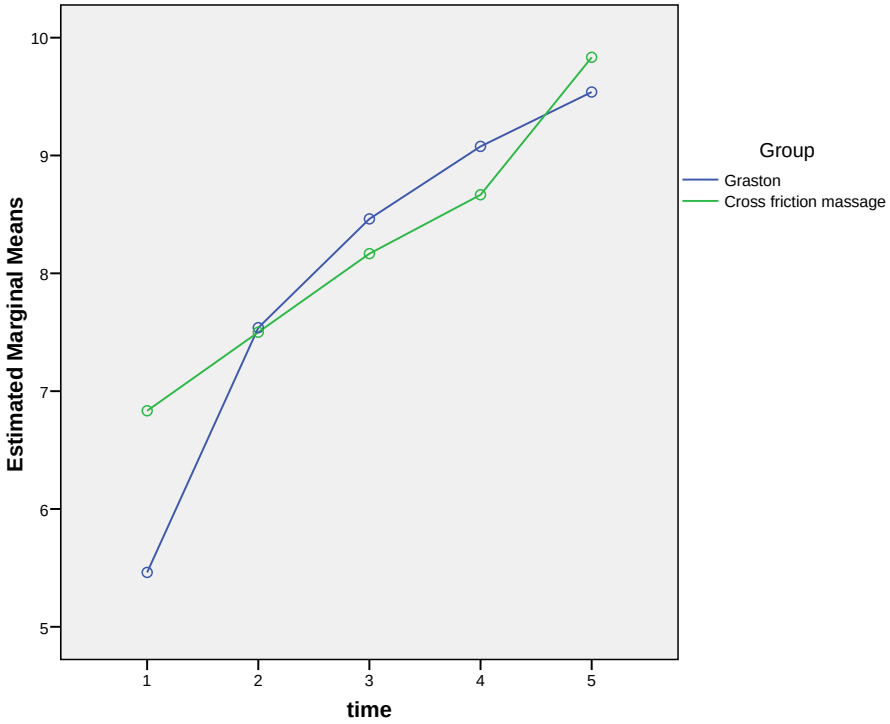

Figure 26: Profile plot of time of mean problems trying to squat for each group

4.5.1.7 Pain that occurs during sport or activity

The pain which occurred during sport or activity improved significantly over time in both groups (p=0.001), but there was no evidence of a differential treatment effect over time (p=0.684) (Table 31). This is shown in Figure 27, where the rates of change in both groups are similar over time.

The GIASTM group did regress during T4-T5. This may be related to the lack of strengthening and stretching in this study which is reported to improve outcomes when used in conjunction with the GIASTM instruments (Carey, 2003:2 and Perle, 2003). Strengthening encourages proper realignment of collagen fibres and promotes further release of scar tissue and develops stability within

the musculoskeletal system (Carey, 2003:64 and Hammer and Pfefer, 2005).

The significant reduction of pain during sport or activity could possibly be as a result of the improved scores for outcome measures for: pain (Figure 1); intensity of pain (Figure 8); frequency of pain (Figure 9); swelling (Figure 10); stability (Figure 11); stiffness (Figure 12); grinding (Figure 13) and locking (Figure 14).

Table 31: Between and within subjects effects for pain which occurs during sport or activity

Effect	Statistic	p value
Time	Wilk's lambda=0.422	0.001
Group	F=0.141	0.711
Time*group	Wilk's lambda=0.897	0.684

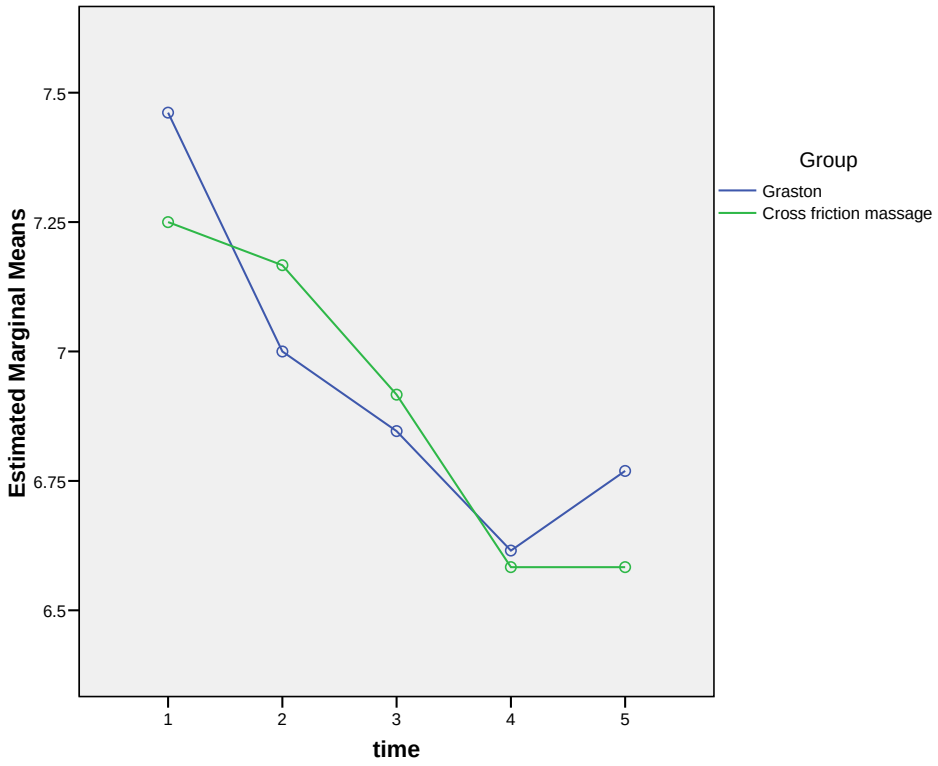

Figure 27: Profile plot of time of mean pain score that occurs during sport or activity for each group

4.5.1.8 Duration of exercise

Scoring for the following question was based on numbers that were randomly allocated by researcher and statistician:

Table 32: Duration of exercise

Duration of Exercise				
Nil	0 – 5 minutes	6 – 10 minutes	11 – 15 minutes	> 15 minutes
17	18	19	20	13

Patellar tendinopathy has traditionally been graded on the Blazina scale (McConnell and Cook, 2007). This scale is empirically based and takes into account pain as related to activity (Brunker and Khan, 2002:481). Clinically these stages can be divided into four (Lian *et al.*, 2003 (Magee, 2002:6, 13).

Table 33: Staging of patellar tendinopathy

Stage 1	Pain at the infrapatellar region after practice or after an event
Stage 2	Pain at the beginning of the activity, disappearing after warm up and reappearing after completion of activity.
Stage 3a	Pain during and after activity, but the patient is able to participate in sports at the same level.
Stage 3b	Pain during and after activity and the patient is unable to participate in sports at the same level.
Stage 4	Complete rupture of the patella tendon

Patients entering this study were required to have had a diagnosis of patellar tendinopathy for more than one month indicating, (as per the Blazina scale) (Lian *et al*., 2003), a greater likelihood of a stage 3 patellar tendinopathy. Therefore patients had the same degree of improvement available to them with respect to the outcome measure of pain during exercise.

For the amount of time they could exercise for, the DTFM group remained constant over time while the GIASTM group showed an improvement. This difference was however not statistically significant (p=0.235) (Table 32).

Patients in this study were not requested to stop their various sport or activity and this may have possibly influenced the significance of this outcome. However, in hindsight this outcome is more likely to reflect reality in clinical practice.

The lack of statistical difference may also be explained by the following factors which may have perpetuated the problem: the omission of a stretching and strengthening programme; lack of correction of sporting technique and the need for further treatments during the two week to one month follow up period. Further research therefore needs to identify factors responsible for limiting the duration of exercise but also at the same time allowing for the reduction in clinical symptoms. The most likely factor is the lack of correction of sporting technique but is subject to further investigation.

Table 34: Between and within subjects effects for how long can exercise

Effect	Statistic	p value
Time	Wilk's lambda=0.877	0.235
Group	F=2.972	0.098
Time*group	Wilk's lambda=0.877	0.235

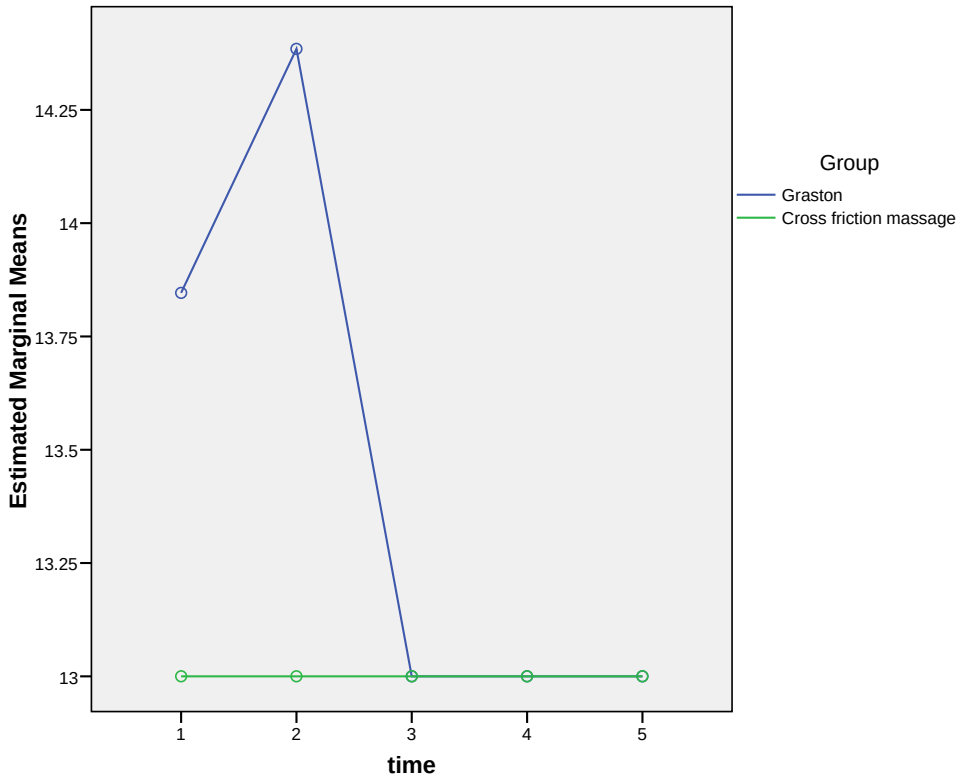

Figure 28: Profile plot of time of mean duration of exercise for each group

4.6 SUMMARY

In summary it has been shown in the results that the demographic factors of gender p=0.671, race p=0.738, age p=0.548 and sporting activities did not influence the outcomes achieved in this study as there was no significant difference between the groups for any of these factors allowing for homogeneity between the DTFM and GIASTM groups.

With respect to objective outcomes, the algometer readings showed a significant improvement overtime in both groups. In contrast, inclinometer readings for hip flexion were borderline significant (Figure 2) and the readings for hip extension were insignificant between the groups (Figure 3).

Subjective outcomes showed a greater variance in this study compared to objective outcomes.

Significant time improvements occurred in the NRS1 and NRS2.

4.6.1 NRS1 p= 0.002 (Time effect)	**4.6.2** NRS2 p< 0.001 (Time effect)

With regard to the Subjective Knee Score Questionnaire (Time group effect), two outcomes did not show a significant improvement over time: frequency of pain p=0.245 (4.4.1.2.3) and jumping and twisting p=0.670 (4.4.1.2.13).

The following did however show a significant time effect for both groups.

4.6.3 Subjective Knee Score Questionnaire (Time effect):

Swelling	p=0.017	Total	P <0.001
Stability	p=0.027		
Stiffness	p=0.007		
Grinding	p=0.004		
Locking	p=0.049		
Activity	p<0.001		
Walking	p=0.034		
Stairs	p=0.001		
Running	p<0.001		

Three outcomes from the VISA Score Questionnaire did not show a significant improvement overtime (Time group effect): minutes sitting pain free p=0.118 (4.5.1.1); pain walking downstairs p=0.508 (4.5.1.2) and duration of exercise p=0.235 (4.5.1.8).

However the following showed a significant time effect for both groups.

4.6.4 VISA questionnaire (Time effect):

Minutes pain free while sitting	p=0.016	Total	P <0.001
Pain walking downstairs	p<0.001		
Pain at knee with full non active weight bearing knee extension	p=0.013		
Pain with weight bearing lunge	p<0.001		
Single leg hops	p<0.001		
Problems trying to squat	p<0.001		
Pain with sport or activity	p=0.001		
Duration of exercise	p=0.235		

The objective outcomes as well as the subjective outcomes generally showed statistically significant improvements over time. Therefore, as hypothesised in Chapter One, GIASTM was no more effective than DTFM in treating patellar tendinopathy but GIASTM nevertheless has been shown to be as effective as DTFM in treating patellar tendinopathy. Therefore the results of this study show that GIASTM is an effective conservative form of therapy as compared to DTFM that can be used as a treatment intervention for patellar tendinopathy and that patients can expect successful results when treated with the GIASTM instruments. The only limitation identified by this research to hamper the success of GIASTM is the omission of a strengthening and stretching programme (Carey, 2003:2 and Perle, 2003).

4.6.5 In conclusion the Hypotheses state:

The first null hypothesis as outlined in chapter one stated that the use of GIASTM would be no more effective than DTFM in treating patellar tendinopathy in terms of objective findings.

Based on the results of the study we accept the first null hypothesis of this study.

The second null hypothesis as outlined in chapter one stated the use of GIASTM would be no more effective than DTFM in treating patellar tendinopathy in terms of subjective findings

Based on the results of the study we accept the second null hypothesis of this study.

CHAPTER FIVE

5.1 CONCLUSION

The focus of any conservative management programme in treating patellar tendinopathy should be to encourage collagen synthesis, maturation and strength and decrease loading of the patella tendon (Warden and Brunker, 2003:752). It may take up to six months for symptoms of patellar tendinopathy to improve with the goal of treatment being the reduction of pain and restoration of function (Wilson and Best, 2005).

The aim of this study was to determine the relative effectiveness of GIASTM and DTFM in terms of subjective and objective outcomes in treating patellar tendinopathy. For all objective and subjective outcomes measured in this study, there was no difference in the treatment effect of the GIASTM group compared with the DTFM group, and both treatments were equally effective at reducing pain and symptoms.

In terms of the first objective (objective data):

- Significant improvements in the patients' pain threshold were seen in both the GIASTM and DTFM group.
- No improvement in the flexibility of the hamstring and quadriceps femoris muscles were noted.
- No statistically significant improvement in treatment effect between the GIASTM and DTFM groups were noted.

In terms of the second objective (subjective data):

- There was a statistically significant improvement in both the GIASTM and DTFM groups.
- There was no statistically significant improvement in treatment effect between the GIASTM and DTFM groups.

Clinical experience suggests a twelve-week recovery period from patellar tendinopathy as an optimal management strategy to obtain the best clinical results (Cannel *et al.*, 2001). Therefore in conclusion the objective and subjective results of this study demonstrate that GIASTM can play a promising role in the conservative treatment and management of patellar tendinopathy.

Results of this study did not provide any statistically significant evidence that GIASTM offered more effective objective and subjective outcomes compared to that of DTFM and therefore both the first and second hypotheses were accepted.

Nevertheless the researcher can confirm anecdotal evidence, reported by most clinicians, that the decreased treatment time necessary to administer soft tissue mobilization via GIASTM, greatly decreases strain on the interphalangeal joints of the fingers (Perle, 2003). This is encouraging as standard techniques such as DTFM can cause a lot of discomfort for the provider (Perle, 2003). This together with the results discussed in Chapter Four may provide a possible solution for both the clinician and patient (Perle, 2003).

In addition the researcher can also confirm that GIASTM creates a significant vibration that helps in detecting scar tissue by providing tactile diagnostic feedback to both the clinician and patient (Perle, 2003). Both clinician and patient are able to identify when the instrument moves over a problem area in the patella tendon. Anecdotal evidence suggesting that the GIASTM instruments allow for greater specificity (Perle, 2003) is therefore supported by the researcher in this study.

5.2 RECOMMENDATIONS

1. Although vital that treatment be administered by the same person to promote reliability and reproducibility of clinical results, it is always better to have an independent person responsible for recording of data. This ensures that the researcher cannot both treat and record data to ensure that researcher bias is eliminated.

2. Self-administration of the Subjective Knee Questionnaire is recommended. The researcher recommends not being involved in explaining the questionnaire or guiding the patients when they were confused with the questions at any stage of the research. In their study on the mode of data collection with regard to subjective knee score questionnaires it was stated by Hoher *et al.,* (1997) that the presence of an interviewer may be a significant influence in reported patient outcomes.

3. A larger sample size would have strengthened the reliability of results. It is reported that results obtained from small population sizes cannot be extrapolated onto an entire population because the results obtained rarely provide results better than chance (Hillerman *et al.,* 2005).

4. A more representative sporting population (sports that involve jumping and overuse of the extensor mechanism of the knee) be incorporated into the study.

5. The use of a revised VISA questionnaire is recommended. Taunton *et al.,* (2003) stated that the addition of a visual analogue scale in conjunction with the VISA questionnaire might improve the reliability of this questionnaire in terms of pain relief. Question eight of this questionnaire accounts for thirty percent of the total score, penalizing patients who experience any pain with activity and cannot therefore account for athletes who continue to play through sport (Taunton *et al.,* 2003).

6. A controlled and double blinded clinical trial would establish scientifically accurate reporting of data.

7. Although this study included both unilateral and bilateral presentations of patellar tendinopathy; separate studies on patients suffering from unilateral and bilateral are recommended as data suggests that unilateral and bilateral patellar tendinopathy warrant separate investigation (Gaida *et al*., 2004).

8. Further studies into the exact pathoaetiological mechanism of patellar tendinopathy are recommended as new emerging techniques based on such studies may lead to improved curative and preventative measures (Peers and Lysens, 2005).

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APPENDIX 1

Letter of Information

Dear Patient,

Welcome to my research.

Title of Study: A prospective clinical trial to determine the relative effectiveness of Deep Transverse Friction Massage versus Graston Instrument Assisted Soft Tissue Mobilization in treating patellar tendinopathy.

Names of supervisors: Dr. C. Korporaal 031 3732611
Name of research student: Donna Fraser 031 3732512
Name of institution: Durban University of Technology

Purpose of the study: The purpose of this study is to compare and contrast the results of two similar but different modalities (Deep transverse Friction Massage and Graston Technique Instrument Assisted Soft Tissue Mobilization) in treating patellar tendinopathy.

Procedures: You will fill out questionnaires at the initial consultation. You will be asked to fill these questionnaires out again at certain times during the course of treatment. You will be divided into one of two groups. One group will receive Deep Transverse Friction Massage to the tender region of their knee and the other group will receive Graston Technique Instrument Assisted Soft Tissue Mobilization. During this time you are expected not to receive any other form of therapy for your tendinopathy outside this study, if you do so you will be withdrawn from this study.

Risks/Discomfort: The treatment applied may cause transient pain and discomfort while applied. This should abate within a few hours of the treatment and causes no side effects other than stiffness.

Benefits: You will receive treatment for your patellar tendinopathy. This is expected to benefit you according to the current hypotheses.

New findings: You will be made aware of any new findings during the course of this research. Your contribution to this study will help us as Chiropractors to build on our reserve of knowledge. This will benefit you as a patient as we will be able to provide you with more effective health care in the future.

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

If you are unable to attend your follow up appointments.
If you have changed any lifestyle habits during participation that may affect the outcome of this study (e.g.: medication, supplements or any other form of treatment for your patellar tendinopathy).

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

Remuneration/ costs: You will **NOT** receive a travel allowance or any monetary remuneration for your participation in this research. You will not as a participant in this study be charged for any of the treatments, as long as the consultations are within the parameters of this study.

Confidentiality: All patient information is confidential. The results of this study will be used for research purposes only. Only individuals directly involved in this study, Dr. Korporaal (my supervisor) and I will have access to these records.

Persons to contact should you have any problems or questions:

Should you have any questions you would prefer to have answered by an independent individual, you can contact my supervisor on the number provided. Should you have any queries or complaints you are also welcome to contact a representative from the Faculty of Health Sciences Research and Ethics Committee – Mr. Vikesh Singh – 031 3732701.

Thank you for participating in my research.

……..	……
DONNA FRASER	**DR CHARMAINE KORPORAAL**
Sixth Year Intern	**Supervisor**

APPENDIX 2

INFORMED CONSENT FORM

Date: 1 /02/2006

TITLE OF RESEARCH:

A prospective clinical trial to determine the relative effectiveness of Deep Transverse Friction Massage versus Graston Instrument Assisted Soft Tissue Mobilization in treating patellar tendinopathy.

Name of supervisor: Dr. C. Korporaal
Tel: 031 3732611

Please circle the appropriate answer **YES/NO**

1. Have you read the research information sheet?
2. Have you had an opportunity to ask questions regarding this research?
3. Have you received satisfactory answers to your questions?
4. Have you had an opportunity to discuss this study?
5. Have you received enough information about this study?
6. Do you understand your implications of your involvement in this study?
7. Do you understand you are free to withdraw from this study, at any time without having to give any reason for withdrawing and without affecting your future healthcare?
8. Do you agree to voluntarily participate in this study?
9. Who have you spoken to?

Please ensure that the researcher completes each question 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 name:
Signature:

Witness name:
Signature:

APPENDIX 3
DURBAN UNIVERSITY OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: _____ Date: _____

File #: _____ Age: _____

Sex: _____ Occupation: _____

Intern: _____ Signature: _____

FOR CLINICIANS USE ONLY:

Initial visit

Clinician: Signature:

Case History:

Examination:

Previous: Current:

X-Ray Studies:

Previous: Current:

Clinical Path. lab:

Previous: Current:

CASE STATUS:

PTT:	Signature:	Date:

CONDITIONAL:

Reason for Conditional:

Signature: Date:

Conditions met in Visit No:	Signed into PTT:	Date:
Case Summary signed off:		Date:

Intern's Case History:

1. Source of History:

2. Chief Complaint : (patient's own words):

3. Present Illness:

	Complaint 1	**Complaint 2**
< 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. x-rays
< Environmental Hazards (Home, School, Work)
< Exercise and Leisure
< Sleep Patterns
< Diet
< Current Medication
Analgesics/week:
< Tobacco
< Alcohol
< Social Drugs

7. Immediate Family Medical History:

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

8. Psychosocial history:

< Home Situation and daily life
< Important experiences
< Religious Beliefs

9. Review of Systems:

< General

< Skin

< Head

< Eyes

< Ears

< Nose/Sinuses

< Mouth/Throat

< Neck

< Breasts

< Respiratory

< Cardiac

< Gastro-intestinal

< Urinary

< Genital

< Vascular

< Musculoskeletal

< Neurologic

< Haematologic

< Endocrine

< Psychiatric

9. Review of Systems:

< General

< Skin

< Head

< Eyes

< Ears

< Nose/Sinuses

< Mouth/Throat

< Neck

< Breasts

< Respiratory

< Cardiac

< Gastro-intestinal

< Urinary

< Genital

< Vascular

< Musculoskeletal

< Neurologic

< Haematologic

< Endocrine

< Psychiatric

APPENDIX 4

Durban University of Technology
PHYSICAL EXAMINATION: SENIOR

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

Student : **Signature :**

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

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

SYSTEM SPECIFIC EXAMINATION:

CARDIOVASCULAR EXAMINATION

RESPIRATORY EXAMINATION

ABDOMINAL EXAMINATION

NEUROLOGICAL EXAMINATION

COMMENTS

Clinician: **Signature :**

DURBAN UNIVERSITY of TECHNOLOGY

APPENDIX 5
DURBAN UNIVERSITY OF TECHNOLOGY
KNEE REGIONAL EXAMINATION

Patient:_____ File:_____ Date:_____
Intern:_____ Signature:_____
Clinician:_____ Signature:_____

OBSERVATION (Standing, Seated and during gait cycle).

A. Anterior view
Genu Varum:_____
Genu Valgum:_____
Patellar position:_____
Tibial Torsion:_____
Skin:_____
Swelling:_____

B. Lateral view
Genu Recurvatum:_____
Patella Alta:_____
Patella Baja:_____
Skin:_____

C. Posterior view
Swelling:_____
Skin:_____

D. General
Movement symmetry:_____
Structures symmetry:_____

ACTIVE MOVEMENTS
Flexion (0 - 135E)_____
Extension (0 - 15E)_____
Medial Rotation (20 - 30E)_____
Lateral rotation (30 - 40E)_____

PASSIVE MOVEMENTS
Tissue approx_____
Bone-bone_____
Tissue stretch_____
Tissue stretch_____
Patellar movement_____

RESISTED ISOMETRIC MOVEMENTS
Knee: Flexion:_____
Extension:_____
Internal rotation:_____
External rotation:_____

Ankle: Plantarflexion_____
Dorsiflexion_____

LIGAMENTOUS ASSESSMENT

One-Plane Medial Instability
Valgus stress (abduction)
Extended_____
Resting Position_____

One-Plane Lateral Instability
Varus stress (adduction)
Extended_____
Resting Position_____

One-Plane Anterior Instability
Lachman Test (0-30°)_____
Anterior Drawer Sign _____

One-Plane Posterior Instability
Posterior "sag" Sign_____
Posterior Drawer Test_____

Anterolateral Rotatory Instability
Slocum Test_____
Macintosh Test_____

Anteromedial Rotatory Instability
Slocum Test_____

Posterolateral Rotatory Instability
Jacob _____
Hughston's Drawer Sign_____
Reverse pivot shift test_____

Posteromedial Rotatory Instability
Hughston's Drawer Sign_____

TESTS FOR MENISCUS INJURY

McMurray _____ Anderson med-lat grind _____
"Bounce Home" _____ Apley=s _____

PLICA TESTS

Mediopatellar Plica _____ Hughston's Plica _____
Plica "Stutter" _____

TESTS FOR SWELLING

Brush/Stroke Test _____ Patellar Tap Test _____

TESTS FOR PATELLA FEMORAL PAIN SYNDROME

Clarke's Sign _____ Passive patella tilt test _____
Waldron test _____

OTHER TESTS

Wilson's _____ Quadriceps Contusion Test _____
Fairbank's _____ Leg Length Discrepancy _____
Noble Compression _____

JOINT PLAY

Movement of the tibia on the femur P | A: _____ A | P: _____
Translation of the tibia on the femur M | L: _____ L M: _____
Long axis distraction of the tibiofemoral joint _____
Inf, sup, lat, + med glide of the patella _____
Movement of the inf. tibiofibular joint A | P: _____ P | A: _____
Movement of the sup. tibiofibular joint A | P: _____ P | A: _____
Movement of the sup. tibiofibular joint S | I : _____ I | S _____

PALPATION

Tenderness _____ Swelling _____
Joint line _____ Nodules/exostoses _____
Ligaments _____ Muscles: thigh: _____
Patella: _____ Leg : _____
Patella tendon: _____ Popliteal artery: _____
Bursae: _____

REFLEXES AND CUTANEOUS DISTRIBUTION

	R	L
Patellar Reflex (L3,L4)		
Medial Hamstring Reflex (L5,S1)		

DERMATOMES

	R	L		R	L
L2			S1		
L3			S2		
L4			S3		
L5					

07/02/2007

APPENDIX 6

DURBAN UNIVERSITY OF TECHNOLOGY

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

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

S: ***Numerical Pain Rating Scale (NRS1)*** ***Intern Rating*** ***A:***
Least 0 1 2 3 4 5 6 7 8 9 10 Worst

0: ***P:***

E:

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

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

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

O: ***P:***

E:

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

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

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

O: ***P:***

E:

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

APPENDIX 7

Patents

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

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

APPENDIX 8A

ALGOMETER READINGS

	PRE TREATMENT 1			PRE TREATMENT 9			PRE VISIT 13			TWO WEEK FOLLOW UP			FOURTH WEEK FOLLOW UP		
DATE															
READINGS															
AVERAGE READING															

APPENDIX 8B

INCLINOMETER READINGS

	PRE TREATMENT 1			PRE TREATMENT 9			PRE VISIT 13			TWO WEEK FOLLOW UP			FOURTH WEEK FOLLOW UP		
DATE															
READINGS															
AVERAGE READING															

APPENDIX 9

Numerical Rating Scale (NRS2)

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

Please write only one number.

0 _____ 100

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

Please write only one number.

0 _____ 100

Are you between the ages

25 - 45

& have suffered with

KNEE

PAIN?

(lower knee, for longer than 1 month)

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

FREE TREATMENT

is available to those who qualify to for this study

For more information contact Donna on 307 2205 / 2512

APPENDIX 11

SUBJECTIVE KNEE SCORE QUESTIONNAIRE (Please check the statement that best describes the condition of your knee)

Pain

20	I experienced pain in my knee
16	I have occasional pain with strenuous sport or heavy work. I don't think that my knee is entirely normal. Limitations are mild and tolerable
12	There is occasional pain in my knee with light recreational sports or moderate work
8	I have pain brought on by sports, light recreational activities, or moderate work. Occasional pain is brought on by daily activities such as standing or kneeling
4	The pain I experience is a significant problem with activities as simple as walking. The pain is relieved by rest. I can't participate in sports
0	I have pain in my knee at all times, even during walking, standing, or light work

Intensity	A mild	15	B Moderate	17	C Severe	18
Frequency	A Constant	19	B Intermittent	20		

Swelling

10	I experience no swelling in my knees
8	I have occasional swelling in my knee with strenuous sports or heavy work
6	There is occasional swelling with light recreational activities or moderate work
4	Swelling limits my participation in sports and moderate work
2	My knee swells after simple walking activities and light work. Rest relieves the swelling
0	I have severe swelling with simple walking activities. Rest does not relieve the swelling

Stability

20	My knee does not give out
16	My knee gives out only with strenuous sports or heavy work
12	My knee gives out with light recreational activities or moderate work; it limits my vigorous activities, sports, or heavy labour occasionally, only with strenuous sports or heavy work
8	Because my knee gives out, it limits all sports and moderate work. It occasionally gives out with walking or light work
4	My knee gives out frequently with simple activities such as walking. I must guard my knee at all times
0	I have severe problems with my knee giving out. Can't turn or twist without my knee giving out

Stiffness	A None	33	B Occasional	34	C Frequent	35	D Constant	36
Grinding	A None	37	B Mild	38	C Moderate	39	D Severe	40
Locking	A None	41	B Occasional	42	C Frequent	43	D Constant	44

***15 to 44 (16 excluded) = These are the numbers that were allocated randomly for the corresponding answers

SUBJECTIVE KNEE SCORE QUESTIONNAIRE (Please check the statement that best describes the condition of your knee)

Overall Activity

20 No limitations. I have a normal knee and am able to do everything including strenuous sports and/or heavy labour
16 I can partake in sports including strenuous ones but at a lower level. I must guard my knee and limit the amount of heavy labour or sports
12 Light recreational activities are possible with rare symptoms. I am limited to light work
8 No sports or recreational activities are possible. Walking activities are possible with rare symptoms. I am limited to light work
4 Walking activities and daily living cause moderate problems and persistent symptoms
0 Walking and other activities cause severe problems.

Walking

10 Normal, unlimited
8 Slight, mild problems
6 Moderate problems, flat surface up to half a mile
4 Severe problems, only 2 - 3 blocks
2 Severe problems, need cane or crutches

Stairs

5 Normal, unlimited
4 Slight, mild problems
3 Moderate problems, only 10 - 15 steps possible
2 Severe problems, require banister for support
1 Severe problems, only 1 - 5 steps without support

Running

10 Normal, unlimited, fully competitive
8 Slight, mild problems, run at half speed
6 Moderate problems, only 1 - 2 miles possible
4 Severe problems, only 1 - 3 blocks possible
2 Severe problems, only a few steps possible

Jumping and Twisting

5 Normal, unlimited, fully competitive
4 Slight, mild problems, some guarding
3 Moderate problems, gave up strenuous sports
2 Severe problems, affects all sports, always guarding
1 Severe problems, only light activity possible (pool/swim)

***15 to 44 (16 excluded) = These are the numbers that were allocated randomly for the corresponding answers

APPENDIX 12

Purpose: To use the VISA score as an index for the severity of symptoms in a patient with Patellar Tendinosis (Jumper's Knee)

Data	**Enter**
Do you have symptoms of Jumper's Knee (Patellar Tendinopathy)? (Y or N)	
How many minutes can you sit pain-free?	
On a scale of 0 to 10 indicating no pain and 0 indicating strong, severe pain:	
Pain when walking downstairs with a normal gait, cycle (enter 0 to 10)	10
Pain at the knee with full active, non weight-bearing knee extension (enter 0 to 10)	10
Pain when doing a full weight bearing lunge (enter from 0 to 10)	10
Do you have pain during or immediately after doing 10 single leg hops?	10
On a scale of 0 to 10, with 10 indicating no problems and 0 completely unable to do so:	
Problems when trying to squat (enter 0 to 10)	10

Pain or Problems	Points to assign
None/Nil	10
Mild	8
Mild to Moderate	6
Moderate to Severe	4
Severe	2
Excruciating	0

Enter an "x" in the appropriate column for each question (only 1 answer per row)

Are you currently undertaking sport or other physical activity, and if so, at what level compared to that before symptoms began?	Not at all	Training and/or competition	Competition but not at same level	Competing at same or higher level	
When undertaking sport or activity, indicate which occurs.	No pain	Pain that does not stop completion	Pain that stops completion of activities		
How long can you exercise for?	Nil	0 – 5 minutes	6 – 10 minutes	11 -15 minutes	>15 minutes

Calculate	**Result**
Data complete?	
Evaluation appropriate?	
Total VISA score – out of 100 points	

APPENDIX 13

DURBAN UNIVERSITY OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC

CONFIDENTIAL PATIENT INFORMATION/INDEMNITY FORM

Date: .....

Male/ Female: .....

Surname: ..... Title: .....

First name: ..... Initials: .....

Birthdate: ..... I.D..number: .....

Occupation: ..... Marital status: .....

Medical aid: ..... M/A number: .....

Med doctor: ..... Last visit: .....

Chiropractor: ..... Last visit: .....

Postal address: ..... Residential address:

....

....

Tel - work: ..... Tel - home: .....

Cell number: .....

Employer:.....

Employer's address:

.....

NB: Please ensure that you supply your Medical Aid No for refund purposes

FINANCIAL INFORMATION

The current fee schedule of the Chiropractic Day Clinic is :

Student (5th Year Students)		**Student Intern (6th Year Students)**	
Initial visit:	**R 50.00**	Initial visit:	**R 70.00**
Subsequent visits	**R 40.00**	Subsequent visits	**R 60.00**

All consumables (e.g. needles) : Prices are available on request at the reception desk.

Medical Aid schemes pay in varying degrees for coverage of Chiropractic Services. This coverage is therefore medical aid dependant and we request that you check with your medical aid in this respect. **The DIT Chiropractic Day Clinic is contracted out of medical aid**, which means that we run on a **strictly cash only basis**, whereby you are requested to pay cash in advance of services rendered. You will be sent a monthly statement which you must submit to your medical aid for them to refund you directly. This statement will be sent out at the **end of each month.**

Charges are **not** applicable to **research patients**

Medico-Legal Reports:
As the Chiropractic Day Clinic is a teaching facility we are not in a position to generate any reports required for medico-legal purposes**,** claims that relate to injury on duty (IOD) or workman's compensation

Report of findings:
It is imperative that the student / student intern treating you explains fully your diagnosed condition, both as an educational requirement for the intern but also, **and more importantly**, such that you are able to make an informed decision about the type of treatment that you wish to receive.

Treatment options:
It is imperative that the student / student intern explains all treatment options that are available for you based on the diagnosed condition/(s) that was/ were given to you in respect of the above.

Risks/Benefits:
The student / student intern must explain to your satisfaction / understanding all risks and benefits in relation to treatment of your reported diagnosis / condition(s).

As a Patient at this, the Chiropractic Day Clinic, I understand that I am attending an educational facility and I give my permission to allow observation, and if necessary the video recording of supervised examination and treatment by Doctors of Chiropractic and Interns. In addition I, as the patient note, that information generated through my attendance of the clinic, may be used for research purposes (either through my direct participation in the research or alternatively through data collected in my patient file).

By signing this form I agree that

a) I understand and take full financial responsibility for consultations.
b) I understand that I cannot request records for medico legal reasons.
c) I understand that should I be on medical aid, that my diagnosis and treatment information will be shared for the purposes of medical aid reimbursing me according to that which I am contractually bound in terms of my medical cover (and that only a written request or instruction from myself will be accepted in terms of discontinuing this practice by my health care provider – the Chiropractic Day Clinic).
d) The student / student intern has discussed with me to my satisfaction, and I fully understand, my / my minor child's diagnosed condition(s) that I have.
e) The student / student intern has discussed with me to my satisfaction, and I fully understand all treatment and/or non treatment options and their relative successes and/or failures as applicable to the diagnosed condition(s).
f) I am making an informed decision with regard to, and will submit to / consent to my minor child being submitted to, the treatment protocol as explained.

Date: ..... **Patient Signature:** .....

Date: ..... **Parent / legal guardian signature:** .....
(in the case of patient's who are under the age of 21 years)

Relationship of guardian to the minor: .....

Date: ..... **Intern Signature:** .....

Date: ..... **Clinician Signature:** .....