THE SHORT-TERM EFFECT OF GRASTON INSTRUMENT-ASSISTED SOFT TISSUE MOBILIZATION (GISTM) ON SUPRASPINATUS TENDINOSIS AND IT'S CONCOMITANT FINDINGS

By

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I, Grant Michael Harper, do declare that this dissertation represents my own work in both conception and execution.

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Dedication

To my dad, mom and bro, thank you for all your love and support!

Acknowledgments

The Lord, for his unconditional love and peace in my life.

Dr Charmaine Korporaal. Mentor, friend and true inspiration!

My beautiful girlfriend, Bianca, thank you for loving me like you do

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To all my Chiro mates for all the laughs and good times, we will meet again someday.

Abstract

Shoulder injuries, which account for 8% to 20% of volleyball injuries, are usually rotator cuff and / or biceps tendinosis caused by overuse (Briner et al.1999); in addition 38-75% of competitive swimmers have had a history of shoulder pain, while 9 - 35% of these swimmers were currently experiencing pain (McMaster and Troup, 1993). Rotator cuff tendinosis is also found in laborers involved in repeated overhead activities (i.e. among shipyard welders and steel plate workers), with a prevalence of 18, 3% and 16, 2% respectively (Herberts et al. 1984).

Fricker and Hoy (1995), suggest that the principal cause of tendinosis of the rotator cuff muscles is repetitive microtrauma, due to overfatigued muscles and / or weakening of the rotator cuff and scapulothoracic muscles.

The etiology of impingement syndrome is therefore multifactorial and is commonly associated with other clinical entities such as weak or dysfunctional scapular musculature, posterior glenohumeral capsule tightness, inflammation of tendons (viz. supraspinatus and long head of biceps), bursal inflammation and glenohumeral instability (Michener et al., 2003). Shoulder syndromes are often related to the development and perpetuation of myofascial trigger points (TrPs) as found by Hains (2002), who suggested that these TrPs become activated during mechanical stress and overload of the involved shoulder musculature.

Hammer (1991), suggests that the most valuable modality to treat chronic overuse soft tissue syndromes (irrespective of muscular or tendinous in origin) is friction massage to both regions. Cyriax (1984) and Prentice (1994) state the effect of frictions to include the breakdown of adhesions (scar tissue), as well as preventing the formation of further adhesions.

In this respect the Graston Technique instrument-assisted soft tissue mobilization (GISTM) is a form of soft tissue mobilization that is used to detect and release scar tissue, adhesions and fascial restrictions much like that of the work of Cyriax (Carey-Loghmani, 2003:31, 51-62; Hammer, 2001). However these statements have been made in the absence or appropriate research in order to support such statements, therefore the aim of this study was to assess the short-term effect of Graston instrument-assisted soft tissue mobilization (GISTM) on supraspinatus tendinosis and its concomitant clinical findings.

Objective measures included: Isokinetic testing which was used to evaluate changes in both strength (peak torque) and endurance (total work) of the cuff muscles, whilst goniometer readings were taken to evaluate the associated changes in range of motion that may have taken place.

Lastly, a brief clinical assessment of associated clinical conditions was done. Subjective measurements included: the Numerical Rating Scale – 101 (NRS-101) and the Shoulder Pain And Disability Index (SPADI), which was subdivided into 2 categories; namely, pain and function.

The study consisted of a total of 40 subjects (randomly split into two equal groups of 20) each diagnosed with supraspinatus tendinosis. Participants were mainly involved in overhead activities and were between the ages of 18 and 40.

Group 1 received 6 treatments (with GISTM) over a 3 week period, whereas group 2 received only a single treatment (with GISTM) over the same 3 week period. Subjects underwent a clinical assessment (objective and subjective findings) pre-initial treatment and 24-hour follow up assessment to note any immediate changes. Both groups were clinically assessed (objectively and subjectively) at the end of the 3 week period to note changes.

Data analysis was done in SAS version 9.1 (SAS Institute Inc., Cary, NC). Baseline comparisons between the categorical baseline variables and the group to which the participant was assigned were done using Fisher's exact test. Continuous baseline variables that were not normally distributed were compared between groups using a non-parametric Wilcoxon Mann-Whitney test. Continuous normally distributed baseline data were compared using the two sample t-test.

In accordance with past research, this pilot study revealed that with respect to GISTM, it has yet to be shown whether or not a series of treatments is needed or whether the hypothesized clinical effects are best achieved with one treatment in terms of the individual outcomes measured in this study.

Correlations were noted in the treatment group between the objective outcomes (Age; Goniometer abduction, internal rotation, external rotation; Peak torque adduction, internal rotation and Total work abduction, adduction, internal and external rotation) with the subjective outcome SPADI (pain). And within the measurement group, which showed correlations (Table 9) between the objective outcomes (Goniometer abduction; Peak torque abduction, adduction, internal and external rotation; Total work abduction, adduction, internal and external rotation) with the subjective outcomes SPADI (pain) and (function).

However with respect to the hypotheses the following discussions are apparent:

Hypothesis one indicated that a single treatment is insufficient in treating patients presenting with supraspinatus tendinosis, in terms of subjective findings. This hypothesis was *rejected* with regard to the p-values obtained in this study.

Similarly hypothesis two, which Indicated that a single treatment is insufficient in treating patients presenting with supraspinatus tendinosis, in terms of objective findings; was *rejected* with regard to the p-values obtained in this study.

In terms of hypothesis three, which indicated that multiple treatments are necessary in treating tendinopathies. The hypothesis was *rejected* with regard to the p-values obtained in this study, as no evidence significantly supported this hypothesis.

The fourth and last hypothesis which indicated that patients with an increase in the number of concomitant conditions may take longer to improve, in terms of subjective an objective measurements was *rejected* with regard to the p-values obtained in this study.

It is recognized that the results observed indicate that the use of gender stratification, a power analysis (e.g. larger sample size), use of a control group as well as the use of predetermined markers as points to measure significant improvement (with respect to the clinical outcomes), may have assisted in determining group significant differences.

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List of Abbreviations

GISTM: Graston Technique Instrument-assisted Soft Tissue Mobilisation

NRS-101: Numerical Rating Scale – 101

GT: Graston technique

TrP: Myofascial trigger point

Definition of Terms:

Active myofascial trigger point

An active myofascial TrP, as defined by Travell and Simons (1999), causes a clinical pain complaint. It is a focus of hyperirritability in a muscle or its fascia that is symptomatic with respect to pain. It is always tender, prevents full lengthening of the muscle, weakens the muscle, refers a patient – recognized pain on direct compression, mediates a local twitch response of muscle fibers when adequately stimulated, and, when compressed within the patients pain tolerance, produces referred motor phenomena, and often autonomic phenomena, generally in its pain reference zone, and causes tenderness in the pain reference zone.

Agonist

Muscles, or portions of muscles, so attached anatomically that when they contract they develop forces that complement or reinforce each other. (Travell and Simons, 1999:1)

Antangonist

Muscles, or portions of muscles, so attached anatomically that when they contract they develop forces that oppose each other (Travell and Simons, 1999:2).

Functional Unit

A group of agonist and antagonist muscles that function together as a unit because they share common spinal-reflex responses (Travell and Simons, 1999:3).

Graston Technique Instrument-assisted Soft Tissue Mobilisation (GISTM)

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

Latent myofascial trigger point

A focus of hyperirritability in a muscle or its fascia that is clinically quiescent with respect to pain, but may have all the other clinical characteristics of an active trigger point (Travell and Simons, 1999).

Myofascial trigger point (TrP)

A trigger point is a hyperirritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The activation of a TrP is usually associated with some degree of mechanical abuse of the muscle in the form of muscle overload, which may be acute, sustained and/or repetitive. The spot is painful on compression and can give rise to characteristic referred pain, autonomic phenomena and motor dysfunctions, (Travell, Simons and Simons, 1999:5). These motor dysfunctions, as defined by Travell and Simons (1983:21),

includes spasm of other muscles, weakness of the involved muscle function, loss of co-ordination and decreased work tolerance of the involved muscle

Peak torque

Defined as the highest muscular torque produced by the muscle. Peak torque indicates the muscle's maximum strength capability. Peak torque can be evaluated specific to time (Torque at .20sec) or to ROM (Torque at 30º). Peak torque is an absolute value, when used alone peak torque is difficult to assess the strength specific to a person (Marrule, 1996:110-114).

Tendinitis

Is defined according to Clancy (1990), as symptomatic degeneration of the tendon with vascular disruption and inflammatory repair response. Histologically, degenerative changes as noted in tendinosis, with superimposed evidence of a tear, including fibroblastic and myofibroblastic proliferation, haemorrhage and organizing granulation tissue.

Tendinosis

Is defined according to Clancy (1990), as intratendinous degeneration (commonly due to ageing, microtrauma, vascular compromise). Histologically, there is collagen disorientation, disorganization and fibre separation by an increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularization and focal necrosis or calcification.

Total work

The total volume of work under the torque curve with each repetition regardless of speed, range of motion or time (Marrule 1996:110-114).

Work

Defined as the maximum work produced in a single repetition. This is a better indicator of the function of a muscle group than peak torque (Marrule 1996: 110-114).

Ultrasonography

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

Chapter One

1.1 Introduction

Shoulder injuries, which account for 8% to 20% of volleyball injuries, are usually rotator cuff and / or biceps tendinosis caused by overuse (Briner et al., 1999). In addition, 38-75% of competitive swimmers have had a history of shoulder pain, while 9 - 35% of these swimmers were currently experiencing pain (McMaster and Troup, 1993). Rotator cuff tendinosis is also found in laborers involved in repeated overhead activities (i.e. among shipyard welders and steel plate workers), with a prevalence of 18, 3% and 16, 2% respectively (Herberts et al., 1984).

According to Hains (2002), rotator cuff syndrome and supraspinatus tendinosis, impingement syndrome and subacromial pain syndrome are readily interchangeable terms when diagnosing patients presenting with pain in the anterolateral region of the deltoid, which then radiates into the upper arm. Fricker and Hoy (1995), suggest that the principal cause of tendinosis of the rotator cuff muscles is repetitive microtrauma, due to overfatigued muscles and / or weakening of the rotator cuff and scapulothoracic muscles.

The etiology of impingement syndrome is therefore multifactorial and is commonly associated with other clinical entities such as weak or dysfunctional scapular musculature, posterior glenohumeral capsule tightness, inflammation of tendons (viz. supraspinatus and long head of biceps), bursal inflammation and glenohumeral instability (Michener et al., 2003).

In addition to this, shoulder syndromes are often related to the development and perpetuation of myofascial trigger points (TrPs) as found by Hains (2002), who suggested that these TrPs become activated during mechanical stress and overload of the involved shoulder musculature.

Often, these 2 clinical entities are treated separately to achieve a clinical outcome yet Hammer (1991), suggests that the most valuable modality to treat chronic overuse soft tissue syndromes (irrespective of muscular or tendinous in origin) is friction massage to both regions.

In this respect Graston Technique instrument-assisted soft tissue mobilization (GISTM) is a form of soft tissue mobilization that is used to detect and release scar tissue, adhesions and fascial restrictions much like that of the work of Cyriax, who pioneered transverse friction massage (Carey-Loghmani, 2003).

It is therefore hypothesized that GISTM could address both components within the complex shoulder syndrome (i.e. myofascial and tendinopathy that occur concurrently). However past research, with respect to GISTM, is yet to show whether or not a series of treatments is needed or whether the hypothesized clinical effects are best achieved with one treatment therefore, this study aims to determine the short-term effects of GISTM in the treatment of the patients presenting with supraspinatus tendinosis. The results thereof will also be compared with the number and type of concomitant diagnoses that the patient presents with.

1.2 Aim of the Study

The aim of this study was to assess the short-term effect of Graston instrument-assisted soft tissue mobilization (GISTM) on supraspinatus tendinosis and its concomitant clinical findings.

The four main objectives were:

1.2.1 The first objective is to analyze and compare pre- (initial treatment) and 24 hour follow-up data, in patients with supraspinatus tendinosis after having received a single treatment with GISTM.

The first hypothesis:

Indicated that a single treatment is insufficient in treating patients presenting with supraspinatus tendinosis, in terms of subjective findings.

1.2.2 The second objective is to analyze and compare post- (initial treatment) and 24 hour follow-up data, in patients with supraspinatus tendinosis after having received a single treatment with GISTM.

The second hypothesis:

Indicated that a single treatment is insufficient in treating patients presenting with supraspinatus tendinosis, in terms of objective findings.

1.2.3 The third objective is to compare a single treatment to multiple treatments with GISTM in terms of subjective and objective clinical findings.

The third hypothesis:

Indicated that multiple treatments are necessary in treating tendinopathies.

1.2.4 The fourth objective is to assess patient improvement clinically with respect to the presence / absence in number and type of concomitant conditions.

The fourth hypothesis:

Indicated that patients with an increase in the number of concomitant conditions may take longer to improve, in terms of subjective an objective measurements

1.3 Rationale for the Study

Fascia or dense irregular connective tissue, is densely innervated by mechanoreceptors and when stimulated leads to tonus changes of related skeletal motor units (Schleip 2003: 11-19). The suprascapular nerve (SSN) provides the motor innervation of the supraspinatus and infraspinatus muscles, and the sensory and proprioceptive innervation of the glenohumeral and acromioclavicular joints, subacromial bursa, scapula, and posterior aspect of the shoulder joint (Reeser, 2003). Therefore, one could hypothesize that by stimulating the SSN there may be reflex changes that occur in the structures supplied by this nerve.

Hammer (1991) suggests that the most valuable modality to treat chronic overuse soft tissue syndromes is transverse friction massage, which acts to break down adhesions, inhibit nociception and reduce edema by releasing potent chemical mediators. It therefore stands to reason that GISTM, which is used to detect and release scar tissue, adhesions and fascial restrictions (Carey-Loghmani, 2003) and seems to address treatment in a similar fashion to that of transverse friction massage, would be an effective clinical modality.

However, there is little evidence available to support the use of any common intervention, in the management of a patient with shoulder pain (Green et al., 1998), in particular rotator cuff tendinosis, and specifically with the use of GISTM.

1.4 Limitations

GISTM is a relatively new form of treatment in the management of soft tissue injuries therefore, patient awareness and knowledge about its use is very limited. Hence, one needs to assume that patient expectations may be somewhat different to that of standard treatment methods, for example ultrasound therapy or dry needling, that are both frequently used in the treatment/management of soft tissue injuries. Also those subjects in the single treatment group may not think that one treatment is sufficient, and therefore the resultant outcome may be affected.

1.5 Conclusions

The aim of this study therefore, was to assess the short-term effect of Graston instrument-assisted soft tissue mobilization (GISTM) on supraspinatus tendinosis and its concomitant clinical findings.

Chapter two consists of a brief review of literature, followed by the research methodology and materials used (chapter three), and lastly the results and interpretation thereof in chapters four and five, resepectively.

Chapter Two

REVIEW OF RELATED LITERATURE

2.1 Introduction and overview of the chapter

The following chapter aims to review and describe: the relevant anatomy of the shoulder girdle, the most common soft tissue pathology found in the girdle, as well as the most current treatment methods available. Thereafter hypotheses will be presented on the effects of GISTM on supraspinatus tendinosis, in terms of objective and subjective findings, as well as an analysis of the number and type of concomitant pathologies.

2.2 Anatomy and function of the cuff

The supraspinatus, infraspinatus, teres minor and subscapularis muscles are referred to as the rotator cuff of the shoulder joint, because all the muscles (except supraspinatus) are rotators of the humerus at the shoulder joint (Moore, 1992; Prescher, 2000).

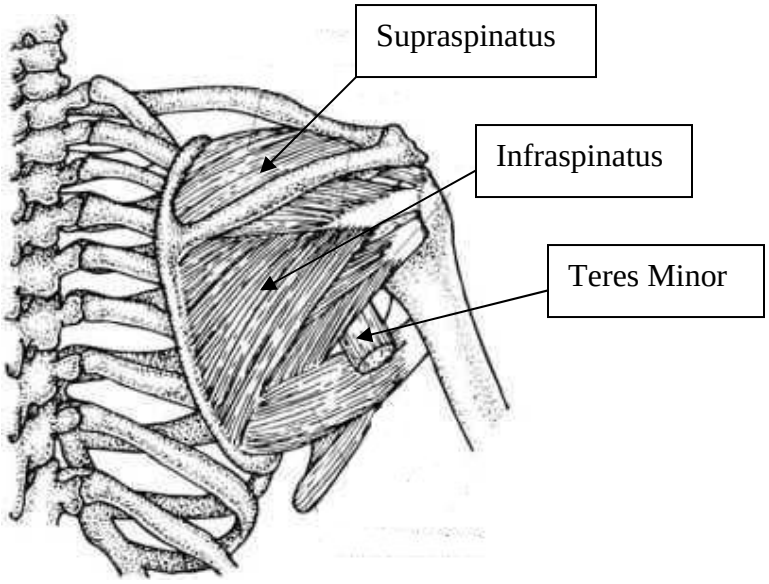


Image 1: Anatomy of the cuff (subscapularis omitted from the picture)
By Kibler, W.B (2005).

Table 1: The following table illustrates the attachments of these rotator cuff muscles:

Muscle	Origin	Insertion
Subscapularis	Arises from the medial and lower two-thirds of the axillary border of the subscapular fossa.	Fibres converge in a tendon, which is inserted into the lesser tubercle of the humerus.
Infraspinatus	Arises from fibers on the medial two-thirds of the infraspinatous fossa.	Fibres converge to a tendon and are inserted into the middle impression on the greater tubercle of the humerus.
Teres Minor	Arises from the dorsal surface of the axillary border of the scapula.	Fibres end in a tendon, which is inserted into the lowest of the three impressions on the greater tubercle of the humerus**.**
Supraspinatus	Arises from the medial two-thirds of the supraspinatous fossa.	Fibres converge to a tendon, which is inserted into the highest of the three impressions on the greater tubercle of the humerus.

(Table information from Gray, Pickering Pick & Howden, 1974; Moore, 1992 and Kieser & Allen, 1999)

Principally the supraspinatus assists the deltoid to abduct the arm and in conjunction with the other rotator cuff muscles (viz. infraspinatus, teres minor and subscapularis), helps hold the humeral head in the glenoid cavity (Norkin and Levangie, 1992; Moore, 1992 and Moore and Dalley, 1999). When the arm is elevated the cuff muscles work to depress the head of the humerus to prevent the deltoid muscle from jamming the upper end of the humerus against the acromion process (Nicholas and Hershman, 1995; Thomson A. et al., 1991).

The suprascapular nerve (SSN) arises from the superior trunk of the brachial plexus mainly at the C5 - C6 level, whereby it passes obliquely outwards beneath the trapezuis muscle in the posterior triangle of the neck (Gray, 1998; Moore, 1992). The nerve passes through the suprascapular notch, whereby it enters the supraspinous fossa giving off two motor branches to the supraspinatus muscle. The SSN also gives off a superior articular branch, proximal to the suprascapular notch, which travels with its fellow nerve through the notch before proceeding laterally to innervate the acromioclavicular joint and its associated bursa and the coracoclavicular and coracohumeral ligaments.

The suprascapular nerve therefore provides the motor innervation of the supraspinatus and infraspinatus muscles, and the sensory and proprioceptive innervation of the glenohumeral and acromioclavicular joints, subacromial bursa, scapula, and posterior aspect of the shoulder joint (Gray, 1998; Reeser, 2003).

From the above it is plausible to assume that there is a neurological link between the numerous structures supplied by the SSN, and could therefore hypothesize that stimulating the SSN may have an effect on all structures (as stated in the previous paragraph) supplied by the SSN (Reeser, 2003). In order to understand these structures and the pathologies that occur, each will be discussed in sequence below.

2.3 The shoulder pathologies

2.3.1 Incidence and prevalence

Shoulder pain is a common complaint with an estimated annual incidence of 6.6 to 25 cases per 1000 patients seen by primary health care practioners in general practice (Van der Windt et al., 1995). In support of this, Brukner (1996) and Arroyo et al., (1997) reported that rotator cuff tendonitis is a common cause of pain and dysfunction in the shoulder girdle, and is the most common shoulder problem in sports medicine, particularly in overhead throwing athletes.

Van der Windt et al. did a survey in 1995, whereby 35150 patients participated in an observational study by eighteen practitioners in a Dutch general practice, to determine the incidence and management of intrinsic shoulder disorders. Seven hundred and fifty four consultations took place over a year period, 472 of those patients presenting with shoulder complaints. Of this number, 392 patients presented with an incident complaint (defined as the number of new cases of a disease that occur in a population each year (www.legacyhealth.org/body.cfm, 2005)). Thus, there was an estimated 14.7 per 1000 per annum cumulative incidence of shoulder complaints. The peak age category was between 45 and 64 years of age, with complaints being greater in females than males. The disorder most frequently diagnosed amongst practitioners was subacromial impingement syndrome.

This concurs with the reporting of shoulder injuries, which accounted for 8% to 20% of volleyball injuries, where most were rotator cuff and/or biceps tendinosis caused by overuse (Briner et. al., 1999). In addition and concurrence to this, 38-75% of competitive swimmers reported having a history of shoulder pain, while 9-35% of these swimmers were currently experiencing pain (McMaster and Troup, 1993).

Furthermore, rotator cuff tendinosis is also found in laborers involved in repeated overhead activities (i.e. among shipyard welders and steel plate workers), were the prevalences of 18, 3% and 16, 2% exist respectively (Herberts et al., 1984).

2.3.2 Aetiology of rotator cuff tendinosis

Even in light of the relatively high incidence and prevalence, and according to Dela Rosa et al. (2001) the exact cause and pathogenesis of rotator cuff tendinosis is still unclear however, there are three main theories that exist, which include:

- ***Overuse activity.***
  The overuse mechanism, especially seen in athletes involved in repetitive overhead motions that causes microscopic failure of the tendon fibrils leading to injury (Dela Rosa et al., 2001).
- ***The intrinsic mechanism and***
  The intrinsic mechanism involves injury with in the tendon itself from either direct tendon overload and/or breakdown of the cuff (Budoff et al., 1998; Uhthoff et al., 1998).
- ***The extrinsic mechanism***
  Lastly, the extrinsic mechanism involves tendon damage by compression against surrounding structures (Neer, 1972; Bigliani and Levine, 1997).

2.3.2.1 Overuse

According to Dela Rosa et al. (2001), chronic overloading of the affected tendon leads to microscopic failure of the fibrils and eventual tendon injury.

Fricker and Hoy (1995) suggest that the principal cause of tendinosis of the rotator cuff muscles is repetitive microtrauma due to overfatigued muscles or weakening of the rotator cuff and scapulothoracic muscles.

2.3.2.2 Intrinsic causes

The main contributing factors to rotator cuff disease include: cuff tendon degeneration, avascularity, direct tendon overload or repetitive trauma (Hertling and Kessler, 1996; Faber, et al., 1998).

Many authors have described a “critical” zone of relative hypovascularity within the supraspinatus tendon (Hawkins and Kennedy, 1980; Loehr et al., 1990; Souza, 1994 and Dela Rosa et al. 2001). This area is located nearby the tendon’s insertion onto the greater tuberosity. It is this “critical” zone that gets compressed under the coracoacromial arch when the arm is abducted. Therefore those participating in repetitive overhead activities, whether it is recreational or work-related, who constantly place the arm in the abducted position, do not allow the critical zone to be able to repair itself properly leading to process a continuous injury and eventual tendon degeneration (Hertling and Kessler, 1996; Souza 1994).

This cycle is depicted in the theoretical cycle below.

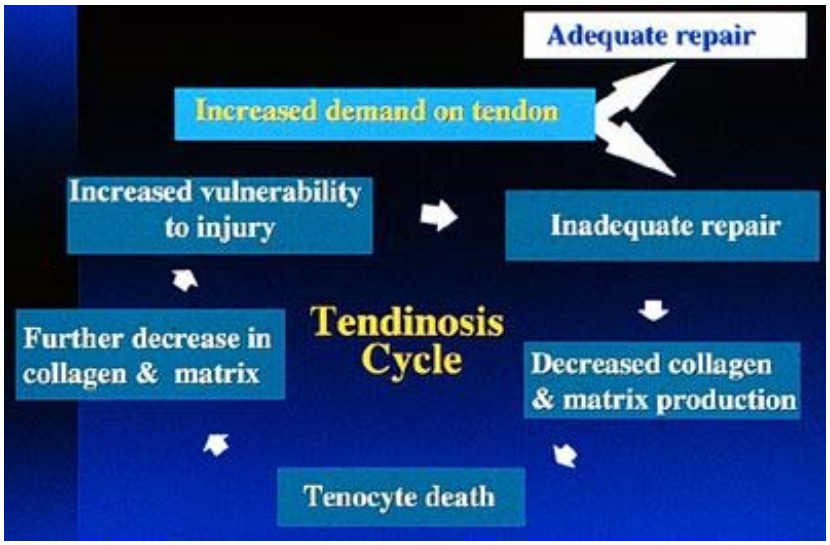


Figure A: A theoretical model of the tendinosis cycle taken from Khan et al., 1999

2.3.2.3 Extrinsic causes

Tendon injury here is mainly through compression of the supraspinatus tendon against a surrounding structure, namely the coracoacromial arch (Neer, 1983; Dela Rosa et al., 2001). The arch is composed of the coracoid process, the coracoacromial ligament and the anterior part of the acromion. Contents of the supraspinatus outlet include: the rotator cuff tendon, the subacromial bursa and the long head of the biceps brachii muscle (Michener et al., 2003; Dela Rosa et al., 2001). Therefore any abnormalities in this outlet can result in an impingement syndrome and rotator cuff tendonitis (Chang, 2004).

Acromial morphology has been also been implicated as a possible cause of impingement, and are classified into three main types; flat (type 1), curved (type 2) and hooked (type 3). Most commonly associated with rotator cuff disease is the type 3 or hooked-type of acromion (Neer, 1983; Bigliani and Levine, 1997; Chang, 2005).

In addition to the above structural anatomic constraints in terms of the structures that lie within the area, their interaction in terms of movement, especially with repetitive overhead activity, also plays a role. Here the dynamic and static stabilizers of the shoulder joint are placed under great stress, therefore predisposing the shoulder to injury (Chang, 2005; Malanga, 2004). The dynamic stabilizers are consequently fatigued sooner and there is altered movement between the involved humerus and scapula, resulting in a narrowing of the suprapspinatus outlet and hence causing impingement of various structures (Bigliani et al. 1997; Faber, et al., 1998).

Thus it would seem that the 3 theories are interrelated and affect one another, as tendon degeneration cannot happen without impingement of the tendon and vice versa and muscle fatigue could be a cause or a result of the development of a tendinosis. With this in mind the following will discuss the pathology of tendinosis.

2.3.3 Pathology of rotator cuff tendinosis

The inflammatory cascade that is initiated and results in rotator cuff tendinopathy, is brought about by mechanical impingement whether it is a primary (direct trauma) or secondary cause (repetitive microtrauma to surrounding structures), as stated by Fu et al., (1995) and Myers (1999). The end result of the inflammatory cascade is granulation or scar tissue formation (Hammer, 1991).

The scar is primarily produced by fibroblasts that lay down collagen and increase the strength of the wound. The random orientation in which the fibres are laid down often hinder function but do restore the structure of the tendon itself (Hunter, 1994). Due to this loss of function, the resultant tissue may be irritated / overloaded again and a vicious cycle of microtearing – inflammation – scarring – microtearing – inflammation –scarring occurs (Hammer, 1991).

Therefore Hunter (1994) suggests the orientation of the collagen fibres of the wound is vitally important and should resemble that of the undamaged tissue in order to maximize the tensile strength of the wound and prevent the cyclical degeneration.

2.3.4 Examination and presentation

Rotator cuff tendonitis can clearly be diagnosed and classified on the basis of the history and physical examination (Faber et al., 1998).

According to Faber et al. (1998), patients often localize the pain in the front and to one side of the shoulder, which often radiates down the upper arm to the elbow, but not past it. Patients may also report pain, particularly at night and when sleeping on the affected side. Pain is usually aggravated by activity where the elbow is level with the shoulder.

Chang (2004) states that the MRI is the imaging study of choice for shoulder pathology. The MRI is able to detect intrasubstance tendon degeneration or partial rotator cuff tears. Neer (1983) stated that the most reliable method for detecting full rotator cuff tears is arthrography. More recently, ultrasonography is being widely accepted (Blankstein et al., 2005) and used amongst practitioners due to its accuracy in demonstrating full-thickness tears (Faber et al., 1998). However, these are not always practical in terms of their use especially with respect to accessibility, affordability and thus most diagnoses are based on clinical acumen.

Table 2: Neer's (1983) stages of Rotator cuff disease include the following:

Stage 1	"Edema and hemorrhage." Characteristically observed in patients younger than 25 years of age. Result of repetitive overhead use in work or sports.
Stage 2	"Fibrosis and tendonitis." Characteristically found in the older athlete, 25 – 40 years of age, whereby the shoulder becomes symptomatic after excessive overhead activity. There is increase fibrosis and thickening of the subacromial bursa.
Stage 3	"Tears of the rotator cuff, biceps rupture, and bone changes." These lesions are found almost always in patient's older than 40 years of age. With further impingement wear, incomplete or complete tears of the rotator cuff, biceps lesions, and bone alterations at the acromion and greater tuberosity may occur.

On examination, tenderness of the supraspinatus tendon is found when palpated (Hammer, 1991). In this respect, Hammer (1991) suggested the two most common sites of involvement are just before insertion on the greater tuberosity and the musculotendinuous junction. In addition there is pain on full passive abduction to 180° whereby the tenoperiosteal junction is compressed against the roof of the glenoid.

Performing manual muscle testing and the relevant orthopaedic tests also reproduces symptoms.

These include (Hammer, 1991):

- Painful arc between 60° and 120° of abduction
- Pain and relatively strong on resisted abduction
- Possible pain on resisted lateral rotation
- Possible pain on active and passive abduction to 180°
- Possible pain on stretching the supraspinatus muscle

Whilst the patient is being assessed with regard to the clinical symptomatology, it is recommended that the following differentials be excluded in order to arrive at an appropriate diagnosis. Furthermore, these differentials are included in order to assist the reader of this study in understanding the possible concomitant diagnoses that the patient may have presented with and which will be discussed in chapter four with the results obtained in this study.

2.4 Differential diagnoses may include (Chang, 2004):

2.4.1 Acromioclavicular Joint Injury

Acromioclavicular (AC) joint injury is usually as a direct result of trauma to the shoulder, particularly the acromion. Patients present with an acute or chronic episode of anterior or superior shoulder pain (Quillen et al., 2004). Many individuals experience nocturnal pain and awaken when rolling onto the involved shoulder much the same as seen in rotator cuff pathology. Clinically, there may be swelling, bruising and localized tenderness over the AC joint (Byran, 2002).

2.4.2 Bicipital Tendonitis

Patients typically complain of anterior shoulder pain, which is exacerbated with overhead activity or with lifting heavy objects. Most are as a result of impingement or as an isolated inflammatory injury but also may be linked to rotator cuff disorders, labral tears, and intra-articular pathology. Passive abduction of the arm in a painful arc maneuver typical of impingement syndrome may elicit pain; however, this finding may be negative in isolated biceps tendonitis (Durham, 2005).

2.4.3 Brachial Plexus Injury

Usually the direct result of trauma to the brachial plexus can lead to the "stinger" or "burner" syndrome, which is classically characterized by a burning sensation that radiates down an upper extremity. Cervical spine injuries are common in contact sports, and may lead to motor and / or sensory deficits to various areas of the upper limb depending on the nerve root/s that are affected (Vaca, 2004).

2.4.4 Cervical Disc Injuries

These injuries are often the result of axial applied forces, with secondary forces of hyperflexion, hyperextension, and rotation adding to the overall injury pattern. Cervical disc injuries are relatively common in athletes involved in both contact and non-contact sports. Athletes with symptomatic cervical disc injuries commonly present with segmental neck pain, muscle spasm, loss of ROM, and referred pain in both radicular and nonradicular distribution. Nerve root involvement leads to radicular upper extremity pain, weakness, and sensory changes (Windsor, 2005).

2.4.5 Cervical Radiculopathy

Cervical radiculopathy is a dysfunction of a nerve root of the cervical spine usually as result of a disk herniation or an acute injury causing foraminal impingement of an exiting nerve. Patients report a reduction in their radicular

symptoms by abducting their shoulder and placing their hand on / behind their head (Malanga, 2004).

2.4.6 Cervical Spine Sprain/Strain Injuries

Strain refers to an injury to a muscle that occurs when a muscle-tendon unit is stretched or overloaded (www.bd.com/elastics/glossary/, 2005). Sprain refers to a ligamentous injury (www.back.com/s.html, 2005), and the diagnosis of cervical sprain implies that the ligamentous and capsular structures connecting the facet joints and vertebrae have been damaged. Clinically, it may be difficult to differentiate a sprain from a strain as these 2 entities may occur simultaneously in this region.

Neck motion is often painful with pain referral especially to the occipital area or the shoulder girdle; however, there is no radiation of pain or paresthesia in any of the patient's extremities (Malanga, 2004).

2.4.7 Clavicular Injuries

Clavicle fractures may be caused by direct trauma, such as a fall directly onto the bone, or from indirect trauma, such as falls onto the lateral shoulder causing a compressive force across the clavicle. There is usually a clear history of trauma and the pain and deformity is obvious to the practitioner (Ertl, 2004).

2.4.8 Infraspinatus Syndrome

Infraspinatus syndrome is defined as a condition of frequently painless atrophy of the infraspinatus muscle caused by a suprascapular (SSN) neuropathy (Reeser, 2005). Symptoms mimic that of rotator cuff tendinopathy, and the diagnosis is often overlooked. The typical patient is a young athlete who reports vague posterior shoulder pain that is aggravated by activities that involve overhead motions or sport-specific skills (Reeser, 2005).

2.4.9 Myofascial Pain in Athletes

Patients with active myofascial trigger points (TrPs) usually complain of poorly localized, regional, aching pain in subcutaneous tissues, including muscles and joints. The myofascial pain is often referred away from the TrP in a pattern that is characteristic for each muscle (Travel and Simmons, 1999). Sometimes, the patient is aware of numbness or paresthesia rather than pain (Travel and Simmons, 1999). Patients may have other associated clinical symptomatology such as rotator cuff pathology and cervical radiculopathy (Facco et al., 2005) that may be mistaken for a myofascial pain and dysfunction syndrome (Bruno, 2004).

2.4.10 Rotator Cuff (RC) Injury

RC injury represents a spectrum of diseases, ranging from acute reversible tendonitis to massive tears involving the supraspinatus, infraspinatus, and subscapularis muscles. Pain is often felt over the anterolateral part of the shoulder and is exacerbated by overhead activities. Pain, weakness, and loss of shoulder motion are common symptoms reported with rotator cuff pathology. Night pain is a frequent symptom, especially when the patient lies on the affected shoulder (Malanga, 2004).

2.4.11 Shoulder Dislocation

Up to 96% of shoulder dislocations are traumatic in nature, with 85% of these being anterior displacements. An acutely dislocated shoulder is severely painful and the patient may not be able to move the joint (Bryan, 2004).

2.4.12 Shoulder Impingement Syndrome

Neer (1983) first introduced the concept of rotator cuff impingement to the literature, stating that it results from mechanical impingement of the rotator cuff tendon beneath the anteroinferior portion of the acromion, especially when the shoulder is placed in the forward-flexed and internally rotated position.

Impingement and rotator cuff disease are increasingly more common in athletes whose sport involves repetitive overhead motions (Chang, 2005).

2.4.13 Superior Labrum Lesions

Superior labrum anterior posterior (SLAP) lesions are described as a labral detachment, originating posterior to the long head of biceps insertion and extending anteriorly. Patients often present with nonspecific shoulder pain with overhead or cross-body activities. Popping, clicking, or catching at the shoulder joint are often commonly reported. Patients may describe a deep, vague pain within the shoulder joint in association with weakness or stiffness (Williams, 2005).

2.4.14 Swimmer's Shoulder

Swimmer's shoulder is the term used to describe the problem of shoulder pain in the competitive swimmer. A spectrum of overuse injuries is seen in the swimmer's shoulder, the most common of which is rotator cuff tendinitis**.**
The patient may be able to localize the area of pain to the posterior shoulder, may describe the pain as being deep. The patient less commonly localizes the pain anteriorly, as well as at the deltoid insertion area of the upper arm (Ho, 2005).

2.5 Treatment opportunities for rotator cuff tendinosis

2.5.1 Relative rest

Non-operative management of rotator cuff tendinosis begins with rest and cessation of activities that cause pain, allowing the injured tendon sufficient time to repair itself (Dela Rosa et al., 2001). When dealing with athletes in particular, this period should be emphasized as an active form of rest. Khan et al., (1999), explain in their extensive research into the histopathology of common tendinopathies that in a tendinosis there is structural damage to the tendon that

may include partial tearing of the collagen, which may require a longer healing period. Tendons have a slow metabolic rate, therefore it is postulated that tissue repair may take months rather than weeks. The remodelling phase of tissue healing lasts from three weeks to twelve months or more. This stage involves fibroblastic activity and fibrosis (Carey-Loghmani, 2003:31; Vizniak, 2003:165).

However, complete immobility of the affected limb should be avoided due to the increase likelihood of developing TrP's in the shoulder girdle musculature (Travell and Simons, 1999).

2.5.2 Strengthening

Recent research by Hömlich et al. (1999) shows that there is definite scientific evidence to support the clinical effectiveness of eccentric strengthening programs. Manual loading of soft tissue is said to increase fibroblastic proliferation, which synthesizes and maintains collagen, fibronectin, proteoglycans, and other proteins of connective tissue matrix (Hammer, 1991).

On the converse however, a complete tear or disruption of the tendon should undergo surgical intervention prior to active use (DeLee and Drez, 1994).

2.5.3 NSAIDs and Steroid injections

The use of anti – inflammatory drugs is quite commonly used to control pain and inflammation however, their effectiveness is questionable because they have no therapeutic effect in tendinosis, which is a non-inflammatory condition (Khan et al., 1999). A further question asked, is to whether or not the antiprostaglandin and COX – 2 inhibition actions of the drugs actually helps or hinders the healing process (Khan et al., 2000).

The same, therefore, can be said about the use of corticosteroid injections, knowing that tendinosis is not an inflammatory condition; the use of steroid medication is questionable (Khan et al., 2000). According to Dela Rosa et al., (2001), the use of steroid injections is a common practice, yet intratendinous

injections are thought to have no place in the management of tendon disease, as steroid injections are known to have deleterious effects such as inhibition of collagen synthesis, weakening of the tendon and tissue necrosis, especially when injected directly into the affected tendon.

2.5.4 Modalities

Van der Heijden et al., (1997) in a systematic computerised literature search, evaluated previous articles in which patients were treated with physiotherapy for disorders of soft tissue of the shoulder. Trial sizes were small: only six trials included intervention groups of more than 25 patients. Ultrasound therapy, evaluated in six trials, was not shown to be effective. Four other trials favoured physiotherapy (laser therapy or manipulation), but the validity of their methods was unsatisfactory. In conclusion, there is evidence that ultrasound therapy is ineffective in the treatment of soft tissue shoulder disorders.

Thus, is would seem that the recommendations by DeLee and Drez (1994), where they suggested that electrotherapeutic modalities such as ultrasound, transcutaneous nerve stimulation, muscle stimulation and laser therapy should only be instituted according to a positive patient response to these modalities, is more appropriate. They are mainly pain control modalities and should be incorporated into the therapist's treatment protocol accordingly.

2.5.5 Cryotherapy

Souza (1994) states the use of cold therapy is important for three major reasons:

1) A cold compress is effective in reducing swelling. The inflammatory process is slowed down by the effect of the cold on the local metabolic rate.
2) There is a reduction in pain through a decrease in neural conduction.
3) It is theorized that there is reflex inhibition, whereby cold allows muscles to be stretched further with resetting of the muscle spindle.

Due to the chronicity of most overuse shoulder injuries/disorders, the application of cold therapy is mostly used for pain inhibition rather than its anti – inflammatory properties. Cryotherapy is used as an adjunct to other treatments and can also be done effectively at home by the patient for pain control.

2.5.6 Surgery

Surgery must be considered as a last resort for tendinopathies, and in this case, tendinosis. Tissue affected by tendinosis can be excised, but there seems to be no evidence to support the fact that surgery stimulates collagen synthesis or maturation (Khan et al., 2000). In advanced cases surgery is often indicated and an arthroscopy is done to evaluate the extent of tear, if any, in order to treat associated pathology and decompress the subacromial region (Curtis and Wilson, 1996). In an article by Arroyo et al., (1997) it was found those athletes that had undergone subacromial decompression, that they had significant relief of pain; however they were unable to return the same level of activity. This subsequently has limitations in terms of return to sport / complications.

Thus all the above modalities have a restriction or complication in respect of the application of the modality either as a single entity or in a combined manner.

However, with cross friction these limitations are not as apparent, although it is recognized that cross friction stimulates the inflammatory process and can be associated with ecchymosis or some clinical discomfort to the patient. This modality was discussed in chapter one, however it is discussed in further detail below.

2.5.7 Cross – friction massage or Soft tissue mobilization

Hammer (1991) suggests that the most valuable modality to treat chronic overuse soft tissue syndromes is friction massage. Deep friction massage has long been supported by the work of Cyriax (1984). Cyriax (1984) believed that friction massage induced:

- Local hyperemia, causing the release of chemical mediators namely bradykinin and histamine, resulting in vasodilatation and reduced edema (Hammer, 1991).
- The breakdown of old disorganized scar tissue and adhesions (Cyriax, 1984 and Hertling and Kessler, 1996).
- Local anaesthesia, thought to be due to gate control inhibition (Melzack and Wall, 1965) with large fibre proprioceptor stimulation inhibiting the small fibre nociceptors. Friction anaesthesia is also thought to be due to a counter-irritation response of central inhibition through endorphin release (Souza, 1994).

Hammer (1991) states that a mechanical stress applied to injured tissue is required for remodelling the weak scar tissue with fibres parallel to the line of tension. Hertling and Kessler (1996) likened the effect of friction massage to that of rolling your hand over an unorganized pile of toothpicks. The toothpicks eventually all become orientated perpendicular to the direction in which the hand moves. Cyriax (1984) believed that moving across the fibres as in deep (transverse) friction massage would not injure the normal healing but would however break down scar tissue and prevent adhesion formation.

Graston Technique instrument-assisted soft tissue mobilization (GISTM) is a form of soft tissue mobilization that is used to detect and release scar tissue, adhesions and facial restrictions. GISTM is also used to induce local hyperemia and mechanically mobilize scar tissue much like that of the work of Cyriax (1984). Davidson et al., (1997) concluded that in their study on rat tendons, the group that received friction massage with a hand-held tool, proliferation of fibroblasts was promoted and hence the tendon healed faster. Wilson et al.,

(2000) also concluded in their study on patellar tendinitis, that 100% of the augmented soft tissue mobilization (ASTM) group and 60% of the traditional friction massage group had resolution of the problem at the end of the study, hence concluding that ASTM therapy is superior to traditional friction massage in treating patellar tendonitis.

Therefore it is hypothesized that GISTM introduces:

- a small amount of trauma, which induces the inflammatory reaction to start over again. The inflammatory response is responsible for fibroblast proliferation, which increases collagen synthesis (Carey-Loghmani, 2003). The aim being, to correct the orientation of the collagen fibres so that they resemble original tissue, hence regaining maximal tensile strength and optimal functioning (Hunter, 1994). Davidson et al., (1997) concluded that in their study on rat tendons, the group that received friction massage with a hand-held tool, proliferation of fibroblasts was promoted and hence the tendon healed faster,
- a neurological effect is also hypothesized, as the stimulation of mechanoreceptors having an end result of a decrease in sympathetic activity via reflex spinal, supraspinal reflexes and feedback loops (Schleip, 2003:104-116).

As the GISTM is not only applied over the tendon, but also the muscle belly of the supraspinatus muscle, the following discussion centres around pathologies that are associated with the muscle in patients that have supraspinatus tendinosis.

2.6 Short summary of shoulder pathologies

A common complaint seen by Chiropractors in general practice is that of shoulder pain and dysfunction (Hains, 2002). Pathology in the rotator cuff muscles (supraspinatus, infraspinatus, subscapularis and teres minor) is always a possibility when a patient presents with shoulder pain (Evans et al., 1998,

Faber et al., 1998), in addition the rotator cuff tendinosis is second to acromioclavicular joint disorders, as the most common cause of shoulder pain (Dela Rosa et al., 2001).

According to Hains (2002), rotator cuff and supraspinatus tendinosis, impingement syndrome and subacromial pain syndrome are readily interchangeable terms when diagnosing patients presenting with pain in the anterolateral region of the deltoid. This pain then radiates into the upper arm, commonly from myofascial pain and dysfunction in one of the muscles associated with the supraspinatus tendinosis.

2.7 Myofascial trigger points

In this respect, Myofascial pain syndrome (MPS) or pain caused by myofascial TrPs, is defined as a regional muscular disorder (Lee et al.,1997), and represents a leading cause of musculoskeletal pathologies (Chaitow and Delany, 2002; Hains, 2002). It usually occurs as a result of acute overload, chilling of the area, overworked or fatigued muscles and gross trauma (Travell and Simons, 1999), which are similar aetiological factors that present in patients with supraspinatus tendonosis. A muscular or fascial lesion, which heals by fibroblastic proliferation together with the irregular laying down of collagen (if left untreated) leads to the maturation stage of tissue healing. Myofibroblasts are responsible for scar shrinkage and contracture (Carey-Loghmani, 2003). This hypertonic fascia then traps and squeezes nerve receptors, along with blood and lymph vessels. This creates and activates trigger points (Cowie, 2003).

Therefore it is hypothesized that myofascial trigger points (TrP's) will be evident in the shoulder musculature in patient's presenting with an overuse or repetitive strain injury (e.g. supraspinatus tendinosis). According to Travell and Simons (1999: 546), patients presenting with suprapinatus TrP's may have associated Trp's in muscles in the corresponding functional unit (i.e. the infraspinatus, upper trapezius, deltoid and lattismus dorsi muscles).

2.7.1 Incidence and prevalence

Individuals of any age or gender can develop myofascial TrPs, (Travell and Simons, 1983: 13), as found in a study by Sola et al., (1955), where among 200 asymptomatic young adults; he found that there were latent trigger points in the shoulder girdle muscles of 54% of the females and 45% of the males.

The prevalence with regard to MPS in the general population can only be estimated in the general population (Bruce, 1995). This is due to the fact that most of the research conducted at the moment on the epidemiology of MPS has been completed in a clinical setting.

The cause of overuse muscle pain is thought to be microtrauma that outpaces the capacity of the muscle for repair. The end result of hypertonic muscles leads to hypoxia, acidosis, and metabolic depletion, followed by calcium-mediated cellular damage. In overhead athletes and laborers, continued or repetitive use of fatigued muscles causes mechanical damage that is directly related to the heaviness of the work (Sola et al., 1955; Bruno, 2004).

2.7.2 Presentation

Clinically the TrP is painful on compression and can give rise to characteristic referred pain, autonomic phenomena and motor dysfunctions - including (Travell, Simons and Simons, 1999:5 and 21):

- Spasm of other muscles,
- Weakness of the involved muscle function,
- Loss of co-ordination and
- Decreased work tolerance of the involved muscle.

The most reliable diagnostic criteria of MTrp's on examination of the muscle is the presence of exquisite tenderness at a nodule in a palpable taut band (Travell,

Simons and Simons, 1999:117). The weakness results from a “guarding mechanism” reflex motor inhibition and characteristically occurs without atrophy of the affected muscle (Travell and Simons, 1999:21), but results in dysfunction and faulty movement patterns (Lawrence et.al., 1996:74).

2.7.3 Treatment

2.7.3.1 Trigger point release (Travell and Simons, 1999:126)

In order to obtain long-term relief from MPS, a treatment protocol must take into account both the contributing and perpetuating factors (Esenyl, et al, 2000:51).

There are many different forms of treatment for MPS and these include:

- Spray and stretch – (Travell and Simons, 1999:127)
- Voluntary contraction and release/relaxation techniques – (Travell and Simons, 1999:138)
- Trigger point pressure release – (Travell and Simons, 1999:140)
- Deep stroking and massage – (Travell and Simons, 1999:141)
- Myofascial release – (Travell and Simons, 1999:143)
- Modalities e.g. T.E.N.S – (Travell and Simons, 1999:146)

Therefore, the main goals of conservative management, for a patient with myofascial pain syndromes, are to eliminate muscle spasm, weakness, loss of coordination and decreased work tolerance in order to avoid pain and disability that results when the impingement syndrome manifests (Travell and Simons, 1999:21). This approach also allows for the restoration of shoulder function in the shortest possible time.

Inflammation, active range of motion deficits, and neuromuscular control, should also be addressed (Myers, 1999), in order to avoid further regression to an impingement syndrome.

2.7.4 Short summary

Many authors (Chang, 2004 and Myers, 1999) have suggested that secondary impingement can be caused by soft tissue or rotator cuff overload and muscle imbalances that may exist. Consequently, one could postulate that myofascial TrP's could be related to the development of impingement syndrome, and it would be necessary to inactivate these trigger points as a part of the practitioner's treatment protocol. Treating the TrPs alone will not be sufficient as both entities are related, so therefore both need to be addressed.

There is still little evidence to support the use of any common intervention (especially those with limited side effects) in the management of patients with shoulder pain (Green et al., 1998), specifically that of GISTM. Van der Heijden et al., (1997), recently found that the evidence to support the use of ultrasound therapy is ineffective in the treatment of soft tissue shoulder disorders; in fact, Johansson et al., (2005) reported that acupuncture is more effective than ultrasound when applied in addition to home exercises, in patients diagnosed with impingement syndrome.

2.8 The purpose of this research.

Thus in part due to the lack of research and also due to the clinical need for improved management of supraspinatus impingement (tendinosis) syndromes, the researcher has been unable to establish whether, like cross friction massage a series of treatments are needed or whether the hypothesized clinical effects are best achieved with one treatment with GISTM. In addition to this, the application of the GISTM occurs over both the muscle and the tendon(s) involved and therefore it is possible that by resolving the 2 aspects of the syndrome concurrently that the patients experience an improved recovery.

Therefore this research aimed at determining the short-term improvement of patients presenting with supraspinatus tendinosis when treated with Graston

instrument-assisted soft tissue mobilization (GISTM) and assess how this improvement may be influenced by the presence or absence of concomitant conditions.

Chapter Three

MATERIALS AND METHODS

This chapter aims to describe the research methodology as well as the materials used in collecting data and their analysis. Patients were assessed and both subjective and objective measurements were recorded at three separate intervals over a 3-week period. In addition the subjects' improvement was noted with regard to the concomitant number, and type of associated clinical findings.

3.1 Design

This study was designed as a pilot pragmatic clinical investigation based on comparison between a "treatment group" and a "measurement group." The treatment group received six treatments using the Graston tools, whereas the measurement group received a single treatment using the Graston tools and no further treatment.

Key variables

A comparison between a single treatment and several treatments was drawn from the following variables:

- Torque ratios
- Range of motion
- Pain
- Associated clinical findings (e.g. biceps tendinosis, instability, MFTP's, etc.),

Which constituted the primary data and secondary data constituted that found in the literature as published in books, journal articles, specialist internet search sites (e.g. Pubmed, Mantis).

3.2 Sampling

3.2.1 Method

Advertisements were posted around the D.I.T campus, in the local newspaper and placed at various sporting clubs in the greater Durban area (Appendix A). Thus patients were obtained by means of consecutive convenience sampling (Mouton, 2002). Random allocation was utilised to allocate the patients into either of the two groups before or after the assessment for the inclusion and exclusion criteria (see 3.3.1 and 3.3.2) (Mouton, 2002:143).

Treatment commenced on a “first-come, first-serve” basis as soon as it was convenient for both researcher and patient.

3.2.2 Size and Allocation:

Being a pilot study, all subjects accepted into the study were randomly split into two equal groups of 20 patients each, giving a total of 40 patients. This is in congruence with the study of Cowie (2003) and the noted epidemiology reported in that study (Cowie, 2003). Twenty labels representing “treatment group” and twenty representing “measurement group” were placed in an envelope and each patient was asked to draw out a label to determine which group they were assigned to (Mouton, 2002: 143). In congruence with the study being a pilot study, it was noted that the numbers of patients accepted into the study presented an inherent limitation in ability of the research to be able to detect small changes (in terms of statistical significance).

3.3 Research Methodology

Patients, who presented to the D.I.T Chiropractic Clinic with shoulder pain, were considered as potential candidates for the study and underwent a face-to-face interview in order to determine their eligibility for the study.

Patients telephonically contacting the clinic underwent a telephonic interview in order to rule out unsuitable candidates.

In both instances questions pertaining to age, duration and area of pain, disability from the injury, occupation, etc. were asked in order to ensure maximal compliance with the inclusion and exclusion criteria.

At the initial consultation, the patients were then screened for inclusion by conducting a case history (appendix D), physical examination (appendix E) and shoulder regional examination (appendix F) to determine whether they were suitable subjects for this study.

3.3.1 Inclusion Criteria:

- All subjects chosen were between the ages of 18 and 40 years of age. Firstly to avoid the need for parental consent, and secondly to help rule out causes of rotator cuff pathology found exclusively in the older population (older than 40 y.o.a) (Neer, 1983:70-77), in addition to assisting the researcher to achieve sample homogeneity (Mouton, 2002)
- Only those with subacute (more than 48 hours after initial injury), and chronic (more than 5 days after the initial injury) were considered (Reid, 1992) in order to achieve sample homogeneity (Mouton, 2002).
- Only patients diagnosed by the researcher as having rotator cuff tendinosis were considered. This was evaluated according to the following tests:
    - Positive Orthopedic tests: Hawkins-Kennedy and Empty can. (Vizniak, 2002).
- Functional testing of the patient had to indicate supraspinatous tendinosis, which concurred with and included Hammer's (1991) criteria of:
    - Pain and relatively strong on resisted abduction
    - Possible pain on resisted lateral rotation
    - Possible painful arc (between 60° –120° of abduction)

- Possible pain on active and passive abduction to 180°
- Possible pain on stretching the supraspinatus muscle

For the purpose of this study, patients had to have at least 4 out of 5, of the above criteria to be included in the study, in order to ensure an accurate diagnosis of supraspinatus tendinosis was made. As no one orthopaedic test has been shown to be useful and no one test guarantees a diagnosis or rates severity consistently (Laslett and Williams, 1994), the researcher was tasked to make an informed decision regarding the clinical relevance of a particular test, its validity, reliability, specificity and sensitivity (Walsh, 1998). Thus, in this research, it was suggested that multiple tests triangulated to arrive at a clinical diagnosis was the best and most accurate in the clinical situation (Walsh, 1998).

- In addition to the above patients, had to have an NRS rating of between 6 and 10, to further achieve sample homogeneity (Mouton, 2002:136), in terms of the clinical symptomatology therefore allowing more accurate recordings of patient improvement and therefore comparisons between the two patient groups.
- An informed consent form (appendix C) had to be signed by the patient in order to allow them entrance into the study.
- Participants were also required to stop all pain and anti-inflammatory medication at least 48 hours before the study (Poul et al., 1993), and not use any medication during the course of the study, in order that these interventions did not result in skewed data and to achieve accurate results.
- A total of 40 patients were included in this study. If a participant fell out due to other commitments and was unable to continue with this study, they were then replaced until data from 40 participants had been collected. Exclusions for reasons of worsening clinical symptoms were however noted.

3.3.2 Exclusion Criteria:

1) Patients had to meet the inclusion criteria in order to be accepted into the study.
2) Those who did not sign the consent form were not able to take part in the study, due to the lack of consent provided.
3) Patients with contraindications to GITSM as stated by Carey-Loghmani (2003) were excluded:

   Red flags (absolute contraindications) included:

   Open wounds – unhealed suture sites, unhealed fractures, thrombophlebitis, uncontrolled hypertension, patient intolerance / hypersensitivity, haematoma, osteomyelitis, myositis ossificans.

   Yellow flags (relative contraindications) included:

   Anti- coagulant medications, cancer, varicose veins, burn scars, acute inflammatory reactions, kidney dysfunction, inflammatory condition secondary to infection, Rheumatoid Arthritis, Osteoporosis.
4) Further clinical tests where utilised to exclude differential diagnoses that would also presented as shoulder pain and included amongst others C5/6 radiculopathy and brachial plexus injuries (Travell and Simons, 1999:544).
5) If there was a history of traumatic shoulder dislocation or if there was a positive drop arm test that could indicate a rupture of the rotator cuff, the patient was excluded.
6) If there was a history of shoulder surgery.

Those subjects who had not met the inclusion criteria were referred to other interns at the Chiropractic Day Clinic for treatment of their condition, as paying outpatients.

3.4 Intervention

Following the applicant telephonic screen, clinical assessment and group allocation, the patient received GISTM to the involved tendon, musculotendinous portion and muscle (supraspinatus muscle) with the desired GISTM tool for 30 seconds to a maximum of 1 minute.

The tool of choice to treat the cuff region is the GT-3 or the “tongue depressor”, whereby a pencil grip is adopted and either brush stroke or strumming is recommended (Hyde, 2003) over the region.

The patient was seated with the area exposed and the affected arm placed supported in a prolonged horizontal adduction position during the treatment session (Carey-Loghmani, 2003).

Treatment is recommended twice a week with two days between sessions and to maintain maximum benefit, treatment over period of 10 – 14 days is suggested (Carey-Loghmani, 2003). However there is very little evidence in the literature to support the above treatment protocol, hence a need for this study and the choice of the following treatment schedule outlined in table 1:

Week	Visit	Graston – single treatment	Graston – multiple treatments
1	1	Tx	Tx
	2	-	Tx
2	3	-	Tx
	4	-	Tx
3	5	-	Tx
	6	-	Tx

Tx = treatment

3.5 Measurements

Patients were required to attend the following:

The "measurement group" was only seen in "Week 1", and with a follow up reading at "Week 3", whereas the "intervention group" was required to attend all 8 of the consults scheduled over the 3 week period as indicated in table 2.

Week	Visit	Graston – single treatment – measurement group	Graston – multiple treatments – intervention group
1	1 DIT	Clinical assessment	Clinical assessment
	2 (King's Park Medical Centre)	Readings **Tx**	Readings **Tx**
	3 (King's Park Medical Centre)	24 hour readings -	24 hour readings **Tx**
2	4 DIT	-	**Tx**
	5 DIT	-	**Tx**
3	6 DIT	-	**Tx**
	7 DIT	-	**Tx**
	8 (King's Park Medical Centre)	Readings	Readings

Tx = Treatment

Readings = both subjective and objective findings (as detailed under 3.5.1)

DIT = Durban Institute Chiropractic Day Clinic

Clinical assessment = Included initial case history, physical examination and shoulder regional examination (see Appendix D, E and F respectively).

3.5.1 Objective measurements

3.5.1.1. Torque ratios

The movements that were measured included;

- Internal and External rotation (Moore 1992, Reid, 1992),
- Abduction in keeping with the prime movements of the rotator cuff muscles (Moore 1992, Reid, 1992),
- As well as adduction, this is a required movement to create a torque ratio with abduction (Appendix J: cybex testing protocol).

Before starting the isokinetic testing, the subjects were asked to complete a numerical pain rating scale (appendix H). They would then undergo a 3 to 5 minute rotator cuff warm up including internal, external rotation as well as abduction and adduction. They were then positioned onto the cybex where would undergo a 'practice round' in order to familiarize them with the procedure.

According to Wilk (1990:123-150) and Davies (1987) the procedure for the Concentric – Concentric test is as follows:

- Sub-maximal warm-up repetitions at 90 degrees/sec
- 1 min rest
- 2 trial repetitions of maximal effort at 60 degrees/sec
- 1 min rest
- 3-5 repetitions of maximal effort at 60 degrees/sec
- 4 min rest

Peak torque values for concentric internal/external rotation, as well as abduction/adduction were recorded. For statistical purposes, single repetition work values for individual subjects were combined and an average was taken (total work) for each individual subject in each concentric test and expressed as a ratio.

This protocol was adapted from: Davies (1992:43-4), Perrin (1993:48) Wilk et al., (1991:63-70) and Wright (2003).

For over 20 years, independent clinical research has proven Cybex isokinetic testing to be accurate, objective, reproducible and safe. More than a 1000 published articles, studies and presentations have shown Cybex systems to provide objective measurement of impairment and documentation of rehabilitation effectiveness (Cybex, 1996:1-9).

Chan and Maffulli (1996:22-3) report correlation co-efficients between 0.93 and 0.99 when using an Isokinetic Dynomometer (no p-value stated). Davies (1992:35) states that several studies have been conducted confirming the reliability and validity of the Cybex (no p-value stated).

3.5.1.2. Shoulder range of motion - the goniometer

Goniometric readings of the shoulder joint, which include abduction, internal and external rotation were also taken by the researcher in order to assess range of motion (Appendix I).

The procedure for the use of the goniometer was as follows:

- The patient was asked to stand with both feet positioned flat on the floor and the arms positioned in the anatomical position at the sides.
- Abduction was measured as the angle between the arm and the thoracic rib cage as the arm moves upwards and outwards towards the ear.
- External rotation was measured with the patient lying supine, the elbow bent to 90º, arm held at 90º abduction, and the palm facing outwards.
- Internal rotation was measured with the patient lying supine, the elbow bent to 90º, arm held at 90º abduction, and the palm facing inwards.

A total of three readings (i.e. abduction, internal and external rotation) were taken on three separate consultations. Measurements were taken from the above-mentioned postions. The results were recorded in degrees (º) of range of motion.

Hayes et al., (2001) showed that examiners from various clinical backgrounds were more consistent in measuring full elbow flexion and full wrist extension positions with a goniometer than by estimating the same angles by eye. While there is some evidence to suggest that the goniometer can be used reliably for measuring certain shoulder joint movements (Boone et al., 1978).

3.5.1.3. Associated clinical findings e.g. Instability, Myofascial Trigger Points and Tendinopathy of other muscles.

Clinical findings associated with the supraspinatus tendinosis were also assessed and recorded appropriately at each consult to note any possible changes that may occur throughout the duration of the study (Appendix I).

These were listed and noted on the data sheet (Appendix I), then translated onto the SPSS spreadsheet. The different clinical entities were numerically coded for statistical purposes in order to allow for evaluation of their influence on the clinical outcomes of the study.

3.5.2 Subjective readings

3.5.2.1 The **Numerical Pain Rating Scale** (NRS 101) was used to determine the subjective pain intensity experienced by the patient. The patient was then to record their perceived level of pain on a numerical scale ranging from 0 to 100, with 0 representing no pain at all and 100 representing pain at its worst (Jensen et al., 1986). (See Appendix H).

This scale was chosen because of the ease with which it can be administered and scored. Jensen et al., (1986) established its validity and reliability when proving subjective information about the levels of pain perceived by the patient. It was used to monitor the patient's progress with a decrease in pain intensity indicating improvement. For statistical purposes, the data collected from the NRS scales were added together and divided by two in order give an average score to get a single number for statistical analysis.

3.5.2.2 The **Shoulder Disability Scale** (Appendix G) will be used to determine the subjective disability experienced by the patient. Scores are allocated to both the amount of activity the patient is able to perform and the pain associated with the activity (Gartsman, 1990).

Williams et al., (1995) measured shoulder dysfunction using the Shoulder pain and disability index (SPADI). The SPADI was shown to accurately distinguish between patients who improved, those who stayed the same and those who worsened.

For statistical purposes the data collected from the disability scale was added up, first according to pain, and then function. Both the pain and function subscale have 12 items. The difference lies in the fact that pain is rated out of a score between 0 (complete disability) and 5 (no pain). Function on the other hand, is rated out of a score between 0 (unable) and 4 (normal). Therefore the pain subscale has a total of 60, whereas the function subscale gives a total of 48. The higher the score for each category indicates the less pain and better the function of the shoulder region there is.

3.6 Statistical analysis

Data analysis was done in SAS version 9.1 (SAS Institute Inc., Cary, NC). Baseline comparisons between the categorical baseline variables and the group to which the participant was assigned were done using Fisher's exact test. Continuous baseline variables that were not normally distributed were compared between groups using a non-parametric Wilcoxon Mann-Whitney test. Continuous normally distributed baseline data were compared using the two-sample t-test.

The follow-up measures were summarised by group and visit. The change from Visit 1 to Visit 3, as well as the change from Visit 1 to Visit 2, is compared using the t-test for paired observations. The collected data was analysed using the SPSS package, whereby the patient population was described utilizing descriptive analysis (frequency tables), which may be represented visually as bar charts, pie charts or frequency tables.

All data was tested for normality. If the data collected followed a normal distribution, parametric testing was applied. The Paired T-Test was used for intra-group analysis and the Independent Paired T-Test for inter-group analysis. If the data follows a non-normal distribution, non-parametric testing was applied. Intra-group analysis was assessed using the Wilcoxin test for matched pairs, whilst the Mann Whitney U test was used for inter-group analysis. The Pearsons Product Moment Correlation Coefficient was applied to test for the significance of a relationship across both the subjective and objective tests. The significance level was set at $\alpha = 0.05$. The appropriate p-values were used for decision-making.

Logistic regression analysis and covariance statistics were utilized to ascertain the association between the various factors. Contingency co-efficients were done to assess the strength of the relationships.

Chapter Four

RESULTS

4.1) Introduction

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

Primary Data

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

Secondary Data

This particular data came from many different sources, which included:
Textbooks, Journal articles and specialist internet search sites (e.g. Pubmed, Mantis)

Key of symbols

N = number
% = percentage
SD = standard deviation
p-value = probability value

4.2) Demographics

Forty participants who met eligibility criteria were randomized into two groups: a "treatment group" and a "measurement group." All patients that were excluded from the study where patients that where excluded on the basis of the inclusion and exclusion criteria and not for purposes worsening clinical symptomatology. All participants in the study that entered the research study completed the research programme and thus there were no drop outs in either of the groups that needed to be accounted for.

The treatment group received six treatments using the Graston tools, whereas the measurement group received a single treatment using the Graston tools and no further treatment:

Group 1 (N = 20) "treatment group"

Group 2 (N = 20) "measurement group"

Demographic characteristics were compared between the two groups to ensure that the randomization process was complete in eliminating confounding variables between the groups.

4.2.1) Gender

There were 35 males (87, 5%) and 5 females (12, 5%). There were significant differences between the proportions of each gender in each group (p = 0.0471[1] – See Table 1).

Table 3: Gender and group

		Group 1		Group 2		
		N	**%**	**N**	**%**	**p-value**
Gender	Male	20	100.0	15	75.0	0.0471[1]
	Female	0	0	5	25.0	

Fisher's exact test for comparison between Group 1 and Group 2

The only baseline variable that showed a difference between the groups was gender, with all five females being enrolled in Group 2 and none in Group 1.

A possible reason for a greater number of male participants in this particular study is that there is a greater population of male athletes when it comes to water sport activities, viz. swimming, waterpolo and canoeing (Fricker and Hoy, 1995: 412).

*It is therefore noted that there may be a difference between the groups based on the M: F difference (table 1).

4.2.2) Race

The majority of the participants in the study were White (95%), with only one Indian (2, 5%) and one Colored participant (2, 5%).

This ratio does not accurately reflect the race distribution of South Africa, and in particular the province of Kwa-Zulu Natal (http://www.statssa.gov.za/census2001/digiAtlas/index.html, 2006).

The inversed ethnic ratio demonstrated may be due to a number of different reasons.

Firstly, the African population of KZN may not be familiar with Chiropractic, which is derived from a western culture. Secondly, participants in this study were required to make a number of trips to the Chiropractic clinic and the Kings Park Medical Centre, where the treatments and objective measurements were conducted. This may have resulted in a sample that is representative of only the highly mobile portion of the population, rather than the population in general.

Nevertheless, there was no significant difference in the racial distribution between the groups ($p = 1.000$ – see Table 2) and therefore the possibility that

the racial distribution between the groups would affect the outcomes is limited if present at all.

Table 4: Racial distribution by group

		Group 1		Group 2		
		N	**%**	**N**	**%**	**p-value**
Race	Indian	0	0	1	5.0	1.000[1]
	Caucasian	19	95.0	19	95.0	
	Coloured	1	5.0	0	0	

Fisher's exact test for comparison between Group 1 and Group 2

4.2.3) Age, weight and height

Table 5: Comparison of mean age, weight and height between the two groups

	N	Mean	SD	Minimum	Median	Maximum	p-value
Age (years)							
Group 1	20	28.3	6.85	18	26	40	0.8709[1]
Group 2	20	28.0	7.01	19	25	39	
Weight (kg)							
Group 1	20	84.9	11.1	68	86	115	0.4092[2]
Group 2	20	80.8	19.2	51	83.5	112	
Height (cm)							
Group 1	20	185.2	8.4	170	186.5	204	0.0642[2]
Group 2	20	179.9	9.2	159	180	194	

[1] Wilcoxon Mann-Whitney test for comparison between Group 1 and Group 2

[2] t-tests for independent groups for comparison between Group 1 and Group 2

According to Arroyo et al., (1997) and Jobe and Kvitne, (1989) one can categorize shoulder injuries into two groups, namely:

1. The *young overhead athlete* (18 to 35 years of age)
2. The *older overhead athlete* (older then 35 years of age)

The *young overhead athlete* is susceptible to problems involving micro-trauma to the glenohumeral joint static stabilizers, which may lead to instability and subluxation, secondary impingement and eventually rotator cuff tears.

The *older overhead athlete* suffers more from degenerative processes, which can result in mechanical impingement on the coracoacromial arch and associated coracoacromial ligament thickening and spur formation, which can cause narrowing of the subacromial space.

The mean age of participants was 28 yrs, although the ages of the participants in the study ranged from 18 to 40. It is therefore most likely that the individuals in this study would have had shoulder pain from micro-traumatic injury because the mean age was below 35 years of age. This implies that both groups tended towards the same stages in the pathogenesis of their respective conditions, which would have assisted in achieving homogeneity between the groups, thereby strengthening the outcomes reached at the end of the study (Mouton, 2002).

Similar trends are seen in respect of height and weight in this study, however there is no comparative data in the literature with which to compare these outcomes.

Table 6: Comparison of involved/injured side and sporting activity.

		Group 1		Group 2		
		N	**%**	**N**	**%**	**p-value**
Side/Shoulder	Left	4	20.0	6	30.0	0.7164[1]
	Right	16	80.0	14	70.0	
Sporting activity	Swimming/water polo	9	45.0	6	30.0	0.4273[1]
	Paddler or canoeist	2	10.0	5	25.0	
	Tennis or squash	1	5.0	1	5.0	
	Weight training	3	15.0	6	30.0	
	Baseball or softball	1	5.0	1	5.0	
	Cricket	3	15.0	0	0	
	Occupational	1	5.0	1	5.0	

[1] Fisher's exact test for comparison between Group 1 and Group 2

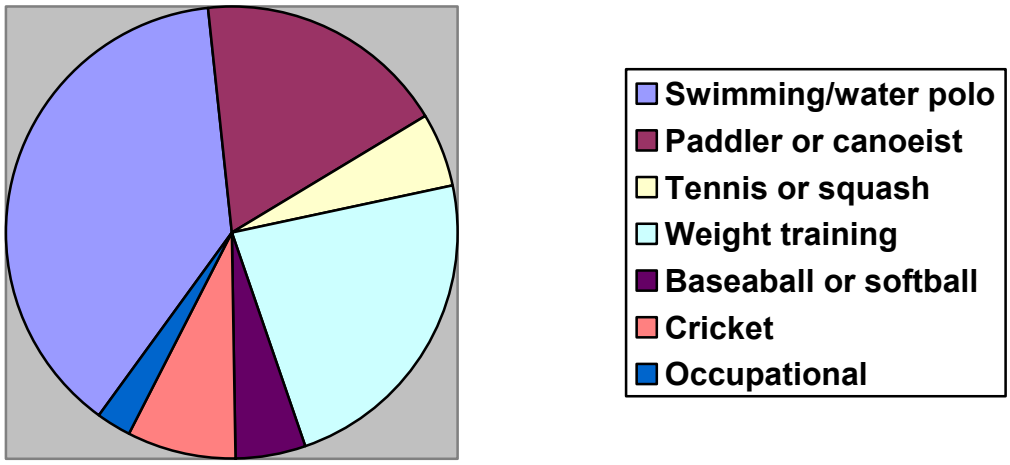


Figure B: Sporting activities of participants (N=40)

Seventy-five percent of the participants were right shoulder dominant (N=30) while 25% (N=10) were left shoulder dominant.

The main sporting activity the subjects took part in was waterpolo/swimming (37, 5%), followed by weight training (22, 5%). This is shown in Figure B.

Several of the participants competed at a provincial and/or national level for their sport and are required to do strength training and cardiovascular training to maintain optimum fitness. Therefore, besides their main sporting activity, many did numerous overhead activities. Owing to the high demands placed on the shoulder muscles of these athletes, the dynamic and static stabilizers of the shoulder joint are placed under great stress (Chang, 2005; Malanga, 2004). They may therefore develop a painful shoulder (Kennedy, 1980), where the dynamic stabilizers are fatigued sooner and thus altered movement between the involved humerus and scapula was affected, resulting in a narrowing of the suprapspinatus outlet and hence causing impingement of various structures (Bigliani et al., 1997; Faber, et al., 1998).

In addition, swimming requires repetitive internal rotation of the shoulder. This involves a high-resistance pull-through phase evident in most strokes, which leads to strengthening of the internal rotators without specific training of the external rotators. This creates an imbalance between these two groups of muscles and results in early fatigue of the external rotators. This fatigue may become chronic and the athlete then becomes susceptible to an overuse injury such as impingement as a result of the dysfunctional scapular musculature, (Souza, 1994:118).

As a result of the above, it would appear that the only significant difference between the groups would have been that of the participants' handedness. The participation in sports (although the principle sport was noted) indicated that the majority cross trained in order to maintain cardiovascular fitness, strength and / or endurance, thus predisposing the majority of the groups to very similar conditions in terms of the sports participation.

There is no current literature to state that the handedness of an individual will have an effect on outcome on the results.

Please see Appendix M for a summary of outcome measures obtained from both groups.

4.3) Subjective Data

4.3.1) NRS-101

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	38.75	11.22	20.00	40.00	65.00
2	37.13	9.08	20.00	40.00	60.00
3	28.13	11.15	5.00	25.00	50.00
Change Visit 1 to 2	-1.63	4.46	-17.50	0.00	5.00
Change Visit 1 to 3	-10.63	11.69	-40.00	-10.00	15.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	36.75	10.64	15.00	35.00	55.00
2	35.50	10.41	15.00	35.00	55.00
3	25.00	9.87	10.00	25.00	45.00
Change Visit 1 to 2	-1.25	5.53	-10.00	0.00	15.00
Change Visit 1 to 3	-11.75	10.76	-40.00	-10.00	5.00

The mean pain measurements in Group 1 decreased from 38.8 at Visit 1 to 28.1 at Visit 3, and in Group 2 decreased from 36.8 at Visit 1 to 25.0 at visit 3. The p-value for the change from Visit 1 to Visit 3 is 0.7533 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.8147 (paired t-test), which does not indicate a significant group effect.

There is an overall decrease in pain levels experienced by both groups. The mechanism by which GISTM works is by reducing scar tissue, aiding

inflammation and allowing healing and the tissue remodeling process to begin (Carey-Loghmani, 2003:31, 51-62).

By aiding the inflammatory process, GISTM allows the injury to progress to the later stages of healing and finally to resolution. In doing so there is an improvement in the level of the patients' pain (Carey-Loghmani, 2003:31; Hammer, 2001).

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

A possible reason for the reduction in pain in group 2 only after a single treatment could be that the scar tissue or adhesions that were detected in the supraspinatus tendon were broken down and the patient was able to regain increased mobility. According to Hammer (2001), once the vicious cycle of microtearing-inflammation-scarring is broken, there will be an increase in mobility and extensibility of the tissue. An increase in extensibility means an increase in functional ability and stimulation of the "Gate control theory" (Melzack and Wall, 1965:971).

The reduction that occurred in both groups may also be accounted for by natural history, which involves the natural resolution of an injury over time (Lachmann and Jenner, 1994:28).

4.3.2) The Shoulder Disability Scale (Shoulder pain and disability index (SPADI))

Scores are allocated to both the amount of activity the patient is able to perform (function) and the pain associated with the activity (Gartsman, G.M. 1990).

4.3.2.1) SPADI (function)

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	42.15	4.16	33.00	43.00	47.00
2	41.80	3.89	33.00	43.00	47.00
3	43.40	4.58	31.00	45.00	48.00
Change Visit 1 to 2	-0.35	2.58	-10.00	0.00	5.00
Change Visit 1 to 3	1.25	4.04	-11.00	1.50	8.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	43.25	3.64	34.00	45.00	47.00
2	43.85	3.13	34.00	45.00	47.00
3	45.35	2.35	39.00	46.00	48.00
Change Visit 1 to 2	0.60	1.73	-1.00	0.00	7.00
Change Visit 1 to 3	2.10	3.57	-4.00	1.00	12.00

The mean SPADI function in Group 1 increased from 42.2 at Visit 1 to 43.4 at Visit 3, and in Group 2 increased from 43.3 to 45.4. The p-value for the change from Visit 1 to Visit 3 is 0.4848 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.1795 (paired t-test), which does not indicate a significant group effect.

The overall increase in scores, even as slight as they may be, indicates that patients reported an increase in function. A possible reason for the slight decrease (-0.35) that occurred in Group 1 may be that the treatment given may have potentially caused some bruising and discomfort (Carey-Loghmani, 2003: 37) as stated in the letter of information (Appendix B).

This is as a result of the GT aiming to correct the orientation of the collagen fibres so that they resemble original tissue, hence regaining optimal functioning and with time, maximal tensile strength utilizing the appropriate rehabilitation techniques as pertinent to the patient's ability (Hunter, 1994). In order to achieve this effectively the GT induces inflammatory response, which is responsible for fibroblastic proliferation, which increases collagen synthesis (Carey-Loghmani 2003). According to Hertling and Kessler (1996) and Hammer (1991), friction massage may assist in restoring mobility between tissue interfaces or may increase the extensibility of individual structures. This may be said to be directly linked to functional ability of the involved structures, which in this case, refers to the improved function of shoulder complex (as indicated by the trends even though they are insignificant).

There is, however, a slight discrepancy (Group 1 increased from 42.2 at Visit 1 to 43.4 at Visit 3, and in Group 2 increased from 43.3 to 45.4) in the overall improvement in functional ability reported between the groups. A possible reason for this may be due to repeated treatments received by Group 1, whereby the inflammatory cycle is re-initiated at every follow up visit. As a result the patient may have increased pain and tenderness as a known possible side effect of GISTM (Carey-Loghmani, 2003: 36) thus reporting their improvement as smaller increments over time.

It is suggested that a future study consider a follow up one month after the cessation of the treatment to assess the SPADI (function), as this time period would allow for the inflammatory responses induced by treatment to subside and

may therefore give a more accurate indication of the functional improvement present in the patient.

4.3.2.2) SPADI (pain)

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	49.15	7.92	28.00	52.00	57.00
2	49.40	7.11	34.00	52.00	57.00
3	51.85	7.03	34.00	52.50	59.00
Change Visit 1 to 2	0.25	1.48	-2.00	0.00	6.00
Change Visit 1 to 3	2.70	6.67	-14.00	2.00	15.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	52.20	5.73	39.00	55.00	57.00
2	52.85	5.01	39.00	55.00	57.00
3	54.70	4.75	44.00	57.00	60.00
Change Visit 1 to 2	0.65	2.48	-2.00	0.00	10.00
Change Visit 1 to 3	2.50	3.12	-4.00	2.00	11.00

The mean SPADI pain in Group 1 increased from 49.2 at Visit 1 to 51.9 at Visit 3, and in Group 2 increased from 52.2 to 54.7. The p-value for the change from Visit 1 to Visit 3 is 0.9043 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.5399 (paired t-test), which does not indicate a significant group effect

There is an overall decrease in pain levels experienced by both groups (noted by an increase in SPADI scores). The reduction that occurred in both groups may be accounted for by natural history, which involves the natural resolution of an injury over time (Lachmann and Jenner, 1994:28).

It would seem that the effect that GISTM has on pain and function could be attributed to a number of factors. Firstly, by breaking down adhesions in the supraspinatus tendon, GISTM may assist in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62).

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

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

As a result of the inclusiveness of the changes between with groups with respect to the pain levels, it is suggested that the following be considered in follow up studies:

- One month follow up to re-evaluate the changes that have been effected.
- An increased period of treatment to optimise the degree of scar tissue breakdown (suggested by Carey-Loghmani (2003:28) is 4 to 8 sessions for acute / subacute conditions, with chronic conditions taking longer, as many as 9 – 14 treatments.)
- An increased time between treatments to decrease the effect of the previous treatment (i.e. inflammation induction by the treatment with GT may have resulted in elevated pain levels that where indicated, which reflected more closely the induced inflammation as opposed to the treatment effect).

4.4) Objective data

Table 7: The following outlines the muscles that make up the functional unit of shoulder and their innervation: (Travell and Simons, 1983: 555-556 and Moore, 1999: 710-711 and Magee, 1997:235-236).

Actions	Muscles Acting	Nerve Supply	Nerve Root Derivation
External rotators	• Infraspinatus • Posterior Deltoid • Teres Minor	• Suprascapular • Axillary • Axillary	• C5-C6 • C5-C6 • C5-C6
Internal rotators	• Pectoralis Major • Anterior Deltoid • Subscapularis	• Lateral Pectoral • Axillary • Subscapular	• C5-C6 • C5-C6 • C5-C6
Abductors	• Deltoid • Supraspinatus	• Axillary • Suprascapular	• C5-C6 • C5-C6
Adductors	• Pectoralis Major • Latissimus dorsi • Teres major	• Lateral Pectoral • Thoracodorsal • Lower Subscapular	• C5-C6 • C6-C7 • C6-C7

4.4.1 Isokinetic Values

Both Peak Torque and Total Work values for each individual movement will be discussed below.

4.4.1.1) Peak torque: Abduction

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	45.35	13.97	25.00	45.00	84.00
2	48.85	13.82	30.00	46.00	76.00
3	52.80	15.72	33.00	49.50	84.00
Change Visit 1 to 2	3.50	9.05	-15.00	3.00	19.00
Change Visit 1 to 3	7.45	9.27	-11.00	7.50	24.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	41.50	20.91	6.00	44.00	98.00
2	45.25	23.20	8.00	43.00	98.00
3	43.30	21.13	14.00	40.00	90.00
Change Visit 1 to 2	3.75	9.32	-13.00	2.00	30.00
Change Visit 1 to 3	1.80	11.10	-17.00	1.00	34.00

The mean Peak Torque abduction in Group 1 increased from 45.4 at Visit 1 to 52.8 at Visit 3, and in Group 2 increased from 41.5 to 43.3. The p-value for the change from Visit 1 to Visit 3 is 0.0886 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.9318 (paired t-test), which does not indicate a significant group effect.

4.4.1.2) Total Work: Abduction

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	42.90	17.73	17.00	43.00	78.00
2	44.15	18.71	15.00	44.00	78.00
3	48.45	19.64	17.00	50.00	86.00
Change Visit 1 to 2	1.25	9.19	-16.00	0.00	21.00
Change Visit 1 to 3	5.55	8.62	-11.00	6.50	22.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	33.70	18.16	3.00	35.50	85.00
2	38.30	20.45	7.00	35.00	77.00
3	38.20	21.78	9.00	35.50	94.00
Change Visit 1 to 2	4.60	10.37	-11.00	1.00	28.00
Change Visit 1 to 3	4.50	10.17	-11.00	3.00	26.00

There was a large difference in total work abduction between the two groups at the start of the study. A possible reason for this is that all five of the female participants were in group two. According to Fricker and Hoy (1995), males have an increased muscle mass-producing more force during activity.

This makes the comparison of change during the study between the groups difficult. The mean total work abduction in Group 1 increased from 42.9 at Visit 1 to 48.5 at Visit 3, and in Group 2 increased from 33.7 to 38.2. The p-value for the change from Visit 1 to Visit 3 is 0.7266 (paired t-test), which does not indicate a significant group effect. Although the change from Visit 1 to Visit 3 was almost the same in both treatments, the values at Visit 1 was much higher for Group 1, indicating that the groups were not comparable at the start of treatment. The p-value for the change from Visit 1 to Visit 2 is 0.2863 (paired t-test), which does not indicate a significant group effect.

Nevertheless it is important to note that the supraspinatus and deltoid muscles are equally responsible for producing abduction at the shoulder joint (Jacobson et al., 1989), whereby Arroyo et al., 1997, suggest that the supraspinatus merely assists the deltoid in abducting the arm.

For research purposes the involved supraspinatus muscle belly, the musculotendinous portion and its tendonous insertion were treated and changes were noted.

After injury, tensile strength is significantly reduced; therefore a major role in the healing process is to restore this new tissue to full strength so that the patient is able to return to full activity. Hunter (1994) suggests the key aim in the treatment of soft tissue lesions is to encourage the damaged tissue to regain tensile strength. The tensile strength gained is not due to the new collagen formation but it is rather due to the intra- and extra- molecular cross-linkage between the collagen fibres (Hunter, 1994). Alleviating the supraspinatus of adhesions and scar tissue formation with GISTM, the inflammatory cascade is initiated and fibroblastic proliferation takes place, and hence new collagen is synthesized (Hammer, 1991), facilitating the renewal of cross linkages between the newly developed collagen fibres.

There is an increase in both peak torque and total work values for both groups; however, Group 1 tends to show a better overall improvement with sustained effects. The improvement can be hypothesized to be caused by breaking the neural reflex circuit that is responsible for maintaining the patient's signs and symptoms (Leach, 1994:99). This is supported by research indicating that other possible causes for this decline could include the presence/ formation of a neural scar from a chronic overuse condition that causes lasting muscle inhibition (Leach, 1994:101).

Group 2, seems to show a slow decline after an initial rise. One would then question the sustained efficacy of a single treatment of GISTM on peak torque and total work values.

It is suggested that future studies consider utilising the EMG to measure the amount of muscle activity with respect to correlating this with the improved outcomes of peak torque and total work. This may allow for the researchers to more conclusively indicate whether the treatment affects the efficacy of neurological recruitment patterns of the motor end plates and thus the work or peak torque, or both.

4.4.1.3) Peak torque: Adduction

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	83.85	32.63	29.00	87.50	153.00
2	91.00	24.05	46.00	93.50	128.00
3	100.20	22.21	59.00	109.50	138.00
Change Visit 1 to 2	7.15	23.11	-32.00	9.00	62.00
Change Visit 1 to 3	16.35	19.57	-15.00	17.50	55.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	74.55	36.18	24.00	71.50	178.00
2	86.45	33.01	39.00	88.00	164.00
3	83.85	34.76	19.00	83.00	164.00
Change Visit 1 to 2	11.90	19.55	-14.00	4.50	67.00
Change Visit 1 to 3	9.30	20.03	-21.00	4.50	59.00

The mean Peak Torque adduction in Group 1 increased from 83.9 at Visit 1 to 100.2 at Visit 3, and in Group 2 increased from 74.5 to 83.9. The p-value for the change from Visit 1 to Visit 3 is 0.2673 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.4871 (paired t-test), which does not indicate a significant group effect

4.4.1.4) Total Work: Adduction

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	65.85	30.44	20.00	61.50	152.00
2	76.45	33.58	22.00	67.50	135.00
3	75.00	35.95	23.00	71.00	164.00
Change Visit 1 to 2	10.60	19.12	-17.00	7.00	44.00
Change Visit 1 to 3	9.15	18.77	-14.00	8.00	47.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	75.75	34.05	26.00	72.00	129.00
2	82.45	31.31	34.00	80.00	132.00
3	91.20	31.52	45.00	91.50	152.00
Change Visit 1 to 2	6.70	22.93	-24.00	3.50	76.00
Change Visit 1 to 3	15.45	18.49	-15.00	13.50	48.00

The mean total work adduction in Group 1 increased from 74.8 at Visit 1 to 91.2 at Visit 3, and in Group 2 increased from 65.9 to 75.0. The p-value for the change from Visit 1 to Visit 3 is 0.2916 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.5625 (paired t-test), which does not indicate a significant group effect.

There was a large difference in peak torque and total work adduction between the two groups at the start of the study. A possible reason for this is that all five of the female participants were in group two. According to Fricker and Hoy (1995), males have an increased muscle mass- producing more force during activity.

Notwithstanding the above, the main muscles involved in adduction at the shoulder are: pectoralis major, latissimus dorsi and teres major (see table 5). These muscles work as antagonists to the abductors (agonists).

Therefore it is important to note that there is an increase in both peak torque and total work values for both groups; however, Group 1 tends to show a better overall improvement with sustained effects. The improvement can be hypothesized to be caused by breaking the neural reflex circuit that is responsible for maintaining the patient's signs and symptoms (Leach, 1994:99). This is supported by research indicating that other possible causes for this decline could include the presence/ formation of a neural scar from a chronic overuse condition that causes lasting muscle inhibition (Leach, 1994:101).

Also, the removal of AMI in the agonists will allow the antagonists to work more effectively as a result of decreased pain and improved movement

Group 2 seems to show a slow decline after an initial rise. One would then again question the sustained efficacy of a single treatment of GISTM on peak torque and total work of the antagonist muscle group.

These results would imply that the effects as discussed above (4.4.1.1, 4.4.1.2, 4.4.1.3 and 4.4.1.4) for abduction and adduction are similar, which stands to reason as these muscles work as an agonist – antagonist unit (Travell and Simons (1999:546), in which the effect on one muscle would have a corresponding effect on the opposing muscle. It would therefore seem to suggest that the possible causes for the changes stem from the same or similar source.

4.4.1.5) Peak Torque: External Rotation

Group 1 (N=20)

Visit	Mean	SD	Minimum	Median	Maximum
1	27.25	5.60	16.00	27.50	38.00
2	27.55	6.79	16.00	28.00	40.00
3	29.95	8.12	12.00	29.50	49.00
Change Visit 1 to 2	0.30	4.67	-9.00	0.00	12.00
Change Visit 1 to 3	2.70	5.86	-9.00	2.50	21.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	26.00	10.77	6.00	26.00	47.00
2	24.65	10.02	8.00	25.00	47.00
3	26.15	11.79	9.00	25.00	55.00
Change Visit 1 to 2	-1.35	5.14	-11.00	0.00	6.00
Change Visit 1 to 3	0.15	4.15	-9.00	0.00	8.00

The mean Peak Torque external rotation in Group 1 increased from 27.3 at Visit 1 to 30.0 at Visit 3, and in Group 2 increased from 26.0 to 26.2. The p-value for the change from Visit 1 to Visit 3 is 0.1204 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.2948 (paired t-test), which does not indicate a significant group effect.

4.4.1.6) Total Work: External Rotation

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	30.30	9.63	14.00	29.50	59.00
2	31.10	9.48	12.00	33.00	54.00
3	30.75	10.37	8.00	31.00	49.00
Change Visit 1 to 2	0.80	5.71	-5.00	0.00	14.00
Change Visit 1 to 3	0.45	7.38	-17.00	1.00	16.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	28.20	12.95	7.00	24.50	57.00
2	26.60	10.81	7.00	28.00	45.00
3	27.40	15.26	10.00	24.00	76.00
Change Visit 1 to 2	-1.60	6.19	-12.00	0.00	10.00
Change Visit 1 to 3	-0.80	6.57	-9.00	-0.50	19.00

The mean total work external rotation in Group 1 increased from 30.3 at Visit 1 to 30.8 at Visit 3, and in Group 2 increased from 28.2 to 27.4. The p-value for the change from Visit 1 to Visit 3 is 0.5750 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.2103 (paired t-test), which does not indicate a significant group effect.

Spinal motor neurons are arranged into groups called motor neuron pools[1] and it is these motor neuron pools that are responsible for the force of muscle contraction of the group of muscles they supply (Hopkins and Ingersoll, 2000).

[1] The spinal motor neuron and the muscle fibres it innervates are called a motor unit and thus due to the generalised inhibitory effect of interneuronal activity, there is a reduction in motor unit recruitment and a reduction in force of muscle contraction

The main agonists of external rotation of the shoulder are the infraspinatus, teres minor and the posterior deltoid muscles (see table 5).

Notwithstanding the above, the external rotators are mainly innervated from the cervical level of C5-6. Due to the fact that the infraspinatus muscle and the supraspinatus muscle share the same motor innervation (Moore, 1999: 710-711 and Magee, 1997: 235-236), the suprascapular nerve (SSN), one could postulate that by stimulating the SSN at the supraspinatus muscle, a possible neurologically mediated effect may have taken place in the infraspinatus muscle.

It was Hamilton et al., (2004) who related the number of motor-units innervating a muscle positively to the strength[2] of that muscle. Thus it could be hypothesized that by stimulating a particular spinal nerve could have a positive effect on the motor units, by applying the theories proposed by Homewood (1977), Korr (Leach,1994) and Vernon et al., (2005). This could in turn imply that soft tissue mobilization, more in particular GISTM, may have an affect on the strength of the muscle innervated by those motor units.

In saying this, this could be the reason for the increase in peak torque values gained in group 1, as compared to group 2.

However the total work values did not report the same improvement. There are a number of factors that could have contributed to the weakening or inhibition of the external rotators:

- If the agonists had more TrPs then the antagonists initially, this would decrease their stretch range of motion and weaken them, which would

[2] Strength is defined by Lewis *et al.* (1991) as being a function of muscle cross-sectional area, motor-unit recruitment and neuromuscular coordination, which has the ability to develop force in a maximal-effort voluntary contraction of rested muscle. Isokinetic testing, using the Cybex Orthotron II, may be performed as a screening technique to determine any weakness or imbalance of the torque (force (in Newton's) x radius (in meters)) ratios of any of the major peripheral joints (Krukner *et al.* 2001, Maffulli 1996, Siqueira *et al.* 2002). Additionally it has provided valuable information for the evaluation of shoulder strength assessment (Scotville *et al.* 1997).

reflexively inhibit their antagonists, thereby weakening them further (Lawrence et al, 1996:73-74).

- The weakness may also be a type of guarding mechanism, in which the muscle is reflexely inhibited from full contraction because of pain (Schneider, 1995: 74).

Travell and Simons (1999:20-22) go on to say that TrPs cause disturbances in motor functions, which may include spasm of other muscles, weakness of involved muscle function, loss of coordination and decreased work tolerance.

4.4.1.7) Peak Torque: Internal Rotation

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	64.20	14.76	40.00	64.50	91.00
2	64.75	11.98	45.00	65.50	90.00
3	66.95	15.85	40.00	70.00	97.00
Change Visit 1 to 2	0.55	6.89	-10.00	-0.50	16.00
Change Visit 1 to 3	2.75	5.49	-5.00	3.00	12.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	56.65	21.43	19.00	51.00	99.00
2	58.30	20.83	22.00	57.00	98.00
3	57.90	20.10	24.00	62.00	101.00
Change Visit 1 to 2	1.65	7.59	-13.00	1.00	18.00
Change Visit 1 to 3	1.25	8.57	-18.00	0.00	20.00

The mean Peak Torque internal rotation in Group 1 increased from 64.2 at Visit 1 to 67.0 at Visit 3, and in Group 2 increased from 56.7 to 57.9. The p-value for the change from Visit 1 to Visit 3 is 0.5137 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.6340 (paired t-test), which does not indicate a significant group effect.

4.4.1.8) Total Work: Internal Rotation

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	71.75	20.32	40.00	69.50	124.00
2	76.35	18.49	40.00	78.00	114.00
3	74.55	24.48	34.00	76.50	128.00
Change Visit 1 to 2	4.60	9.94	-10.00	4.00	27.00
Change Visit 1 to 3	2.80	14.11	-24.00	6.00	28.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	63.90	28.09	22.00	54.00	126.00
2	64.65	25.62	21.00	66.50	110.00
3	63.35	26.92	29.00	62.00	134.00
Change Visit 1 to 2	0.75	12.54	-32.00	2.00	27.00
Change Visit 1 to 3	-0.55	11.48	-30.00	1.00	20.00

The mean total work internal rotation in Group 1 increased from 71.8 at Visit 1 to 74.6 at Visit 3, and in Group 2 decreased from 63.9 to 63.4. The p-value for the change from Visit 1 to Visit 3 is 0.4154 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.2887 (paired t-test), which does not indicate a significant group effect.

When compared to external readings, it can be seen that these values are somewhat higher.

There is once again quite a substantial difference at the start, mainly due to the fact of the presence of the female participants in Group 2. Another possible reason for this could be that the Cybex Isokinetic Dynomometer used at the Kings Park Medicine Centre, does not offer gravity correction for this specific test position (Wright, 2003). The weight of the lever handle would be a factor in elevating these ratios to a certain extent, although, according to Wright (2003) this would be extremely difficult to calculate.

The chief internal rotators are the pectoralis major, anterior deltoid and subscapularis muscles (see table 5). Numerous authors (Alderink and Kluck, 1986:163; Ivey et al., 1984:127) state that normal shoulder internal / external rotation ratios established through isokinetic testing are 3:2 or 150%. The ratio may also be expressed as an external / internal rotation ratio, giving a normative value of 2:3 or 66, 6% (Ivey et al., 1985:384-386). The most commonly used parameter in isokinetic testing is peak torque (Davies, 1992:53).

Owing to the fact that 37, 5% of the participants were swimmers or waterpolo players their repetitive internal rotation which entails a high-resistance pull-through phase in most strokes has lead to strengthening of the internal rotators. This is also evident in the peak torque and total work values of the Adductor muscle group.

Group 1 showed an increase in overall peak torque values which could have resulted from the decrease or disruption of scar tissue and adhesion formation, with a resultant improvement in the Hawkins-Kennedy test position (Vizniak, 2002). Anatomically, a reduction in scar tissue / inflammatory would allow smooth movement of the humeral head in the internally rotated position allowing the underlying structures to move freely as before the injury (Vizniak, 2002).

However, the total work values did not report the same improvement. There are a number of factors that could have contributed to the weakening or inhibition of the external rotators:

- If the agonists had more TrPs then the antagonists initially, this would decrease their stretch range of motion and weaken them, which would reflexively inhibit their antagonists, thereby weakening them further (Lawrence et al., 1996:73-74).
- The weakness may also be a type of guarding mechanism, in which the muscle is reflexely inhibited from full contraction because of pain (Schneider, 1995:74).

Travell and Simons (1999:20-22) state that trigger points within a muscle can cause disturbances in motor functions, which may include spasm of other muscles, weakness of involved muscle function, loss of coordination and decreased work tolerance.

Group 2, seems to show a slow decline after an initial rise. One would then again question the sustained efficacy of a single treatment of GISTM on peak torque and total work of the agonist muscle group.

This is supported by research indicating that other possible causes for this decline could include the presence/ formation of a neural scar from a chronic overuse condition that causes lasting muscle inhibition (Leach, 1994:101).

Based on the baseline difference between the groups where all five of the female participants were in group two. It is possible that the results achieved (Fricker and Hoy, 1995) reflect a bias where males have an increased muscle mass-producing more force during activity. Therefore the trends observed and ultimate hypothesis discussions need to be treated with caution.

4.4.2) Shoulder range of motion - the goniometer

According to Magee (1992: 97), the average ranges of motion are as follows:

Abduction	170-180 degrees
External Rotation	80-90 degrees
Internal Rotation	60-100 degrees

A decrease in the above mentioned ranges could be associated with rotator cuff impingement (Reid 1992:934), while an increased range could be associated with varying degrees of instability from the repetitive activity and overuse of both dynamic and static stabilizers.

4.4.2.1) Shoulder Abduction

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	123.00	17.50	80.00	130.00	150
2	122.50	17.51	80.00	130.00	150
3	126.00	18.18	80.00	130.00	160
Change Visit 1 to 2	-0.50	2.76	-10.00	0.00	5
Change Visit 1 to 3	3.00	9.79	-20.00	0.00	20

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	122.75	18.60	90.00	120.00	160
2	121.00	19.51	90.00	120.00	160
3	122.75	18.60	90.00	120.00	160
Change Visit 1 to 2	-1.75	5.45	-20.00	0.00	5
Change Visit 1 to 3	0.00	8.58	-20.00	0.00	20

The mean shoulder abduction in Group 1 increased from 123º at Visit 1 to 126º at Visit 3, and in Group 2 showed no change. The p-value for the change from Visit 1 to Visit 3 is 0.3092 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.3678 (paired t-test), which does not indicate a significant group effect.

Group 1 and 2 both reported a decrease in abduction from visit 1 to 2, -0.50 and -1.75 respectively which is a very slight decrease to comment on, however a possible cause for this decrease could be the initiation of the inflammatory cycle and slight discomfort after the first treatment (Carey-Loghmani, 2003: 36).

Receptors are divided into four groups according to their neurological properties, which include three corpuscular mechanoreceptors and one nociceptor - these are Type I, Type II, Type III and Type IV mechanoreceptors respectively (Wyke, 1985, as cited by Schafer in Schafer 1987 and Bergmann et al., in Bergmann et al., 1993 and Leach, 1995) and described as follows:

- Type I receptors are confined to the outer layers of the joint capsule and are stimulated by active or passive joint motions. They have a low threshold, making them very sensitive to movement and they are slow adapting, making the effects of movement long lasting.
- Type II receptors are found within the deeper layers of the joint capsule. They are also low threshold and are stimulated even with minor changes in tension. Unlike Type I receptors, however, type II receptors are very rapidly acting and cease firing when the joint stops moving.
- Type III receptors are absent from all the synovial spinal joints but are found in the intrinsic and extrinsic ligaments of the peripheral joints.
- Type IV receptors are composed of a network of free nerve endings as well as unmyelinated fibres. They are associated with pain perception and include many different varieties and wide ranges of sensations. They are present throughout the fibrous portions of the joint capsule and ligaments.

Golgi tendon organs (Type I) are found in all dense connective tissue. They are sensory receptors that respond to slow stretch by influencing (alpha) motor neurons via the spinal cord to lower their firing rate, and hence cause reduced muscle tone (Schleip 2003: 104-116).

Ninety percent of all Golgi tendon organs are situated in the muscular portion of the musculotendinous junction (Schleip 2003:104-116). By stimulating these sensory receptors with the GISTM, one could possibly influence a change in muscle tonus of the supraspinatus muscle and therefore affect range of motion within the shoulder joint, in particular shoulder abduction.

According to Hertling and Kessler (1996) and Hammer (1991), friction massage may assist in restoring mobility between tissue interfaces or may increase the extensibility of individual structures. This may be said to be directly linked to functional ability of the involved structures.

The above two mechanisms may have been the basis for the overall improvement in Group 1, where it seems that multiple treatments given, are better at addressing these mechanisms than a single intervention. The initial breakdown of scar tissue in group 2 may have resulted in regaining some range of motion, even as slight as the change may have been.

4.4.2.2) Shoulder External Rotation

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	85.50	14.23	50.00	90.00	110.00
2	86.50	13.68	50.00	90.00	110.00
3	91.00	12.73	65.00	92.50	110.00
Change Visit 1 to 2	1.00	2.62	0.00	0.00	10.00
Change Visit 1 to 3	5.50	8.09	-10.00	2.50	20.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	83.75	12.34	50.00	90.00	100.00
2	84.50	12.24	50.00	90.00	100.00
3	89.75	11.06	70.00	90.00	110.00
Change Visit 1 to 2	0.75	4.06	-10.00	0.00	10.00
Change Visit 1 to 3	6.00	10.08	-10.00	0.00	30.00

The mean external rotation in Group 1 increased from 85.5 at Visit 1 to 91.0 at Visit 3, and in Group 2 increased from 83.8 to 89.8. The p-value for the change from Visit 1 to Visit 3 is 0.8636 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.8183 (paired t-test), which does not indicate a significant group effect.

According to Travell and Simons (1999:546), both the supraspinatus and the infraspinatus muscles frequently develop TrPs, and the trapezius muscle may be become involved as part of the functional unit. The deltoid muscle may also develop satellite TrPs due to the fact that it lies within the pain reference zone of the supraspinatus muscle (Travell and Simons, 1999:546).

When the TrP is inactivated and its taut band released, its range of motion and therefore its contraction strength will return to normal (Travell and Simons 1999: 22). In saying that, by inactivating supraspinatus and trapezius TrPs, one could potentially have an effect on both infraspinatus and deltoid (posterior) muscles, which in turn will affect the range of motion of the external rotators.

4.4.2.3) Shoulder Internal Rotation

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	83.25	18.30	45.00	82.50	110.00
2	83.25	18.30	45.00	82.50	110.00
3	88.00	16.97	60.00	87.50	120.00
Change Visit 1 to 2	0.00	0.00	0.00	0.00	0.00
Change Visit 1 to 3	4.75	6.38	0.00	0.00	20.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
1	83.00	18.17	50.00	80.00	110.00
2	85.50	16.13	60.00	85.00	110.00
3	88.50	14.70	70.00	85.00	110.00
Change Visit 1 to 2	2.50	4.44	0.00	0.00	10.00
Change Visit 1 to 3	5.50	7.59	0.00	0.00	30.00

The mean internal rotation in Group 1 increased from 83.2 at Visit 1 to 88.0 at Visit 3, and in Group 2 increased from 83.0 to 88.5. The p-value for the change from Visit 1 to Visit 3 is 0.7371 (paired t-test), which does not indicate a significant group effect. The p-value for the change from Visit 1 to Visit 2 is 0.0210 (paired t-test), which indicates a significant difference in the two groups immediately after treatment.

These results would imply that the effects as discussed above (4.4.2.2 and 4.4.2.3) for external and internal rotation are similar, which stands to reason as these muscles work as a agonist – antagonist unit, in which the effect on one muscle would have a corresponding effect on the opposing muscle. It would therefore seem to suggest that the possible causes for the changes stem from the same or similar source.

4.4.3) Associated clinical findings e.g. Instability, Myofascial Trigger Points and Tendinopathy of other muscles.

4.4.3.1) Myofascial Trigger Points

According to Travell and Simons (1999: 546), both the supraspinatus and the infraspinatus muscles frequently develop TrPs, and the trapezius muscle may become involved as part of the functional unit. The deltoid muscle may also develop satellite TrPs due to the fact that it lies within the pain reference zone of the supraspinatus muscle. Lastly, the latissimus dorsi muscle might also become involved because it works as an antagonist to the abductor (supraspinatus).

As a result patients were examined for TrPs in the above-mentioned muscles (either active or latent). They were clinically noted and recorded (Appendix I) at subsequent visits to establish whether or not the number of TrPs had any influence on outcome results in those patients presenting with, and being treated for, supraspinatus tendinosis with GISTM.

Image 2: The supraspinatus muscle

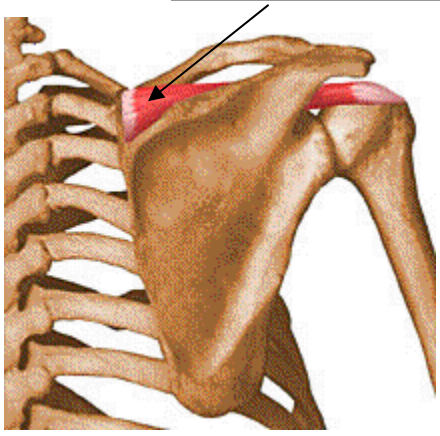
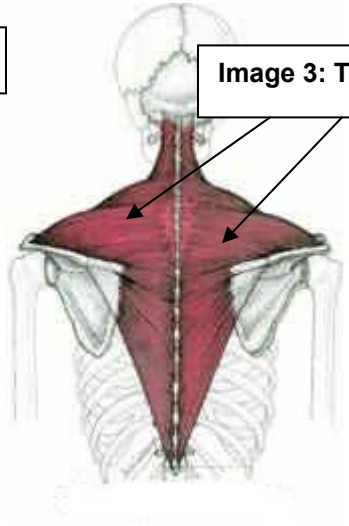


Image 3: The trapezius muscle



According to Carey-Loghmani (2003: 10), GISTM is indicated for the treatment of MPS and fascial restrictions within a muscle. The patient received GISTM to the supraspinatus muscle, musculotendinous portion and involved tendon.

Theoretically then, from the above statement, GISTM will have an effect on the myofascial component of the supraspinatus muscle and may even have an hypothesized effect on the other muscles as mentioned above that may be involved with the supraspinatus.

a) <u>Number of active trigger points at each visit</u>

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
Visit 1	3.50	1.15	1.00	3.50	6.00
Visit 2	3.65	1.31	1.00	3.50	6.00
Visit 3	1.80	1.40	0.00	2.00	6.00
Change Visit 1 to Visit 2	0.15	0.93	-1.00	0.00	3.00
Change Visit 1 to Visit 3	-1.70	1.42	-4.00	-2.00	1.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
Visit 1	4.05	1.23	2.00	4.00	6.00
Visit 2	3.85	1.35	1.00	4.00	6.00
Visit 3	2.00	1.81	0.00	2.00	6.00
Change Visit 1 to Visit 2	-0.20	1.01	-3.00	0.00	1.00
Change Visit 1 to Visit 3	-2.05	2.01	-5.00	-2.00	2.00

The p-value comparing the change in number of active trigger points between the two groups is 0.4323 (Wilcoxon two sample test), which does not indicate a significant group effect.

b) <u>Total number of all trigger points (active and latent) at each visit</u>

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
Visit 1	3.80	0.95	2.00	4.00	6.00
Visit 2	3.90	1.07	2.00	4.00	6.00
Visit 3	3.35	1.09	2.00	3.00	6.00
Change Visit 1 to Visit 2	0.10	0.55	-1.00	0.00	1.00
Change Visit 1 to Visit 3	-0.45	1.00	-3.00	0.00	1.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
Visit 1	4.25	1.02	2.00	4.00	6.00
Visit 2	4.25	1.21	2.00	4.00	6.00
Visit 3	3.35	1.14	0.00	3.00	6.00
Change Visit 1 to Visit 2	0.00	0.56	-1.00	0.00	1.00
Change Visit 1 to Visit 3	-0.90	1.37	-5.00	-0.50	1.00

The p-value comparing the change in number of active trigger points between the two groups is 0.2875 (Wilcoxon two sample test), which does not indicate a significant group effect.

4.4.3.2) Concomitant findings

Group 1 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
Visit 1	2.15	0.99	1.00	2.00	4.00
Visit 2	2.15	0.99	1.00	2.00	4.00
Visit 3	1.40	0.94	0.00	1.50	3.00
Change Visit 1 to Visit 2	0.00	0.00	0.00	0.00	0.00
Change Visit 1 to Visit 3	-0.75	0.55	-2.00	-1.00	0.00

Group 2 (N = 20)

Visit	Mean	SD	Minimum	Median	Maximum
Visit 1	2.30	1.03	1.00	2.50	4.00
Visit 2	2.30	1.03	1.00	2.50	4.00
Visit 3	1.25	1.16	0.00	1.00	4.00
Change Visit 1 to Visit 2	0.00	0.00	0.00	0.00	0.00
Change Visit 1 to Visit 3	-1.05	0.83	-3.00	-1.00	0.00

The p-value comparing the change in number of concomitant findings between the two groups is 0.2530 (Wilcoxon two sample test), which does not indicate a significant group effect.

There was no change in concomitant conditions between Visit 1 and Visit 2; however there was a slight improvement in both groups at the visit 3. The application of the technique may cause varying degrees of discomfort, but this discomfort should be localized to the area of the lesion (Carey-Loghmani, 2003:37).

The etiology of impingement syndrome is multifactorial and is commonly associated with other clinical entities such as weak or dysfunctional scapular musculature, posterior glenohumeral capsule tightness, inflammation of tendons (viz. supraspinatus and long head of biceps), bursal inflammation and glenohumeral instability (Michener et al., 2003). Travell and Simons (1999:545-546), state that TrPs can cause an increase in muscle tension, incoordination, and inhibition of muscles in the same functional unit. This then provides a potential source of scapulohumeral muscular dysfunction and imbalance in the dynamic stability provided by these muscles.

The referral pain patterns of a few muscles involved will briefly be described:

- TrPs in the supraspinatus muscle cause a deep ache in the shoulder, concentrating in the mid-deltoid region (Travell and Simons (1999: 538). Pain can also be felt generally over the upper trapezius area and then refer distally to the lateral epicondyle.

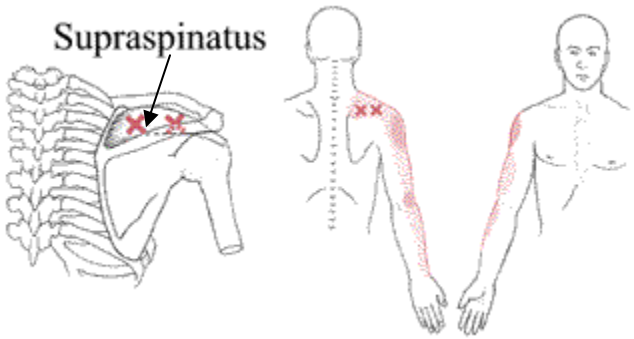


Image 4

- TrPs in the infraspinatus muscle like the supraspinatus also causes a deep ache in the anterior deltoid region. Pain is occasionally referred to the suboccipital and posterior cervical muscles (Travell and Simons (1999: 552).

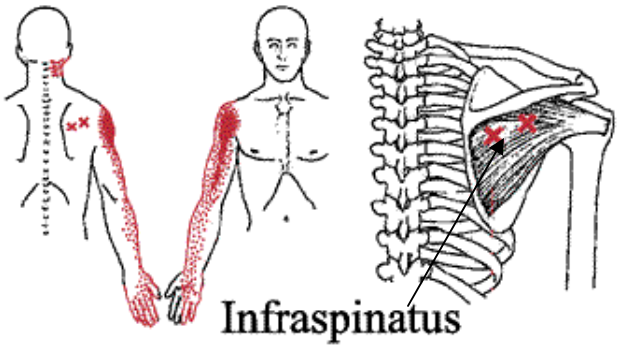


Image 5

- TrPs in the upper trapezius fibers are known to refer pain and tenderness mainly to the posterolateral aspect of the neck, behind the ear and to the temple. Fibers of the middle trapezius, less commonly are known to project pain toward the vertebrae and to the interscapular region (Travell and Simons 1999: 78).

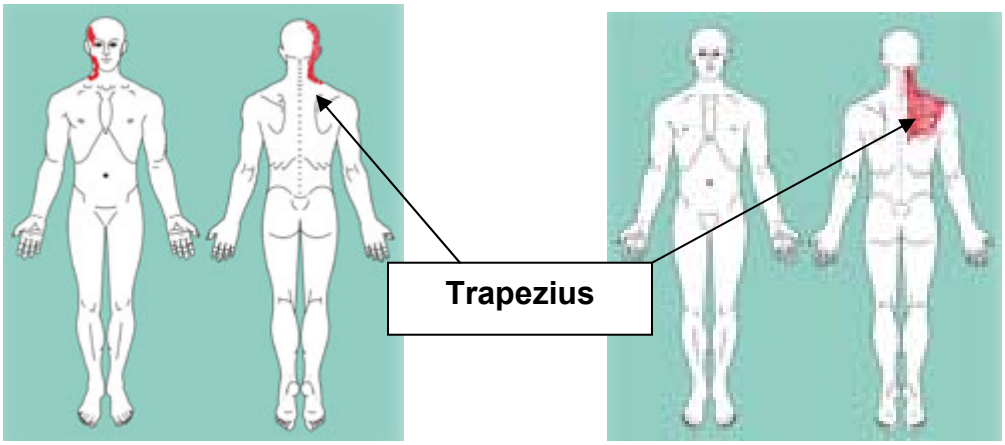


Image 6

Therefore, patients reporting concomitant neck pain or upper back pain might actually be reporting the referred pain patterns of active TrPs from one/many of the muscles in the shoulder girdle, as described above.

The rhomboid muscle is said to act synergistically with the supraspinatus during abduction of the arm (Travell and Simons 1999: 616). Musculoskeletal impairment can significantly alter the coordinated shoulder complex motion; (Andrews and Wilk, 1994: 15) hence potentially allowing for the perpetuation and activation of TrPs in the rhomboid muscle. Travell and Simons (1999: 618) also state there is articular dysfunction that is associated with the rhomboid TrPs, and can involve any of the spinal segments from the 7th cervical vertebra down to the 5th thoracic vertebra.

Therefore, patients complaining of associated upper back pain may not be purely of myofascial origin, but may also involve articular structures which may require spinal manipulative therapy to restore joint mechanics at the involved level.
Past research, with respect to GISTM is yet to show whether or not a series of treatments is needed or whether the hypothesized clinical effects are best achieved with one treatment. With regard to concomitant findings the results show that there is an overall decrease, however there are a few possible reasons for this:

1. Breaking down adhesions in the supraspinatus tendon, GISTM may assist in restoring mobility between tissue interfaces and increasing extensibility of structures (Hertling and Kessler, 1996:134; Carey-Loghmani, 2003:31, 51-62).
2. The restoration of normal function of the agonists (abductors) and antagonists (adductors) and stopping the inhibitory effect of the agonists, therefore, allowing for normal function (Lawrence et al., 1996:73-74)
3. Breaking the neural reflex circuit that is responsible for maintaining the patient's signs and symptoms (Leach, 1994:99).

4.5) Intra-group correlations between changes in objective and subjective findings

KEY

Age (mean) – mean age of participants (N=20)

∆NRS – Change in NRS-101

∆goniabd – Change in Goniometer Abduction

∆goni ext rot – Change in Goniometer External Rotation

∆goni int rot – Change in Goniometer Internal Rotation

∆pt abd – Change in Peak Torque Abduction

∆pt add – Change in Peak Torque Adduction

∆pt ext rot – Change in Peak Torque External Rotation

∆pt int rot – Change in Peak Torque Internal Rotation

∆SPADI (f) – Change in SPADI Function

∆SPADI (p) – Change in SPADI Pain

∆tw abd – Change in Total Work Abduction

∆tw add – Change in Total Work Adduction

∆tw ext rot – Change in Total Work External Rotation

∆tw int rot – Change in Total Work Internal Rotation

* Please see Appendix L for Pearson Correlation Coefficient of the above data.

Table 8: Significance of correlation between objective and subjective outcomes in the "treatment group" (p-values).

All p-values

	Age (mean)	ΔNRS	Δgoni abd	Δgoni ext rot	Δgoni int rot	Δpt abd	Δpt add	Δpt ext rot	Δpt int rot
Age(mean)	-	0.1786	0.0458	0.1823	0.0046	0.7448	0.2218	0.8112	0.0694
ΔNRS	0.1786	-	0.9808	0.2415	0.7889	0.3666	0.0748	0.3346	0.0624
Δgoni abd	0.0458	0.9808	-	0.0057	0.0257	0.9284	0.4058	0.7089	0.2162
Δgoni ext rot	0.1823	0.2415	0.0057	-	0.1862	0.6794	0.2173	0.8271	0.6276
Δgoni int rot	0.0046	0.7889	0.0257	0.1862	-	0.7029	0.4251	0.6301	0.3601
Δpt abd	0.7448	0.3666	0.9284	0.6794	0.7029	-	0.2187	0.2502	0.4916
Δpt add	0.2218	0.0748	0.4058	0.2178	0.4251	0.2187	-	0.4386	0.0173
Δpt ext rot	0.8112	0.3346	0.7089	0.8271	0.6301	0.2502	0.4386	-	0.3964
Δpt int rot	0.0694	0.0624	0.2162	0.6276	0.3601	0.4916	0.0173	0.3964	-
ΔSPADI (f)	0.7122	0.1234	0.1695	0.1039	0.5992	0.4254	0.9096	0.7056	0.9267
ΔSPADI (p)	0.2847	0.9522	0.7026	0.2308	0.0252	0.4365	0.7753	0.5363	0.1924
Δtw abd	0.0401	0.7541	0.5514	0.7387	0.0202	0.3940	0.8020	0.8673	0.8787
Δtw add	0.8629	0.5864	0.4640	0.1571	0.5082	0.5879	0.0007	0.6518	0.1645
Δtw ext rot	0.9627	0.0593	0.8596	0.9838	0.8738	0.8568	0.1790	0.0605	0.1228
Δtw int rot	0.3183	0.0997	0.1760	0.5785	0.3747	0.4422	0.1234	0.7697	0.0131

Table 8: Significance of correlation between objective and subjective outcomes in the "treatment group" (p-values) continued.

	ΔSPADI (f)	ΔSPADI (p)	Δtw abd	Δtw add	Δtw ext rot	Δtw int rot
Age (mean)	0.7122	0.2847	0.0401	0.8629	0.9627	0.3183
ΔNRS	0.1234	0.9522	0.7541	0.5864	0.0593	0.0997
Δgoniabd	0.1695	0.7026	0.5514	0.4640	0.8596	0.1760
Δgoni ext rot	0.1039	0.2308	0.7387	0.1571	0.9838	0.5785
Δgoni int rot	0.5992	0.0252	0.0202	0.5082	0.8738	0.3747
Δpt abd	0.4254	0.4365	0.3940	0.5879	0.8568	0.4422
Δpt add	0.9096	0.7753	0.8020	0.0007	0.1790	0.1234
Δpt ext rot	0.7056	0.5363	0.8673	0.6518	0.0605	0.7697
Δpt int rot	0.9267	0.1924	0.8787	0.1645	0.1228	0.0131
ΔSPADI (f)	-	0.2936	0.7646	0.5407	0.3031	0.5762
ΔSPADI (p)	0.2936	-	0.1438	0.3115	0.8101	0.8943
Δtw abd	0.7646	0.1438	-	0.0688	0.7628	0.5381
Δtw add	0.5407	0.3115	0.0688	-	0.2704	0.4613
Δtw ext rot	0.3031	0.8101	0.7628	0.2704	-	0.0004
Δtw int rot	0.5762	0.8943	0.5381	0.4613	0.0004	-

4.5.1) Table 9: Correlation between subjective and objective data in the treatment group

Reading 1	*Relationship*	*Reading 2*	*Discussion*
Age	**-** **-** **-**	∆goniabd ∆goni int rot ∆tw abd	Most of the younger participants have underlying instability, and generalized ligamentous laxity which results in increase range of motion. Decrease in internal rotation related to possible concomitant findings (viz. biceps and bursal involvement, AC joint dysfunction), older patients may have already entered early stages of DJD esp. in the AC joint, therefore preventing increase movement, in particular internal rotation. Older participants have reduced work tolerance esp. abduction, possibly not as physically active/fit as younger participants; they may not have as much muscle mass as the younger participants.
∆goniabd	**+** **+**	∆goni int rot ∆goni ext rot	There may be decreased inflammation and a reduction in scar tissue as a known effect of GISTM, allowing greater tissue extensibility and ultimately increased movement in the Hawkins-Kennedy test position. Deactivating TrPs allows for restoration of normal function, in particular that of the agonists and antagonists.
∆goni int rot	**+** **+**	∆SPADI (p) ∆tw abd	GISTM works is by reducing scar tissue, aiding inflammation by reducing pain and allowing healing of tissue to begin. Deactivating TrPs can affect both strength and work tolerance.
∆pt add	**-** **+**	∆pt int rot ∆tw add	Internal rotation might be inhibited by pain (AMI), due to joint dysfunction and inability to recruit all motor units of the muscle group to their full extent during a maximal effort voluntary muscle contraction. Deactivating TrPs can affect both strength and work tolerance.
∆pt int rot	**+**	∆tw int rot	Deactivating TrPs can affect both strength and work tolerance.
∆tw ext rot	**+**	∆tw int rot	Deactivating TrPs would allow for smooth uninhibited movement between agonists and antagonists.

Table 10: Significance of correlation between objective and subjective outcomes in the "measurement group" (p-values).

All p-values

	Age (mean)	ΔNRS	Δgoni abd	Δgoni ext rot	Δgoni int rot	Δpt abd	Δpt add	Δpt ext rot	Δpt int rot
Age (mean)	-	0.6290	0.4164	0.9751	0.4787	0.6558	0.5804	0.3699	0.7974
ΔNRS	0.6290	-	0.5492	0.4243	0.1130	0.8710	0.8257	0.4117	0.4838
Δgoni abd	0.4164	0.5492	-	0.3012	0.4963	0.5301	0.7582	0.2301	0.0164
Δgoni ext rot	0.9751	0.4243	0.3012	-	0.1946	0.6340	0.3512	0.4156	0.8383
Δgoni int rot	0.4787	0.1130	0.4963	0.1946	-	0.1046	0.0510	0.1807	0.3414
Δpt abd	0.6558	0.8710	0.5301	0.6340	0.1046	-	0.4248	0.0884	0.5513
Δpt add	0.5804	0.8257	0.7582	0.3512	0.0510	0.4248	-	0.9854	0.9954
Δpt ext rot	0.3699	0.4117	0.2301	0.4156	0.1807	0.0884	0.9854	-	0.0494
Δpt int rot	0.7974	0.4838	0.0164	0.8383	0.3414	0.5513	0.9954	0.0494	-
ΔSPADI (f)	0.4725	0.2325	0.8856	0.7498	0.6186	0.4537	0.3009	0.2735	0.6940
ΔSPADI (p)	0.1465	0.0506	0.8050	0.9163	0.4826	0.6974	0.2415	0.8314	0.6467
Δtw abd	0.2196	0.5373	0.9598	0.6824	0.2387	0.0362	0.0069	0.2249	0.6455
Δtw add	0.7758	0.1952	0.7738	0.6179	0.4704	0.1389	0.0008	0.7822	0.8778
Δtw ext rot	0.5039	0.6217	0.5832	0.2889	0.2985	0.5201	0.3497	<0.0001	0.4421
Δtw int rot	0.8545	0.9918	0.3418	0.9454	0.6310	0.0551	0.8992	0.0071	0.0087

Table 10: Significance of correlation between objective and subjective outcomes in the "measurement group" (p-values).

	ΔSPADI (f)	ΔSPAD (p)	Δtw abd	Δtwadd	Δtw ext rot	Δtw int rot
Age (mean)	0.4725	0.1465	0.2196	0.7758	0.5039	0.8545
ΔNRS	0.2325	0.0506	0.5373	0.1952	0.6217	0.9918
Δgoniabd	0.8856	0.8050	0.9598	0.7738	0.5832	0.3418
Δgoni ext rot	0.7498	0.9163	0.6824	0.6179	0.2889	0.9454
Δgoni int rot	0.6186	0.4826	0.2387	0.4704	0.2985	0.6310
Δpt abd	0.4537	0.6974	0.0362	0.1389	0.5201	0.0551
Δpt add	0.3009	0.2415	0.0069	0.0008	0.3497	0.8992
Δpt ext rot	0.2735	0.8314	0.2249	0.7822	<0.0001	0.0071
Δpt int rot	0.6940	0.6467	0.6455	0.8778	0.4421	0.0087
ΔSPADI (f)	-	0.0002	0.1274	0.2952	0.5432	0.9704
ΔSPADI (p)	0.0002	-	0.0939	0.0371	0.9401	0.6505
Δtw abd	0.1274	0.0939	-	0.0078	0.3904	0.0589
Δtw add	0.2952	0.0371	0.0078	-	0.9952	0.3492
Δtw ext rot	0.5432	0.9401	0.3904	0.9952	-	0.0051
Δtw int rot	0.9704	0.6505	0.0589	0.3492	0.0051	-

4.5.2) Table 11: Correlation between subjective and objective data in the measurement group

Reading 1	*Relationship*	*Reading 2*	*Discussion*
∆goniabd	**-**	∆pt int rot	Internal rotation might be inhibited by pain (AMI), due to joint dysfunction and inability to recruit all motor units of the muscle group to their full extent during a maximal effort voluntary muscle contraction.
∆pt abd	**+** **+**	∆tw abd ∆tw int rot	There may be decreased inflammation and a reduction in scar tissue as a known effect of GISTM, allowing greater tissue extensibility and ultimately increased movement in the Hawkins-Kennedy test position. Deactivating TrPs allows for restoration of normal function of the agonists and antagonists, and in this case with respect to work tolerance of the internal rotators.
∆pt add	**+** **+**	∆tw abd ∆tw add	Deactivating TrPs can affect both strength and work tolerance. Improvement in agonist vs. antagonist, affect on one will affect the other.
∆pt ext rot	**+** **+** **+**	∆pt int rot ∆tw ext rot ∆tw int rot	Improvement in agonist vs. antagonist, affect on one will affect the other. Deactivating TrPs can affect both strength and work tolerance.
∆pt int rot	**+**	∆tw int rot	Deactivating TrPs can affect both strength and work tolerance.
∆SPADI (f)	**+**	∆SPADI (p)	Breaking down adhesions reduces pain and restores mobility between tissue interfaces, increasing extensibility of structures and improving function.
∆SPADI (p)	**-**	∆tw add	A single treatment with the GT may result in adhesion formation (from induced inflammatory reaction – micro trauma). These adhesions can cause restriction of motion of the wound tissue, therefore making it more vulnerable to re-injury (Hunter, 1994). Patients may report some pain relief from GISTM, however other concomitant conditions for eg. TrPs in the Latissimus dorsi muscle may prevent full/maximal effort during isokinetic testing.
∆tw abd	**+**	∆tw add	Improvement in agonist vs. antagonist, affect on one will affect the other.
∆tw ext rot	**+**	∆tw int rot	Improvement in agonist vs. antagonist, affect on one will affect the other.

4.6) Summary and Conclusions

In accordance with past research, with respect to GISTM, it has yet to be shown whether or not a series of treatments is needed or whether the hypothesized clinical effects are best achieved with one treatment.

Thus even though Graston Technique instrument-assisted soft tissue mobilization (GISTM) is a form of soft tissue mobilization that is used to detect and release scar tissue, adhesions and facial restrictions; as well as to induce local hyperemia and mechanically mobilize scar tissue much like that of the work of Cyriax, indicated as follows:

> Breaking down the fibrotic tissue and remodeling the collagen to the correct alignment assists in restoring mobility between tissue interfaces, increasing extensibility of structures and improving function (Hertling and Kessler, 1996: 134).
>
> GISTM, similarly to frictions, aids the inflammatory process. GISTM initiates inflammation allowing the process to complete itself and for healing and the tissue remodelling to occur, ultimately decreasing the patient's pain and improving function (Carey-Loghmani, 2003:31, 51-62; Hammer, 2001).
>
> Transverse friction massage, and so GISTM, can also improve pain levels by increasing large-fibre input and, therefore, decrease nociceptive transmission through the 'Gate Control Theory' put forward by Melzack and Wall (Lynch and Kessler, 1990: 48; Melzack and Wall, 1965: 971).

It remains unknown if these parameters are actually clinically manifested in the patient for each of the outcomes measured.

The treatment group showed correlations (Table 7) between the objective outcomes (Age; Goniometer abduction, internal rotation, external rotation; Peak torque adduction, internal rotation and Total work abduction, adduction, internal and external rotation) with the subjective outcome SPADI (pain).

The measurement group showed correlations (Table 9) between the objective outcomes (Goniometer abduction; Peak torque abduction, adduction, internal and external rotation; Total work abduction, adduction, internal and external rotation) with the subjective outcomes SPADI (pain) and (function).

However with respect to the hypotheses the following discussions are apparent:

4.6.1) The first hypothesis

Indicated that a single treatment is insufficient in treating patients presenting with supraspinatus tendinosis, in terms of subjective findings.

The *hypothesis is rejected* with regard to the p-values obtained in this study.

4.6.2) The second hypothesis

Indicated that a single treatment is insufficient in treating patients presenting with supraspinatus tendinosis, in terms of objective findings.

The *hypothesis is rejected* with regard to the p-values obtained in this study.

4.6.3) The third hypothesis

Indicated that multiple treatments are necessary in treating tendinopathies.

The *hypothesis is rejected* with regard to the p-values obtained in this study.

4.6.4) The fourth hypothesis

Indicated that patients with an increase in the number of concomitant conditions may take longer to improve, in terms of subjective an objective measurements.

The *hypothesis is rejected* with regard to the p-values obtained in this study.

It is recognized that the results observed indicate that a larger sample size may have been more appropriate in order to determine group significant differences and that gender stratification may have assisted in achieving comparable outcome.

Chapter Five

Conclusions and Recommendations

5.1) Introduction

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

5.2) Conclusions

The aim of this pilot study was to assess the short-term effect of Graston instrument-assisted soft tissue mobilization (GISTM) on supraspinatus tendinosis and its concomitant clinical findings.

It was noted that there were no significant differences as per the attained p-values in this study (appendix M), thus it cannot be conclusively concluded that the presence of statistical insignificance would automatically translate or not translate into a clinically presentable change. Thus no clinical conclusions can be drawn from this study in terms of the effects on individual outcomes of GISTM in the clinical setting.

It is however noted that the M:F ratio, which showed a significant difference between the groups, would have modified the outcomes of the results in this study. Therefore the trends may be indicative of this ratio as opposed to clinical improvement and further research should include a stratification of gender.

The gender stratification should be considered in conjunction with larger sample size in order to improve the ability of the statistical analysis to detect significant changes in order to improve strength of the study in this regard.
As in this study, the small sample size could have allowed for an outlier to change the average significantly, in addition to which group 2 had a higher

proportion of female participants thus confounding the ability of the results to be compared between the groups, as a consequence of differing responses measured between the male and female participants in respect of the cybex measures.

5.3) Recommendations

Further recommendations include:

The inclusion of a control group within a future study would be recommended in order to assist with and highlight the following two recommendations.

The completion of an analysis prior to future studies in order to determine the optimum number of participants for the future study (ies) to be able to interpret more accurately the statistical and clinical significances is recommended.

Furthermore this could be enhanced by setting clinical and statistical significance levels or barriers prior to the study in order to more accurately determine the outcomes in terms of the stated levels / barriers, such that the significances are not based solely on comparison between groups, but also within the groups by virtue of comparison to the predetermined level / barrier.

Having a peer intern or clinician to take objective and subjective measures may result in more reliable readings and limit researcher bias.

As a result of the inconclusiveness of the changes between with groups with respect to the pain levels, it is suggested that the following be considered in follow up studies:

- One month follow up to re-evaluate the changes that have been affected.
- An increased period of treatment to optimise the degree of scar tissue breakdown (suggested by Carey-Loghmani (2003: 28) is 4 to 8

sessions, chronic conditions may take longer, as many as 9 – 14 treatments.)

- An increased time between treatments to decrease the effect of the previous treatment (i.e. inflammation induction by the treatment with GT may have resulted in elevated pain levels that where indicated, which reflected more closely the induced inflammation as opposed to the treatment effect).

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

The Graston Technique (GT) = GISTM + exercise. For the purposes of this research, participants did not receive the full GT protocol. It is important to note, that GISTM should be used in conjunction with a cardio warm-up, targeted stretching and strengthening exercises and post treatment cryotherapy. By following this comprehensive treatment approach, the full benefit/effect of GISTM can be realized (Carey-Loghmani, 2003: 64).

APPENDICES

A – advertisement

B – letter of information

C – informed consent form

D – case history

E – physical examination

F – shoulder regional

G – shoulder disability scale

H – NRS-101

I – table of readings

J – cybex measurement protocol

K – SOAPE note

L – Correlation coefficient tables

M - Summary of outcome measures

APPENDICES

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SHOULDER PAIN?

Do you participate in overhead sports/activities i.e. swimming, baseball pitcher, waterpolo player and between the ages of 18-40?

You may qualify for research being conducted at Durban Institute of Technology

CHIROPRACTIC DAY CLINIC
WHICH INCLUDES:

A FREE ISOKINETIC SHOULDER STRENGTH ASSESSMENT ON A CYBEX(II) ISOKINETIC DYNAMOMETER.

FREE TREATMENT
Is available to those who qualify to take part in this study

For more information contact:

Grant
on
031-2042205/2512

Appendix B

Letter of information

Title:

"The short-term effect of Graston instrument-assisted soft tissue mobilization (GISTM) on supraspinatus tendinosis and it's concomitant clinical findings."

Supervisor: Dr. C. Korporaal (031) 2042611
Research student: G. Harper (031) 5638163

Introduction:

Graston Technique Instrument – assisted Soft Tissue Mobilization (GISTM) is an advanced form of soft tissue mobilization that is used to detect and release scar tissue, adhesions and fascial restrictions. The Graston technique instruments are made of stainless steel which can be likened to a tuning fork and are shaped to treat the various contours of the body.

Procedure:

60 participants will be required to complete this study. All participants will undergo a brief case history, physical examination and shoulder regional. Participants will then be assessed (i.e. strength, range of motion, pain and neuromuscular control), receive treatment and re-assessed to determine the immediate effects of the treatment. The participants will then be randomly allocated in two groups: a treatment group and a non-intervention group. Those in the treatment group will be required to attend six consultations and a follow-up consultation, whereas those in the non-intervention group will receive a single treatment without any further intervention; however they will be required to attend the follow-up session.

Those patients in group two will be given the opportunity to receive five free treatments from an intern at the Durban Institute of Technology Chiropractic Day Clinic at the conclusion of the study.

Risks and benefits:

There are no major risks involved with GISTM. Minor things that you may experience include mild discomfort, slight tenderness and bruising. The benefits far outweigh the risks and include: accelerated healing rates which means faster, improvement, decreased treatment time and overall increasing patient satisfaction.

Your participation in this study is greatly appreciated.

Yours faithfully

Research Student: _____ Date: _____

Appendix C

Informed Consent Form

Title of research project: “The short-term effect of Graston instrument-assisted soft tissue mobilization (GISTM) on supraspinatus tendinosis and it’s concomitant clinical findings.”

Name of Supervisor: Dr. C. Korporaal (031) 2042611

Name of research student/s: G. Harper (031) 5638163

Date: _____

Please circle the appropriate answer

1. Have you read the research information sheet? Yes / No
2. Have you had an opportunity to ask questions regarding this study? Yes / No
3. Have you received satisfactory answer to your questions? Yes / No
4. Have you had an opportunity to discuss this study? Yes / No
5. Have you received enough information about this study? Yes / No
6. Who have you spoken to anyone? , if yes, who? Yes / No

_____

7. Do you understand the implications of your involvement in this study? Yes / No
8. Do you understand that you are free to withdraw from this study? Yes / No
   a) at any time
   b) without having to give any reason for withdrawing, and
   c) without effecting your future health care.
9. Do you agree to voluntarily participate in this study? Yes / No

If you have answered no to any of the above, please obtain the information before signing

_____ _____

Patient Signature Guardian Signature

_____ _____

Date Date

DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: _____ Date: _____

File # : _____ Age: _____

Sex : _____ Occupation: _____

Intern : _____ Signature _____

FOR CLINICIANS USE ONLY:

Initial visit

Clinician: Signature :

Case History:

Examination:

Previous: Current:

X-Ray Studies:

Previous: Current:

Clinical Path. lab:

Previous: Current:

CASE STATUS:

PTT:	Signature:	Date:

CONDITIONAL:

Reason for Conditional:

Signature: Date:

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

Intern's Case History:

1. **Source of History:**

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

3. **Present Illness:**

	Complaint 1	**Complaint 2**
▸ Location ▸ Onset : Initial: Recent: < Cause: ▸ Duration ▸ Frequency ▸ Pain (Character) ▸ Progression ▸ Aggravating Factors ▸ Relieving Factors ▸ Associated S & S ▸ Previous Occurrences ▸ Past Treatment ▸ **Outcome:**		

4. **Other Complaints:**

5. **Past Medical History:**

- General Health Status
- Childhood Illnesses
- Adult Illnesses
- Psychiatric Illnesses
- Accidents/Injuries
- Surgery
- Hospitalizations

6. Current health status and life-style:

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

7. Immediate Family Medical History:

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

8. Psychosocial history:

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

9. Review of Systems:

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

Appendix E **DURBAN INSTITUTE OF TECHNOLOGY**
CHIROPRACTIC DAY CLINIC
PHYSICAL EXAMINATION

Patient:_____ File#:_____ Date:_____

Clinician:_____ Signature:_____

Student:_____ Signature:_____

1. VITALS

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

2. GENERAL EXAMINATION

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

3. CARDIOVASCULAR EXAMINATION

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

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

Palpation: - Apex Beat (character + location):

- Right or left ventricular heave:
- Epigastric Pulsations:
- Palpable P2:
- Palpable A2:

Pulses:
- General Impression:
- Radio-femoral delay:
- Carotid:
- Radial:
- Dorsalis pedis:
- Posterior tibial:
- Popliteal:
- Femoral:

Percussion: - borders of heart

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

4. RESPIRATORY EXAMINATION

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

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

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

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

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

5. ABDOMINAL EXAMINATION

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

Inspection
- Shape:
- Scars:
- Hernias:

Palpation
- Superficial:

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

Percussion - Rebound tenderness:
- Ascites:
- Masses:

Auscultation - Bowel sounds:
- Arteries (aortic, renal, iliac, femoral, hepatic)

Rectal Examination - Perianal skin:
- Sphincter tone & S4 Dermatome:
- Obvious masses:
- Prostate:
- Appendix:

6. G.U.T EXAMINATION

External genitalia**:**
Hernias:
Masses:
Discharges:

7. NEUROLOGICAL EXAMINATION

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

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

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

Evidence of head trauma:

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

Cranial Nerves:

I Any loss of smell/taste:
Nose examination:

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

- Fundoscopy findings:

III Ocular Muscles:
Eye opening strength:

IV Inferior and Medial movement of eye:

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

b. Motor - Masseter:
- Jaw lateral movement:

c. Reflexes - Corneal reflex
- Jaw jerk

VI Lateral movement of eyes

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

b. Taste - Anterior two-thirds of tongue:

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

IX & Gag reflex:
X Uvula deviation:
Speech quality:

XI Shoulder lift:
S.C.M. strength:

XII Inspection of tongue (deviation):

Motor System:

a. Power

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

= Toe (Plantarflexion & Dorsiflexion):

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

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

Sensory System:

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

b. Joint position sense
- Finger:
- Toe:

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

Cerebellar function:

Obvious signs of cerebellar dysfunction:
= Intention Tremor:
= Nystagmus:
= Truncal Ataxia:
Finger-nose test (Dysmetria):
Rapid alternating movements (Dysdiadochokinesia):
Heel-shin test:
Heel-toe gait:
Reflexes:
Signs of Parkinsons:

8. <u>SPINAL EXAMINATION:</u>(See Regional examination)

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

9. <u>BREAST EXAMINATION:</u>

Summon female chaperon.

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

Palpation - masses:
- tenderness:
- axillary tail:
- nipple:
- regional lymph nodes

SHOULDER REGIONAL EXAMINATION

Patient: ……. File No: …….. Date: ….

Intern: ……Signature: ……

Clinician: ……Signature: ……...

Observation

Posture		S-C Joints	
Skin		Clavicles	
Swelling		A-C Joints	
Shoulder levels		Scapulae	
Comments			

Palpation

S-C Joint:		SCM:	Scalenes:
Sternum:		Ribs and costal cartridge:	
Clavicle:		Coracoid process:	
A-C Joint:		Acromion:	
Greater Tuberosity:			
Lesser Tuberosity:			
Intertubercular (bicipital groove):			
Trapezius:		Deltoid:	
Biceps:		Triceps:	
Supraspinatus insertion:			
Musculotendinous portion of supraspinatus:			
Axilla:	Lymph nodes:		
	Brachial artery:		
	Serratus anterior (medial wall):		
	Pectoralis major (anterior wall):		
	Lattisimus dorsi (posterior wall):		
Scapula	Borders:	Spine:	
	Supraspinous fossa:		

	Infraspinous fossa:
Cervico-thoracic spine:	

Active Movements (note ROM and pain)

Elevation through abduction (170-180°):	
Painful arc with abduction:	
Elevation through forward flexion (160-180°):	
Elevation through scapula plane (170-180°):	
Lateral rotation (80-90°):	Medial rotation (60-100°):
Extension (50-60°):	Adduction (50-75°):
Horizontal adduction/abduction (130°):	
Circumduction (200°):	
Apley's Scratch:	

Passive movements (note end-feel, ROM and pain)

Elevation through abduction (bone to bone or tissue stretch)……

Elevation through forward flexion (tissue stretch)……..

Lateral rotation (tissue stretch)……..

Medial rotation (tissue stretch)……..

Extension (tissue stretch)…….

Adduction (tissue approximation) ……..

Horizontal adduction (tissue stretch or approximation)……...

Horizontal abduction (tissue stretch)……

Quadrant Test…….

..

Resisted Isometric Movements (note strength and pain)

Flexion		Medial rotation	
Extension		Lateral Rotation	
Adduction		Elbow flexion	
Abduction		Elbow extension	

Joint Play Movements (and motion palpation)

SC Joint	Supero-inferior (shrug shoulder with arm at side):
	Horizontal add/abduction (arm abducted 90°):
AC Joint	A-P Shear:
	Supero-inferior shear:
Scapula	Normal scapulo-humeral rhythm?:
	General mobility of scapula:

Glenohumeral Joint

Lateral movement of humeral head _____

Inferior movement of humeral head (Caudal glide)(50°) _____

Anterior movement of humeral head (P-A glide) (25°) _____

Posterior shear of humeral head (A-P glide) **>50%** At 10° flexion _____

At 90° flexion _____

Backward glide of humeral head in abduction _____

Long-axis distraction of humeral head in abduction _____

Downward and backward (S-I → A-P) _____

Outward and backward (med-lat → A-P _____

External rotation of humeral head _____

Internal rotation of humeral head _____

Instability Tests

1. Anterior Instability Tests

	Right			Left		
	Pos	Neg	n/a	Pos	Neg	n/a
Anterior drawer Test						
Rowe Test						
Fulcrum Test						
Apprehension (crank) Test						
Clunk Test (tear of labrum)						
Rockwood Test						

2. Posterior Instability Tests

	Pos	Neg	n/a	Pos	Neg	n/a
Posterior Apprehension Test						
Norwood Stress Test						

Push-pull Test						
Jerk Test						

3. Inferior and Multi-directional instability tests

	Pos	Neg	n/a	Pos	Neg	n/a
Inferior Shoulder Instability Test						
Feagin Test (antero-inferior instability)						

A-C Joint Stress Test: _____

S-C Joint Stress Test: _____

Tests for Muscle or Tendon Pathology

1.	Speed's Test (bicipital tendonitis)	
2.	Gilchrest Sign (bicipital tendonitis)	
3.	Supraspinatus Test (supraspinatus tendonitis)	
4.	Hawkins-Kennedy Impingement Test (supraspinatus tendonitis)	
5.	Drop –arm Test (rotator cuff tear)	
6.	Impingement Test	
7.	Pectoralis Major Contracture Test	
8.	Ludington's Test (rupture of long head of biceps)	

Tests for neurological function

Brachial Plexus Tension Test			Radial Nerve										
			Median Nerve										
Tinel's Sign (Scalene triangle)													
Dermatones	C4		C5		C6		C7		C8		T1		T2	
Reflexes	Biceps(C5/6)						Triceps (C7/8)						

Thoracic Outlet Syndrome Tests

Adson's Test		Halstead's Test	
Costoclavicular Test		Eden's Test (cervical rib)	
Hyperabduction Test		Roos Test	
Allen's Test			

Appendix G

Rating scale of the American Shoulder and Elbow Surgeons

Name: _____ Date:_____ Age: _____ Score: _____

Pain: (5 = none; 4 = slight; 3 = after unusual activity; 2 = moderate; 1 = marked;
0 = complete disability: _____)

Function: (4 = normal; 3 = mild compromise; 2 = difficulty; 1 = with aid;
0 = unable, NA not available)

Activity	Score	Activity	Score
1. Use back pocket		7.Carry 10 to 15 lbs with arm at side	
2. Reach behind back, fasten bra		8. Dress	
3. Wash opposite underarm		9. Sleep on the affected side	
4. Eat with a utensil		10. Pulling	
5. Comb hair		11. Use hand overhead	
6. Use hand with arm at shoulder level		12. Lifting	
		TOTAL	

SPORTS: **Points**

Same overhead sport, equal performance (normal)	4
Same non-overhead sport, equal performance (mild compromise)	3
Same sport, decreased performance (difficult)	2
Different sport (with aid)	1
Sports not possible (unable)	0

Taken from: Yeomans, S.G. 2000.pp550. The Clinical Application of Outcomes Assessment.Appleton & Lange. ISBN 0-8385-1528-2

Modified from Gartsman G.M. Arthroplastic acromioplasty for lesions of the rotator cuff. Journal of Bone and Joint Surgery.1990; 72A: 169-180.

Appendix H

Numerical Rating Scale –101 Questionnaire

Patient name: _____ File No.: _____

Date: _____

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

0_____100

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

0_____100

Appendix I **TABLE OF READINGS**

OBJECTIVE DATA

READINGS	1	2	3

GONIOMETER READINGS (IN DEGREES)	
SHOULDER ABDUCTION	
INTERNAL ROTATION	
EXTERNAL ROTATION	

MYOFASCIAL TRIGGER POINTS														
	Yes	No	Act	Lat	Number of Trigger points									
					1	2	3	4	5	6	7	8	9	10
Upper Trapezius														
Deltoid														
Infraspinatus														
Latissimus Dorsi														

ASSOCIATED CLINICAL FINDINGS				
	Present	Absent	Structures involved	
Instability (Ant, Post or MDI)				
Impingement Σ				
Tendinopathy (specify muscle)				
Bursitis				
Scapulo-humeral dysfunction				
Spinal pathology				
Other				

APPENDIX J:

CYBEX TESTING PROTOCOL

Subjects are to be given standardised, scripted verbal encouragement while performing the test.

Subjects are to complete a 5 minute warm up cycle.

Concentric-Concentric Test

- 4 Sub-maximal warm-up repititions at 90 degrees/sec
- 1 min rest
- 2 trial repetitions of maximal effort at 60 degrees/sec
- 1 min rest
- 3-5 repititions of maximal effort at 60 degrees/sec
- 4 min rest

Eccentric-Eccentric Test

- 2 Sub-maximal warm-up repititions at 90 degrees/sec
- 1 min rest
- 2 trial repetitions of maximal effort at 60 degrees/sec
- 1 min rest
- 3-5 repititions of maximal effort at 60 degrees/sec

Should subjects experience pain that prevents them from completing the test they will be excluded from the study.

Adapted from:Davies (1992: 43-4), Perrin (1993: 48) Wilk et al. (1991 :63-70) and Wright (2003).

Measurement procedure:

1. Strength Measurements

Internal rotation

The patient is seated upright with arm abducted to 45 degrees at the shoulder, elbow flexed to 90 degrees and hand is neutral (midway between pronation and supination). The patient is then asked to apply their greatest effort internally.

External rotation

The patient is seated upright with arm abducted to 45 degrees at the shoulder, elbow flexed to 90 degrees and hand is neutral (midway between pronation and supination). The subject is asked to apply their greatest effort against the machine but this time externally.

Abduction

The patient is seated with the chair declined by 35 degrees from the upright position. The subject is asked to lift the arm away from the body with the greatest possible effort.

Adduction

The patient is seated with the chair declined by 35 degrees from the upright position. The subject is asked to pull the arm towards the body with the greatest possible effort.

DURBAN INSTITUTE OF TECHNOLOGY

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

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

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***

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

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

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***

Appendix L: Intra-group correlations between changes in objective and subjective findings

Table ??: Pearson correlation coefficients and of objective and subjective outcomes in the "treatment group".

	Age (mean)	ΔNRS	Δgoni abd	Δgoni ext rot	Δgoni int rot	Δpt abd	Δpt add	Δpt ext rot	Δpt int rot
Age(mean)	1	-0.31328	-0.45135	-0.31082	-0.60645	0.07769	0.285849	-0.05704	-0.41419
ΔNRS	-0.31328	1	-0.00575	0.274515	-0.06391	-0.2133	-0.40714	0.227568	0.424059
Δgoni abd	-0.45135	-0.00575	1	0.59457	0.497183	-0.02146	-0.19672	-0.08903	0.28919
Δgoni ext rot	-0.31082	0.274515	0.59457	1	0.308205	0.098538	-0.28851	-0.05216	0.115562
Δgoni int rot	-0.60645	-0.06391	0.497183	0.308205	1	0.090965	-0.18889	-0.11473	0.216125
Δpt abd	0.07769	-0.2133	-0.02146	0.098538	0.090965	1	0.287698	-0.26967	-0.16326
Δpt add	0.285849	-0.40714	-0.19672	-0.28851	-0.18889	0.287698	1	-0.18354	-0.52562
Δpt ext rot	-0.05704	0.227568	-0.08903	-0.05216	-0.11473	-0.26967	-0.18354	1	0.200605
Δpt int rot	-0.41419	0.424059	0.28919	0.115562	0.216125	-0.16326	-0.52562	0.200605	1
ΔSPADI (f)	-0.088	-0.35599	0.319646	0.374397	0.125112	-0.18877	-0.02714	0.090107	0.02198
ΔSPADI (p)	-0.25153	-0.01433	0.091056	0.280589	0.498662	0.184347	0.068137	0.146993	0.304068
Δtw abd	-0.46247	-0.07475	0.141656	-0.0796	0.514665	0.201628	-0.05987	0.039926	0.036463
Δtw add	-0.04125	-0.12949	-0.17367	-0.32868	0.157156	0.12897	0.69457	-0.10754	-0.32321
Δtw ext rot	0.01119	0.428762	0.04226	0.004845	-0.0366	-0.04312	-0.31303	0.42686	0.35652
Δtw int rot	-0.23514	0.378646	0.315083	0.132206	0.209773	0.182128	-0.356	0.069882	0.544484

	ΔSPADI (f)	ΔSPADI (p)	Δtw abd	Δtw add	Δtw ext rot	Δtw int rot
Age(mean)	-0.088	-0.25153	-0.46247	-0.04125	0.01119	-0.23514
ΔNRS	-0.35599	-0.01433	-0.07475	-0.12949	0.428762	0.378646
Δgoniabd	0.319646	0.091056	0.141656	-0.17367	0.04226	0.315083
Δgoni ext rot	0.374397	0.280589	-0.0796	-0.32868	0.004845	0.132206
Δgoni int rot	0.125112	0.498662	0.514665	0.157156	-0.0366	0.209773
Δpt abd	-0.18877	0.184347	0.201628	0.12897	-0.04312	0.182128
Δpt add	-0.02714	0.068137	-0.05987	0.69457	-0.31303	-0.356
Δpt ext rot	0.090107	0.146993	0.039926	-0.10754	0.42686	0.069882
Δpt int rot	0.02198	0.304068	0.036463	-0.32321	0.35652	0.544484
ΔSPADI (f)	1	0.247086	0.071473	-0.14543	-0.24241	-0.13299
ΔSPADI (p)	0.247086	1	0.338892	0.238353	0.057382	0.031737
Δtw abd	0.071473	0.338892	1	0.414983	0.072037	0.146343
Δtw add	-0.14543	0.238353	0.414983	1	-0.25887	-0.17472
Δtw ext rot	-0.24241	0.057382	0.072037	-0.25887	1	0.718347
Δtw int rot	-0.13299	0.031737	0.146343	-0.17472	0.718347	1

Table ??: Pearson correlation coefficients and of objective and subjective outcomes in the "measurement group".

	Age (mean)	∆NRS	∆goni abd	∆goni ext rot	∆goni int rot	∆pt abd	∆pt add	∆pt ext rot	∆pt int rot
Age(mean)	1	-0.11507	-0.19239	0.007448	0.1681	-0.10622	0.131551	-0.21185	-0.06131
∆NRS	-0.11507	1	-0.14241	-0.18921	0.365531	0.038781	0.05261	-0.19432	-0.16616
∆goni abd	-0.19239	-0.14241	1	0.243342	0.161532	-0.1492	0.073469	-0.28101	-0.52938
∆goni ext rot	0.007448	-0.18921	0.243342	1	0.302668	-0.11342	0.220051	-0.19272	0.048742
∆goni int rot	0.1681	0.365531	0.161532	0.302668	1	-0.37365	0.442022	-0.31189	-0.22447
∆pt abd	-0.10622	0.038781	-0.1492	-0.11342	-0.37365	1	0.189033	0.390849	0.141678
∆pt add	0.131551	0.05261	0.073469	0.220051	0.442022	0.189033	1	-0.00437	-0.00138
∆pt ext rot	-0.21185	-0.19432	-0.28101	-0.19272	-0.31189	0.390849	-0.00437	1	0.444738
∆pt int rot	-0.06131	-0.16616	-0.52938	0.048742	-0.22447	0.141678	-0.00138	0.444738	1
∆SPADI (f)	0.170445	-0.27962	0.034375	-0.07612	-0.11855	-0.17764	-0.24352	-0.2573	-0.09381
∆SPADI (p)	0.336797	-0.44269	0.058948	0.025103	-0.16664	-0.09273	-0.27453	0.050857	0.10922
∆tw abd	-0.28714	0.146651	0.012058	0.097565	0.2761	0.470626	0.583965	0.284005	0.109595
∆tw add	-0.068	0.302288	-0.06861	0.11881	0.171217	0.342864	0.688031	0.065987	0.036727
∆tw ext rot	-0.15871	-0.11752	-0.13057	-0.24942	-0.24466	0.152816	-0.22071	0.833069	0.18214
∆tw int rot	-0.0438	0.002449	-0.22428	0.016373	-0.11442	0.435336	0.030278	0.582301	0.56997

	Δ SPADI (f)	Δ SPADI (p)	Δ w abd	Δtw add	Δtw ext rot	Δtw int rot
Age(mean)	0.170445	0.336797	-0.28714	-0.068	-0.15871	-0.0438
ΔNRS	-0.27962	-0.44269	0.146651	0.302288	-0.11752	0.002449
Δgoniabd	0.034375	0.058948	0.012058	-0.06861	-0.13057	-0.22428
Δgoni ext rot	-0.07612	0.025103	0.097565	0.11881	-0.24942	0.016373
Δgoni introt	-0.11855	-0.16664	0.2761	0.171217	-0.24466	-0.11442
Δpt abd	-0.17764	-0.09273	0.470626	0.342864	0.152816	0.435336
Δpt add	-0.24352	-0.27453	0.583965	0.688031	-0.22071	0.030278
Δpt ext rot	-0.2573	0.050857	0.284005	0.065987	0.833069	0.582301
Δpt int rot	-0.09381	0.10922	0.109595	0.036727	0.18214	0.56997
ΔSPADI (f)	1	0.737582	-0.35253	-0.24628	-0.14452	-0.00887
ΔSPADI(p)	0.737582	1	-0.38479	-0.46868	0.017959	0.10797
Δtw abd	-0.35253	-0.38479	1	0.576465	0.203102	0.429323
Δtw add	-0.24628	-0.46868	0.576465	1	0.00145	0.220946
Δtw ext rot	-0.14452	0.017959	0.203102	0.00145	1	0.600454
Δtw int rot	-0.00887	0.10797	0.429323	0.220946	0.600454	1

Appendix M: Summary of outcome measures.

		Treatment group (group 1)			Measurement group (group 2)		
Outcome measurements	p-values	Mean	Change 1 to 2	Change 1 to 3	Mean	Change 1 to 2	Change 1 to 3
NRS-101	0.7533	38.75	-1.63	-10.63	36.75	-1.25	-11.75
SPADI (F)	0.4848	42.15	-0.35	1.25	43.25	0.60	2.10
SPADI (P)	0.9043	49.15	0.25	2.70	52.20	0.65	2.50
PT: abduction	0.0886	45.35	3.50	7.45	41.50	3.75	1.80
TW:abduction	0.7266	42.90	1.25	5.55	33.70	4.60	4.50
PT:adduction	0.2673	83.85	7.15	16.35	74.55	11.90	9.30
TW:adduction	0.2916	65.85	10.60	9.15	75.75	6.70	15.45
PT:external rotation	0.1204	27.25	0.30	2.70	26.00	-1.35	0.15
TW:external rotation	0.5750	30.30	0.80	0.45	28.20	-1.60	-0.80
PT:internal rotation	0.5137	64.20	0.55	2.75	56.65	1.65	1.25
TW:internal rotation	0.4154	71.75	4.60	2.80	63.90	0.75	-0.55
Goni:abduction	0.3092	123.00	-0.50	3.00	122.75	-1.75	0.00
Goni:external rotation	0.8636	85.50	1.00	5.50	83.75	0.75	6.00
Goni:internal rotation	0.7371	83.25	0.00	4.75	83.00	2.50	5.50
No. of active TrPs	0.4323	3.50	0.15	-1.70	4.05	-0.20	-2.05
Total no. of TrPs	0.2875	3.80	0.10	-0.45	4.25	0.00	-0.90
Concomitant findings	0.2530	2.15	0.00	-0.75	2.30	0.00	-1.05

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