

**THE EFFECTIVENESS OF A HOME PROGRAMME OF ISCHAEMIC  
COMPRESSION COMPARED WITH SELF APPLIED ISCHAEMIC  
COMPRESSION UNDER CLINICAL OBSERVATION FOR THE  
TREATMENT OF MYOFASCIAL PAIN SYNDROME OF THE LEVATOR  
SCAPULAE MUSCLE**

**By  
Quinton Webb**

A dissertation submitted to the Faculty of Health in partial compliance with the requirements for a Masters Degree in Technology: Chiropractic at the Durban Institute of Technology.

I, Quinton Leslie Webb, do hereby declare that this dissertation represents my own work both in concept and execution.

**Quinton Leslie Webb**

**Date**

**Approved for final submission**

**Dr. Andrew Jones, M.Dip.C, CCSP, CCFC**

**Date**

## DEDICATION

This work is dedicated to my parents **Rosemary** and **Leslie** whom I love very much.

Thank you both for your love, understanding, support and encouragement throughout difficult times.

To **Victoria**, thank you for coming home. Your patience and love have made you the incredible person you are. I love you.

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## ABSTRACT

The purpose of this study was to determine the effectiveness of a home programme of ischaemic compression in the treatment of Myofascial Pain Syndrome.

The study was a prospective, unblinded, randomised, clinical trial. The sample size used was 40 patients selected from the Durban Metropolitan Area. Only patients diagnosed with active trigger points in the Levator Scapulae muscle were accepted into the study.

The sample was divided into two groups of 20 patients each. One group performed a home programme of ischaemic compression using a Thera Cane device, whilst the other group performed ischaemic compression under the observation of the researcher using the same Thera Cane device. Each patient performed five treatments over five consecutive days and then returned for a one-week follow-up for data collection only.

Data was obtained from the patients at the first and fifth consultations, prior to treatments, as well as at the one-week follow-up consultation. Objective data was obtained from pressure threshold algometry and the Myofascial Diagnostic Scale. Subjective data was obtained with the Numerical Pain Rating Scale (NRS 101).

Statistical analysis of the data involved both parametric and non-parametric testing. Initially a kolmogorov-Smirnov test was performed in order to test for normality of data.

Intra-group comparisons were made using the Friedmans test followed up by a Wilcoxon test for matched pairs. Analysis of Variance (ANOVA) was used to test for a significant difference. If the difference was significant a paired t-test was performed. Inter-group comparisons were made using the Mann Whitney

U-test and the independent paired t-test. All statistical analyses were completed at the 95% level of significance.

Evaluation of the intra-group statistical analysis revealed significant improvements with regards to subjective (NRS 101) and objective (MDS) data for both groups. With regards to the objective algometer readings, although an improvement in pressure threshold was evident, this improvement was not a statistically significant one. This may be related to post treatment soreness after performing ischaemic compression for five consecutive days.

Evaluation of the inter-group statistical analysis revealed no statistically significant difference between the groups except with regards to the NRS 101 where the clinic group showed a greater reduction in perceived pain intensity. This group being under observation may have performed the treatment more diligently or may indicate a positive Hawthorne effect.

Due to the fact that both groups tended to show significant improvement it was concluded that a home programme of ischaemic compression is an effective form of treatment for active trigger points of Myofascial Pain Syndrome, in terms of both subjective and objective clinical findings. Suggestions were to have patients perform treatments on alternate days so as to reduce the discomfort of post treatment soreness. It is hoped that the observations made by the author with regards to home based treatment programmes will encourage patients to take responsibility as the primary pain manager in adopting a wellness approach.

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# CHAPTER ONE: INTRODUCTION

## **1.1 THE PROBLEM AND ITS SETTING**

According to Hong et al. (1993) MPS is considered to be one of the most common muscular dysfunctions found in patients. The syndrome is one of the least understood yet commonly encountered problems in the out patient setting. Unfortunately, the condition often goes unrecognised, mis-diagnosed, mistreated, leading to unnecessary pain, suffering and disability (Auleciems, 1995). The fact that myofascial TrPs have been described in the literature for acupuncturists, anaesthesiologists, chronic pain managers, dentists, family practitioners, gynaecologists, neurologists, nurses, orthopaedic surgeons, paediatricians, physical therapists, physiologists, rheumatologists and veterinarians is evidence of the syndromes clinical importance. (Travell, Simons and Simons (1999 1:13).

Myofascial Pain Syndrome is a regional muscular disorder that results from myofascial trigger points (Lee et al., 1997) and may be “defined as the sensory, motor and autonomic symptoms caused by these myofascial trigger points (TrPs), or hyperirritable spots within skeletal muscles that are associated with hypersensitive palpable nodules in a taut band” (Travell, Simons and Simons, 1999 1:5).

In the clinical setting it becomes important to differentiate between active and latent trigger points. According to Travell, Simons and Simons (1999 1:1) an active myofascial trigger point is always tender, prevents full lengthening of

the muscle, weakens the muscle, mediates a local twitch response and when directly compressed refers pain within a specific pattern. It is this referred pain that distinguishes an active TrP from a latent TrP. A latent trigger point may have all the characteristics of an active trigger point in the absence of spontaneous pain (Travell, Simons and Simons (1999 1:4).

The literature reviewed advocates the application of stretch and spray, stretch and ice, deep massage, ischaemic compression, myofascial release, medication as well as exercises (Auleciems 1995:25-28), behavioral therapy, cold, heat, myofascial trigger point injection, spinal adjustments and electrical modalities (Hubbard1998: 23-26) According to Andersen (1998) the choice of treatment is often a personal one due to the lack of sufficient clinical evidence to support one particular technique over another. Despite the fact that a wide variety of treatment modalities for myofascial pain already exist, there is still agreement amongst authors that more studies that aid in the efficacy of treatment are required (Han and Harrison, 1997).

## **1.2 NEED FOR A SOLUTION TO THE PROBLEM**

This research study is designed to test one of the above treatment modalities namely, ischaemic compression, by offering it to patients as a self-administered home programme.

Ischaemic compression can be defined as the application of slowly increasing, non-painful pressure over a TrP until a barrier of tissue resistance is encountered. Contact is then maintained until the tissue barrier releases, and pressure is increased to reach a new barrier to eliminate the TrP tension and tenderness (Travell, Simons and Simons 1999 1:8).

Ischaemic compression is thought to cause a localised stretch of the contracted fibres thereby producing a mechanical separation of the actin-myosin cross fibres links (Schneider 1996). Schneider (1996) also suggests that prolonged deep pressure may induce a so-called "nerve block" by inhibiting the reflex pathways that perpetuate the TrP activity.

Ischaemic compression has been found to be more effective than other modalities in two independent studies by Garvey (1989) and Hong et al. (1993). Garvey (1989) found ischaemic compression to be more effective than lidocaine injection and dry needling in a randomised, double-blinded, prospective study involving 63 patients, while Hong et. al. (1993) found ischaemic compression to be more effective than spray and stretch, moist heat packs and ultrasound.

Hanten et. al. (2000) found a home programme, consisting of ischaemic pressure and sustained stretching to be effective in reducing TrP sensitivity and pain intensity in 40 individuals with neck and upper back pain. However, it was not clear whether the ischaemic pressure or the sustained stretching produced these results independently.

At present, it is not known whether ischaemic compression performed on its own by the patient as a home programme, will prove effective in the treatment of MPS. Ischaemic compression is a simple and easily taught, effective, inexpensive and non-invasive alternative to numerous follow-up sessions with patients suffering from difficult to treat Myofascial TrPs. Many of the other popular treatments for Myofascial TrPs can be costly, time consuming and invasive in nature.

It is essential to carry out a clinical trial to determine whether a home programme, which encourages a wellness approach and serves to involve the

patient as the primary pain manager, will prove effective in the treatment of Myofascial Pain Syndrome.

### **1.3 AIM OF THE STUDY**

The purpose of this investigation is to determine the effectiveness of a home programme of ischaemic compression, in terms of subjective and objective clinical findings, for the treatment of myofascial pain syndrome.

Objective 1 - to determine the effectiveness of a home programme of ischaemic compression, in terms of subjective clinical findings, for the treatment of myofascial pain syndrome.

Objective 2 - to determine the effectiveness of a home programme of ischaemic compression, in terms of objective clinical findings, for the treatment of myofascial pain syndrome.

### **1.4 HYPOTHESES**

It is hypothesised that a home programme of ischaemic compression will be effective in the treatment of patients with myofascial pain syndrome, in terms of both subjective and objective clinical findings, and that the home programme of ischaemic compression will be more effective than the clinic programme, for the treatment of this condition.

## **1.5 BENEFITS OF THE STUDY**

The hope is that this study will provide valuable information on the management of neck and upper back pain associated with TrPs through a home programme of ischaemic compression. The aim is to have minimal patient – clinician contact while still providing effective treatment and symptomatic relief in this age of managed health care, where emphasis is placed on shorter treatment times and decreased number of clinic visits. This study may also form the basis for further research into home treatment protocols for other muscles affected by TrPs with periodic monitoring by a clinician.



## **CHAPTER TWO: LITERATURE REVIEW**

### ***2.1 INTRODUCTION***

“Myofascial pain syndrome may be defined as the sensory, motor and autonomic symptoms caused by myofascial trigger points (TrPs), or hyperirritable spots within skeletal muscles that are associated with palpable nodules in a taut band. These trigger points are extremely common and become a distressing part of nearly everyone’s life at one time or another.” (Travell, Simons and Simons, 1999 1:5,12)

“Myofascial pain syndrome is one of the least understood, yet commonly encountered problems in the outpatient settings. Unfortunately, the condition often goes unrecognized, misdiagnosed, mistreated, leading to unnecessary pain, suffering and disability” (Auleciems, 1995). It is the opinion of Bruce (1995) that despite remarkable advances in modern health care, there is a lack of knowledge with regards to the understanding, evaluation and management of everyday musculoskeletal pain. The following chapter is an overview of the current literature and concepts in TrP aetiology, pathogenesis, diagnosis and management.

### ***2.2 PREVALENCE AND INCIDENCE***

The most recent studies on the incidence and prevalence of Myofascial Pain Syndrome seem to have been carried out since the 1980’s.

A study conducted by Skootsky et al. (1989) showed myofascial pain to be the single most common reason for a patient with pain to visit a physician. In this study, 172 consecutive patients presenting to a university primary care general internal medicine practice were examined. Thirty percent of the 54 patients whose reason for the visit was pain, were diagnosed with myofascial pain, representing the prevalence of this condition.

Han and Harrison (1997) found that the incidence of Myofascial Pain Syndrome varied between thirty and eighty five percent of people presenting to pain clinics and quote Taylor's Nuprin report (1985) which states that as many as 53% of the American population suffer with muscle pain, while 33% of these people experience pain lasting longer than 11 days, while 10% of these people experience pain lasting longer than 100 days.

(With the condition being more prevalent in women than in men).

Myofascial Pain Syndrome occurs in both sexes although it appears to be more common in females as found in a study where 107 out of the 119 patients treated for Myofascial Pain Syndrome, were female. Hou et al (2002:1411-1412). Two further studies by Walker and Wilks respectively, substantiate the fact that myofascial pain syndrome appears to be more common in females. Walker (2002) found that 72% of the 60 patients treated were female while Wilks (2003) found that 60% of the 60 patients treated for myofascial TrPs were female.

Four further studies by Gerwin (1995), Fishbain (1996), Chaiamnuay et. al. (1998) and Banks et. al. (1998) respectively, substantiate the high prevalence of Myofascial Pain Syndrome, Gerwin found that 93% of the 96 patients presenting at a community pain medical center had at least part of their pain caused by TrPs. Fishbain made an accurate diagnosis of Myofascial Pain Syndrome in

85% of 283 admissions to a pain centre. Chaiamnuay et al. found that 36,2% of the 2436 rural Thailand subjects examined and interviewed had musculoskeletal pain of which MPS was the second most common diagnosis. Banks et al. reports that patients with Myofascial Pain Syndrome in the United States account for over 70 million visits to physicians and 425 million visits to Chiropractors per annum.

People of any age can develop myofascial trigger points, which leads to Myofascial Pain Syndrome. Travell, Simons and Simons (1999 1:3) indicate that individuals in their mature years (31-50) are more likely to suffer from the condition. In a population of hospitalised and ambulatory Physical Medical patients with myofascial TrPs, the greatest numbers were between the ages of 31 and 50 years (Travell, Simons and Simons, 1999 1:3). Chettiar (2001) found that of the 60 patients treated for myofascial pain, 52% were between the ages of 32-55 and 43% were between 20-31. Van Aardenne (2002) found that of the 60 patients treated for myofascial pain in this study, 48.2% were between 31-50 while 36.6% were between 20-31. These two studies show the greatest number of sufferers being between the ages of 20-50 years.

## **2.3 AETIOLOGY**

According to Travell, Simons and Simons, (1999) acute injuries may cause immediate symptoms, while chronic stresses are more likely to cause a gradual onset of symptoms. The latter has a tendency to perpetuate the activation of trigger points.

The mechanical stresses that tend to activate Myofascial Trigger Points acutely include stresses such as wrenching movements, automobile accidents, falls, fractures, joint sprains, dislocations, a direct blow to the muscle, or an episode of excessive, unusual exercise. Sustained postural overload, prolonged immobilization and poor work ergonomics may lead to TrP formation by way of gradual onset or chronic stress aetiologies. Travell, Simons and Simons, (1999) indicate that TrPs within the levator scapulae muscle become active due to sustained elevation of the shoulders or due to cramped positioning, particularly when the muscle is fatigued and exposed to cold.

Orthopaedic abnormalities that place the muscles in prolonged abnormal function may activate TrPs e.g. TrPs occurring in muscles that lie within the sclerotogenous referred pain zones of inflamed joints, or the dermatomal referred pain zone of an inflamed nerve root (Schneider, 1995). Chu (1997) and Travell, Simons and Simons (1999) agree that TrPs may develop in muscles innervated by a compressed nerve.

Gatterman (1990) believes that the following factors predispose patients to developing TrPs: poor fitness, inadequate nutrition, allergies, metabolic abnormalities and physical, sexual and psychological abuses.

Indirect activation of TrPs can also occur as a result of visceral diseases, arthritic joints, other existing TrPs and by emotional distress (Travell, Simons and Simons, 1999) these authors also mention iatrogenic causes of TrPs e.g. intramuscular injection of medicinal substance and therapeutic interventions such as spray and stretch may activate latent TrPs.

According to Gay *et al.* (1994) the currently accepted aetiology of MFPS is that of a cycle of muscle hyperactivity, which leads to muscle spasm, pain and finally constant chronic muscle fatigue.

Hong and Simons (1998) proposed the spinal cord mechanism of TrP activation where “input from nociceptors in an original receptive field persists (pain from an active TrP), central sensitisation in the spinal cord may develop and the receptive field corresponding to the original dorsal horn neuron may be expanded (referred pain). Through this mechanism, new TrPs, or satellite TrPs, may develop in the referred zone of the original TrP.”

## ***2.4 PERPETUATING FACTORS***

According to Auleciems (1995) the events that activate TrPs can be quite different from the factors that perpetuate them. Travell, Simons and Simons, (1999 1:178) are of the opinion that the long-term prognosis of Myofascial Pain Syndrome improves drastically when the factors that perpetuate the condition are corrected. The following is a list of perpetuating factors outlined by Travell, Simons and Simons, (1999 1: 110-112).

Mechanical stresses such as skeletal asymmetry (short leg or small hemi pelvis) and disproportion (long second metatarsal and short upper arms). Misfitting furniture, poor posture and prolonged immobility are listed as other significant contributing factors. People who work at a desk, computer or typewriter for long periods at a time, e.g. secretaries, according to Travell, Simons and Simons (1999) are more susceptible to developing levator scapulae TrPs. Typing with the

head and neck turned to look at the computer, making long telephone calls without using a headset and sleeping without adequate pillow support, so that the neck is in a tilted position are factors which commonly perpetuate levator scapulae TrPs. (Travell, Simons and Simons 1999)

Nutritional inadequacies, metabolic and endocrine disturbances, psychological factors, (depression, tension, anxiety), chronic infection, allergy, impaired sleep and nerve entrapment are all factors which could aggravate the condition of MFPS.

## **2.5 CLINICAL FEATURES**

### 2.5.1 Symptoms

According to Han and Harrison (1997) patients will typically present with a history of regional pain which ranges from a mild ache to excruciating pain, that is either described as being sharp, dull, burning or heavy and often associated with general fatigue and decreased range of motion and muscle strength. The pain is usually constant, reproducible and does not follow a dermatomal or nerve root distribution.

Patients will usually present with pain related to a traumatic injury e.g. muscle strain or overload, or, in chronic cases related to repetitive strain or other cases of chronic muscular tension e.g. poor posture (Schneider, 1995). According to Travell, Simons and Simons (1999) when patients present with markedly limited

rotation or “stiff neck” it is usually as a result of TrPs in the levator scapulae muscle.

Additional symptoms as described by Travell, Simons and Simons, (1999 1:21) are as follows:

- Disturbances of autonomic functions and proprioceptive disturbances.
- Motor disturbances including weakness of involved muscles, spasm of other muscles (synergistic and/or antagonistic muscles) and decreased muscle power or work tolerance.

Myofascial pain may present as referred pain to a distant site from the TrP, in a characteristic pattern for that muscle with some patients being aware of numbness or parasthesia rather than pain (Travell, Simons and Simons, 1999).

The referred pain from the levator scapulae muscle usually concentrates in the angle of the neck and along the vertebral border of the scapula and sometimes projects to the posterior shoulder joint.

### 2.5.2 Signs

Upon examination of a patient suffering with Myofascial Pain Syndrome, certain physical findings are necessary before a correct diagnosis can be made. The most characteristic physical sign in MPS is the presence of trigger points (Travell, Simons and Simons, 1999)

Auleciems (1995) states that TrPs exist in a variety of forms namely active and latent. An active TrP is painful when compressed and will often cause

characteristic referred pain and autonomic phenomena. A latent TrP may result in muscle stiffness, weakness, limited range of motion and dysfunction, but without persistent pain of the affected muscle and no referred pain.

The method most frequently used to locate TrPs is palpation of the affected muscle by applying sustained deep pressure (Han and Harrison, 1997). According to Travell, Simons and Simons, (1999), active TrPs are identified when patients recognize the pain that was induced by applied pressure to a TrP, as the pain they experience at rest. These TrPs are always located within a taut band of hypersensitive muscle fibres, and snapping palpation across the muscle fibres may elicit a local twitch response. Local twitch response (LTR) is a brisk contraction of the muscle fibres in and around the taut band. The same effect is achieved by rapid needle insertion into the TrP (Hong and Simons, 1998). Often when TrPs are severely tender patients may withdraw from the examiner exhibiting a reaction known as a “jump sign” (Han and Harrison, 1997).

## ***2.6 DIAGNOSIS OF MYOFASCIAL PAIN SYNDROME***

Schneider (1995) outlines a set of recommended diagnostic criteria for Myofascial Pain Syndrome:

### Major Criteria

Regional pain complaint

Pain pattern in the expected distribution of muscular referred pain.

Palpable taut band in accessible muscles.

Exquisite spot tenderness at one point or nodule within a taut band.



Some degree of restricted range of motion or slight muscle weakness

### Minor Criteria

Manual pressure on the TrP nodule reproduces the clinical pain complaint.

Local twitch response caused by either snapping, palpation or injection of the tender spot.

Pain is diminished or eliminated by muscular therapy e.g. therapeutic stretch, ischaemic compression or needle injection of the TrP.

To diagnose MPS, all five major criteria should be present and at least one of the three minor criteria.

Travel, Simons and Simons (1999), suggest that the minimum acceptable criteria for identifying a trigger point are a combination of the spot tenderness in a palpable band and patient recognition of the pain.

#### 2.6.1 Confirmatory Diagnosis

The Myofascial Diagnostic Scale Chettiar (2001) was made up of four indicators. The first indicator consisted of five grades of soft tissue tenderness. Each grade was scored as follows:

Grade 0 - no tenderness = 0

Grade 1 - tenderness to palpation without grimace or flinch = 1

Grade 2 - tenderness with grimace and or flinch to palpation = 2

Grade 3 - tenderness with withdrawal = 3

Grade 4 - withdrawal to non-noxious stimuli = 4

The second and third indicators represented the presence of the local twitch response and the taut band respectively. These indicators were given a value of 4 each. The fourth indicator was the presence of referred pain. Since this sign is considered the strongest indicator of an active trigger point, this indicator was given a value of 5. These signs were assessed and scored by the researcher. Total values of 9 or more were indicative of an active trigger point and only these patients were accepted into the study. The data was collected at the initial, the fifth and one week follow up visits, which allowed the researcher to establish intra-group and inter-group changes in terms of clinical signs.

## ***2.7 PATHOPHYSIOLOGY OF TRIGGER POINTS***

The following is an overview of muscle structure, function and the formation of TrPs. This is a summary from Travell, Simons and Simons, (1999 1:45-60)

A muscle consists of a bundle of fascicles, each of which is made up of muscle fibres. These fibres each contain numerous myofibrils surrounded by a sac-like structure called the sarcoplasmic reticulum. The sarcoplasmic reticulum is the source of the contractile force of muscle. Calcium is released from the sarcoplasmic reticulum thereby stimulating the actin and myosin of the myofibrils to contract in the presence of ATP (adenosine triphosphate). Action potentials are responsible for this release of calcium and the contraction is maintained until the ATP is depleted or until the free calcium is returned to the sarcoplasmic reticulum.

A motor unit consists of the cell body, its axon and multiple motor endplates of an alpha motorneuron in the anterior horn of the spinal cord. The action potential

begins in the cell body, travels along the axon and is then transmitted chemically across the synaptic cleft of the motor endplate thereby causing a muscle contraction.

Travell, Simons and Simons, (1999) suggest that "a TrP is a cluster of minute loci of intense abnormality found throughout the trigger point and that this abnormality is a neuromuscular dysfunction of the motor endplate." Events such as trauma or prolonged mechanical stress may result in an excessive release of acetylcholine from the nerve terminal. This causes a sustained release of calcium from the sarcoplasmic reticulum resulting in maximal contracture of the muscle fibre. This sustained contraction produces a local ischaemia, which prevents oxygen and ATP from entering the area and therefore the calcium pump is unable to return calcium to the sarcoplasmic reticulum. The continuous contact with calcium causes further contraction and a vicious cycle is set up. This process is known as the "energy crisis theory". Histologically these areas of contraction are visible as contraction knots. A group of these contraction knots within a taut band of muscle constitute a TrP and give it its nodular feel. [Travell, Simons and Simons, 1999]

The "energy crisis" resulting in these areas may stimulate the production of vasoreactive substances that can sensitise local nociceptors known as sensitive loci. It is believed that these sensory nociceptors or sensitive loci elicit pain, referred pain and latent twitch responses. These sensitive loci are found throughout the entire muscle but are in higher concentrations within the TrP region. When a sensitive locus and an active locus are in close proximity, a myofascial TrP locus develops. When the input from the sensitive loci persists, central sensitisation in the spinal cord may develop, resulting in referred pain corresponding to the receptive field of the original dorsal horn neuron. [Travell, Simons and Simons 1999]

## **2.8 TREATMENT OF MYOFASCIAL PAIN SYNDROME**

Myofascial pain syndrome can range from simple cases with single muscle syndromes to complex cases involving multiple muscles and numerous interrelating factors. It is the opinion of Friction (1994) that the difficulty in managing myofascial pain lies in the need to match the level of complexity of the management program with that of the patient. Fischer (1999) believed that treatment of myofascial pain should aim at reducing pain quickly thereby enabling patients to cope with this pain and that the removal of aetiological factors is necessary to prevent recurrence of pain.

A wide variety of treatment modalities including spray and stretch, trigger point injection, dry needling, exercise, TENS, ultrasound, massage, ischaemic compression, biofeedback and psychological intervention among others are available according to preference of the clinician (Han and Harrison, 1997: 95; Hubbard, 1998:23). Hou et al (2002:1406) state that despite all the research done on Myofascial Pain Syndrome, the clinical efficacy of the numerous techniques has not been well established.

According to Gatterman (1990: 296) treatment of myofascial pain syndrome is aimed at breaking the pain – spasm – pain cycle by disrupting the reverberating neural circuits. This may be achieved by inactivation of active TrPs by mechanically releasing the taut bands within the muscle by means of localised stretching of this taut band (Hong et al. 1993). The energy crisis theory by Travell, Simons and Simons, (1999 1:72) explains the effectiveness of any treatment that essentially stretches the TrP portion of the muscle by decreasing the energy consuming contractive activity between actin-myosin filaments. Schneider (1995) believes that the common factor among all TrP modalities is that, in some way, they all release the contracture of taut bands within the skeletal muscle. Some of the many treatment methods are discussed below.

Several treatment methods including trigger point injection, dry needling, spray and stretch and transcutaneous electrical nerve stimulation (TENS) have been studied for effectiveness. (Han and Harrison 1997)

### 2.8.1 Dry needling and TrP injection

Several authors have reported the effectiveness of injection of saline or local anaesthetics as well as dry needling in the treatment of Myofascial Pain Syndrome. (Hameroff et. al. 1981, Garvey et. al. 1989, Hong 1994, Broome 1996). However, this treatment involves an invasive procedure and often produces post-injection soreness as well as muscle necrosis (Hong 1994). Consideration must be given to the contraindications that may arise. Contraindications to TrP injections such as allergy to anaesthesia agents, bleeding disorders, local or systemic infection and those on anti-coagulation therapy would exclude this form of treatment (Han and Harrison 1997). It is the opinion of these authors that TrP injection following acute muscle trauma should not be attempted.

Garvey et al.(1989) compared injection of a local anaesthetic, injection of a local anaesthetic plus steroid, dry needling and acupuncture with vapocoolant spray. The authors found that the acupuncture plus vapocoolant spray, their control procedure and the only non-invasive procedure, was the most effective at relieving pain.

### 2.8.2 Modalities

The TENS modality has been successfully employed in the treatment of trigger points as demonstrated by Hutchings (1998). However, Han and Harrison (1997) and Graff-Radford et al (1989) in two independent studies found that although TENS showed a reduction in myofascial pain intensity there was no change in trigger point sensitivity, hence the questionable effect in producing sufficient long term benefits.

In a randomised controlled trial done by Gam et al (1998) they found that ultrasound gave no pain reduction and was ineffective in the treatment of Myofascial Pain Syndrome.

A study by Christie (1995) compared dry needling to interferential current for 30 patients with TrPs in the shoulder girdle. Both groups shared an equal improvement in symptoms and Christie concluded that interferential current was a viable alternative treatment for Myofascial Pain Syndrome.

### 2.8.3 Spray and Stretch

Spray and stretch using a vapocoolant spray, is performed by passively stretching the involved muscle while applying a cooling agent. Jaeger, Reeves and Graff-Radford (1986) found that TrP sensitivity and pain intensity decreased following spray and stretch of patients affected by Myofascial Pain Syndrome. Travell, Simons and Simons, (1999) hypothesized that the decrease in TrP pain, utilizing spray and stretch is due to the elongation of the muscle to its full length.

Hanten *et. al.* (2000), Travell, Simons and Simons, (1999) and Lewit and Simons (1984) all agree that muscle lengthening or muscle stretch is the process which plays the major role in TrP pain relief.

#### 2.8.4 Soft Tissue Therapies.

Schneider (1996: 78) suggests the use of manual soft tissue therapies, as they do not require any sophisticated or expensive equipment, they are simple and easy to use, quick to apply and are non-invasive in nature. These manual therapies include spray and stretch, post-isometric relaxation, ischaemic compression and active release techniques.

The most widely recognized form of manual therapy for the treatment of myofascial pain syndrome is ischaemic compression or trigger point pressure release (Travel, Simons and Simons, 1999 1:26) Ischaemic compression has been shown to be effective in treating Myofascial TrPs by Hong *et al.* (1993) who showed that deep pressure, where the palpable taut bands were firmly compressed to the extent the patient could tolerate, by two digits of the therapist, and then stretched by moving these two digits towards the distal portions of the muscle (i.e. a modified form of ischaemic compression) was more effective in reducing pain levels of active TrPs than ultrasound, spray and stretch or hydrocollater heat packs in a study involving 83 patients with active TrPs in the upper Trapezius muscles. Garvey *et. al.* (1989) found ischaemic compression to be more effective than lidocaine injection and dry needling in a randomised, double-blinded, prospective study involving 63 patients. Hanten *et al.* (2000) found a home program of ischaemic compression followed by sustained stretch to be more effective than a control treatment of active range of motion in reducing both TrP sensitivity and pain intensity in 40 individuals with active TrPs.

They did not examine effectiveness relative to any other outcome such as functional limitation or disability and indicated that it was not clear whether the ischaemic compression or the sustained stretch was responsible for the favourable results.

Schneider (1996) describes ischaemic compression as a firm, non-painful, direct pressure to the centre of the TrP which causes a specific localized stretch of the contracted fibres and may mechanically separate actin-myosin cross fibre links. He further states that prolonged deep pressure may induce a so-called “nerve block by inhibiting the reflex pathways that perpetuate the TrP activity.” Schneider (1996) also mentions reflex vasodilation and hyperstimulation analgesia as possible mechanisms of action of ischaemic compression. Sandman (1981) and Schneider (1996) both agree that applying ischaemic compression with excessive force or duration will cause the patient to respond with muscle tightening, thereby increasing the patients pain or causing muscle bruising.

In a study by Hanten (2000) a device called a Thera Cane was successfully used to perform a home programme of ischaemic pressure. The Thera Cane is a J-shaped cane with 6 knobs placed at various points on the cane. It is a non-invasive passive device, which was designed to allow minimal exertion by the user to create sustained pressure in hard-to-reach areas.

## ***2.9 AN OVERVIEW OF THE LEVATOR SCAPULAE MUSCLE***

The Levator Scapulae muscle is one of the most commonly involved shoulder-girdle muscles, with respect to myofascial pain syndrome (Travell, Simons and



Simons, 1999). TrPs within the Levator Scapulae muscle develop at two locations. The primary TrP at the angle of the neck where the muscle emerges beneath the anterior border of the upper trapezius, and a secondary TrP slightly above the muscles attachment to the superior angle of the scapula (Travell, Simons and Simons, 1999). Referred pain from these TrPs is concentrated at the angle of the neck with a spillover along the medial border of the scapula and the posterior aspect of the shoulder.

When patients suffer from a “stiff neck” it is often due to active TrPs within the Levator Scapulae muscle which limits neck rotation due to pain (Travell, Simons and Simons, 1999)

## ***2.10 SUMMARY OF THE LITERATURE***

It is quite clear from the above literature review that Myofascial Pain Syndrome is a common condition encountered in the field of musculoskeletal medicine. Although many forms of treatment have been shown to be beneficial in the treatment of Myofascial Pain, many authors agree that more studies into the efficiency of treatments are required (Han and Harrison 1997). Since ischaemic compression has been shown to be a safe, inexpensive, effective and easily taught non-invasive procedure for self-treatment it's important to determine it's value with respect to a home protocol of treatment for Myofascial Pain Syndrome.

## **CHAPTER THREE: METHODOLOGY**

### **3.1 INTRODUCTION**

The details of the research study are discussed in this chapter. This includes a detailed description of the study design, subjects used, data measurement and procedures used for intervention. The methods of statistical analysis used for evaluation of data are also discussed.

The objective of this study was to determine the effectiveness of a home programme of ischaemic compression for the treatment of myofascial pain syndrome of the levator scapulae muscles.

3.1.1 The first objective was to evaluate the effectiveness of a home programme of ischaemic compression for the treatment of myofascial pain syndrome (of the levator scapulae muscles) in terms of subjective clinical findings.

3.1.2 The second objective was to evaluate the effectiveness of a home programme of ischaemic compression for the treatment of myofascial pain syndrome (of the levator scapulae muscles) in terms of objective clinical findings.

## **3.2 STUDY DESIGN AND PROTOCOL**

This study was a prospective, randomized clinical trial involving 40 patients divided into 2 groups of 20 individuals each. Local notice boards, flyers and newspapers were used to inform subjects of the study (Appendix J). On presenting to the Chiropractic Day Clinic patients were randomly assigned to either the home group (group A), or the clinic group, (group B). Only myofascial trigger points in the levator scapulae muscle were treated in an attempt to achieve sample stratification.

### **3.2.1 Standard of Acceptance**

Only patients presenting to the Chiropractic Day Clinic (Durban) were considered for the study. At the initial consultation the patient underwent a full case history (Appendix C), physical examination (Appendix D) and a regional examination (Appendix E). The patient was screened for myofascial pain syndrome of the levator scapulae muscles and assessed for meeting the inclusion and exclusion criteria below. Only patients who met these criteria were accepted into the study.

### **3.2.2 Diagnostic Criteria**

Only patients with one or more active trigger points (TrPs) in the levator scapulae muscle were accepted into the study. Both TrPs are known to have an essential reference zone at the angle of the neck, with a spill over zone along the vertebral border of the scapula and to the posterior shoulder of the lateral deltoid area. In addition TrP 2 is known to project pain to the inferior angle of the scapula. (Travell, Simons and Simons, 1999).

An active trigger point is one that shows the characteristics as outlined by Travell, Simons and Simons, (1999), including:

Taut band of muscle fibres palpated by snapping or rolling the muscle under the finger.

Tender nodule palpated within this taut band of muscle fibres.

Local twitch response of the taut band fibres to snapping palpation.

Pain reference to the reference zone specific to the muscle involved. (Travell, Simons and Simons, 1999 1:21/22).

### 3.2.3 Inclusion and Exclusion Criteria of Patients

#### **Inclusion criteria –**

Only patients between the ages of 18 - 55 years were accepted into the study. According to Travell, Simons and Simons (1999 1:12) individuals in their mature years (up to 55 years) are most likely to suffer from the pain syndromes of active myofascial TrPs. Both male and female volunteers were accepted.

Patients were only accepted into the study on exhibition of varying degrees of the characteristics ranked in the Myofascial Diagnostic Scale (APENDIX I). The Myofascial Diagnostic Scale (Chettiar, 2001) uses a scoring system where each of the four signs of an active trigger point, according to Travell, Simons and Simons, (1999), is given a value. Face validity for the Myofascial Diagnostic Scale has been completed.

## **Exclusion Criteria –**

Those patients that exhibited any of the contra indications to massage and massage type therapies were excluded from the study. Those include infection due to bacterial action: rheumatoid, infective or gouty arthritis: bursitis and calcification in soft tissue structures as outlined by Basmajian (1985: 284 - 285).

Patients who exhibited the signs of fibromyalgia syndrome were excluded from the study. Fibromyalgia syndrome is diagnosed by a history of wide spread pain for at least three months (pain on both sides of the body, above and below the waist) and the pain in 11 of 18 tender point sites on digital palpation (Schneider 1995). For the purpose of this study only patients with active myofascial trigger points were accepted.

Participants were not to receive any other form of treatment for Myofascial Pain Syndrome or related musculoskeletal conditions for the entire duration of the study.

Patients using Cipromil medication were excluded from the study.

If any new medication had to be started while involved with the study, that participant was excluded from the study.

If any major lifestyle changes (exercise) were made while involved with the study, that participant was excluded from the study.

### ***3.3 DETAILED PATIENT PROCEDURE***

At the initial consultation the researcher explained the nature and importance of the study to the subjects who had been accepted. In addition each patient

was given a letter of information (Appendix A) and was asked to complete an informed consent form (Appendix B), before commencement of the treatments. Each patient received 5 treatments over 5 consecutive days and then reported to the clinic for a one-week follow-up where measurements were taken but no treatment given.

Patients assigned to group A (home) received a home programme of ischaemic compression using a Thera Cane device, whilst patients assigned to group B (clinic) received a clinic programme of ischaemic compression using a Thera Cane device.

### 3.3.1 Clinic Programme of Ischaemic Compression

The Thera Cane is a passive non-invasive device, which is used to apply pressure to active myofascial trigger points. It is a plastic J-shaped cane with 6 knots placed at various points on the cane. The cane was designed to allow minimal exertion by the user to create sustained pressure in hard-to-reach areas (Hanten et. al. 2000).

The primary trigger point within the levator scapulae muscle was located and marked, using henna dye, by the researcher. The patient was then given verbal and written instructions (Appendix F) followed by a demonstration on how to perform ischaemic compression using a Thera Cane device. The patient then placed the Thera Cane over his or her primary TrP, applied gradually increasing pressure to the TrP and held that pressure for up to one minute. The researcher observed this procedure in the clinic on 5 consecutive days. The patients returned to the clinic one-week after the last treatment for data collection only.

### 3.3.2 Home Programme of Ischaemic Compression

The primary trigger point within the levator scapulae muscle was located and marked, using henna dye, by the researcher. The patient was then given verbal and written instructions (Appendix F) followed by a demonstration on how to perform ischaemic compression using a Thera Cane device. These patients were then instructed to perform this procedure at home once per day for five consecutive days. These patients presented to the clinic on the first, fifth and again for a one-week follow up for the collection of data. These visits took place before the treatments were performed for those days. Patients in this group were asked to keep a diary of their treatment sessions to ensure compliance (Appendix K).

## **3.4 THE DATA**

### 3.4.1 The Primary Data

The primary data included the following information for each patient:

- Case history (Appendix C).
- Physical examination (Appendix D).
- Cervical spine regional examination (Appendix E).
- Subjective data: Numerical pain rating scale 101 (NRS 101) (Appendix G).
- Objective data: Algometer reading (Appendix H).  
Myofascial diagnostic scale (Appendix I).

### 3.4.2 The Secondary Data

Secondary data was collected from related literature found in journal articles, textbooks and the Internet.

## **3.5 METHODS OF MEASUREMENT**

The subjective and objective measurements were obtained from each patient at the initial consultation, prior to the first treatment, and again at the fifth consultation (prior to the fifth treatment) and at a one-week follow-up consultation where no treatment was performed.

### 3.5.1 Subjective Measurements

#### 3.5.1.1 Numerical Pain Rating Scale (NRS-101)

The NRS-101 assesses the perceived level of pain intensity of the patient. The questionnaire consists of a numerical scale from 0 – 100, where 0 = no pain and 100 = pain at its worst. Jensen et al (1986) examined the usefulness of six different pain intensity measures in a group of 75 chronic pain patients and the NRS 101 proved to be the most practical. It is simple to administer and score in written or verbal form. The NRS-101 is not associated with incorrect responding more than any other scale and the difficulty is not associated with age. A mean percentage was obtained for each consultation by adding the two scores (for pain at its least and pain at its worst)



## 3.5.2 Objective Measurements

### 3.5.2.1 Pressure Threshold Algometry

Fischer (1987: 207) refers to pressure threshold, as the minimum pressure required causing pain or discomfort. Fischer (1987) performed a study on the pressure threshold measurement for diagnosis and evaluation of treatment results of trigger points and he concluded that pressure algometry is an accurate method for diagnosis of trigger points and useful in their clinical management and assessment of treatment results. The reliability of the pressure algometer has been demonstrated in studies by Reeves et al. (1986). The algometer used was FDK20 force dial manufactured by Wagner Instruments: P O Box 1217, Greenwich, CT 06836. The pressure range of the algometer was 11 kilograms.

The algometer was used as follows:

- The dial on the gauge was set to zero.
- The 1cm rubber disc was applied to the point of maximum tenderness by placing the gauge perpendicular to the surface.
- The pressure was gradually increased at a rate of 1kg/second, as recommended by Fischer (1986).
- The patient was told to say “now” at the point of which they first perceived pain.
- The pressure was stopped at this point by removing the gauge from the skin.
- The reading on the dial was recorded.

### 3.5.2.2 Myofascial Diagnostic Scale

The Myofascial Diagnostic Scale (Appendix I) was designed and used to evaluate the clinical signs of Myofascial Pain Syndrome by Chettiar (2001). According to Travell Simons and Simons (1999 1: 35-35) the signs of a trigger point are the following: referred pain in the zone of reference, local twitch response, palpable taut band, and focal tenderness.

The Myofascial Diagnostic Scale as outlined by Chettiar (2001) is made up of 4 indicators. The first indicator consisted of 5 grades of soft tissue tenderness:

- 0 = no tenderness (0 points).
- 1 = tenderness to palpation without grimace or flinch (1 point).
- 2 = tenderness to palpation with grimace or flinch (2 points).
- 3 = tenderness with withdrawal (3 points).
- 4 = withdrawal to non-noxious stimulus (4 points).

The second and third indicators represented the presence of the local twitch response and the taut band respectively. These were each given a value of 4. The fourth indicator is the presence of referred pain due to trigger point compression. This indicator was given the value 5 as it is deemed the strongest indicator of active trigger points. Any patient scoring a total of 9 or more points was considered to have an active myofascial trigger point and hence accepted into the study (Chettiar, 2001).

### **3.6 ETHICAL CONSIDERATION**

- The rights and the welfare of the patients were protected.
- Informed consent was obtained (Appendix B).
- Patients were not coerced into participating in the study.
- Information was given to patients in an understandable language.
- Confidentiality was maintained.
- Participation was voluntary and did not involve financial benefits.
- Patients were free to withdraw from the study at any stage.

### **3.7 TREATMENT OF DATA**

The subjective data was treated as follows:

- The questionnaires were checked by the researcher for correctness.
- The scores obtained from the NRS-101 were expressed as mean percentages for each consultation.
- The data were then statistically analysed.

The objective data were treated as follows:

- The algometric readings were recorded in kg/sq. cm.
- The scores obtained from the myofascial diagnostic scale were recorded as whole numbers, with the highest possible score being 17.
- The data were then statistically analysed.

### **3.8 STATISTICAL ANALYSIS**

Statistical Analysis was conducted using the SPSS (version 11.5) software suite. This Statistical software program is manufactured by SPSS Inc, 444N. Michigan Avenue, Chicago, Illinois, USA. Various descriptive and inferential statistical techniques were used. The descriptive procedures used were tables, graphs and summary statistics including but not limited to means, proportions and percentages. Various hypothesis tests were used in the inferential procedures. Throughout we tested for normality of the appropriate random variate and based on the results we either applied a parametric or non-parametric test. All hypothesis tests set the type 1 error at 5%, or mentioned differently  $\alpha = 0.05$ . If the p value, as reported, was less than 0.05 a significant result was declared and the null hypothesis was rejected and alternatively if the p – Values were greater than 0.05 the null hypothesis was not rejected.

#### 3.8.1 Exploratory testing of the Data

Initially, and throughout all the analyses, the Kolmogorov Smirnov test statistic was used to test normality of data. If the p- values were less than 0.05 the null hypothesis was rejected concluding there was sufficient evidence to reflect that the data was significantly different from a normal distribution. In this case the suitable non-parametric testing techniques were performed. If the p – values were on the borderline i.e. slightly larger than 0.05, then both the parametric and non-parametric testing techniques were performed.

### 3.8.2 Parametric Tests

For all the Intra Group tests the Analysis of Variance (Anova) test was performed to test if there was a significant difference between three or more population means. If this difference was significant this test was followed up by a Paired T Test for all possible combination of pairs of population means to see where this difference was occurring. If Anova was not significant no follow up testing was necessary.

For the inter group tests dealing with only 2 groups of population means the Independent Paired T Test was used to test for significant difference of 2 population means and this was done for all pairs.

The same tests were applied to both the objective tests (Myofascial Diagnostic Scale and the Algometer readings) and the subjective test (The Numerical Rating Scale).

### 3.8.3 Non - Parametric Tests

For all the intra group tests the Friedman`s Test was performed to test if there was a significant difference between three or more population means. If this difference was significant, a Wilcoxon Test for matched pairs was performed. This second test was run for all possible combination of pairs of population means to see where this difference was occurring. If Friedman`s was not significant, no follow up testing was necessary.

For the inter group tests dealing with only 2 groups of population means, the Mann Whitney U –Test was used to test for significant difference of 2 population means and this was done for all pairs.

The same tests were applied to both the objective tests (Myofascial Diagnostic Scale and the Algometer readings) and the subjective test (The Numerical Pain Rating Scale).

### **3.9 TEST 1:THE KOLMOGOROV SMIRNOV TEST**

$H_0$  : The random variable under observation follows a normal distribution

$H_1$  : The random variable under observation does not follow a normal distribution.

$\alpha = 0.05$

Note:  $\alpha$  = probability of rejecting  $H_0$  when is true (Type 1: error)

The test is two tailed.

The test statistic is:

The Kolmogorov-Smirnov Test procedure compares the observed cumulative distribution function for a variable with a specified theoretical distribution, in this case normal. The Kolmogorov-Smirnov Z is computed from the largest difference (in absolute value) between the observed and theoretical cumulative distribution functions. This goodness-of-fit test tests whether the observations could reasonably have come from the specified distribution

The tabulated value is obtained from Tables.

Note: The p – value = The probability of  $H_0$  being true.

If the p-value is  $< \alpha = 0.05$  then  $H_0$  is rejected.

### **3.10 TEST 2: ANALYSIS OF VARIANCE**

$H_0$  : All three population means are equal.

$H_1$  : At least one of the population means is unequal.

$\alpha = 0.05$

Note:  $\alpha$  = probability of rejecting  $H_0$  when is true (Type 1: error)

The test is two tailed.

The test statistic is:

$$F \text{ Test Statistic} = \frac{\text{Sum of Squares (Treatments) / Degrees of Freedom for treatments}}{\text{Sum of Squares (Error) / Degrees of Freedom for Error}}$$

The tabulated value obtained from F Tables.

Note: The p – value = The probability of  $H_0$  being true.

If the p-value is  $< \alpha = 0.05$  then  $H_0$  is rejected.

### **3.11 TEST 3: PAIRED T TEST**

$H_0$  :  $\mu_1 = \mu_2$

$H_1$  :  $\mu_1 \neq \mu_2$

$$\alpha = 0.05$$

Note:  $\alpha$  = probability of rejecting  $H_0$  when is true (Type 1: error)

The test is two tailed.

The test statistic is:

$$T \text{ Test Statistic} = \frac{\sqrt{(n-1)}\Sigma d}{\sqrt{n\Sigma d^2 - (\Sigma d)^2}}$$

where  $d$  = the difference between the 2 columns.

And  $n$  = the number of pairs.

The tabulated value obtained from T Tables.

Note: The  $p$  – value = The probability of  $H_0$  being true.

If the  $p$ -value is  $< \alpha = 0.05$  then  $H_0$  is rejected.

### **3.12 TEST 4: INDEPENDENT PAIRED T TESTS**

$$H_0 : \mu_1 = \mu_2$$

$$H_1 : \mu_1 \neq \mu_2$$

$$\alpha = 0.05$$

Note:  $\alpha$  = probability of rejecting  $H_0$  when is true (Type 1: error)

The test is two tailed.



The test statistic is:

$$T \text{ Test Statistic} = \frac{x_1 - x_2 - 0}{\sqrt{s_1^2/n_1 + s_2^2/n_2}}$$

The tabulated value obtained from T Tables.

Note: The p – value = The probability of Ho being true.

If the p-value is  $< \alpha = 0.05$  then Ho is rejected.

### **3.13 TEST 5: FRIEDMANS TEST**

H<sub>0</sub> : All three population means are equal.

H<sub>1</sub> : At least one of the population means is unequal.

$\alpha = 0.05$

Note:  $\alpha$  = probability of rejecting Ho when is true (Type 1: error)

The test is two tailed.

The test statistic is:

$$\chi^2 = \frac{\sum(Rg)^2 - 3n(k+1)}{nk(k+1)}$$

where n = number of observations

k = number of columns

$R_g$  = the sum of ranks for column  $g$

The tabulated value obtained from  $\chi^2$  Tables.

Note: The  $p$  – value = The probability of  $H_0$  being true.

If the  $p$ -value is  $< \alpha = 0.05$  then  $H_0$  is rejected.

### **3.14 TEST 6: WILCOXON TEST FOR MATCHED PAIRS**

$H_0$  :  $\mu_1 = \mu_2$

$H_1$  :  $\mu_1 \neq \mu_2$

$\alpha = 0.05$

Note:  $\alpha$  = probability of rejecting  $H_0$  when is true (Type 1: error)

The test is two tailed.

The test statistic is:

The Wilcoxon test considers information about both the sign of the differences and the magnitude of the differences between pairs. It tabulates the data into 2 columns, creates a third column which calculates both the magnitude and sign of the difference between the first 2 columns. A forth column then ranks the magnitudinal differences ignoring the signs and a fifth column then totals the ranks from the less frequent sign in column three. This total is then compared to the Wilcoxon Tables. If the totaled value is larger than the critical value from tables then  $H_0$  is rejected.

### 3.15 TEST 7: MANN WHITNEY U TEST

$$H_0 : \mu_1 = \mu_2$$

$$H_1 : \mu_1 \neq \mu_2$$

$$\alpha = 0.05$$

Note:  $\alpha$  = probability of rejecting  $H_0$  when is true (Type 1: error)

The test is two tailed.

The test statistic is:

The two groups are arranged in Joint Rank Order in each of their two separate columns. The ranks are then totalled for each column. Two statistics are then calculated, one for each group using the two separate formulae below:

$$U_1 = \frac{n_1 n_2 + n_1(n_1 + 1) - \sum R_1}{2}$$

$$U_2 = \frac{n_1 n_2 + n_1(n_1 + 1) - \sum R_2}{2}$$

The smaller of these two values is identified and compared to the critical value from the Mann – Whitney Tables. If the smallest value is less then the value from tables then  $H_0$  is rejected.

### **3.16 TEST 8: DESCRIPTIVE STATISTICS**

#### IMPORTANT POINTS OF HYPOTHESIS TESTING

Confidence intervals are closely connected to another useful statistical decision making technique called hypothesis testing. Hypotheses are just statements about parameters of probability distributions. The objective is to make decisions about these statements. Often these decisions can be made by examining the range of reasonable values for a parameter from a confidence interval.

## CHAPTER FOUR: RESULTS

### ***4.1 INTRODUCTION***

The following chapter contains the tables obtained from the statistical analysis of the primary data collected over the duration of the research programme. The data collected was from those patients who met the research criteria and who participated for the entire duration of the research programme.

The measurement criteria included:

Subjective data: - Numerical pain rating scale

Objective data: - Myofascial Diagnostic Scale  
- Algometer readings

**Note:** Each group (home and clinic) received five treatments over five consecutive days. Subjective and objective measures were taken at treatment one, treatment five and again at a one week follow-up. For the purpose of the following statistical analysis measurement one represents treatment one, measurement two represents treatment five and measurement three represents the one-week follow-up.

## 4.2 TABLES OF DEMOGRAPHIC DATA

**Table 1: Gender distribution**

Gender	Group A (home)	Group B (clinic)	Total % of patients
No. of males	12	8	50
No. of females	8	12	50

**Table 2: Age distribution**

Age group	Group A (home)	Group B (clinic)	Total % of patients
16-20	1	2	7.5
21-25	5	4	22.5
26-30	2	3	12.5
31-35	2	3	12.5
36-40	4	2	15
41-45	2	4	15
46-50	3	0	7.5
51-55	1	2	7.5

**Table 3: Race distribution**

Race	Group A (home)	Group B (clinic)	Total % of patients
White	16	18	85
Indian	4	2	15

**Table 4: Patient occupations**

Occupation	Group A (home)	Group B (clinic)	Total % of patients
Student	6	6	30
Lecturer	1	3	10
Computer / IT	2	3	12.5
Housewife	5	0	12.5
Sales/Marketing	2	0	5
Bodyguard	1	0	2.5
Business	2	3	12.5
Travel Consultant	0	1	2.5
Unemployed	0	1	2.5
Secretary	0	1	2.5
Estate agent	0	1	2.5
Management	1	1	5

**Table 5: Pain aggravating activities**

Activity	Group A (home)	Group B (clinic)	Total % of patients
Working at PC/desk	6	10	40
House work	4	0	10
Sport (golf/weights)	3	1	10
Driving	3	1	10
Telephone use	1	2	7.5
Bartending	1	3	10
Emotional stress	2	2	10
Poor sleeping posture	0	1	2.5

### ***4.3 TABLES OF STATISTICAL RESULTS***

#### **4.3.1 Tests Relating to the Myofascial Diagnostic Scale**

Intra-group Tests were performed to determine any significant difference in population means within Group 1 and Group 2.

Initially, the various normality tests were performed.



**Table 6: Kolmogorov-Smirnov test for normality of data**

**Tests of Normality**

ID		Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
		Statistic	df	Sig.	Statistic	df	Sig.
1.00	MFDS1	.356	20	.000	.626	20	.000
	MFDS2	.219	20	.013	.851	20	.006
	MFDS3	.363	20	.000	.826	20	.002
2.00	MFDS1	.268	20	.001	.897	20	.037
	MFDS2	.187	20	.066	.901	20	.043
	MFDS3	.294	20	.000	.755	20	.000

a. Lilliefors Significance Correction

There was sufficient evidence to suggest that all readings were from non-normal populations based on the p Values which were all  $\leq 0.05$  (except for one), therefore non-parametric tests were applied in this analysis.

**Table 7: The appropriate descriptive statistics**

	<b>First Reading</b>	<b>Second Reading</b>	<b>Third Reading</b>
<b>Group 1</b>	14.15	9.1	6.25
<b>Group 2</b>	13.35	9.55	6.8

The appropriate test therefore was the Friedmann Test to test if there was a significant difference between all three means in the first group and thereafter in a separate test in the second group.

The results of the Friedmann test for both the first and second group are portrayed below:

**Table 8: The Friedman test for intra-group analysis of the MFDS**

**Ranks**

ID		Mean Rank
1.00	MFDS1	2.93
	MFDS2	1.90
	MFDS3	1.18
2.00	MFDS1	2.73
	MFDS2	1.93
	MFDS3	1.35

**Test Statistics<sup>a</sup>**

1.00	N	20
	Chi-Square	35.343
	df	2
	Asymp. Sig.	.000
2.00	N	20
	Chi-Square	20.622
	df	2
	Asymp. Sig.	.000

a. Friedman Test

In Both cases the p Values were 0.000, which was  $< 0.05$ , implying that the three means at all stages of measurement were significantly different across both groups. The Wilcoxon Test for matched pairs was applied to calculate all the permutations of pairs with each group. The results of such tests are outlined below:

**Table 9: The Wilcoxon tests for matched pairs for the intra-group analysis of MFDS**

**Ranks**

ID			N	Mean Rank	Sum of Ranks
1.00	MFDS2 - MFDS1	Negative Ranks	17 <sup>a</sup>	9.00	153.00
		Positive Ranks	0 <sup>b</sup>	.00	.00
		Ties	3 <sup>c</sup>		
		Total	20		
2.00	MFDS2 - MFDS1	Negative Ranks	16 <sup>a</sup>	11.44	183.00
		Positive Ranks	4 <sup>b</sup>	6.75	27.00
		Ties	0 <sup>c</sup>		
		Total	20		

a. MFDS2 < MFDS1

b. MFDS2 > MFDS1

c. MFDS2 = MFDS1

**Test Statistics<sup>b</sup>**

ID		MFDS2 - MFDS1
1.00	Z	-3.644 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.000
2.00	Z	-2.917 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.004

a. Based on positive ranks.

b. Wilcoxon Signed Ranks Test

**Ranks**

ID			N	Mean Rank	Sum of Ranks
1.00	MFDS3 - MFDS1	Negative Ranks	20 <sup>a</sup>	10.50	210.00
		Positive Ranks	0 <sup>b</sup>	.00	.00
		Ties	0 <sup>c</sup>		
		Total	20		
2.00	MFDS3 - MFDS1	Negative Ranks	18 <sup>a</sup>	10.25	184.50
		Positive Ranks	1 <sup>b</sup>	5.50	5.50
		Ties	1 <sup>c</sup>		
		Total	20		

a. MFDS3 < MFDS1

b. MFDS3 > MFDS1

c. MFDS3 = MFDS1

**Test Statistics<sup>b</sup>**

ID		MFDS3 - MFDS1
1.00	Z	-3.937 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.000
2.00	Z	-3.615 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.000

a. Based on positive ranks.

b. Wilcoxon Signed Ranks Test

### Ranks

ID			N	Mean Rank	Sum of Ranks
1.00	MFDS3 - MFDS2	Negative Ranks	13 <sup>a</sup>	7.00	91.00
		Positive Ranks	0 <sup>b</sup>	.00	.00
		Ties	7 <sup>c</sup>		
		Total	20		
2.00	MFDS3 - MFDS2	Negative Ranks	12 <sup>a</sup>	8.54	102.50
		Positive Ranks	3 <sup>b</sup>	5.83	17.50
		Ties	5 <sup>c</sup>		
		Total	20		

a. MFDS3 < MFDS2

b. MFDS3 > MFDS2

c. MFDS3 = MFDS2

### Test Statistics<sup>b</sup>

ID		MFDS3 - MFDS2
1.00	Z	-3.204 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.001
2.00	Z	-2.425 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.015

a. Based on positive ranks.

b. Wilcoxon Signed Ranks Test

In all three cases the p Values were < 0.05, implying that at each reading point the values drop significantly from the previous reading and this is apparent across both groups.

**Comparison to show if a significantly different drop rate was shown between group 1 and group 2 for this testing technique.**

Initially, the normality tests are performed.

**Table 10: Kolmogorov-Smirnov test for normality of data**

Tests of Normality							
ID		Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
		Statistic	df	Sig.	Statistic	df	Sig.
1.00	MFDS1_2	.166	20	.149	.890	20	.027
	MFDS2_3	.223	20	.011	.834	20	.003
2.00	MFDS1_2	.105	20	.200*	.952	20	.396
	MFDS2_3	.132	20	.200*	.947	20	.323

\*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

In most cases the p value was > 0.05 therefore there was not conclusive evidence to reject normality of the data and as a result for the purposes of being thorough both the parametric and non-parametric tests were conducted here.

**Table 11: The appropriate descriptive statistics.**

	First Reading-Second Reading	Second Reading-Third Reading
<b>Group1</b>	5.05	2.85
<b>Group2</b>	3.8	2.75

A parametric testing procedure namely: an independent paired T-Test was performed on the First Reading – The Second Reading. The results are reflected below:

**Table 12: The Independent paired T-test for the inter-group comparison of the MFDS**

**Group Statistics**

ID	N	Mean	Std. Deviation	Std. Error Mean
MFDS1_2 1.00	20	5.0500	3.06894	.68624
2.00	20	3.8000	4.62943	1.03517

**Independent Samples Test**

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
MFDS1_2	Equal variances assumed	4.447	.042	1.006	38	.321	1.2500	1.24197	-1.26425	3.76425
	Equal variances not assumed			1.006	32.997	.322	1.2500	1.24197	-1.27683	3.77683

The p-value is greater than 0.05, therefore the null Hypothesis is accepted (which concludes that there is a difference in both population means at a 5% significance level).

A non-parametric testing procedure namely: the Mann Whitney U – Test was performed on the First Reading – The Second Reading. The results are reflected below:

**Table 13: The Mann Whitney U Test for the inter-group comparison of the MFDS**

**Ranks**

	ID	N	Mean Rank	Sum of Ranks
MFDS1_2	1.00	20	21.70	434.00
	2.00	20	19.30	386.00
	Total	40		

**Test Statistics<sup>b</sup>**

	MFDS1_2
Mann-Whitney U	176.000
Wilcoxon W	386.000
Z	-.653
Asymp. Sig. (2-tailed)	.514
Exact Sig. [2*(1-tailed Sig.)]	.529 <sup>a</sup>

a. Not corrected for ties.

b. Grouping Variable: ID

These results concur exactly with the results of the parametric test above. Therefore there was no significant difference in the drop rate between the first and second readings for both groups 1 and 2 at the 5% level of significance.

A parametric testing procedure namely: an independent paired T-Test was performed on the Second Reading – The Third Reading. The results are reflected below:



**Table 14: Independent paired T- test for the inter-group comparison of the MFDS**

**Group Statistics**

ID	N	Mean	Std. Deviation	Std. Error Mean
MFDS2_3 1.00	20	2.8500	3.13344	.70066
2.00	20	2.7500	4.44706	.99439

**Independent Samples Test**

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
MFDS2_3	Equal variances assumed	2.681	.110	.082	38	.935	.1000	1.21644	-2.36256	2.56256
	Equal variances not assumed			.082	34.135	.935	.1000	1.21644	-2.37175	2.57175

From the results of the p value we can see that it is greater than 0.05, therefore the null Hypothesis was accepted (which concludes that there is a difference in both population means at a 5% significance level).

A non-parametric testing procedure namely: the Mann Whitney U – Test was performed on the Second Reading – The Third Reading. The results are reflected below:

### Ranks

	ID	N	Mean Rank	Sum of Ranks
MFDS2_3	1.00	20	20.48	409.50
	2.00	20	20.53	410.50
	Total	40		

### Test Statistics<sup>b</sup>

	MFDS2_3
Mann-Whitney U	199.500
Wilcoxon W	409.500
Z	-.014
Asymp. Sig. (2-tailed)	.989
Exact Sig. [2*(1-tailed Sig.)]	.989 <sup>a</sup>

a. Not corrected for ties.

b. Grouping Variable: ID

These results concur exactly with the results of the parametric test above therefore there was no significant difference in the drop rate between the second and third readings across both groups 1 and 2 at the 5% level of significance.

#### 4.3.2 Tests relating to the algometer readings

The Intra-group Tests testing for significant difference in population means within Group 1 and Group 2.

Initially, the various normality tests were performed.

**Table 15: The Kolmogorov-Smirnov test for normality**

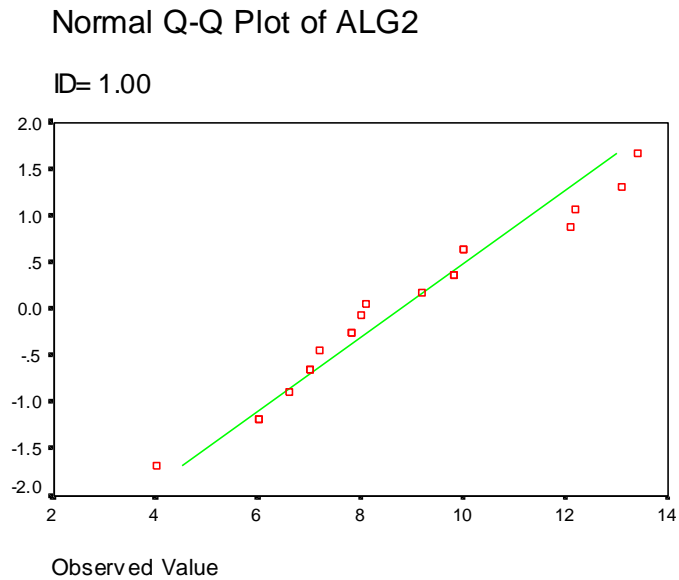
Tests of Normality							
ID		Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
		Statistic	df	Sig.	Statistic	df	Sig.
1.00	ALG1	.184	20	.075	.936	20	.203
	ALG2	.152	20	.200*	.956	20	.475
	ALG3	.209	20	.022	.928	20	.141
2.00	ALG1	.142	20	.200*	.912	20	.069
	ALG2	.120	20	.200*	.942	20	.264
	ALG3	.182	20	.081	.911	20	.067

\*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

The results above indicate that the data in a few cases is definitely non-normal as the p value is less than 0.05, but in other cases there is not enough evidence to suggest the non-normality of the data. As a result of these figures both parametric and non-parametric analysis of the data was conducted where appropriate. A normal probability plot of the data was drafted which indicates that the more the dots fall along the straight line the more probability that the data follows a normal distribution.

**Graph 1: A normal probability plot of the data**



**Table 16: The appropriate descriptive statistics**

	<b>First Reading</b>	<b>Second Reading</b>	<b>Third Reading</b>
<b>Group 1</b>	8.07	8.755	9.145
<b>Group 2</b>	7.095	7.595	8.455

It is obvious without even doing any further statistics that the mean trends indicate very little change over readings within both groups.

A parametric testing procedure namely: an analysis of variance or alternatively referred simply as ANOVA was performed for both groups. The results are reflected below:

**Table 17: The ANOVA or analysis of variance for the analysis of the algometer readings**

**ANOVA**

ALG

ID		Sum of Squares	df	Mean Square	F	Sig.
1	Between Groups	2.028	2	1.014	.141	.868
	Within Groups	408.529	57	7.167		
	Total	410.557	59			
2	Between Groups	18.928	2	9.464	1.267	.289
	Within Groups	425.649	57	7.468		
	Total	444.576	59			

A non- parametric testing procedure namely the Friedman Test was performed for both groups. The results are reflected below

**Table 18: A non- parametric testing procedure namely the Friedman Test**

**Ranks**

ID		Mean Rank
1.00	ALG1	1.93
	ALG2	1.98
	ALG3	2.10
2.00	ALG1	1.80
	ALG2	2.00
	ALG3	2.20

**Test Statistics<sup>a</sup>**

1.00	N	20
	Chi-Square	.333
	df	2
	Asymp. Sig.	.846
2.00	N	20
	Chi-Square	1.641
	df	2
	Asymp. Sig.	.440

a. Friedman Test

Both the 2 test results concur. All p values are greater than 0.05 therefore the Ho was accepted at a 5% level of significance and indicates that there exists no evidence to suggest a significant difference amongst means (from all three readings) for both groups.

**Comparison to show if a significantly different drop rate has occurred between group 1 and Group 2 for this testing technique:**

**Table 19: The Kolmogorov-Smirnov test for normality of data**

Tests of Normality							
ID		Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
		Statistic	df	Sig.	Statistic	df	Sig.
1.00	ALG1_2	.226	20	.009	.876	20	.015
	ALG2_3	.190	20	.056	.927	20	.134
2.00	ALG1_2	.179	20	.091	.918	20	.089
	ALG2_3	.183	20	.079	.925	20	.123

a. Lilliefors Significance Correction

The data above reflect evidence that the data in this case is non-normal, as the p value is less than 0.05 in a few cases and just over in other cases. To be safe apply both parametric and non-parametric testing procedures were performed.

The appropriate non-parametric test namely the Mann Whitney Test was applied.

**Table 20: The Mann-Whitney U Test for inter-group analysis of the algometer readings**

**Ranks**

ID	N	Mean Rank	Sum of Ranks
ALG1_2 1.00	20	19.98	399.50
2.00	20	21.03	420.50
Total	40		

**Test Statistics<sup>a</sup>**

	ALG1_2
Mann-Whitney U	189.500
Wilcoxon W	399.500
Z	-.284
Asymp. Sig. (2-tailed)	.776
Exact Sig. [2*(1-tailed Sig.)]	.779 <sup>a</sup>

a. Not corrected for ties.

b. Grouping Variable: ID

**Ranks**

ID	N	Mean Rank	Sum of Ranks
ALG2_3 1.00	20	21.30	426.00
2.00	20	19.70	394.00
Total	40		



**Test Statistics<sup>b</sup>**

	ALG2_3
Mann-Whitney U	184.000
Wilcoxon W	394.000
Z	-.433
Asymp. Sig. (2-tailed)	.665
Exact Sig. [2*(1-tailed Sig.)]	.678 <sup>a</sup>

a. Not corrected for ties.

b. Grouping Variable: ID

In all cases the p values are greater than 0.05. Therefore Ho was accepted, indicating no significant difference in drop rate between all readings across both groups.

The corresponding parametric test procedure, namely the independent T –test was applied below and the results are exactly the same as the results in the test above.

**Table 21: The independent paired T-test for the inter-group comparison of the algometer readings**

**Group Statistics**

ID	N	Mean	Std. Deviation	Std. Error Mean
ALG1_2 1.00	20	-.6850	2.23448	.49965
2.00	20	-.5000	1.94503	.43492

**Independent Samples Test**

	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
ALG1_2	.221	.641	-.279	38	.782	-.1850	.66242	-1.52600	1.15600
Equal variances assumed			-.279	37.291	.782	-.1850	.66242	-1.52684	1.15684
Equal variances not assumed									

**Group Statistics**

ID	N	Mean	Std. Deviation	Std. Error Mean
ALG2_3 1.00	20	-.3900	2.45441	.54882
2.00	20	-.8600	2.53759	.56742

**Independent Samples Test**

	Levene's Test for Equality of Variances		t-test for Equality of Means						
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
								Lower	Upper
ALG2_3	.475	.495	.595	38	.555	.4700	.78941	-1.12808	2.06808
Equal variances assumed			.595	37.958	.555	.4700	.78941	-1.12814	2.06814
Equal variances not assumed									

4.3.3 Tests relating to the NRS 101

The Intra Group Tests testing for significant difference in population means within Group 1 and Group 2 were performed.

**Table 22: The Kolmogorov-Smirnov test for normality of data**

**Tests of Normality**

ID	Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk			
	Statistic	df	Sig.	Statistic	df	Sig.	
1.00	NRS1	.174	20	.114	.949	20	.351
	NRS2	.115	20	.200*	.950	20	.366
	NRS3	.117	20	.200*	.965	20	.640
2.00	NRS1	.137	20	.200*	.941	20	.248
	NRS2	.205	20	.027	.908	20	.059
	NRS3	.184	20	.076	.853	20	.006

\*. This is a lower bound of the true significance.

a. Lilliefors Significance Correction

There was sufficient evidence to suggest that all readings are from non-normal populations based on the p Values which are all > 0.05 (except for one), therefore non-parametric tests are applied in this analysis.

The appropriate descriptive statistics are summarised below.

**Table 23: The appropriate descriptive statistics**

**Descriptive Statistics**

ID	N	Mean	Std. Deviation	Minimum	Maximum	
1.00	NRS1	20	43.9875	12.60912	12.50	67.50
	NRS2	20	36.1250	12.68222	10.00	55.00
	NRS3	20	24.0500	14.36269	.00	50.00
2.00	NRS1	20	47.8250	12.64315	15.00	75.00
	NRS2	20	35.7750	12.44512	5.50	55.00
	NRS3	20	27.6000	15.49414	5.00	45.00

**Table 24: The Friedman test was used to calculate the Intra-group results of the NRS scores**

**Ranks**

ID		Mean Rank
1.00	NRS1	2.60
	NRS2	2.10
	NRS3	1.30
2.00	NRS1	2.63
	NRS2	1.98
	NRS3	1.40

**Test Statistics<sup>a</sup>**

1.00	N	20
	Chi-Square	21.500
	df	2
	Asymp. Sig.	.000
2.00	N	20
	Chi-Square	19.705
	df	2
	Asymp. Sig.	.000

a. Friedman Test

The p value in all cases was less than 0.05, indicating a difference between treatment means across both groups 1 and 2.

The following tests were performed in order to isolate where those differences are occurring.

Initially the normality tests were performed.

**Table 25: The Kolmogorov-Smirnov test for normality of data**

<b>Tests of Normality</b>							
ID		Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
		Statistic	df	Sig.	Statistic	df	Sig.
1.00	NRS1_2	.183	20	.078	.940	20	.240
	NRS2_3	.174	20	.116	.894	20	.032
2.00	NRS1_2	.164	20	.167	.909	20	.061
	NRS2_3	.190	20	.058	.915	20	.079

a. Lilliefors Significance Correction

There was strong evidence that the data is non-normal as p values are either less than 0.05 or very close to it. The appropriate hypothesis test namely, the Wilcoxon Test was performed.

**Table 26: The Wilcoxon test for intra- group analysis of the NRS scale**

**Ranks**

ID	N	Mean Rank	Sum of Ranks
1.00 NRS2 - NRS1	Negative Ranks	12 <sup>a</sup>	9.29
	Positive Ranks	3 <sup>b</sup>	2.83
	Ties	5 <sup>c</sup>	
	Total	20	
2.00 NRS2 - NRS1	Negative Ranks	12 <sup>a</sup>	8.96
	Positive Ranks	3 <sup>b</sup>	4.17
	Ties	5 <sup>c</sup>	
	Total	20	

a. NRS2 < NRS1

b. NRS2 > NRS1

c. NRS2 = NRS1

**Test Statistics<sup>b</sup>**

ID	NRS2 - NRS1
1.00	Z
	Asymp. Sig. (2-tailed)
2.00	Z
	Asymp. Sig. (2-tailed)

a. Based on positive ranks.

b. Wilcoxon Signed Ranks Test

### Ranks

ID			N	Mean Rank	Sum of Ranks
1.00	NRS3 - NRS2	Negative Ranks	14 <sup>a</sup>	8.46	118.50
		Positive Ranks	1 <sup>b</sup>	1.50	1.50
		Ties	5 <sup>c</sup>		
		Total	20		
2.00	NRS3 - NRS2	Negative Ranks	11 <sup>a</sup>	8.18	90.00
		Positive Ranks	3 <sup>b</sup>	5.00	15.00
		Ties	6 <sup>c</sup>		
		Total	20		

a. NRS3 < NRS2

b. NRS3 > NRS2

c. NRS3 = NRS2

### Test Statistics<sup>b</sup>

ID		NRS3 - NRS2
1.00	Z	-3.326 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.001
2.00	Z	-2.360 <sup>a</sup>
	Asymp. Sig. (2-tailed)	.018

a. Based on positive ranks.

b. Wilcoxon Signed Ranks Test

In all analysis above, the p value was less than 0.05, indicating that within Group 1 there was a significant drop in means between both treatments 1 and 2 and also between treatments 2 and 3. The same situation is evident in Group 2.

**Comparison to show if a significantly different drop rate has occurred between group1 and Group 2 for this testing technique.**

**Table 27: The Kolmogorov-Smirnov test for normality of data**

Tests of Normality						
	Kolmogorov-Smirnov <sup>a</sup>			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
NRS1_21	.226	20	.009	.876	20	.015
NRS1_22	.164	20	.167	.909	20	.061
NRS2_31	.174	20	.116	.894	20	.032
NRS2_32	.190	20	.058	.915	20	.079

a. Lilliefors Significance Correction

There was sufficient evidence to suggest that all readings are from non-normal populations based on the p Values which are all > 0.05 (except for one), therefore non-parametric tests are applied in this analysis.

The descriptives are highlighted below.



**Table 28: The appropriate descriptive statistics**

	N	Minimum	Maximum	Mean	Std. Deviation
NRS1_21	20	-5.20	2.50	-.6850	2.23448
NRS1_22	20	-10.00	49.00	12.0500	16.93323
NRS2_31	20	-2.50	35.00	12.0750	12.08552
NRS2_32	20	-15.00	40.00	8.1750	14.01153
Valid N (listwise)	20				

**Table 29: The Mann Whitney U Test for the inter- group comparison of the NRS 101 scale**

GROUP	N	Mean Rank	Sum of Ranks
NRS1_21 1	20	15.88	317.50
2	20	25.13	502.50
Total	40		

	NRS1_21
Mann-Whitney U	107.500
Wilcoxon W	317.500
Z	-2.505
Asymp. Sig. (2-tailed)	.012
Exact Sig. [2*(1-tailed Sig.)]	.011 <sup>a</sup>

a. Not corrected for ties.

b. Grouping Variable: GROUP

**Ranks**

	GROUP	N	Mean Rank	Sum of Ranks
NRS2_31	1	20	22.58	451.50
	2	20	18.43	368.50
	Total	40		

**Test Statistics<sup>b</sup>**

	NRS2_31
Mann-Whitney U	158.500
Wilcoxon W	368.500
Z	-1.135
Asymp. Sig. (2-tailed)	.256
Exact Sig. [2*(1-tailed Sig.)]	.265 <sup>a</sup>

a. Not corrected for ties.

b. Grouping Variable: GROUP

Based on the p values there has been a significantly different drop rate between treatments 1 and 2 for both groups, this trend was not apparent between treatments 2 and 3. Group 2 has shown the greater drop between treatments 1 and 2.

## **CHAPTER FIVE: DISCUSSION OF THE RESULTS**

### **5.1 INTRODUCTION**

This chapter involves the discussion of the demographic data and the results of the statistical analysis of the objective and subjective data. The problems encountered throughout the research program are also discussed in detail.

The results of the statistical analysis are discussed under objective and subjective results and further evaluated in terms of intra- and inter- group comparisons. All the statistical tests were performed at a 5% level of significance.

Evaluation of the intra-group results between the first and sixth consultations (overall measurement interval) gives an indication of the overall effectiveness of the treatment regimens. Evaluation of the results between the first and fifth consultations (first measurement interval) gives an indication of the progression of the treatment regimen. Evaluation of the results between the fifth consultation and the one-week follow up gives an indication of any changes which occurred one week after the final treatment.

Evaluation of the inter-group results of the first consultation reveals any variance in the subjective and objective findings between the two groups at the beginning of the study. Evaluation of the inter-group results at consultation five and the one-week follow-up reveal any difference in the overall improvement as well as the rate of improvement between the two groups.

## **5.2 DISCUSSION OF THE DEMOGRAPHIC DATA**

Of the forty patients that participated in the research programme, twenty were male and twenty were female (table 1). Han and Harrison (1997:90) state that Myofascial Pain Syndrome is more common in females, thus, this study does not show a high correlation with the literature regarding the sex distribution of the above condition. However, small sample size may well be the reason for this.

The age group chosen as an inclusion criterion for this study was between 18 and 55 years. The age group of greatest prevalence in this study was 21-25 years, (22,5%) (Table 2). It is the opinion of the author that this was the age group of greatest prevalence because the study was carried out at a tertiary education institution where students had the greatest exposure to the study. The age group between 36-40 and 41-45 accounted for 15% each, therefore 30% of patients combined, which seems to correlate with the statement by Travell, Simons and Simons (1999 1:13) that individuals in their mature years (between 31-50) are most likely to suffer from the condition.

Evaluation of the race groups represented by this study show the majority of patients to be Caucasian (85%) and the remainder of the patients being Indian (15%) while no Black or patients of mixed race took part in the study. (Table 3) This does not give a true representation of the race distribution of the South African population. The most likely explanation for the above results is that the advertisements for the study were in English and posted in the area surrounding the Durban Institute of Technology, which consists of mainly White and Indian communities.

Of those patients that were accepted into the study, 30% were students, 12,5% were business men/women, 12,5% were house wives, 12,5% were involved with computers and 10% were lecturers (Table 4). These occupations correlate with the high percentage (40%) of patients that said working at a desk or in front of a computer was the most common activity associated with aggravating their pain (Table 5). Han and Harrison (1997:92) suggested that poor posture associated with prolonged sitting at a desk might explain the high prevalence of the condition in these patients.

### **5.3 DISCUSION OF THE OBJECTIVE RESULTS**

#### 5.3.1 Myofascial Diagnostic Scale

##### Intra- group comparison

Evaluation of the statistical results of the Friedmann's test and the Wilcoxon tests for matched pairs for both groups revealed a statistically significant improvement between measurements 1&2, 2&3 with regards to the Myofascial Diagnostic Scale. (Table 8 & 9)

These findings suggest that both home and clinic groups show a reduction in the clinical signs outlined in the Myofascial Diagnostic Scale, over the course of the study. This is partly due to the fact that essentially both groups were receiving similar treatments with the only difference being that group A performed the treatment at home while the group B performed the treatment under the observation of the researcher at the DIT clinic. However, these results may also give an indication that the Myofascial Diagnostic Scale is

somewhat subjective and its validity as a measurement tool needs to be confirmed. It is the opinion of the author that there was no obvious clinical difference between the two groups with respect to the Myofascial Diagnostic Scale.

#### Inter- group comparison

A parametric test (Independent paired T-Test) and a non-parametric test (Mann Whitney U-Test) for inter- group comparison of the Myofascial Diagnostic Scale data showed no difference between groups at measurements one, two or three. This result showed that there was minimal variance between the home and clinic groups with regard to the data collected over the entire research program (Table 12,13 & 14)

Both groups received essentially the same treatment therefore each group showed an improvement as expected and thus there was no statistically significant difference between groups 1 and 2 with regards to the Myofascial Diagnostic Scale. It is the opinion of the author that there was no obvious clinical difference between the two groups with respect to the Myofascial Diagnostic Scale.

#### 5.3.2 Algometer Readings

#### Intra- group comparisons

A parametric test (ANOVA) and a non-parametric test (Friedman) were performed on both groups (Table 17&18). Both the test results concur. All p-values were greater than 0.05 therefore the null hypothesis, that there was no statistical difference amongst means (from all three readings) for both groups 1 and 2, was accepted.

These findings suggest that both groups showed an increase in pressure threshold levels over the research programme, although this increase was not a statistically significant one, both groups did in fact improve clinically. These results highlight the fact that ischaemic compression is an effective tool for a home treatment programme. A possible explanation as to why the increase was not a significant one may be that patients in both groups received ischaemic compression for five consecutive days and this may have contributed to some degree of post treatment soreness. The author did observe that those patients in the clinic group tended to use excessive levels of pressure during treatment sessions. This could possibly explain why patients in this group responded less favorably in terms of clinical improvement and reported a higher level of post treatment soreness.

#### Inter- group comparisons

The appropriate non-parametric test (Mann Whitney) as well as the corresponding parametric test (Independent Paired T-Test) was used for statistical comparison of groups 1 and 2. In all cases the p-values were greater than 0.05. (Table 20 & 21) Therefore the null hypothesis was accepted, indicating no significant difference between all readings across both groups.

## **5.4 DISCUSSION OF THE SUBJECTIVE RESULTS**

### 5.4.1 Numerical Pain Rating Scale 101

#### Intra- group comparison

Evaluation of the results of intra-group comparison using the Friedmann and Wilcoxon test for the NRS 101 scores revealed a statistically significant improvement between measurements 1 & 2 and 2 & 3 for both the home and clinic groups (Tables 24 & 26).

These findings suggest that both the home and clinic groups showed a statistically significant reduction in pain intensity over the course of the research programme.

With regard to clinical improvement it is the opinion of the author that the clinic group did report feeling less neck pain and/or stiffness towards the end of the study. This may be due to a more diligent approach to the treatments by the patients in this group because they were being observed. This result may also be due to aspects of the Hawthorne effect, which states that observation itself will have some effect on the outcome of the study.

#### Inter- group comparison

Comparison between the home and clinic group, using the Mann Whitney U test revealed that the clinic group showed a significantly higher rate of improvement than the home group between measurements 1 & 2 (Table 29).



These findings suggest that the clinic group showed greater reduction in perceived pain intensity than the home group over the research programme. This may indicate a positive placebo effect as the clinic group did have contact with the researcher for five consecutive days. The Hawthorne effect (as mentioned above) is another possible explanation for the results observed.

## **5.5 SUMMARY OF THE CLINICAL FINDINGS**

The hypothesis that a home programme of ischaemic compression would be an effective form of treatment for patients with Myofascial Pain Syndrome (hypothesis 1) was supported by this study. However, the hypothesis that a home programme of ischaemic compression would be more effective than the clinic group (hypothesis 2) was not supported by this study.

Intra- group analysis of data obtained from the home group revealed significant improvements between measurements 1 & 2 and 2 & 3 in terms of subjective and objective data. These results suggest that not only was the treatment regime effective over the research programme, but that the improvement continued at the one-week follow-up.

However, similar results were obtained for the clinic group, in terms of both subjective and objective data. This was due to both groups essentially receiving the same treatment with the home group performing ischaemic compression as a home programme while the clinic group performed ischaemic compression under observation in the clinic.

Inter- group analysis of data obtained from the home and clinic groups revealed no significant differences between the groups at the end of the research programme, in terms of algometric measurements and Myofascial Diagnostic Scale scores. The clinic group showed a significantly higher rate of improvement than the home group between measurements 1 & 2. These results suggest that the home and clinic groups responded equally in terms of algometric measurements and Myofascial Diagnostic Scale scores. However, the clinic group responded more favorably than the home group, in terms of the NRS 101 questionnaire between measurements 1 & 2. The clinic group performed five treatments of ischaemic compression over five consecutive days under the observation of the researcher. It is the opinion of the researcher that the Hawthorne effect or possible positive placebo effect (contact with the researcher) was a possible reason for this difference.

Another possibility is that the clinic group, being under observation, performed the ischaemic compression diligently. The patients in the home group were instructed to complete a diary of their treatment programme and were telephoned regularly to improve compliance. There is however no accurate way of determining the level of honesty and thus compliance of patients within this group.

## **5.6 PROBLEMS ENCOUNTERED WITH THE DATA**

### 5.6.1 Objective Data

With regards to the Myofascial Diagnostic Scale, the author tends to agree with Walker (2002) that it may have been moderately subjective.

There was no accurate way of ensuring that the degree of pressure used to elicit tenderness over the TrP area was the same for each patient and this

may have lead to researcher bias in favor of a particular treatment regimen. The validity of this statement could be tested in an independent study of inter-examiner reliability.

No problems were encountered with the use of the Algometer.

### 5.6.2 Subjective Data

The NRS 101 questionnaire was easy to explain and the author believes that all patients had a good understanding of how to complete the form.

## **CHAPTER SIX: CONCLUSION AND RECOMMENDATIONS**

### **6.1 CONCLUSION**

This study consisted of 40 patients, divided into two groups of 20 each. After a diagnosis of active Myofascial TrPs of the Levator Scapulae muscles was confirmed, each patient underwent a full case history, general physical examination and regional examination. The patients were then randomly allocated to either the home group or the clinic group. Those patients in the home group performed a home programme of ischaemic compression for active TrPs in the Levator Scapulae muscles, whilst those in the clinic group performed ischaemic compression for active TrPs in the Levator Scapulae muscles under observation of the researcher. Each patient performed five treatments over five consecutive days and data was collected at the initial, fifth and one-week follow-up consultations.

Evaluation of the statistical results showed that both groups responded favorably in terms of subjective findings and objective Myofascial Diagnostic Scale scores. Both groups experienced an improvement in pressure threshold as indicated by the objective algometer readings, however this improvement was not statistically significant and this result may be due to the presence of post treatment tenderness. The clinic group also showed a statistically significant reduction in perceived pain intensity in terms of subjective NRS 101 scores when compared to the home group. This may indicate a heightened placebo effect as a result of interaction of the researcher. It is the opinion of the author that due to the favorable response by both groups it can be concluded that a home programme of ischaemic

compression using a Thera-Cane device is an effective treatment for patients suffering with active TrPs of Myofascial Pain Syndrome.

This study provides practitioners with a simple, effective, non-invasive alternative to numerous follow-up sessions with patients suffering from difficult to treat Myofascial TrPs. A home programme will encourage a wellness approach and serve to actively involve the patient in his or her treatment, acting as the primary pain manager. Thus reducing the number of visits to practitioners and enabling patients to reduce medical expenses. It is important to note that this home programme should be included as an adjunct to periodic visits to Chiropractors for re-assessment and/or any other standard therapy for the trigger points.

## **6.2 RECOMMENDATIONS FOR FUTURE STUDIES**

The sample size for this study was relatively small. A larger sample size is recommended in order to allow for more accurate statistical analysis.

The South African population was not well represented in this study. This problem should be addressed in any future studies by advertising to a broader community and in varied languages.

In order to minimize the effect of post treatment soreness as a result of ischaemic compression it is recommended that any future studies look at performing treatments on alternate days rather than consecutive days.

A follow-up consultation was used in this study, however this follow-up was conducted at one-week following the final treatment. It may prove more beneficial to look at a one-month follow-up session to obtain more accurate data of this programme.

It is recommended that the Myofascial Diagnostic Scale is researched further to determine its value as an objective measure.

Since a home programme of ischaemic compression has been shown to be an effective treatment for Myofascial Pain Syndrome, further study suggestions include:

- Comparison of the home programme to other forms of treatment for Myofascial Pain Syndrome (dry needling)
- Using a home programme of ischaemic compression as an adjunct to Chiropractic manipulation, stretching and education protocols.
- Using a home programme of ischaemic compression to treat trigger points occurring in other muscle groups i.e. Quadratus Lumborum

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